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# CAUSAL ATTRIBUTION AND DEPRESSION

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CAUSAL ATTRIBUTION AND DEPRESSION

BY

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Academic Awards

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## ABSTRACT

### CAUSAL ATTRIBUTION AND DEPRESSION, BY A IKHLEF

This thesis reports a series of experiments conducted to elucidate the nature of the relationship between attribution and depression.

After an extended review of the literature on attribution and depression it was concluded that further research is needed to evaluate the aetiological importance of depressogenic attributions, and also to elucidate the nature of the relationship they may entertain with depression.

A series of experiments and studies were performed to address these and other issues. In experiments 1-3 subjects' attributions for success and failure were manipulated and their effects on subsequent mood, expectations and psychomotor performance were assessed. The results showed that depression and its correlates are causally influenced by negative self-attributions. A further study, in which depressed and non-depressed patients' attributions were assessed, also provided evidence in support of the aetiological importance of these kinds of attributions.

Experiments 5 and 6 were designed to clarify the nature of the relationship between attributions and depressed mood. Experiment 6 showed that mood can affect attributions, suggesting that the relationship between these two variables is at least reciprocal. The implications of this finding for cognitive formulations of depression were discussed.

Another part of the programme was concerned with the determinants of depressogenic attributions. Two studies investigated ways in which depressed and non-depressed subjects used information to formulate attributions. The results suggest that depressed subjects' maladaptive attributions may develop as a result of a tendency to use personal rather than environmental information.

Finally, a multifactorial model was proposed, and its implications for the understanding of the aetiology and development of depression were discussed.

## ABSTRACT

### CAUSAL ATTRIBUTION AND DEPRESSION, BY A IKHLEF

This thesis reports a series of experiments conducted to elucidate the nature of the relationship between attribution and depression.

A review of the literature bearing on the issues of diagnosis, nosology, and aetiology of depression showed that such issues are still unsettled. After a subsequent review of the more relevant attribution-depression literature it was concluded that further research is needed to evaluate the aetiological importance of the so-called depressogenic attributions, and also to elucidate the nature of the relationship they may entertain with depression.

A series of experiments, in which these and other issues were addressed, were performed. Experiments 1-4 support the claim by learned helplessness theoreticians that negative self-attributions have an aetiological status in depression. Both experimental and clinical evidence were obtained in support of the aetiological importance of these kind of attributions. In addition to producing some informative findings concerning the theoretical status of attribution, the present work also shed light on the nature of the relationship between attribution and depression. The results from experiments 1-6 indicated that the relationship between these two variables is at least reciprocal. The implications of these findings for cognitive formulations of depression (including learned helplessness theory) were discussed.

Another part of this research programme was directed towards a detailed examination of the antecedent conditions of depressogenic attributions. These were found to develop as a result of the tendency to use certain types of information.

In the third part of this thesis, a model that fits the data generated by both the present work and that of others was proposed. The central claim of this model is that depression develops as a result of continuous interplay among various factors. The implications of this multifactorial view for the understanding of the aetiology and development of depression were discussed.

### DECLARATIONS

- (1) While registered for this degree, I have not been a registered candidate for another award of the CNAA or of a university.
- (2) None of the material contained herein has been used in any other submission for an academic award.
- (3) A programme of advanced study has been completed, in partial fulfilment of the requirements for the degree, consisting of guided reading in the area of attribution-depression, attendance at an advanced course on attribution-helplessness-depression (BA Psychology special option, Plymouth Polytechnic) and attendance at relevant research seminars and conferences.

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PART 1

THEORETICAL BACKGROUND

## CHAPTER ONE

### HISTORICAL INTRODUCTION

1.1 INTRODUCTION

1.2 EARLY AETIOLOGICAL CONCEPTIONS

1.3 THE NOSOLOGICAL DEBATE

## 1.1 INTRODUCTION

Depression may be one of the few psychiatric disorders that has seriously threatened man's well being and joie de vivre throughout his long and hazardous history. It has been known since biblical and homeric times - vivid descriptions of depressive symptoms and episodes may be found in the Bible as well as in some of Homer's epic poems. But the first clinical description of manifestations and symptoms characteristic of the depressive syndrome can only be traced back to the classical greek era. In fact the initial step towards the delineation of this clinical syndrome was made when Hippocrates introduced for the first time the term melancholia in the medical terminology.

Hippocrates wrongly believed that melancholia was caused by an accumulation of black bile in the brain. This aetiological view remained unchallenged for a long period of time. It was not until Aretaeus, who is said to have lived in the second century A.D., provided a more exhaustive clinical description of this syndrome that a new conception concerning both the nature and the aetiology of depressive illness evolved. In addition to revealing that manic and depressive states can alternate in the same individual, perhaps the most valuable contribution to the field of psychopathology at that time, Aretaeus recognized the importance of psychological factors in the genesis and development of this syndrome:

"The characteristic appearances, then, are not obscure; for the patients are dull or stern, dejected or unreasonably

torpid, without any manifest cause: such is the commencement of melancholy. And they also become dispirited, sleepless and start up from a disturbed sleep... They are prone to change their minds readily; to become bossy, mean spirited, illeberal, and in a little time, perhaps, simple, extravagant, munificent, not from any virtue of soul, but from the changeableness of the disease. But if the illness becomes more urgent, hatred, avoidance of the haunts of men, vain lamentations are seen; they complain of life and desire to die." (Quoted in Lewis, 1934)

Aretaeus's notion that mania and depression constitute a single clinical entity was embraced by most psychiatric investigators of the nineteenth century. His psychological approach was also revived and adopted after the renaissance by reformers such as Esquirol and Pinel. In his book *Traité médico-philosophique sur l'alienation mentale*, Pinel (1801) clearly revealed his psychological orientation when he refuted organicism and reaffirmed his humane approach to the mentally ill patients.

Once again depression became the focus of the debate when Baillager (1854) introduced the concept of 'folie à double form' and at the same time Falret (1854) coined the term 'folie circulaire'. But the credit went to Kraepelin who soon after elaborated and refined this diagnostic entity that he called manic depressive psychosis.

## 1.2 EARLY AETIOLOGICAL CONCEPTIONS

Although the nineteenth century has seen a scientific revolution, the state of knowledge in psychiatric circles remained at the pre-scientific stage for a long period of time. It was not until Kraepelin, the founder of modern psychiatry, published his *Lehrbuch der Psychiatrie* in 1896 that a renewed interest was shown to the study of the aetiology and nosology of psychiatric disorders in general, and depressive ones in particular.

Kraepelin subdivided mental illnesses into three major categories: dementia praecox, manic-depressive psychosis, and paraphrenia. By introducing this classification, Kraepelin had established a nosological system that gave psychiatry its much needed scientific basis and respect in the medical community.

In his subsequent publications, Kraepelin elaborated his views on both the nosology and the genesis of depressive disorders:

"Manic depressive insanity....includes on the one hand the whole domain of so-called periodic and circular insanity, on the other hand simple mania, the greater part of the morbid states termed melancholia and also a not inconsiderable case of amentia....all the above-mentioned states only represent manifestations of a single morbid process." (1902)

Kraepelin believed that manic-depressive psychosis and indeed all mental illnesses are due to organic causes, although he later conceded that external factors (exogenous) may exert a substantial influence on the prognosis as well as the genesis of certain disorders. In addition to maintaining an organogenesis conception of depressive illness, Kraepelin developed and adopted a classification model whose rigidity and lack of clarity impeded the understanding of the mechanisms underlying most diagnostic entities.

Although Kraepelin's approach won a widespread popularity for its clinical objectivity and nosological innovations, it was also criticised for its lack of flexibility. Theoreticians and clinicians dissatisfied with Kraepelin's system questioned both the underlying theoretical conception and the clinical considerations on which it rests. For Kraepelin did not only maintain his organogenesis conception of mental illness in spite of contrary evidence, he also relied exclusively on the prognosis to define

his 'disease entity'.

While European investigators such as Lange (1928) restricted themselves to making cosmetic changes in Kraepelin's original model, a new school of thought (in America) headed by Adolf Meyer offered a radical view of mental illness in general and of depressive illness in particular. Meyer contested Kraepelin's concept of 'disease entity' and proposed that psychiatric disorders should be viewed as 'reaction types' displayed by an individual to adapt to environmental changes and constraints. Meyer's theory of 'psychobiology', in contrast to the cartesian dualism of mind and body adopted by traditional psychiatry, strongly emphasised the unity of both the psychological and biological structures:

"The apparent disorder of individual organs is merely an incident in a development which we could not understand correctly except by comparing it with the normal and efficient reaction of the individual as a whole, and for that we must use terms of psychology - not of mysterious events, but actions and reactions of which we know they do things, a truly dynamic psychology. There we find the irrepressible instincts and habits at work, and finally the characteristic mental reaction type constituting the obviously pathological aberrations....by dropping some unnecessary shells and traditions, we can see a psychopathology develop without absurd contrast between mental and physical...." (Meyer, 1908)

Meyer's concept of 'psychobiological unit' was enthusiastically embraced and used to approach both the aetiology and nosology of depressive disorders. Those who espoused Meyer's views stressed the importance of personal and social factors in the genesis of depression, thus depression ceased to be a correlate of brain pathology. The Meyerians also rejected the endogenous-reactive (exogenous) dichotomy advocated by Kraepelin and his followers, instead they proposed that depression should be viewed as a single

illness differing not in nature but in severity and chronicity. The Meyerian framework was undoubtedly reflecting the growing influence that psychoanalysis was beginning to have on psychiatric thought since its formulation by Freud.

### 1.3 THE NOSOLOGICAL DEBATE

Kraepelin's and Meyer's divergent views regarding both the nature and classification of depressive disorders gave rise to a long but fruitless debate centred exclusively around the nosological issue, relatively neglecting important questions about the aetiology and treatment of depression. According to Kendell (1976), the reason for this state of affairs is that:

"they (depressions) provide a convenient arena for several disputes about the nature and classification of mental illness as a whole: whether mental illnesses are diseases or reaction types, whether they are independent entities or arbitrary concepts; whether they should be classified on the basis of their symptomatology, their aetiology or their pathogenesis; and whether they should be portrayed by a typology or by dimensions." (p.15)

Whatever the reason for this controversial debate, there is no doubt that the nosological status of depression was at the heart of the dispute. While some have argued for the existence of distinct categories of depressive illnesses (e.g., Gillespie, 1929), others, however, maintained that all depressive illness was the same, and the differences observed in symptomatology were merely quantitative (e.g., Lewis, 1934). Although some areas of agreement have since emerged (cf. Kendell, 1975, 1976), nevertheless the literature on classification of depressive disorders is still confusing. Table 1 illustrates this point.



TABLE 1. Proposed classifications of depression (adapted from Kendell, 1976)

A. Simple typologies	
<u>One category</u>	<u>Lewis</u> (1934) Depressive illness
<u>Two categories</u>	<u>Roth</u> (1965) Endogenous depression Neurotic depression <u>Van Praag</u> (1965) Vital depression Personal depression
<u>Three categories</u>	<u>Overall</u> (1966) Anxious-tense depression Hostile depression Retarded depression
<u>Four categories</u>	<u>Paykell</u> (1971) Psychotic depression Anxious depression Hostile depression Young depressives with personality disorder
B. Dimensional systems	
<u>One dimension</u>	<u>Kendell</u> (1968) Psychotic-neurotic
<u>Two dimensions</u>	<u>Eysenck</u> (1970) Psychoticism and Neuroticism

In this second part of the present chapter, a brief review of the arguments that animated the debate on the nosological status of depression is made, and attempts at evaluating some newly proposed classifications are also made. And finally, the question of whether depressive disorders should be portrayed by a typology or dimensions is asked and a tentative answer is proposed.

### 1.3.1 The unitary conception

The unitary conception of depressive disorders was proposed by Meyer following his sharp criticism of Kraepelin's nosological and aetiological formulations. But it was Lewis (1934) who actively defended and finally established this nosological scheme in modern clinical psychiatry. Clinicians and researchers who subscribe to the unitary view of depressive disorders, argue for their homogeneity, although they recognize that substantial differences may exist in phenomenology, severity, and chronicity of some depressive states. The monists, as they are now known, regard depression as a single illness that occurs in various degrees of severity and chronicity. They argue that the endogenous (psychotic) - reactive (neurotic) dichotomy advocated by Kraepelin and later reiterated by Gillespie (1929) and many others, is neither supported by aetiological studies nor justified by treatment purposes.

While the separatists, those who favour the dichotomy, were actively searching for evidence to substantiate their claim, the monists limited themselves to refuting such evidence. Repeated clinical observations and follow-up studies were soon to reveal that psychotic and neurotic depressive patients exhibit differences not only in clinical symptomatology but also in premorbid personality.

Lewis (1938) was unconvinced by the arguments presented in favour of the discontinuity. He pointed out that the diagnostic categories of psychotic and neurotic depressions:

".... are nothing more than attempts to distinguish between acute and chronic, mild and severe; and where two categories only are presented, the one manic-depressive - gives the characteristics of acute, severe depression, the other of chronic mild depression."

Lewis's unitary approach to the classification of depressive disorders remained unpopular in psychiatric quarters for many years. His views on the nosology and nature of depressive illnesses were not shared by his continental colleagues, although they were eventually espoused and promoted by Henri Ey (1954), a leading French psychiatrist.

### 1.3.2 The endogenous-reactive distinction

Two fundamentally different views of the nature of depression exerted a great influence on discussions about the relationship between endogenous and reactive depressions that started some fifty years ago between the Kraepelinians and the Meyerians. Those loyal to the Kraepelinian tradition adopted a dualistic approach and therefore argued strongly in favour of the distinction between endogenous (psychotic) and reactive (neurotic) forms of depression. In an important paper entitled "The Clinical Differentiation of Types of Depression" Gillespie (1929) reiterated and elaborated on Kraepelin's dichotomy. After a careful study of a group of clinically depressed patients, Gillespie concluded that reactive and autonomous or endogenous depressions are two distinct types of depressive illness. Gillespie's decision to

view endogenous and reactive forms as two distinct disease entities was based on symptomatic data. The results of his study showed that patients diagnosed as reactive depressives were characterised by mood fluctuations and reactivity to environmental changes. Those diagnosed as endogenous depressives, however, displayed a different type of symptomatology whose major characteristic is non-responsiveness to external or environmental influences.

Although Gillespie's notion, that endogenous and reactive depressions can be separated on the basis of reactivity to the environment, may be (theoretically) sound, when applied it failed to discriminate adequately between the two clinical conditions. Despite lack of evidence supporting this symptomatic approach, clinicians and researchers continued to use Gillespie's 'reactivity' as their major diagnostic criterion.

Until some twenty years ago the decision to classify depressive illnesses into endogenous or reactive was based either on clinical symptomatology or on treatment response. However, the refinement of psychometric techniques and the application of sophisticated statistical methods in recent years has offered a sound scientific basis for such nosological classification. Indeed, researchers on both sides of the Atlantic have enthusiastically applied multivariate analytic techniques to all sorts of data (including epidemiological data) to test the classification model inherited from Kraepelin and Gillespie. Although some of their attempts may have been hindered by obvious methodological constraints, their results have not been inconclusive.

In what is now known as the Newcastle school, Roth and his colleagues devoted most of their time to investigating the endogenous/reactive or neurotic issue. In their major study, Carney, Roth, and Garside (1965) subjected a set of data, obtained from a sample of 129 clinically depressed patients diagnosed as endogenous or neurotic, to multiple regression analysis and found evidence supporting the endogenous-neurotic dichotomy. The results of their study clearly showed that the distribution of symptom scores was bimodal, although subsequent attempts to replicate their results have apparently failed, (Kendell, 1968; Post, 1972).

A series of factor analytic studies (Killoh and Garside, 1963; Rosenthal and Klerman, 1966; Hamilton and White, 1958; Rosenthal and Gudeman, 1967; Mendels and Cochrane, 1970; Carney, Roth, and Garside, 1965; Hordern, 1965) reviewed by Mendels and Cochrane (1970) have also reported evidence supporting the distinction between the endogenous and neurotic types of depression. Their review showed that the following symptoms or items loaded positively on the endogenous factor: (a) depth of depression, (b) retardation, (c) loss of interest in life, (d) non-responsiveness to environmental changes, (e) visceral symptoms, (f) absence of precipitating stress, (g) weight loss, and (h) insomnia. It is evident that the clinical picture suggested by the symptoms listed above is that of endogenous depression.

In sum, most factor analytic studies found evidence for the existence of a boundary between 'endogenous' and 'neurotic' depressions. The studies also appeared to have clearly described and positively identified a specific endogenous state. However,

as Costello (1970) and Kendell (1976) noted, relatively a few studies produced factors corresponding to the ill-defined 'neurotic' type of depression. In short, agreement has been reached on the endogenous-neurotic distinction and the existence of an endogenous type of depression. But the definition and classification of 'neurotic' depression is open to debate.

### 1.3.3 The unipolar-bipolar classification

The unipolar-bipolar classification was originally proposed by Leonhard (1959) to reduce the ambiguities and semantic confusion generated by Kraepelin's concept of manic-depressive psychosis. The diagnosis label of bipolar depression is essentially given to patients who have experienced both manic and depressive episodes (alternating mania and depression), and that of unipolar is given to patients who have had successive episodes of either mania or depression (recurrent mania or recurrent depression). Unlike the previous classifications, which are based either on aetiological considerations (endogenous/psychogenic) or on clinical symptomatology (e.g., reactivity to environmental changes and constraints), the unipolar-bipolar classification is made on the basis of anamnestic data. In one of the studies supporting the unipolar-bipolar distinction, Perris (1976) found significant personality and epidemiological differences. The results of this study showed that bipolar depressive patients tend to display a 'syntonic personality pattern', an extrovert type of personality; in contrast, the unipolars were found to be characterised by an 'asthenic personality pattern', an introvert and anxious type of personality. Perris also found that bipolar depression starts

ten years earlier than the unipolar one.

Another important finding reported by Angst (1966) in support of the unipolar-bipolar classification, concerns the incidence and frequency of affective disorders amongst relatives of unipolar and bipolar depressive patients. The results of his study revealed that the risk of developing unipolar depression is higher amongst close relatives of bipolar patients than those of unipolar depressive patients. More recent studies, however, failed to replicate this finding (Reich, Clayton, and Vinokur, 1969; Helzer and Vinokur, 1974). Instead, the studies showed that relatives of bipolar patients are more likely to develop unipolar illness than bipolar illness.

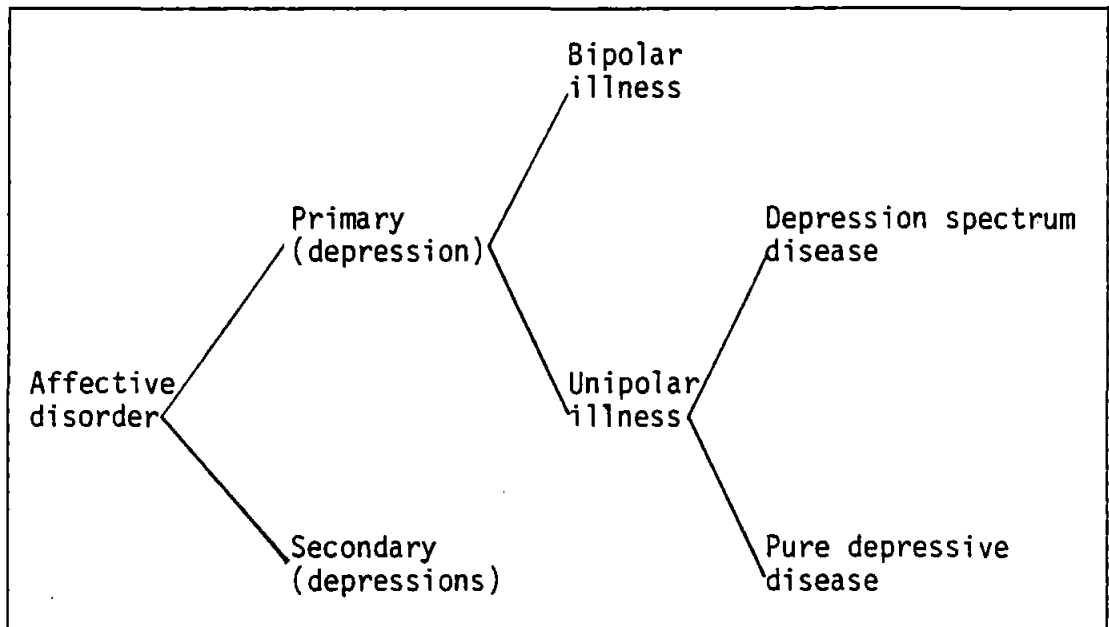
Although significant differences were found in premorbid personality and familial history, more evidence regarding both clinical symptomatology and pharmacological response should be provided if the unipolar and bipolar depressions are to be regarded as separate clinical entities and Leonhard's classification be useful and acceptable.

#### 1.3.4 The primary-secondary classification

The primary-secondary classification may be said to be a refinement of that of Leonhard. Unlike the previous one, it makes a useful distinction between those depressive illnesses preceded by psychiatric disorders and personality disturbances (secondary depression) and those depressions which are not preceded by any known psychiatric or personality disorder (primary depressions).

Table 2 illustrates this classification.

TABLE 2. The primary-secondary classification (adapted from Kendell, 1976)



As can be seen in Table 2, the primary-secondary nosological model disregards all those depressions contaminated by or associated with physical illnesses and major personality disorders. Having separated primary and secondary affective disorders, Robins and his colleagues (1972) then subdivided the former ones into bipolar (consisting of both depression and mania) and unipolar depressions (consisting of depressive illnesses only). By further considering the unipolar type, the authors made a very useful distinction between what they called 'depression spectrum disease' and 'pure depressive disease'.

The distinction between these two sub-types is supported by anamnestic studies. Winokur (1974) found that patients of the 'depression spectrum disease' category experience their first



depressive episode before the age of 40, those of 'pure depressive disease' category develop a depressive illness before the age of forty.

An important feature of this classification model is its clarity and flexibility. More importantly, the model allows precise operational definitions and as such it provides a useful nosological framework for both clinicians and researchers of depression.

### 1.3.5 The dimensional classification

In his initial study on "The Classification of Depressive Illnesses" Kendell (1968) employed a series of multivariate analytic techniques in an attempt to differentiate between the psychotic and neurotic forms of depression. The data obtained from 1,080 patients diagnosed as psychotic, involutional, or neurotic depressives, was subjected first to discriminant function analysis then to factor analysis. The results showed that, although there was a tendency for psychotic depressives to obtain high scores and neurotic depressives to obtain low scores, the distribution of symptom scores was unimodal. Accordingly Kendell concluded:

"Discriminant function analysis provides no support either for the hypothesis that neurotic and psychotic depressions are qualitatively distinct or for the hypothesis that involutional melancholia is an independent entity". (p.31)

Following his first unsuccessful attempt to demonstrate bimodality between psychotic and neurotic depressions, Kendell subjected his clinical ratings to factor analysis. Once again he failed to produce any evidence for the psychotic-neurotic dichotomy. In his final attempt to solve the issue, Kendell employed, in the

same study, Eysenck's criterion analysis method to a set of his data. Here again the analysis showed no clear cut boundaries between the two types of depression.

In spite of repeated failures to separate the psychotic and neurotic depressions, Kendell maintained that 'a valid boundary' between the two types can be demonstrated if only the diagnostic techniques were refined and their reliability enhanced. However, when a subsequent study by Kendell and Gurlay (1970) yielded similar results to the previous one, Kendell (1976) abandoned his arguments for a dichotomy and adopted a continuum view of depressive illness.

Kendell's model offers a sort of compromise (Fowles & Gersh, 1980) in that it relatively satisfies both the separatists and non-separatists:

"Regarding depressive illness as a psychotic/neurotic continuum is a convenient way of acknowledging the apparent lack of any valid boundary between type A (psychotic) and type B (neurotic) illness, yet at the same time acknowledges that the differences - in symptomatology, premorbid personality, treatment response and lifetime course - between the two extremes are too extensive to be regarded as differences in severity and chronicity." (p.19, 1976)

But as Kendell later conceded, a two-dimensional with one dimension expressing psychoticism and the other representing neuroticism (Eysenck, 1970), may even 'do more justice' to the diversity and complexity of depressive symptomatology than one-dimensional model.

#### 1.3.6 Concluding remarks

Research on the nature and classification of depressive disorders

has been the battle-ground for Kraepelinians and Meyerians since the early days of modern psychiatry. This selective review of the relevant literature showed that the disputes between researchers and clinicians of rival schools were more about how depressive disorders should be classified than on how they should be approached or treated. While some based their classification on aetiological considerations, others used clinical symptomatology as their main nosological criterion. These differences in both theoretical orientation and empirical consideration promoted a long debate which confused and at times obscured the nosological status of depression. But despite the early confusion, agreement has been reached that depressive disorders should be classified on the basis of symptoms and history. Agreement has also emerged on the existence of an endogenous type of depression.

With regard to the issue of whether depressive disorders should be portrayed by a typology or dimensions, there seems to be an emerging consensus that the former classification system should be adopted despite its obvious limitations (cf. Kendell, 1976). Part of the reason is that the typological system fits better with most systems adopted in other allied medical and scientific disciplines than the dimensional one. In addition to its familiarity, the typological or categorical classification is easy to understand and use. And as such it facilitates communication between researchers and clinicians of different theoretical persuasions.

## CHAPTER TWO

### DEPRESSION: DIAGNOSIS AND ASSESSMENT

2.1 INTRODUCTION

2.2 RATING SCALES AND ASSESSMENT OF DEPRESSION

2.3 RATING SCALES AS DIAGNOSTIC INSTRUMENTS

2.4 TYPES OF DEPRESSION RATING SCALE

2.5 CONCLUDING REMARKS

## 2.1 INTRODUCTION

Diagnosing depression is recognized to be almost as problematic as classifying it. It is, thus, surprising that clinicians and researchers should devote sufficient attention to the nosological problem but not to the assessment problem. Indeed, the literature on depression is almost devoid of theoretical discussions of the issues relevant to the diagnosis and assessment of depression. The present chapter is concerned with the important problem of the assessment of depression. Specifically, this chapter focuses on the major assessment instruments utilised in measuring depressive disorders and symptomatology.

## 2.2 RATING SCALES AND ASSESSMENT OF DEPRESSION

In recent years a large number of rating scales have been developed and used by clinicians and researchers in the clinical assessment of depression. The construction of these and their introduction to the clinical practice is seen as an attempt to satisfy an urgent need in clinical psychiatry for objective and standard measures of depressive symptomatology and psychopathology. Indeed, the poor reliability and validity of psychiatric diagnoses caused considerable concern and attracted sharp criticism from different quarters (cf. Kendell, 1975). The development of instruments capable of measuring validly and reliably the intensity and severity of disorders is regarded as essential for adequate therapy and research.

Generally, depression rating scales consist of several items covering a wide range of symptoms commonly associated with depression. A common feature to all these scales is that depression is defined and conceptualised in terms of its clinical, phenomenological, and behavioural components. Although depression rating scales may have some limitations, overall they may be said to be psychometrically sound measures of depressive symptoms. Their extensive use in both research and treatment programmes attests to their utility and popularity as assessment instruments for depressive phenomena.

### 2.3 RATING SCALES AS DIAGNOSTIC INSTRUMENTS

Although some of these scales were originally designed to assess the degree of severity or intensity of depressive disorders, they were later modified and refined to perform additional functions. For instance, both the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, and Erbaugh, 1961) and the Zung Self Rating Depression Scale (SD) (Zung, 1965) are being used as screening tools in a systematic manner in most research studies (e.g., human helplessness and depression studies). In spite of the criticism voiced against this use (e.g., Dupue and Monro, 1978), researchers continue to use these scales both for diagnosing depression and measuring the level of its severity and intensity. Authorities such as Beck, Lewinsohn, and Seligman are only a few amongst those who defied such criticism and continued to use these scales as screening tools. While classifying depressive disorders may still be problematic,

diagnosing depression appears to be less so, for a diagnostic decision is based on information which, if not elicited by the traditional interviewing methods, is conveyed by rating scales. Most rating scales describe depression in terms of its cognitive, affective, behavioural, and somatic features. The presence of this cluster of symptoms is interpreted by most clinicians and diagnosticians as an indication that the patient is suffering from a depressive illness of clinical proportions. Although some scales provide a fairly good description of the patient's clinical condition (i.e., detection of symptoms), others, however, fall short of their mission. In an article entitled 'Depression Rating Scales', Carroll, Fielding, and Blashki (1973) listed five reasons that may account for the poor performances of these scales: lack of sensitivity, biased orientation, limited utility, limited specificity, and limited information access.

- (a) The sensitivity of a scale refers to its ability to discriminate between varying degrees of severity of illness i.e., from severe to symptom free. The scales that lack sensitivity often fail to distinguish between adjacent degrees of severity (i.e., between mild, moderate, severe, very severe). Scales lacking this property are not recommended for outcome studies, since they fail to register effectively the changes that may occur following the administration of treatment.
- (b) Orientation is another characteristic of a rating scale that may affect its performance. Some scales have a

biased orientation in that they emphasize one aspect of the illness at the expense of another. For instance, the Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1960) stresses the somatic aspect of depression, whereas the Zung Self-Rating Depression Scale (SDS) (Zung, 1965) emphasizes the subjective component of this illness.

- (c) Information access concerns the amount of information that a scale may convey. More specifically it refers to the inability of a scale to assess some features of an illness. For instance, the items making up some (self) rating scales do not cover the wide range of somatic symptoms.
- (d) The utility of a scale refers to the ease with which the patient (for self-assessment scales) or the clinician (for observer rating scales) uses it. For instance, Carroll and his colleagues (1973) reported that their group of severely depressed patients took between 15 to 20 minutes to complete the SDS. Utility is an important feature that is closely related to information access. Improving the former (utility) may reflect negatively on the latter.
- (e) The specificity of a scale refers to its power to differentiate between two psychiatric or personality disorders. This property is essential for scales that are usually used as screening or diagnostic



instruments. The BDI is reported to have effectively distinguished between anxiety and depressive states (Beck, 1969).

## 2.4 TYPES OF DEPRESSION RATING SCALE

The importance of diagnosis in clinical practice has been recognized since the early stages of the medical discipline. An adequate diagnosis is still considered by most, although not all, clinicians and investigators as an essential pre-requisite to both treatment and research. Although some may persist in claiming objectivity and validity of their clinical judgements and diagnostic evaluations, evidence amassed in the last twenty years tends to refute such claim (cf. Kendell, 1975). The subsequent development and use of different rating scales was an attempt to remedy to this situation. Two types of rating scale have been constructed and employed in the assessment of depressive phenomena: observer rating scales and self-rating scales.

### 2.4.1 Observer-rating

The most popular observer rating scale is that developed by Hamilton (1960, 1967). The Hamilton Rating Scale for Depression (HRSD):

"has been devised for use only on patients already diagnosed as suffering from affective disorder of depressive type". (p.56, 1960).

When used by a trained observer, this scale provides a quantitative assessment of the intensity and severity of the depressive

illness.

The HRSD is composed of 17 items covering the cognitive, behavioural, and somatic aspects of depression. Amongst the symptoms included in this scale are depressed mood, guilt, suicide, different types of insomnia, retardation, agitation, anxiety, hypochondriasis, loss of weight, and other somatic manifestations. Almost half of these symptoms are rated on a 5-point scale (0 - absent, 1 - mild, 2 and 3 - moderate, 4 - severe). The remaining symptoms are rated on a 3-point scale (0 - absent, 1 - slight or doubtful, 3 - clearly present). Hamilton recommends that the scoring should be done by two independent raters or clinicians. In his original study, Hamilton reported a good inter-rater reliability ( $r = .90$ ,  $p < .001$ ).

In their study, Carroll and his colleagues (1973) compared the HRDS and the Zung SDS and found the performance of the former much more superior. They agreed with Hamilton that:

"the scale ... fulfils its purpose of providing a simple way of assessing the severity of a patient's condition quantitatively, and of showing changes in that condition". (p. 276, 1967).

Because of its established validity and good record of inter-rater reliability the HRDS is now widely used, together with self assessment scales such as the BDI, as an outcome measure in therapeutic as well as pharmacological treatment of depression (e.g. Shaw, 1977; Ruch, Beck, Kovacks, and Hollon 1977).

Unlike the HRDS, the Raskin Depression Scale (Raskin, Schulterbrandt,

Reatig, and McKeon, 1970) and the Cutler and Kurland Depression Scale (Cutler and Kurland, 1961) are not widely used in this country. Although their authors claim high rates of inter-rater reliability, their validity and utility both for clinical and research purposes is very limited.

#### 2.4.2 Self-rating

A substantial number of self-rating scales have been specifically developed for use with different categories of depressed patients. The construction of these scales is based on the general assumption that patient's evaluations of himself are congruent with that made by clinicians; that is the patient is considered to be as able as the clinician in describing his symptoms. While this congruence may hold at moderate levels of severity of the illness, it is very difficult to conceive of such a consistency at severe or very severe levels of the illness (Carroll et al, 1973; Hammen, 1981).

Self-rating depression scales have achieved a high level of popularity in the last few years. They have been used with both clinical and non-clinical populations for both therapy and research purposes. Learned helplessness investigators have shown a special preference for this type of instruments; they have been the heavy users of the Beck Depression Inventory (BDI).

##### 2.4.2.1 The Beck Depression Inventory

The Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, and Erbaugh, 1961) is perhaps the most popular of the

self report measures of depression. Like the HRSD, the BDI:

"has been developed to provide a quantitative assessment of the intensity of depression". (p.569, 1961).

Although originally devised to measure the severity of depressive manifestations, the BDI is now also used as diagnostic instrument. There is evidence suggesting that this scale can distinguish quite effectively not only between various degrees of severity (e.g., between severe and very severe clinical condition) (Beck et al 1961; Metcalfe and Goldman, 1965) but also between non-clinically depressed and non-depressed persons (Bumberry, Oliver, and McLure, 1978). The validity and reliability of the BDI have been confirmed by a number of studies carried out in America (Beck, 1967; Beck and Beamesderfer, 1974), in Britain (Metcalfe and Goldman, 1965), and in France (Delay, Pichot, Lemperiere and Mirouze, 1963).

The BDI consists of 21 groups of symptoms. Each symptom is represented by four to five self-evaluative statements and scored on a range from 0 (absence) to 3 (severe). The patient is asked, if not assisted in the completion of the scale, to read each group of statements and then circle the number of the statement(s) that best describes him at the moment of the interview (here and now). The total score of the patient is obtained by summing up the scores of each group of statements. The larger the score the greater the severity of the patient's condition.

The BDI has been praised for its sensitivity and specificity (e.g., (e.g., Delay et al., 1963). Its apparent ability and power in

distinguishing between depression and other clinical conditions (Beck, 1967) make the BDI suitable for use, as a screening tool, with both clinical and non-clinical populations. In spite of its apparent superiority over other instruments, the utility of the BDI may sometimes be limited. Because as Beck and his colleagues concede:

"its applicability depends on the co-operation of the patient as well as his ability to comprehend the items". (p. 569, 1961).

#### 2.4.2.2 The Zung Self-Rating Depression Scale

The Zung Self-Rating Depression Scale (SDS) (Zung, 1965) is not as widely used or as popular as the BDI, partly because it lacks sensitivity (e.g., Carroll et al., 1973). The SDS consists of 20 items which cover the affective, psychological and somatic manifestations of depression. When administered, the patient is asked to indicate on the scale the frequency (not the severity of the illness) with which he experiences the symptoms listed, by rating 'a little of the time', 'some of the time', 'good part of the time', and 'most of the time'. One of the deficiencies of the SDS that has been reported is its low level of sensitivity. Indeed, Carroll and his co-workers reported data suggesting that the SDS is:

"incapable of discriminating effectively between the three sub-groups of (depressed) patients" (p.364, 1973),

although Zung claims the contrary (e.g. Zung, 1965, 1967). The lack of sensitivity may be explained by the fact that the scale is originally designed to assess the frequency and not the

severity of the symptoms. The SDS may also be said to lack specificity because both anxious and depressed patients obtain high scores. This failure to discriminate effectively between two diagnostic entities constitutes one of its major drawbacks. Despite these rather serious limitations, the SDS, when used in conjunction with valid observer or self-rating scales, may be less misleading and may even provide valuable information.

The Minnesota Multiphasic Personality Inventory Depression Scale (MMPI-D) is another instrument which was developed to measure the depth of depression. Originally, the scale was composed of 60 items that cover the following depressive manifestations:

"pessimism of outlook on life and the future, feelings of hopelessness or worthlessness, slowing of thought and action, and frequent pre-occupation with death and suicide".

Factor analytic studies of the MMPI-D-60 revealed that the 60 items making up this scale are lacking homogeneity (e.g., Comrey, 1957). Accordingly the scale was reduced to half its original size. Although psychometrically more sound and acceptable, the MMPI-D-30 still suffers some deficiencies. For instance, the scale does not cover the wide range of somatic symptoms that are usually associated with depression. In addition, to this biased orientation, the MMPI-D-30 fails to discriminate effectively between various degrees of severity of the depressive state.

Other self-assessment scales such as the Depression Adjective Check List (Lubin, 1965) and the Multiple Affect Check List

(MAACL) (Zuckerman and Lubin, 1965) have also been devised to quantify depression. These instruments appear to be more concerned with the subjective aspect of depression than with its clinical features. Although not developed for diagnostic purposes, both the DACL and the MAACL seem to be quite effective in detecting symptoms of depression (e.g., Hammen, 1981).

#### 2.4.3 Observer-rating versus self-rating scales

One of the major deficiencies of self-rating scales is their limited utility. No matter how precisely or clearly the symptoms are described and defined misinterpretation and non-comprehension are bound to occur. It is evident that the performance of this kind of scale and its applicability depend in the first place on whether the patient is willing to co-operate and on whether he is able to comprehend the instructions and the items making up each scale. The performance of a self-assessment scale depends also on how well it is designed, but as Carroll and his colleagues remarked:

"Even the best designed self-rating scale will nevertheless suffer from distortion of information when applied to patients with psychotic illness who have impaired perception and testing of reality". (p. 364, 1973).

The major drawback of observer-rating scales is the bias of a rater. Even a well-trained rater falls victim to his expectations. It is a well-known fact that patients at admission (hospitalisation) are rated as more severely ill than those already hospitalised, not because of differences in clinical condition but because patients are expected to be more ill before than after hospitalisa-

tion (e.g., Snaith, 1981). Despite the problems inherent to both types of scale, they remain very useful. With reasonable assistance to the patient (in his completion of the scale) and with sufficient training of the rater, reliable results may be obtained and adequate assessment of depressive features may be provided by both observer-rating and self-rating scales. It is only by adopting appropriate assessment procedures that more progress could be made in the theoretical understanding and management of depressive disorders.

## 2.5 CONCLUDING REMARKS

Before concluding this brief review perhaps it is worth saying a word on how most of these instruments tend to portray depression. Some of the self-assessment scales seem to describe depression as a mood state, others tend to conceptualise it as a symptom. Although by no means congruent with clinical descriptions of depression, these views are still popular in some quarters. But most rating scales are now concerned with depression not as a symptom but as a clinical syndrome. While there may still be disagreement as to the nature of depression there seems to be a large consensus on its nosological status. A common feature to the assessment measures reviewed here is that they view depression as a unitary phenomenon, a view which is implicitly promoted by contemporary psychological theories of depression (e.g., learned helplessness theory of depression; loss of reinforcement theory of depression).



### CHAPTER THREE

#### DEPRESSION: AETIOLOGY AND DEVELOPMENT

##### 3.1 INTRODUCTION

##### 3.2 EARLY AETIOLOGICAL FORMULATIONS

##### 3.3 CONTEMPORARY PSYCHOLOGICAL THEORIES OF DEPRESSION

### 3.1 INTRODUCTION

Perhaps there is as much disagreement on the aetiological issue of depression as on the question of its nosological status. This may be partly because aetiological inferences were made on the basis of inadequate or arbitrary nosological classifications and vice versa. Another reason that may explain this long dispute is the imprecise definition or meaning of the term depression. Indeed, the term depression denotes different things to clinicians and researchers of different theoretical persuasions. For those in the psychoanalytic tradition, depression refers more to an affect than to a clinical condition; for those with an organic orientation, depression is more than an affect - it refers to a clinical syndrome involving a wide spectrum of symptoms including affective, cognitive, behavioural, and somatic symptoms. Besides the semantic confusion which surrounds this area of affective disorders, there is a lack of consensus on the research strategies that might be adopted in the study of depressive disorders. There are, as Akiskal and McKinney (1975) rightly point out, those who:

"favour 'understanding' depression over objective description of observable signs and symptoms", (p.286),

and there are those who over-emphasize clinical descriptions at the expense of theoretical understanding. These differences in both theoretical orientation and empirical consideration gave

rise to divergent views about the aetiology or causation of depressive illness. Those who adhere to a psychogenic view of depression, emphasize the aetiological significance of psychological factors; those who subscribe to a biological view, in contrast, trace the depressive symptomatology and manifestations to organic or biological events. Although recently reconciled and integrated into a comprehensive psychobiological model (Akiskal, 1980; Akiskal and McKinney, 1975), these two contrasting conceptions of the origin and aetiology of depression still dominate the literature of affective disorders.

A substantial number of psychological hypotheses and theories have been advanced to account for both subjective and clinical manifestations of the depressive illness. As might be expected, theories rooted in the psychoanalytic tradition explain the psychopathology of depression in terms of personality or psychodynamic factors and development events. Although mainly formulated in id psychology terms (libidinal stages), these theories still exert a considerable influence on contemporary thinking about the phenomenon of depression.

More recent cognitive and behavioural formulations of depression appear to enjoy more popularity in both clinical and academic communities. Unlike traditional psychoanalytical theories, the more recent ones are less speculative and more importantly lend themselves easily to experimental and empirical verification or evaluation. But despite this relative superiority, both cognitive and behavioural theories suffer from serious conceptual

inadequacies.

Because of similarities in emphasis and conceptualisation, psychoanalytic theories will be reviewed and evaluated separately. To do them more justice they will be reviewed in the 'historical' context in which they have evolved.

### 3.2 EARLY AETIOLOGICAL FORMULATIONS

Despite its substantial achievements in the nosological domain of mental disorders, clinical psychiatry remained a 'pseudoscience', at least until the birth of psychoanalysis. The emergence of this now different discipline gave psychiatry a new breath. The originality of psychoanalytical theory lies, as Bompard (1980) pointed out, in:

"its insistence that mental illness was not simply the outward manifestation of cerebral pathology, but that its symptoms were psychological in origin and had meaning." (p.15)

Until the birth of psychoanalysis, providing an adequate nosological classification of psychiatric disorders appeared to be the main RAISON D'ÊTRE of most, if not all, psychiatric investigators. Freud, in contrast, devised an analytic technique the aim of which was not only to describe or classify symptoms and syndromes but also, and more importantly, to uncover their causes and meanings. For Freud and his followers, symptoms are more than manifestations of an underlying pathology, they are symbolic representations of latent unconscious conflicts.

The important claim by Freud that mental disturbances could be understood in terms of unconscious mental processes led to

criticism and eventually to the rejection of the widely adopted descriptive psychopathology. Accordingly, Freud's extensive theoretical formulations of anxiety, hysteria, ego defences and unconscious shifted away attention from nosological pre-occupations to more important questions regarding the aetiology and the nature of psychiatric disorders. The subsequent psychoanalytical investigations of unconscious processes and motives resulted in aetiological formulations that facilitated both the understanding and management of psychiatric disorders.

Although depression was not at the top of the list of psychological disorders investigated by psychoanalysts, it has nevertheless received considerable attention in the psychoanalytic literature. Early psychoanalytic writers such as Abraham, regarded depression as an affect resulting mainly from excessive repression of libido. Just like in other disorders (e.g. neurosis) the emphasis was put on libidinal stages. Unlike the traditional libidinal orientation which, as already pointed out, attached great importance to libidinal strivings, the ego psychology orientation emphasised the ego's awareness of its sense of helplessness and its perceived inability to fulfil its narcissic aspirations. Psychoanalysts with ego psychology orientation conceptualise depression as an ego state characterised by its feelings of powerlessness, helplessness, and low self-esteem.

Despite the apparent theoretical attractions of both traditional and more recent or contemporary psychoanalytical formulations of depression, clinicians remained sceptical as to the possibility of these theories fitting the complicated clinical picture of the

depressive condition, and explaining or accounting for the wide spectrum of depressive symptomatology.

### 3.2.1 THE HOSTILITY TURNED-INWARD MODEL

An initial attempt at providing a psychoanalytic formulation of the aetiology of depression was made, not by Freud as some believe, but by Abraham in 1911. In this first psychoanalytic paper on depression, Abraham used two key concepts, borrowed from Freud, to explain the nature and the origin of this affective disorder: the concept of libido and that of repression. In accordance with Freud's theoretical formulations of psychosexual development, Abraham conceptualised depression as a chronic fixation of the libido at an archaic or early developmental stage. More specifically, Abraham regarded depression as an affect resulting mainly from an excessive repression of libidinal desires and instincts. The depressive person is seen in this model as excessively dependent on others and the environment for love, happiness and security.

Although theoretically sound, Abraham's view on depression was not shared by his colleagues. In fact, he was openly criticised by Freud for putting too much emphasis on libidinal stages. Following the publication of Freud's influential paper, 'Mourning and Melancholia' in 1917, Abraham revised and expanded his theoretical propositions vis a vis depression.

Abraham's subsequent work reflected the strong and profound influence that Freud's formulations on melancholia had exerted on him. Accordingly, depression was no more conceived as a

state of retarded or blocked libido, but as an affective state due to the introjection of hostility originally destined to the ambivalently loved object. This is how Abraham described the process leading to the redirection of hostility and anger against the ego:

"When melancholic persons suffer an unbearable disappointment from their love-object they tend to expel that object as though it were feces and to destroy it. They thereupon accomplish the act of introjecting and devouring it - an act which is a specifically melancholic form of narcissitic identification. Their sadistic thirst for vengeance now finds its satisfaction in tormenting the ego". (Abraham, 1924).

Amongst Abraham's other contributions to the theoretical understanding of depression, were his important propositions concerning the predisposing factors to the onset of depression. Abraham viewed oral dependency, a sort of thirst for love, as the characteristic feature of the depressive personality. He postulated that 'primal parathymia', traumatic experiences in childhood, plays an aetiological in the pathogenesis of depression. He maintained that the reactivation of childhood losses later in life is the critical factor in the development of depressive illness:

"In the last resort melancholic depression is derived from disagreeable experiences in the childhood of the patient". (Abraham, 1924).

The depressogenic effects of object loss have also been recognized by Freud. He insisted that the loss need not have happened in childhood, and the lost object need not have died for depression to develop and emerge:

"In melancholia the occasions which give rise to the illness extended for the most part beyond the clear case of loss by death, and include all those situations of being slighted, neglected and disappointed which can impart opposed feelings of love and hate into the relationship or reinforce an already existing ambivalence". (Freud, 1917).

The importance of object loss both as a predisposing and precipitating factor to the development of depression has been stressed in Freud's 'Mourning and Melancholia', his major piece of work on the origin and the nature of depressive disorders. In this classic paper, Freud drew a sharp parallel between the state of mourning and the clinical condition of melancholia. He found similarities not only in antecedent conditions but also in affective or emotional manifestations. A common feature to both mourning and melancholia is that they both develop and emerge as a reaction to a sudden loss of a loved object. More common to both states, are the sorrow and the sadness triggered by loss, the pathological self-reproaches and criticism, the loss of energy, and the lack of interactions and interest in outside world. However, the critical difference, according to Freud, is that in mourning the loss is external, whereas in melancholia the loss is internal (unconscious):

"In grief the world becomes poor and empty; in melancholia it is the ego itself".

Freud identified further differences in the way the loss is handled in both melancholia and mourning. He insisted that in the latter state, the anger arising from feelings of resentment and desertion is directed toward the lost object (the object actually being a person); in melancholia, however, the anger is



directed internally since the lost object is introjected (hence the pathological self-criticisms).

Freud regarded melancholia as a state of pathological mourning. He argued that the libido is the major factor which determines the course that the experience of mourning will take - whether it will be 'healthy' or pathological mourning (melancholia). Freud observed that in normal mourning the free libido (that is the libido previously invested in the lost loved object) is actively re-invested in another subject; in melancholia, however, the free libido is not re-invested in any external object, but introjected or withdrawn into the ego. Freud considered the identification with and the introjection of the lost object as critical to the development and manifestation of depressive disorders. This is how he described the process that eventually provokes depressive illness:

"An attachment of the libido to a particular person, had at one time existed; then owing to a real slight or disappointment coming from this loved person, the object relationship was shattered....the free libido was not displaced on to another object; it was not employed in any unspecified way, but served to establish an identification of the ego with the abandoned object. Thus, the shadow of the object fell upon the ego, and the latter could henceforth be judged by a special agency; as though it were an object, a forsaken object. In this way an object loss is transformed into an ego loss and the conflict between the ego and the critical activity of the ego and the ego as altered by identification". (Freud, 1917).

For Freud, the introjection of the disappointing object and the hostility associated with it breeds depression. Because the hatred and the criticism destined to the love object are now, by means of both mechanisms of identification and projection,

directed against the self. This discharge of anger against the self engenders feelings of dysphoria, inadequacy, guilt, sadness, and eventually depression. For Freud, the introjection or retroflexion of anger is the SINE QUA NON of depression.

The hostility-turned-inward model, initiated by Abraham then expanded and refined by Freud, is regarded as the major psycho-analytical contribution to the theoretical understanding of the phenomenon of depression. Although this model enjoys respectability and popularity in some quarters, it has been criticised both on theoretical and empirical grounds. Critics pointed out that Freud failed to say how depression differs from other affects that also result from the introjection of hostility. Perhaps the major weakness of this model lies in its failure to account for all aspects (subjective as well as clinical) of depression. In fact, some argue that Freud's model has little or no relevant relationship to the clinical picture of depression (e.g. Akiskal & McKinney, 1975).

Attempts at externalising or re-directing hostility toward external objects, have not produced any significant improvement or change in the level of depression (e.g., Klerman and Gershon, 1970; Wadsworth and Baker, 1975). A final point that needs to be made about the conceptual 'inadequacy' of this model, is that contrary to Freud's tendency to assimilate depression to hostility turned inward, contemporary research has provided evidence showing that both depression and hostility are distinct affects that can co-exist within the same person. In other words,

one can experience hostility without feeling depressed.

### 3.2.2 THE LOWERED SELF-ESTEEM MODEL OF DEPRESSION

The lowered self-esteem model of depression abandoned the widely adopted libidinal approach to emphasise the role of the ego in the pathogenesis of depression. In this model, depression is viewed as an affect characterised by the collapse of self-esteem. The self-esteem model was first outlined by Finichel (1945), but it was Bibring (1953) who later developed it and refined it. "Bibring's theory" as Bompomad so rightly described it (1980), is a "paradigm of simplicity and clarity" (p.31).

Rather than conceptualising depression as 'a residue of libidinal strivings', as was the case with the proponents of id psychology, Bibring conceives of depression as an affect arising out of ego contradictions. For Bibring, the conflicts giving rise to depressive disorders are not between the ego and the super ego, but within the ego itself.

Although previous psychoanalytic writers mentioned self-esteem in relation to depression, they failed to recognise its importance in both the aetiology and development of depressive illness. Although Bibring recognises the importance of object loss and developmental events emphasised by both Abraham (1926) and Klein (1948), he maintains that depression is mainly a reaction to a loss or a blow to self-esteem:

"Depression can be defined as the emotional expression of a state of helplessness and powerlessness of the ego, irrespective of what may have caused the breakdown of the mechanisms which established self-esteem". (1953).

Central to this theory is the ego's awareness of its helplessness and powerlessness. For an individual to exhibit an acute sense of helplessness, he must, according to Bibring, perceive a discrepancy between his actual situation and his narcissic aspirations. The perceived inability to achieve or attain aspirations is apparently the mechanism which activates the fall of self-esteem and triggers depression.

Unlike other psychoanalytic theories of depression, the present one has been favourably reviewed by most clinicians and researchers. Part of the reason is that the self-esteem model is much closer to clinical reality in general and to the clinical picture of depression in particular. Another reason as to why Bibring's model was more acceptable is because its conceptual formulation is consistent with contemporary theorising and thinking about the phenomenon of depression. For instance, low self-esteem has been described as a characteristic feature of depressive illness in most clinical observations and reports. Similarly, the component of helplessness is central to one of the most recent and perhaps successful theories of depression (Seligman, 1974; Abramson et al, 1978). Another reason why Bibring's theory has had more success than traditional psychoanalytical formulations, is because it is less speculative and provides more acceptable descriptions and explanations of depressive syndrome.

But despite the apparent adequacy of the lowered self-esteem model of depression, questions arise as to its ability to explain

or account for all aspects of the clinical syndrome of depression. Low self-esteem could perhaps account for the subjective component of depression, but is unlikely to do so for the behavioural and somatic symptoms of this syndrome. In fact, recent theories of depression such as Beck's cognitive model (1967) and Seligman's learned helplessness theory (1974), regarded loss or low self-esteem as an epiphenomenon or a symptom of depression but not as its cause.

It appears after all that the construct of self-esteem is important but perhaps not sufficient to account for the wide range of depressive symptoms and disorders.

### 3.2.3 OBJECT LOSS, STRESS, AND DEPRESSION

There has been a widespread conviction that object loss plays an aetiological role in depressive illness. However, such belief and enthusiasm is hardly matched or justified by the existing empirical findings. The studies carried out so far in this line of research offer little or no support for the hypothesised causal connection between object loss and depression. Attempts at determining the nature of the relationship between these two variables have often been undermined by various methodological problems and constraints (see Tennant et al, 1981, for a detailed discussion of these problems).

Psychoanalysts are, perhaps, unanimous in regarding object loss as an antecedent of depressive complaints. The importance of developmental object loss in the pathogenesis of depression, has been emphasised by both Abraham (1926) and Freud (1917).

In fact, they both postulated a causal link between childhood loss and depression later in life. They regarded depression as an affective reaction to childhood losses.

The initial attempt at assessing the consequences of childhood loss on psychomotor functioning of human infants was made by Rene Spitz in 1945. Spitz reported to have thoroughly examined the reaction of a group of human infants that were separated from their mothers in the second half of the first year of life. The infants' reaction to the separation event, later termed 'anaclitic depression', was found to have similar features to that of a depressive reaction. Specifically, the infants' reaction was characterised by crying, psychomotor retardation, withdrawal, insomnia, anorexia, and weight loss. Although this finding may be regarded as a firm evidence of the depressogenic effects of object loss, some have challenged this interpretation. It was argued that the infants' morbid reaction reflected nothing but the consequences of an abrupt institutionalisation.

Although this may be true for the Spitz study, the syndrome that was soon after described by Robertson and Bowlby (1952) in older children, cannot be accounted for by the sole phenomenon of 'hospitalism' or institutionalisation. The separation syndrome reported by these authors consisted of three phases: Protest Phase - protest is believed to be the initial reaction with which the children respond to the loss of an important attachment bond (mother). In this stage the child is described as restless, agitated, and anxiously searching for his mother. Despair stage - in this stage the child is described as withdrawn,

helpless, and sinking into despair. Detachment stage - in this stage the child loses interest in the outside world; the rejection of the mother is imminent. But despite this seemingly convincing evidence of the relationship between parental loss and depression, clinicians questioned its validity. It was argued that maternal deprivation or separation does not necessarily result in depressive reaction. Given an appropriate maternal substitute, some have argued, most of the symptoms and disorders described by Spitz and Bowlby could be prevented. The work of Bettelheim in the 'kibbutz' in Israel illustrates the point about the prophylactic effects of maternal substitute. Attempts at linking adult depression to early object loss have also failed to provide unambiguous data. A study that managed to establish a connection between these two variables is that of Brown (1961). Brown found that 41% of 216 depressed patients examined reported a loss of a parent before the age of fifteen, compared with only 16% of a sample of medical patients. A more recent study by Brown and Harris (1978) went even further - they linked adult depression to a loss of mother before the age of eleven. Although their data showed that only 10.5% of the depressed women examined and 6% of normal women included in the study ever reported a loss of mother before eleven, they nevertheless emphasised its importance in the aetiology of depression:

"Thus, loss of mother before eleven may well permanently lower a woman's feeling of mastery and self-esteem and hence acts as a vulnerable factor by interfering with the way she deals with loss in adult life". (p. 240, 1978).

While the above two studies may seem to confirm the hypothesised causal relationship between childhood loss and depression later

in life, that of Beck and his co-workers (1963) rejects any aetiological implication for early or childhood loss. The results reported by Beck and his colleagues showed quite clearly that neither maternal nor paternal loss is related to depression. They found that parental loss (loss of mother or father in childhood) failed to distinguish a depressed patients group from a non-depressed patients group. Other authors such as Birchnell (1961; 1970a; 1970b) have linked childhood bereavement and other types of losses not only to depression but also to other forms of psychiatric disorders.

It appears then that neither clinical observations nor research reports support the claim for an aetiological role of childhood losses. In a recent review of the relevant literature, Tennant and his colleagues (1980) arrived at a similar conclusion. These authors refuted any claim for a causal link between parental loss and depression:

"We conclude that the current state of knowledge indicates that parental death in childhood on its own has little impact upon the risk of depressive illness in adult life". (p.298, 1980).

Stressful life events in adult life have also been considered of a great importance in the development of depression. Much of the research carried out in this area, consisted of showing that depressed patients experience more stressor events than non-depressed patients prior to the onset of depression. Most of the studies published so far reported results which showed only a weak association between stress and depression, although some have made wild claims about such relationship. The



most widely quoted study in this line of research is that of Paykel (1974). His results indicated that only 25% of the depressed patients studied experienced stress prior to their episode of depression; although a stronger link was later established between so-called 'exit events' and depression.

Despite the claims for a causal relationship between stressor events and depression, doubt must be expressed as to the possibility of these events playing an aetiological role in depression - there is a lack of evidence concerning the specificity of stress to depressive illness (Tennant et al, 1981). In fact, medically orientated research showed that depression is not the only disorder associated with aversive events.

Medical conditions such as coronary artery disease, myocardial infarction, peptic ulcer, rheumatoid arthritis, and even skin diseases have been linked to stressor events, (e.g., Rahe et al., 1964; Rahe & Lind, 1971). In another review of the studies that claimed to have established a causal relationship between stress and depression, Tennant and his colleagues (1981) once again refuted such claims:

"Our conclusion is that many of the studies from which a causal connection between life events and depressive illness is inferred are so weak methodologically that little can be made of them" (p.387).

It appears then that stress may be important, but other variables such as personality traits, cognitive styles, coping styles may be better predictors of depressive illness.

#### 3.2.4 SUMMARY AND CONCLUSIONS

Although not exhaustive, the present review has pointed to

'deficiencies' inherent in psychoanalytical theorising and thinking about depression. The theories reviewed here were found to be speculative and of little or no predictive value. As noted earlier, their extensive use of metapsychological terms and metaphorical concepts makes their experimental or empirical verification difficult if not impossible. Besides the various deficiencies from which they suffer, these theories have little or no relevant relationship to the clinical reality of depression. Nevertheless, psychoanalysts deserve recognition not only for initiating the psychological research into depression but also for giving respectability to this approach.

### 3.3 CONTEMPORARY PSYCHOLOGICAL THEORIES OF DEPRESSION

In recent years, a substantial number of psychological theories have been proposed to account for the aetiology and development of depressive phenomena. Unlike early psychodynamic formulations, contemporary psychological theories of depression have been almost entirely inspired by clinical practice and experimental research. Although the psychological research on depression and other affective disorders may have been intimidated by the discovery of antidepressant drugs in the fifties, it has regained its confidence and authority in the late sixties. Substantial developments in both learning theory research and cognitive psychology (e.g., Neiser, 1967; Skinner, 1957) have led to the introduction and adoption of new psychological research strategies for the study of clinical depression. Thus, research in this area of affective disorders ceased to be the monopoly of medically trained and minded people.

The adoption, in recent years, of cognitive and behavioural approaches to depression has altered if not revolutionised contemporary thinking about this clinical syndrome. Thus, depression is no longer conceptualized as an affect, but rather as a cognitive or a behavioural phenomenon in which the emotional or affective aspect is a secondary elaboration. The criticism and eventually rejection of the traditional affective approach gave an opportunity to both clinicians and researchers to reconceptualize depression and to adopt new and more fruitful research approaches. Accordingly, interest has been revived and new hypotheses concerning the nature, aetiology, and treatment of depressive illness have been systematically generated and tested.

The emergence in the late sixties of a number of psychological formulations of depression has been the outcome of many years of empirical and experimental work. A characteristic feature of these contemporary theories is that they regard depression not simply as an affect but as a syndrome dominated by its low or pathological mood, reduced motivation, behavioural deficits, and somatic manifestations. Although several theories have been advanced to account for all these depressive phenomena, only a few have survived the scrutiny of experts. The present review is concerned only with these kind of theories.

One of the theories that has been favourably reviewed in the psychological literature on depression is Beck's cognitive theory (1967, 1974). Based on his clinical interventions and observations, Beck argued that the recurrent theme in depressed

patients' verbalisations and dreams is that of negative thinking. Beck also observed that when depression is alleviated such negative thought pattern no longer persists or emerges. These two important observations formed the basis of Beck's theoretical viewpoint. Beck's cognitive conceptualisation suggests that depression is due to maladaptive cognitive processes. Specifically, all other depressive components whether they are affective, motivational, behavioural, or somatic are believed to be secondary to this cognitive disorder.

Behavioural theories such as Lewinsohn's model of reinforcement (1974) emphasise the specific relationships between depressives' behavioural repertoire and external or environmental events. The depressive in this model is seen as socially unskilled and as such he fails to elicit response or reinforcement from his social environment. Lewinsohn seems to refer to a sort of breakdown in the reinforcement system of the depressive. He considers loss of reinforcement to be the major antecedent of depressive states. More specifically, depressive behaviours and manifestations are regarded as the immediate outcome of an individual's low rate of response-contingent positive reinforcement.

A more recent aetiological formulation of depression proposed learned helplessness as a central feature of clinical depression. The learned helplessness model of depression (Seligman, 1974; Abramson, Seligman, and Teasdale, 1978) attributes depressive symptoms and phenomena to perceived response-outcome noncontingency.

According to this model, depression results from an inability to control environmental outcomes due to a previous experience with noncontingent reinforcement (loss of control). Like the previous two models, the present one has generated considerable amounts of empirical and experimental research in the last few years.

Although both cognitive and behavioural formulations claim to account adequately for all depressive components, empirical research shows that such claims are sometimes highly inflated. Although these formulations are more verifiable and less speculative than psychoanalytic formulations, they still have serious drawbacks. For instance, critics have described them as circular, for they have a tendency to account for depression in terms of its symptoms or consequences. These theories have also been criticised for other conceptual irregularities.

### 3.3.1 BECK'S COGNITIVE THEORY

Beck's theory of depression (1967, 1974) refutes the traditional affective approach, giving primary consideration to cognitive factors. Since the early days of modern psychiatry, the emphasis has been exclusively on the affective or emotional aspect of this syndrome. In the sixties Beck vividly questioned the validity of this affective approach and subsequently rejected it to adopt a cognitive framework. Beck's cognitive orientation has been regarded as a reaction to the neglect, by the classical psychoanalytic theory, of conscious cognitive processes.

Beck traces the roots of depressive disorders to a negative cognitive set. He postulated that depressive are characterised

by maladaptive or anomalous cognitive schemas that pre-dispose them to view themselves, the world or the environment, and the future in a negative way; the so-called cognitive triad. In this model, depression is considered as the outcome of this cognitive triad.

Beck argued that this negative cognitive set accounts not only for the patient's low self-esteem, but also for his reduced motivation and interaction with others, low performance, and somatic complaints. For instance, the negative view of the self - that is the depressed patient's belief that he or she is defective, bad and inadequate, accounts for the loss of self-esteem. The patient's negative self-perception derives from his tendency to interpret experiences in terms of his presumed personal deficiencies. Similarly, the negative view of the present and the future accounts for the cognitive and motivational deficits of the depressive states.

According to Beck, all depressive components, whether they are affective, motivational, behavioural or somatic are the outcome of negative conceptualisations (the cognitive triad).

The second important element in Beck's cognitive theory of depression is that of negative schemas or 'silent assumptions'. These specific schemas consist of unspoken rigid rules - the so-called silent rules or assumptions. These rules are believed to give rise to negative conceptualisations of experiences and to inaccurate or biased perceptions of ongoing environmental stimuli.

Beck established that the depressive's unrealistic negative attitudes develop as a result of his tendency to interpret reality in terms of latent anomalous schemas. According to Beck these depressogenic schemas form the basis of the cognitive triad.

Another factor which, according to Beck, plays an important role in the development and maintenance of depressive states, is that of logical errors.

He identified the following logical errors in depressed patients' thinking patterns: arbitrary inference, selective abstraction, overgeneralisation, magnification or minimisation, personalisation and dichotomous thinking. Arbitrary inference means drawing a conclusion from a situation in the absence of evidence to substantiate such conclusion. Selective abstraction refers to a tendency to concentrate on one aspect of the situation (usually the negative aspect), while ignoring the more important features of that situation. Overgeneralisation refers to drawing a conclusion (generally a negative conclusion) on the basis of a single incident or fact. Magnification or minimisation refers to a tendency to draw conclusions about situations on the basis of erroneous evaluations.

Personalisation refers to a tendency to take responsibility for negative events in life in the absence of evidence to support such attitude. Finally, dichotomous thinking refers to a tendency to think in black and white. To summarise, in this model depression is portrayed as a cognitive disorder characterised

by a set of negative attitudes (cognitive triad), specific schemas, and maladaptive thinking patterns (logical errors).

A review of empirical literature on depression shows that Beck's cognitive conceptualisation of depression is supported by both correlational and experimental studies. In a series of studies carried out to evaluate his cognitive formulation, Beck (1974) found a strong evidence for the hypothesised relationship between negative thinking and depression (depression scale) and measures of pessimism (the third component of the triad) ( $r = 0.56$ ) and negative self-concept (the first component of the triad) ( $r = 0.70$ ). Other evidence supporting this cognitive view of depression includes the link established between hopelessness, a central component of the cognitive model, and suicidal intent ( $r = 0.47$ ).

Stronger support for Beck's cognitive model is offered by studies demonstrating the specificity of negative cognitions to depression. In a study designed to test the hypothesis that depressed patients are characterised by negative expectations, Loeb, Beck and Diggory (1971), found evidence in support of this component of the negative cognitive triad. The results obtained showed that, although depressed patients worked as nondepressed patients on a laboratory task, their ratings of the probability of future success were significantly lower. Further evidence that depressives hold negative attitudes toward the future is provided by a recent study carried out by Hammen and Krantz (1976). They reported results which clearly supported Beck's notion that depressives have an unrealistically negative view of the



future. More specifically, it was found that depressed subjects had lower expectations of success than nondepressed subjects.

Relevant to Beck's cognitive viewpoint are also the studies that examined depression in relation to higher cognitive processes such as memory. In one of these studies, Lloyd and Lishman (1975) found evidence that depressives exhibit a recall bias. Specifically, it was found that depressives' recall of negative experiences was quicker and easier than that of positive experiences. A recent study by Teasdale, Taylor, and Fogarty, (1980), however, reported data which showed that such biased recall is more an epiphenomenon than an antecedent of depressive states. Further evidence for Beck's notion that depressives have an exaggerated tendency to misinterpret external or environmental stimuli has been provided by a series of experimental studies. In an important study dealing with this issue, Nelson and Craighead (1977) found that depressives compared to non-depressives, underestimated the percentage of positive feedback they had received following performance on an experimental task, and tended to overestimate the percentage of negative feedback they were given. This finding was later replicated by a study that employed clinically depressed and non-depressed patients (De Monbreun and Craighead, 1977).

Although, as just pointed out, both correlational and experimental studies offered evidence for the hypothesised relationship between cognition and depression, further research demonstrating the primacy of cognitive factors is necessary if negative cognitions are to acquire an aetiological status or role in depression.

One of the studies that indirectly supported Beck's cognitive interpretation of depressive phenomena is that of Velten (1968). To the extent that Velten's study is a valid test of the cognitive model, it confirmed the aetiological role of maladaptive thinking in depression. Using a verbal mood induction procedure, Velten demonstrated that depressed mood could be successfully induced by instructing subjects to read negative or depressing self-referent statements. This finding that depressed mood could be produced by instructing subjects to adopt negative beliefs about themselves supports the cognitive interpretation of self-esteem (e.g. Coleman, 1975). Further evidence for the cognitive basis of depression has been provided by Ludwig (1975). His findings corroborate the view that depression could be induced or alleviated by manipulating cognitive variables such as beliefs, attitudes, and other cognitions.

The cognitive model of depression has been systematically supported by both clinical and experimental studies. Although Beck's account of depressive phenomena may be highly comprehensive and exhaustive, it is weak in some ways. Because of its flexibility and the lack of specificity, Beck's cognitive theory is difficult if not impossible to falsify or disconfirm. In fact Seligman (1981), a leading authority in this field of research, went even further when he questioned the scientific basis of the cognitive formulation:

"The model's main weaknesses", he remarked, "are the looseness of its terms, its descriptive and shallowly explanatory cast, and its loose contact with any

scientific base. Put more badly, the theory is ad hoc" (p.134, 1981).

In spite of these serious difficulties, Beck's cognitive theory has made substantial contributions both to the theoretical understanding and treatment of depressive disorders. In fact, Beck's description of the cognitive functioning of the typical depressive is the most accurate and exhaustive to date. More importantly, the set of therapies that the cognitive model of depression has generated are perhaps the best that psychotherapy can offer in this area of clinical practice.

### 3.3.2 LEWINSOHN'S LOSS OF REINFORCEMENT MODEL OF DEPRESSION

In the last decade a number of behavioural formulations have been developed and advanced to account for a large proportion of depressive complaints and behaviours (e.g. Costello, 1972; Ferster, 1974; Lewinsohn, 1974). An important feature of these behavioural theories is their extensive use of basic learning principles in their attempts to both explain and modify depressive behaviours. Behavioural conceptualisations of depression have, unlike psychodynamic or cognitive theories, focussed almost exclusively upon the depressed patient's overt behaviours and social interaction patterns. A defining characteristic of depression within these operant models is a lack of adequate reinforcement for adaptive behaviour. Specifically, low rate of positive reinforcement is believed to act as an eliciting stimulus for dysphoria and depressive behaviours.

Although all behavioural formulations of depression are unanimous

in hypothesising a link between depressive behaviours and maladaptive patterns of reinforcement, they advance different hypotheses as to the nature of reinforcement that supposedly elicits depressive symptoms and behaviours. For instance, Ferster, (1974) attributes depression to a loss of a major reinforcer, whereas Costello (1972) accounts for this syndrome in terms of a loss of reinforcer effectiveness. Social learning theorists such as Bandura (1971), however, attribute the aetiology of depression to faulty self-reinforcement systems. But only Lewinsohn's formulation concerning both the nature of the depressogenic reinforcement and the characteristics of the depressive's behavioural repertoire appeared to be articulate and comprehensive (Blaney, 1977). Almost all other behavioural models fall short of providing a satisfactory account of the aetiology and development of depressive disorders. Accordingly, only Lewinsohn's theory will be reviewed here.

Lewinsohn's theory concerning both the origin and development of depression stands in sharp contrast to Beck's cognitive viewpoint (1967, 1974). The former appears to be concerned with the interactional and environmental aspects of depression, while the latter attaches more importance to the cognitive basis of depressive phenomena including depressive behaviours. A major advantage of Lewinsohn's theory over Beck's is the reference it makes to the socio-cultural environment in its analysis and explanation of depressive disorders. Its major drawback, however, is its tendency to over-emphasise the role of environmental factors in depression at the expense of

internal cognitive processes (e.g. Hammen and Glass, 1975).

Lewinsohn attributes depressive behaviours to a low rate of response-contingent positive reinforcement. He advanced three reasons as to why the depressed individual may be placed on a reduced positive reinforcement schedule. One important reason is that the social environment fails to provide reinforcement. Another reason is that the individual avoids participation in pleasurable activities that are highly reinforcing, although this social avoidance may be considered as a consequence rather than an antecedent of depression. Another equally plausible explanation as to why the individual may be placed on a prolonged extinction schedule (lack of positive reinforcement) is that he/she lacks the necessary skills (social skills deficit) to elicit reinforcement from his social environment.

In sum, when talking about depression Lewinsohn seems to refer to a sort of breakdown in the reinforcement system. Figure 3 illustrates this operant conception of the aetiology of depression. As Figure 3 indicates, depression is believed to occur when there is a low rate of positive reinforcement. To confirm this aetiological hypothesis one needs not only to demonstrate that the rate of response-contingent positive reinforcement is lower in depressed than in non-depressed persons, but also that the amount of response-contingent positive reinforcement is closely associated with depression (positively correlated). More important if one is to claim a causal link between depression and reduced positive reinforce-

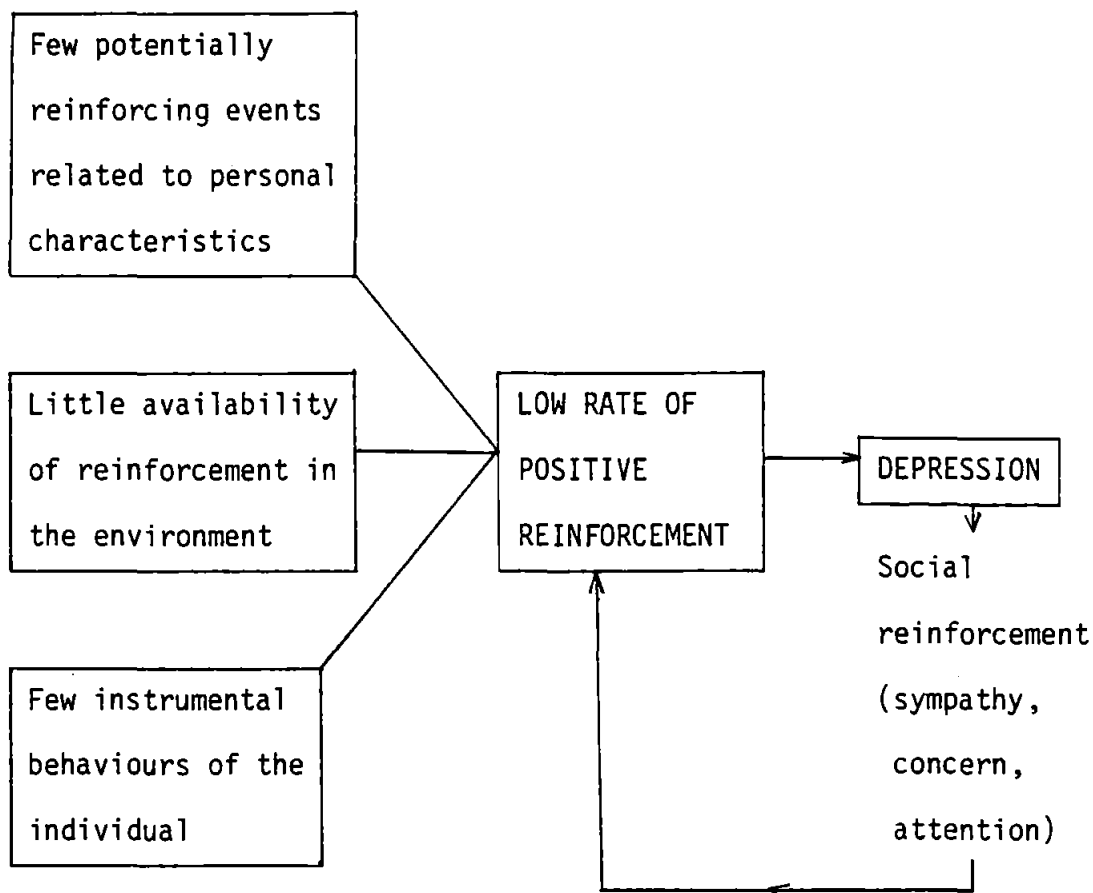


Figure 3. Schematic representation of Lewinsohn's model of depression (adapted from Lewinsohn, 1974)

ment, is the demonstration that depression could be induced or reduced by manipulating the rate of positive reinforcement. The bulk of empirical research that has addressed Lewinsohn's behavioural formulation of depression may be said to have considered all the issues listed above. In a series of correlational studies, Lewinsohn and Libet (1972) and Lewinsohn and Graf (1973) claimed to have established a link between a low

rate of positive reinforcement and depression. Lewinsohn and his colleagues examined the relation between depressed subjects' self-reported mood and participation in pleasant activities (served as a measure of amount of positive reinforcement received). As predicted, they obtained results which showed a positive correlation between the level of depression and the frequency of participation in pleasurable activities. Although this may seem to confirm the aetiological significance of reduced positive reinforcement in depression, it may also be the case that social avoidance or lack of participation in positive activities has little or no aetiological relevance. In other words, the correlational data reported by Lewinsohn and his colleagues leave open the question of direction of causality. What is needed in this context is not only the demonstration that depression is associated with a reduced amount of positive reinforcement, but also that inadequate reinforcement is an antecedent rather than a symptom of depression.

A study that attempted to deal with the direction of causality issue was carried out by Hammen and Glass (1975). Specifically, their study tested Lewinsohn's claim that depression is causally related to a low rate of response-contingent positive reinforcement. In order to test this specific hypothesis, Hammen and Glass instructed three groups of depressed subjects either to increase participation in positive activities (increase activities group), to increase protein intake (attention-placebo group), or not to alter their activities (self-

monitoring group). Following a two week period all subjects were assessed for their level of depression. The comparison failed to show any significant difference in self-reported depression between the group induced to increase the frequency of reinforcing activities and control groups. This failure to find any significant reduction in depression as a result of high rate response-contingent positive reinforcement represents a serious challenge to Lewinsohn's operant conceptualisation of depressive phenomena.

Another study that also assessed the therapeutic implications of increasing the rate of response-contingent positive reinforcement is that of Padfield. In this outcome study, Padfield (1975) assessed the effects of inducing a group of depressed women to engage in positive activities. When compared to another group of depressed women who received no such instruction (control), no significant improvement was found as a result of participation in pleasurable activities. This finding that increased positive reinforcement failed to reduce depression questions the validity of a low rate of response-contingent positive reinforcement as an adequate explanation of depressive behaviours and disorders. Until positive reinforcement is shown to be superior to placebo in alleviating depressive symptoms, any claim for the aetiological significance of reduced positive reinforcement will be unjustified.

To summarise: there is little or no evidence in the empirical literature on depression, suggesting that depression or its



behavioural and affective correlates could be induced or reduced by simply manipulating the amount of response-contingent positive reinforcement. Perhaps Blaney (1977) was right in declaring that Lewinsohn's theory:

"should be treated as a characterisation of the depressed person's interaction with the environment rather than as a hypothesis concerning the causal antecedents of depressive episode" (p. 210).

### 3.3.3 LEARNED HELPLESSNESS MODEL OF DEPRESSION

Recently another model of depression has been proposed to account for various depressive symptoms and manifestations. The learned helplessness model of depression (Seligman, 1974; Abrahamson, Seligman and Teasdale, 1978) invokes environmental as well as cognitive or internal factors to explain the aetiology and development of this clinical syndrome. Unlike cognitive or behavioural models of depression, the present one combines both cognitive and behavioural views to address the aetiological and therapeutic issues of depression. This interactionist approach to explaining emotional upsets and disorders has generated a great deal of research in both areas of social and clinical psychology. Studies examining people's reactions to aversive or stressful life events, such as car accidents, rape, (Janoff-Bulman, 1979; Janoff-Bulman and Wortman, 1977) have particularly benefitted from this framework.

The learned helplessness phenomenon has initially been observed in animals. Cats and dogs exposed to aversive stimulations, such as electric shock, failed to escape when tested in a shuttle

box. This learning deficit later called 'learned helplessness' is believed to result from noncontingency learning; that is the acquisition of a belief (for humans) that responding is independent from reinforcement or outcome. For learned helplessness deficit to occur, the belief in noncontingency should be generalised to situations in which control is available.

Following this important finding, Seligman hypothesised a parallelism between experimental helplessness and clinical manifestations of human depression. He regarded depression and its behavioural and affective components as a consequence of expectations of response-outcome independence. In other words, the apparently complex aetiology of clinical depression is reduced to the so-called associative deficit (response-outcome independence).

Studies that attempted to produce the learned helplessness deficit in humans (e.g. Hanusa and Schulz, 1977; Klein, Fencil-Morse, and Seligman, 1976; Tennen and Eller, 1977; Wortman, Panciera, Shusterman, and Hibscher, 1976) have reported results which were inconsistent with the learned helplessness hypothesis. While some studies (e.g. Hanusa and Schulz, 1977) have observed 'facilitation effects' following experience with uncontrollable outcomes, others however, have only obtained the helplessness effects (performance decrements) in certain circumstances. That is, when previous failure to control outcomes is accounted for in terms of personal shortcomings (internal attributions of failure), such as incompetence, inability and so forth.

The conclusion that emerged from human helplessness research seems to suggest that the laboratory produced helplessness is not only a function of noncontingency learning but also, and more importantly, of the type of attributions involved in the helplessness process (the concept of attribution will be dealt with in the next chapter).

The importance of attributions in mediating and shaping the emotional and behavioural aspects of depression was fully acknowledged and reflected in the recent formulation of the learned helplessness theory (Abramson, Seligman, and Teasdale, 1978). The revised learned helplessness model of depression adopts an attributional framework both to solve some serious conceptual irregularities and to account for the available experimental data generated by the original model. According to the attributional reformulation, the helplessness or depressive reaction depends not merely on the environmental condition of uncontrollability but also on the kind of attribution displayed for loss or lack of control. For instance, the helplessness deficits can only be transferred to situations where control is available, if the attributions made, for uncontrollability or failure in the original situation, were internal and global (internal-global attributions).

The reformulated learned helplessness model of depression predicts that depressives have an exaggerated tendency to make internal, stable, and global attributions for negative outcomes (failure) and external, unstable, and specific attributions for

positive outcomes (success). In brief, the internality, stability, and globality dimensions of attributions are believed to influence respectively the intensity, chronicity, and generality of disorders.

Although the attributional reformulation may have answered important questions about the aetiology and development of depression, it has failed to clarify other relevant issues. As noted by Wortman and Dintzer (1978), the reformulated model (Abramson et al., 1978) is not very specific about the relationship between motivational, affective, and cognitive components of helplessness and depression. Another problem with the Abramson et al. model concerns its failure to specify the conditions under which a given attribution will be displayed (e.g., Wortman & Dintzer, 1978; Jackson & Larrance, 1978). Indeed, in its present form the learned helplessness model provides no basis for understanding why some people make internal, stable, and global attributions when faced with uncontrollable or aversive outcomes, and why some other people display external, unstable, and specific attributions for similar outcomes. Until the determinants of particular attributions will be specified and relevant issues clarified (e.g., the nature of the relationship between motivational, affective, and cognitive components of helplessness and depression), the potential of the helplessness model in increasing theoretical understanding of helplessness and depressive phenomena will be greatly limited.

## CHAPTER FOUR

### ATTRIBUTION: OVERVIEW OF BASIC THEORY AND RESEARCH

- 4.1 INTRODUCTION
- 4.2 THE FORMATION OF CAUSAL ATTRIBUTIONS
- 4.3 THE CLASSIFICATION OF CAUSAL ATTRIBUTIONS
- 4.4 CURRENT ISSUES IN ATTRIBUTION THEORY AND RESEARCH
- 4.5 SUMMARY

#### 4.1 INTRODUCTION

The concept of attribution has recently enjoyed a great deal of popularity and credibility in both areas of social and clinical psychology. It has been evoked and used both by theoreticians and researchers to advance the theoretical understanding of achievement-related behaviours (Weiner, 1974), emotional disorders (Valins and Nisbett, 1972; Johnson et al., 1977), and of psychotherapeutic processes (Kopel and Arkovitz, 1975). The attribution framework has also been adopted to remedy conceptual inadequacies and irregularities inherent to such theories as achievement motivation theory (Atkinson and Feather, 1964; Weiner et al., 1971) and learned helplessness theory (Seligman, 1974; Abramson et al., 1978; Miller and Norman, 1979). The adoption of attributional approaches in these areas and others gave rise to fruitful debates about various conceptual as well as empirical issues in social and clinical psychology (cf. Harvey, Ickes, and Kidds, 1976, 1978).

Attribution refers to the process through which an individual makes judgements about the causes of his/her behaviour and that of others. It also refers to the ways in which people generate explanations for occurrences in everyday life.

Research on people's intuitive perceptions of causality

(attributions) has been initiated by Heider (1958), and elaborated and refined by Jones and Davis (1965) and Kelley (1967). Their writings have, as Semin (1980) recently pointed out:

"laid the necessary groundwork for what is probably the most influential framework in today's social psychology: attribution theory" (p.291).

Attribution theory provides a useful framework for studying and understanding social behaviour in both its adaptive and maladaptive forms. The theory also provides an empirical framework for testing hypotheses concerning the antecedent conditions of specific instances of behaviour.

Despite its apparent conceptual adequacy, attribution theory has been described as lacking the necessary ingredients of a good theory of social cognition (e.g., Semin, 1980). Critics (e.g., Harris and Harvey, 1981) have specifically questioned some basic assumptions made by attribution theorists. One such assumption is that the attributor operates as a statistician or scientist when attempting to explain or interpret things that happen to him/her and to other people. This metaphor has also been challenged by judgement researchers (e.g., Fischhoff, 1976; Hogarth, 1980). The conclusion emerging from judgement research suggests, contrary to the picture projected by attribution theorists, that people's information processing capacity is greatly limited.

But perhaps the most justified criticism of attribution theory concerns its lack of reference to the social context in which attributions are generated and displayed. To paraphrase

Semin (1980), the individual in attribution theory is portrayed as though he/she is operating in 'social vacuum'. Thus, the need for theoretical statements that will capture this social dimension of attribution becomes apparent. Failure to 'socialize' attribution theory could seriously undermine its status as a leading view within social psychology.

#### 4.2 THE FORMATION OF CAUSAL ATTRIBUTIONS

The chief concern of attribution theory and research has been, and still is, the description and explanation of processes that lead to making causal attributions. Early theorists such as Heider, Jones and Davis, and Kelley have written extensively on the antecedent conditions and development of causal attributions and explanations. Specifically, their work involved the identification and descriptions of the heuristics that perceivers use to arrive at causal judgements.

Although Heider's 'naive' analysis of action (1958) and Jones and Davis (1965) correspondent inference formulations are highly influential (in attribution research), Kelley's ANOVA model carries more weight when it comes to describing the phenomenology of the attributor. In particular, Kelley's extensive analyses have led to the identification and specification of attributional 'rules'. Kelley's ANOVA model is believed to provide a more adequate framework for analysing and understanding attributional processes.

Kelley contends that people's intuitive perceptions of causality are governed by a fundamental principle referred to as 'the



covariance principle'. Specifically, the covariance principle states that:

"an effect is attributed to the one of its possible causes with which, overtime, it covaries" (Kelley, 1972, p.3).

Kelley listed four criteria that attributors presumably apply to assess and determine cause-effect covariation (attribution). These are distinctiveness, consensus, consistency overtime, and consistency over modality.

Distinctiveness refers to information concerning the individual's response to the entity (stimulus). Consensus refers to information concerning the responses or reactions of other people to the same entity. Consistency overtime refers to information regarding the individual's response to the entity overtime. Consistency over modality refers to information regarding the individual's response to the entity over modality.

The perceiver or attributor, in Kelley's model, is assumed to obtain information from three different sources: entity (supplies distinctiveness information), persons (supply consensus information), and time/modalities (supply consistency information) - this information is then subjected to a process akin to analysis of variance. This is how Kelley (1972) describes the process leading to attribution of causality:

"given information about a certain effect and two or more possible causes, the individual tends to assimilate it to a specific assumed analysis of variance pattern and from that to make a causal attribution" (p.152).

In sum, Kelley's model suggests that in the pre-attribution stage, perceivers weight, combine, and organize information in the manner of a statistician. This information synthesis is believed to form the basis of the attribution of causality.

Evidence for Kelley's hypothesis that different types of informational cues lead to different kinds of attributions (internal and external attributions) has been provided by McArthur (1972). In a classic study on the determinants of causal attributions, McArthur presented subjects with behavioural information (e.g., 'John laughs at the comedian'). Each behavioural event presented to subjects was accompanied by low or high distinctiveness information (e.g., 'John laughs at hardly any other comedian' - 'John laughs at almost every other comedian'); high or low consensus (e.g., 'almost everyone who hears the comedian laughs at him' - 'hardly anyone who hears the comedian laughs at him'); and high or low consistency (e.g., 'in the past John has almost laughed at the same comedian' - 'in the past John has almost never laughed at the same comedian'). Following the presentation of these informational cues, subjects were asked to indicate the extent to which the person's response to the stimulus (John's reaction to the comedian) was due to (i) something about the person (person attribution), (ii) something about the stimulus (stimulus attribution), (iii) something about the particular circumstances (circumstance attribution), or (iv) some combination of person, stimulus, and circumstance. The results obtained showed, as hypothesised by Kelley, that person attribution was exhibited following the

presentation of low consensus, low distinctiveness, and high consistency. Stimulus attribution was determined by high distinctiveness, high consensus, and high consistency. Circumstance attribution was found to be associated with low consensus. These findings that different attribution are based on different types of information are in line with Kelley's ANOVA conception of the processes of causal attributions.

In his subsequent publications, Kelley (1971, 1972, 1973) described two more schemes that perceivers presumably use to form and generate causal attributions: the augmentation and the discounting methods. These two methods or principles, like the covariation principle, are employed to gather and organize information that serve as raw data for attributional judgements. The augmentation principle states that:

"if for a given effect, both a plausible inhibitory and a plausible facilitative cause are present, the role of the facilitative cause will be judged greater than if it alone were presented as plausible cause of the effect" (Kelley, 1971, p.12).

The discounting principle, in contrast, propose that:

"the role of a given cause in producing a given effect is discounted if other possible causes are also present" (Kelley, 1971, p.8).

Although Kelley's description of these specific judgemental schemes may be theoretically sound, there is as yet no evidence suggesting that people make such a cognitive effort when assessing their behaviour or the behaviour of others. In

fact, a recent study carried out by Hansen (1980) reported evidence suggesting, contrary to Kelley's views, that attributors follow a principle of cognitive economy. In line with the cognitive economy hypothesis, Hansen (1980) concluded that perceivers:

"prefer information allowing for simpler confirmatory inferences over information requiring most sophisticated confirmatory inferences, based on augmentation and discounting" (p.1007).

Finally, Kelley (1972) also discussed the possibility of causal schemata serving as the basis for causal judgements or attributions. According to Kelley, causal schemata are evoked in particular situations in order:

"to make economical and fast attributional analysis" (p.2, 1972).

Here again Kelley's formulation could be criticised on two accounts. Firstly, Kelley failed to identify or specify those situations in which causal attributions are based on causal schemata, and those situations in which causal attributions and explanations are based on consensus, distinctiveness, and consistency information. There are some indications, however, that causal schemata are invoked mainly to account for unusual occurrences and events (e.g., Cunningham and Kelley, 1975). Secondly, Kelley failed to explain how causal schemata affect the search for and the utilization of new data and how the new data might affect the existing schemas. Kelley was apparently aware of this gap when he remarked that:

"further development of attribution theory requires an account of this conflict between existing cognitive structures and new data and the process by which they interact and become reconciled" (p.120, 1973).

#### 4.3 THE CLASSIFICATION OF CAUSAL ATTRIBUTIONS

Considerable progress has been achieved in attribution theory as a result of extensive theoretical and empirical work within the area of achievement motivation. Following the formulation of the attributional model of achievement motivation, Weiner, Frieze, Kukla, Reed, Rest, and Rosenbaum (1971) made a set of propositions vis a vis the underlying properties of causal attributions. Based on the writings of Heider (1958), Rotter (1966), and Kelley (1967), Weiner and his associates (1971) argued quite convincingly that causal attributions may be categorized along the causal dimensions: the dimensions of locus of causality (internal attributions vs. external attributions), and the dimension of stability (stable attributions vs. unstable attributions). The former dimension involves attributions to internal (within the person) and to external (within the environment) factors or causes, while the latter dimension involves attributions to stable (fixed) and unstable (variable) causes.

This two-dimensional analysis of attributions has been inspired by previous work on perceived causes of success and failure. Weiner and his associates (1971) identified four causes that may account for performance in achievement related contexts: ability, effort, task difficulty, and luck. They found that these causal attributions exhibited similarities as well as

differences. For instance, both ability attribution and effort attribution are internal, but the former is stable, whereas the latter is unstable or variable. Similarly, task and luck attributions are both external (outside the person), however, the former is characterised by its stability and the latter by its variability or instability.

Weiner et al.'s decision to subsume causal attributions within the dimensions of locus and stability appears to have been guided by two motives: identifying and specifying individual differences with regard to attributions, and relating performance consequences (e.g., emotions, expectancies) to two different kinds of attributions.

In their attributional model of achievement motivation, Weiner and his associates (1971) hypothesised linkages between attributions and consequences of performance (i.e., success and failure). They proposed that the emotional consequences are influenced by internal and external attributions (internality dimension), while expectancy shifts are related to stable and unstable attributions (stability dimension).

Recently, learned helplessness theorists (Abramson, Seligman, and Teasdale, 1978) also proposed a third attributional dimension to account for some aspects of the learned helplessness phenomenon: "specific-global" - orthogonal to internality and stability, that characterises the attributions of people' (p.57). This third attributional dimension was introduced to account for the causes in which learned helplessness deficits are

limited to the original situation (no generalization across situations), and for the cases in which these deficits occur across situations. Specific attributions imply that helplessness symptoms will be exhibited only in the situation in which they have been induced, global attributions, in contrast, imply that helplessness feelings will be experienced in most if not all stressful situations.

In sum, attribution theory as formulated by Heider (1958), Jones and Davis (1965), Kelley (1967), and extended by Weiner (1974) and Abramson et al. (1978) states that attributions are based on specific judgemental rules and that these attributions shape our feelings and reactions to past as well as future events. Research in this area of social psychology has so far found considerable evidence consistent with both the hypothesised determinants and the predicted consequences of causal attributions (see Antaki, 1981; Harvey, Ickes, and Kidd, 1976, 1978; for a detailed review of this research).

#### 4.4 CURRENT ISSUES IN ATTRIBUTION THEORY

##### AND RESEARCH: THE CASE OF ATTRIBUTIONAL BIASES

An important issue in attribution theory and research has been, and still is, the nature of the biases or errors that frequently distort people's causal judgements and attributions. Recent research has shown that the making of causal attributions does not always involve the use of logical judgemental rules. Specifically, it has been shown that causal attributions are, in some cases, subject to a number of biases. Examples of

attributional biases have been illustrated and documented by the findings reported in actor-observer studies, success-failure studies, and dispositional shift studies.

#### 4.4.1 THE ACTOR-OBSERVER BIAS

Perhaps the most convincing evidence that attributions are biased has been offered by studies examining actor-observer causal attributions (e.g., Jones and Nisbett, 1972). Referring to the issue Jones and Nisbett (1972) stated that:

"there is a pervasive tendency for actors to attribute their actions to situational requirements, whereas observers tend to attribute the same actions to stable personal dispositions" (p.80).

Jones and Nisbett (1972) listed three different factors that may account for actors and observers' attributional biases and differences. They postulated that actors and observers differ in their motivations, their perceptual perspectives, and in their information processing strategies:

- (i) The motivational explanation for actor-observer differential attributions suggests that actors are highly motivated to protect and/or enhance their self-esteem by attributing social desirable behaviours to personal dispositions and undesirable ones are attributed to situational causes or constraints. Studies relevant to this issue reported evidence that suggested that actors' differential attributions for success and failure are motivationally based (e.g., Bradeley, 1978; Miller and Ross, 1975). Consistent



with the motivational hypothesis, are also the studies that found that actors tend to take more credit than observers grant them for successful outcomes (e.g., Snyder, Stephan, and Rosenfield, 1976; Taylor and Koivumaki, 1976). When unsuccessful outcomes are involved, actors assume less responsibility than observers tend to ascribe them (e.g., Harvey, Harris, and Barnes, 1975; Ross et al., 1974; Snyder et al., 1976; Taylor and Kovumaki, 1976).

- (ii) The perceptual or focus of attention explanation suggests that actors focus their visual attention on the surrounding environment, whereas observers' attention is apparently more directed at the actor than to the situation. Consequently, actors adopt situational explanations for their actions and observers favour dispositional explanations for the same actions. The empirical support for the focus of attention hypothesis is equally impressive (e.g., Arkin and Duval, 1975; Nisbett et al., 1973; Storms, 1973).
- (iii) The third hypothesis advanced to account for the divergent causal attributions of actors and observers is informational in nature. The informational explanation of actor-observer bias proposes that actors have a privileged access to many informational sources and cues that are relatively denied to observers. It has been suggested that actors have (historical knowledge' about their past actions and behaviours,

which is not readily available or accessible to observers (e.g., Manson and Snyder, 1977). Consequently, observers will exclusively base their attributions on consensus information (social norm), and actors' attributions will be more based on distinctiveness and consistency information. Studies testing this informational hypothesis reported data that supported the informational basis of attributional biases displayed by both actors and observers (e.g., Eisen, 1979; Hansen and Lowe, 1976). These studies found that actors tend to base their causal attributions on distinctiveness information, and observers rely heavily on consensus information for their causal explanations.

#### 4.4.2 THE SUCCESS-FAILURE CASE

Attributions for achievement-related behaviours is another area of attribution theory and research in which attributional biases have been observed. A common finding reported by the studies dealing with this issue is that people tend to attribute success to internal causes or factors (internal attributions for success) and failure to external causes and constraints (external attributions for failure) (e.g., Snyder et al., 1976; Wolosin et al., 1973). Some theorists attempted to account for this finding in motivational terms (e.g., Snyder et al., 1976; Bradley, 1978), others, however, favoured non-motivational or informational explanations (e.g., Miller and Ross, 1975; Nisbett and Ross, 1980).

Those who subscribe to a motivational view of self-serving biases argue that people are motivated to protect and/or enhance their self-esteem. As a consequence, they take credit for success and deny responsibility for failure. Briefly, this motivational hypothesis suggests that attributions in achievement-related contexts (i.e., attributions for success and failure) are biased by self-serving motives (Bradley, 1978).

Those who subscribe to a non-motivational or informational view of differential attributions for success and failure, in contrast, argue that these attributional biases arise from cognitive or informational sources. In their recent publication, Nisbett and Ross (1980) argued strongly in favour of a non-motivational view of attributional biases. They presented ample evidence that such biases derive from cognitive sources. They concluded that biases in attributions:

"are almost inevitable products of human information-processing strategies" (p.12).

#### 4.4.3 THE DISPOSITIONAL SHIFT CASE

Attributional biases have also been observed in this relatively new area of research. It has been shown that actors' attributions or explanations of their past behaviour are less situational and more dispositional than their explanations for present behaviour (e.g., Moore et al., 1979; Peterson, 1980). This dispositional shift in attributions over time is believed to be due to the predominance of memories about self over memories

about the situation. Specifically, Moore and his colleagues (1979) suggested that memories about the self are more accessible than those about the situation when people engage in attributional activities about past events and experiences. The relatively easy access to memories about the self is, according to the authors, facilitated by well developed self-schemata as compared to weak situation-schemata. A recent study by Peterson (1980) reported evidence consistent with Moore et al.'s hypothesis that the dispositional shift in attributions is related to memory biases.

#### 4.5 SUMMARY

The present chapter has been devoted to a concept of increasing popularity and influence in both areas of social and clinical psychology: attribution. As noted earlier, the concept of attribution has been evoked and used by researchers and clinicians to analyse behaviour both in its adaptive and maladaptive forms. In addition to enriching the literature on social cognition, attribution theory and research have advanced the theoretical understanding of a number of phenomena, including achievement motivation, social interaction (e.g., interpersonal attraction, interpersonal conflict etc.), and learned helplessness. Though many issues, in attribution, remain to be clarified, there is no doubt that the attributional framework will continue to be influential in cognitive social psychology.

PART 2

EXPERIMENTAL AND CLINICAL WORK

## CHAPTER FIVE

### DEPRESSION AND INTERNAL-EXTERNAL ATTRIBUTIONS

- 5.1 INTRODUCTION
- 5.2 EXPERIMENT 1
- 5.3 EXPERIMENT 2
- 5.4 GENERAL DISCUSSION AND IMPLICATIONS

## 5.1 INTRODUCTION

Despite recent advances in psychological research on depression, issues concerning both the aetiology and nosology of depressive disorders remain relatively unsettled.

Three psychological theories (Beck, 1967, 1974; Lewinsohn, 1974; Abramson, Seligman, and Teasdale, 1978) have been advanced to challenge directly the authority of the traditional affective approach to the syndrome of depression.

Beck's cognitive theory (1967, 1974) claims that depression is set off by a negative cognitive set. In this model the depression-prone individual is seen as characterised by negative cognitive schemas. Once activated (usually by an important loss), these maladaptive cognitive schemas or 'silent assumptions' lead the person to develop and maintain a negative view of himself, and of the world, and of the future (the so-called cognitive triad) (see chapter three for further details).

The affective aspect of depression is believed to be secondary to these faulty cognitions. This cognitive view of emotional disorders, equally shared by Ellis (1962) and Valins and Nisbett (1971), is based on the assumption that:

"the affective response is determined by the way an individual structures his experience" (Beck, 1963).

Lewinsohn's behavioural viewpoint (1974), in contrast, attributes depressive disorders to a sort of breakdown in the reinforcement system. He postulates that depression results from a low rate of response-contingent positive reinforcement. The depressed individual is believed to be socially unskilled (social skills deficit) and as such he fails to initiate behaviours that would elicit positive reinforcement from others or the environment (chapter three gives further details).

Although these two theories of depression claim respect and popularity in the psychological literature, some of their assumptions have nevertheless been questioned. But the major criticism addressed to both cognitive and behavioural models of depression concerns their tendency to over-emphasize one aspect of this syndrome at the expense of another, and also their apparent tendency to explain depression in terms of its symptoms and consequences (see Wortman and Dintzer, 1978, for a detailed discussion of these issues).

The reformulated learned helplessness model of depression recently proposed by Abramson and his colleagues (1978) predicts, in contrast to both cognitive and behavioural views, that depression should occur only:

"when highly desired outcomes are believed improbable or highly aversive outcomes are believed probable, and the individual expects that no response in his repertoire will change their likelihood" (p.68).

In their reformulation of the learned helplessness hypothesis, the authors introduced and extensively used attribution theory



constructs to resolve some conceptual inadequacies and other shortcomings inherent to the original hypothesis (Seligman, 1974). Specifically, the adoption of an attributional approach in the reformulation gave them the possibility, which was originally denied, of distinguishing between different types or forms of helplessness. As a result, the attributional analysis successfully differentiated between personal and universal helplessness, between transient and chronic helplessness, and between specific and global or general helplessness. Some characteristic features of helplessness and depression such as generality, chronicity, and self-esteem loss are hypothesised to be related respectively to the globality, stability, and internality of the attribution for uncontrollability or helplessness. Of particular significance in this reformulation, is the importance acquired by causal attribution in the helplessness process. In fact, Abramson and her colleagues regard attribution as a central component of their helplessness theory. Their view at this point is clearly stated:

"The individual first finds out that certain outcomes and responses are independent, then he makes an attribution about the cause. This attribution affects his expectations about future response-outcome relations and thereby determines, the chronicity, generality, and to some degree the intensity of the deficits" (p.56).

Although, as will be seen, the existing experimental data lend some support to this critical aspect of learned helplessness theory, further research is required to investigate directly the relationship between attribution and depression (cf. Wortman and Dintzer, 1978). Particularly, there is a need

to assess the potential role of attribution in the development and maintenance of depressive disorders. This concern together with other related issues prompted the current research.

The present investigation was concerned with the causal relationship between attribution and depression. Specifically, this investigation was an attempt to reveal the extent to which manipulating attributions will affect depression (as measured by the BDI and MAACL). It was also designed to clarify the specificity issue - that is, to determine whether certain types of attributions are more associated with depression than with anxiety, hostility, or other known psychopathological disorders. The study of this issue is of a particular importance, since it bears directly on the aetiological relevance of attribution to depression.

#### 5.1.1 ATTRIBUTIONS OF HELPLESSNESS

Directly relevant to the present investigation are human helplessness studies (Klein et al. 1976; Tennen and Eller, 1977; Wortman et al. 1976) that manipulated subjects' causal attributions of noncontingent outcomes. In an experiment on the alleviation of helplessness deficits, Klein and his colleagues induced depressed and non-depressed students to make either internal or external attributions for failure to control outcomes. When tested on a problem solving task (anagrams), only depressed controls and internal attribution groups (depressed and non-depressed) exhibited the performance deficits predicted by the

helplessness model. As was expected, external attribution groups performed better than internal or control groups.

But despite the differences observed in performance, the results of this study still are not entirely relevant to the etiological or therapeutic issue; because there is little or no evidence suggesting that performance deficit at a laboratory task is indicative of depression. If helplessness investigators are to claim aetiological implication for noncontingency learning they will probably need to present post helplessness data involving not only performance but also depressive affect measures. Surprisingly, the data relevant to this very same issue, was not reported in Klein et al.'s study (1976). Failure to do so was attributed, as one might expect, to some methodological constraints:

"The sliding data indicated that solvable problems decreased sadness and unsolvable problems increased self rated anger, but the scales are at best crude indicator of mood, so the data will not be reported" (p.512).

A further attempt to manipulate causal attributions for non-contingent outcomes, was made by Tennen and Eller (1977). They obtained results which clearly indicated that the helplessness process is strongly influenced by attribution instructions. But here again, the data seem to be more relevant to the helplessness model in general than to the helplessness model of depression.

In sum, human helplessness studies, particularly those referred

to here, showed that non-contingency learning (response-outcome independence) was affected by subjects' beliefs about causality. But they neither established links between attributions and depressive components, nor did they demonstrate a relation between response-outcome independence (the so-called associative deficit) and depression. In fact, the possibility of depressives being sensitive to non-contingency has recently been refuted by Alloy and Abramson (1979). They reported results which showed that depressed subjects were even more accurate in their judgements of contingency than non-depressed subjects were.

However, the possibility that particular types of attributions being involved in depression has already been recognized.

#### 5.1.2 DEPRESSION AND CAUSAL ATTRIBUTIONS FOR SUCCESS AND FAILURE

More relevant to the present investigation are studies that examined the relationship between attribution and depression. In an important study, Rizley (1978) gave either success or failure feedback to depressed and non-depressed college students and instructed them to make causal attributions for their outcomes. He found that depressed subjects tend to ascribe more failure than success to internal or personal factors (inability, incompetence), whereas non-depressed ones attribute more causality to themselves for success than for failure. The tendency for depressives to make depressogenic attributions has also been observed by Kuiper (1978). The

pattern of results obtained from his study seem to suggest the existence of an attributional style specific to depressives. This hypothesis has been successfully examined by Seligman and his colleagues in a recent correlational study (1979). They reported results which clearly confirmed the hypothesis in question. Specifically, it was found that depressives tend to display internal stable attributions for negative outcomes, whereas non-depressives make external unstable attributions or similar outcomes.

The attributional differences observed in this study and others seem to suggest that attributions are operative in the etiology or development of depressive disorders.

## 5.2 EXPERIMENT 1

While human helplessness studies were etiological in nature (or at least as it has been claimed), that is the demonstration that a given condition occasions depression or it correlates, the present study however, was more concerned with the implications that certain attributions may have on depression regardless of its causes or etiology. Considering the fact that attributions are involved in depression, the current study attempted to determine whether manipulating depressed college students' attributions of success and failure on an anagram task would affect their subsequent mood.

In this first experiment, one group of subjects was given instructions designed to elicit internal attributions for

failure (IAF) to solve most of the anagrams making the task. A second group of subjects was induced to make external attributions for failure (EAF) on this task. A third group was exposed to failure (FO) but was not given attribution instructions. A fourth group of subjects was induced to make internal attributions for success (IAS) on this task. A fifth group was induced to make external attributions for success (EAS) on the same task. The sixth and the last group received success feedback (SO) but was not given attribution instructions.

To evaluate the affective consequences of attributions, all subjects completed the Multiple Affect Adjective Checklist (MAACL) both before and after the experiment. Briefly, the MAACL provides measures of three different affects: anxiety, depression and hostility. Thus, this experiment was a 2 (success-failure) x 3 (internal attribution, external attribution and no attribution instructions) factorial design.

Based on the previous research reviewed here, the following predictions were made:

1. As a replication of Rizley's (1978) and Kuiper's (1978) finding that depressives attribute failure but not success to internal factors, FO subjects were expected to make more internal attributions than SO subjects.
2. EAF subjects should report less depression on the MAACL than both IAF and FO subjects. While the former manipulation corrects the depressives' tendency to self-blame, the latter one, however, reinforces this

depressogenic tendency.

3. IAF subjects should report no more depression on the MAACL than their counterparts in failure only (FO) condition.
4. IAS subjects should report less depression than both EAS and SO groups. Because the former treatment is believed to be more esteem enhancing than the latter one.
5. It was also anticipated that both anxiety and hostility affects will vary as a function of success-failure per se.

#### 5.2.1 METHOD

Overview. Depressed undergraduate students selected on the basis of their scores on the Beck Depression Inventory (BDI), participated in a problem solving experiment. Subjects were given either success or failure feedback following performance on an anagram task, and were induced to make internal or external attributions for their outcome. Subjects' mood was assessed before and after the experiment. Mood change scores were obtained by comparing both the initial and subsequent moods as reported on the MAACL.

Subjects. Fifty-four undergraduate students (30 females and 24 males) from Plymouth Polytechnic, participated in this experiment. Subjects were selected on the basis of their scores on the BDI (Beck et al., 1961). Subjects scoring 8 or more on this scale were selected and randomly assigned to one of the six experimental conditions shown above. Studies adopting

similar selection procedure (e.g., Klein et al., 1976; Seligman et al., 1979) indicated that this cutting line offers a useful basis for differentiating between mildly depressed and non-depressed persons. The validity and reliability of the BDI are well established and documented by various research reports (e.g., Beck, 1967; Metcalfe and Goldman, 1967). Although this instrument was initially designed for clinical use, subsequent work (Bumberry et al., 1978) revealed that it could also be used to measure depression in a college student population (see chapter 2 for further details).

Following the administration of the BDI, all subjects completed the MAACL today form (Zuckerman and Lubin, 1965). Briefly, the MAACL scale provides measures of three different affects: depression, anxiety, and hostility. The correlation between BDI and MAACL scores was .36 ( $p < .05$ ). Table 1.1 presents the means and standard deviations for the BDI and MAACL depression scale.

Procedure. A flow chart of the experimental procedure is shown in Table 1.2. All participants were run one at a time. Each subject was seated at a table facing a screen, and was administered both the BDI and MAACL (see Appendix A.1). After completing the mood questionnaires, all subjects were given the following standard instructions for the problem solving task:

"This experiment attempts to identify the strategies that people generally use to solve problems. You will be given 20 anagrams; anagrams are, as you may know, words with the letters scrambled. They will be projected one at a time on the screen in front of you. Your task is to unscramble each of them to form a word in English. When you recognize the word tell me aloud."



TABLE 1.1 - MEANS AND STANDARD DEVIATIONS OF BDI AND  
MAACL SCORES

OUTCOME	INTERNAL ATTRIBUTION		EXTERNAL ATTRIBUTION		NO ATTRIBUTION INSTRUCTIONS	
	M	SD	M	SD	M	SD
SUCCESS						
BDI	11.77	3.38	11.55	2.82	11.00	3.55
MAACL	14.55	4.18	14.77	4.63	16.55	1.94
FAILURE						
BDI	10.88	3.66	11.00	1.63	12.22	4.54
MAACL	12.88	5.19	13.66	4.17	13.66	3.22

NOTE: BDI = Beck Depression Inventory;

MAACL = Multiple Affect Adjective Checklist.

To manipulate subjects' performance on the anagram task (success or failure), it was necessary to use both types of anagrams, solvable and unsolvable anagrams.

In failure conditions, subjects were given 12 unsolvable anagrams (e.g., BNAHE), and 8 solvable ones (e.g., ODELM-MODEL). The order of presentation was random.

In success conditions, subjects were given 20 solvable anagrams of a moderate difficulty. All anagrams had similar letter arrangements (e.g., UMANH-HUMAN) and were selected from Tresselt and Mayer's list (1966).

Prior to commencing the task, all subjects received a training session consisting of 5 anagrams, the aim being the explanation

of the experimental procedure.

Attribution manipulations. Before commencing the task, all subjects received the following instructions:

You may want to know how a sample of Polytechnic students performed on this task, here is a figure showing how they performed.

The figures shown varied according to each experimental condition. In both IAS and EAF conditions, the figure presented merely showed the high percentage of students (80%) failing at this task (task difficulty).

In both EAS and IAF conditions, the figure presented showed the high percentage of students (80%) succeeding at this task (ease of the task).

This information concerning other students' performance at similar task is expected to influence attributions to internal or external factors. A similar procedure has been successfully used by Klein et al. (1976) to manipulate subjects' attributions of their performance on a problem solving task.

In the present experiment, no attempt has been made to manipulate the specific-global dimension of attribution (Abramson et al., 1978).

Following the success or failure feedback on the anagram task, all subjects were asked to estimate as accurately as possible the number of anagrams they had solved. It was stated that subjects solving 10 or more anagrams had succeeded and those

who solved less than 10 had failed. After receiving success or failure feedback (determined by the number of anagrams solved), all subjects were given one of the two attribution questionnaires (see Appendix A.2) adapted from Rizley (1978). Each questionnaire inquired about the causal determinants of either success or failure. All factors known to influence attributions to internal causes (e.g., ability, effort) and attributions to external causes (e.g., task difficulty, luck) were listed and subjects were asked to indicate on 7-point scale (ranging from 1-definitely not a cause of my success or failure; to 7-definitely a cause of my success or failure), the extent to which each of the factors determined their success or failure.

Upon completion of the attribution questionnaire and the MAACL, all subjects were debriefed, paid and thanked for their participation.

### 5.2.2 RESULTS

Manipulation checks. Analysis of the data from the question that asked subjects to estimate the number of anagrams they had solved indicated that subjects in success conditions experienced success and subjects in failure conditions experienced failure. A 2 x 2 analysis of variance of these data showed that the effect for outcome category (success-failure), was as expected, significant,  $F(1,48) = 227.02, p < .0001$ ; such that subjects in success conditions reported that they had solved more anagrams than those in failure conditions (overall

TABLE 1.2 - EXPERIMENTAL PROCEDURE

SELECTION PHASE	TREATMENT PHASE	ASSESSMENT PHASE
Only Ss scoring 8 or more on the BDI were selected. Ss also completed the MAACL as a further index of depression.	Following performance on an anagram task, Ss were told either they succeeded or failed and then were induced to attribute their performance to either internal or external causes. Two groups of Ss received no such instructions (control).	Ss' mood (including anxiety, depression, and hostility) was assessed by means of the MAACL.

NOTE: BDI = Beck Depression Inventory;

MAACL = Multiple Affect Adjective Check List.

M = 13.55 and 5.22 respectively). Table 1.3 presents the means and standard deviations of estimated number of anagrams solved.

An analysis of variance of attribution ratings indicated a significant effect for attribution,  $F(2,48) = 3.65$   $p < .05$ . Further analyses showed that internal attribution groups made more attributions to internal factors (ability, effort) than

to external ones. Table 1.3 presents the means of attribution ratings by condition. Inspection of this table indicates that control groups (FO, SO) ascribed more failure than success to personal causes (M = 4.50 and 3.22 respectively). This result lends further support to the findings (Kuiper, 1978; Rizley, 1978) that depressed persons have an exaggerated tendency to make negative self-attributions.

TABLE 1.3 - MEANS OF NUMBER OF ANAGRAMS SOLVED AND ATTRIBUTION RATINGS

MEASURE	IAS	EAS	SO	IAF	EAF	FO
ANAGRAMS	12.87	14.11	13.33	5.75	5.11	5.44
ATTRIBUTIONS						
INTERNAL	5.00	4.22	4.00	3.33	2.77	4.50
EXTERNAL	3.61	3.27	3.22	2.00	3.56	3.22

NOTE: Attribution ratings could range from 0 to 7. IAS = Internal attribution for success; EAS = External attribution for success; SO = Success only; IAF = Internal attribution for failure; EAF = External attribution for failure; FO = Failure only.

Mood Results. Preliminary analyses of the experimental data from the MAACL indicated that subjects displayed more hostility and reported more anxiety in the failure conditions than in success ones. In addition, the data indicated that depression is influenced not only by outcome (success-failure) but also by the type of attributions made to account for the

outcome in question. In order to compare treatment effects, anxiety and hostility change scores from pre - post administration were computed and subjected to analyses of variance. For the purpose of the experimental hypotheses and because of the nature of present data, an analysis of covariance was performed on depression scores.

Anxiety. A 2(success, failure) x 3(internal attribution, external attribution, no attribution instructions) analysis of variance of the anxiety change scores indicated that the main effect for outcome category (success, failure) was significant,  $F(1,48) = 7.40, p < .01$ . The main effect for attribution was also significant,  $F(2,48) = 3.71, p < .05$ . A simple main effects analysis revealed that subjects in IAF condition experienced more anxiety than those in EAF condition,  $F(2,48) = 3.33, p < .05$ . However, the interaction between outcome and attribution was not obtained ( $F < 1.0$ ).

Hostility. Analysis of the hostility data from the MAACL showed that all subjects displayed more hostility following failure feedback than following success feedback. Analysis of variance of hostility change scores yielded a significant main effect for outcome,  $F(1,48) = 7.75, p < .01$ . Neither the effect for attribution nor the interaction were significant.

Depression. Analysis of the data from the depression scale of the MAACL indicated a substantial change in depression following experimental treatments. The hypothesis that depressed mood will worsen following internal attributions of

failure but not following external attributions of the same outcome, was strongly supported by the present data. The analysis of covariance of depression scores, with pre-treatment scores serving as a covariate, indicated a significant effect for outcome category,  $F(1,47) = 15.16, p < .001$ . The effect for attribution approached statistical significance,  $F(2,47) = 2.47, p > .05 < .10$ . Table 1.4 presents the results of this analysis of covariance. As can be seen in the table, the interaction (outcome x attribution) was significant,  $F(2,47) = 7.63, p < .005$ . Figure 1.1 illustrates this interaction.

TABLE 1.4 - RESULTS OF THE 2 x 3 ANALYSIS OF COVARIANCE

SOURCE OF VARIANCE	SS	DF	MS	F	P
OUTCOME (O)	144	1	144	15.16	<.001
ATTRIBUTION (A)	47	2	23.4	2.47	ns.
O x A	145	2	72.5	7.63	<.005
ERROR	445	47	9.5		

The adjusted means of treatment and control groups were computed and compared by T tests. The treatment comparisons indicated as expected that IAS group reported significantly less depression than IAF group,  $t(16) = 3.53, p < .005$ . Further comparisons indicated that the former groups (IAS) had a lower depression mean ( $M = 12.01$ ) than SO group ( $M = 15.30$ ), although the difference did not reach statistical significance,  $t(16) = 1.54, p > .05 < .10$ . The picture that emerged from this result suggests that IAS treatment initiated changes in the euphoric direction. In

contrast, IAF treatment appeared to have caused more dysphoria (pre  $M = 12.89$  post  $19.56$ ). As predicted IAF group reported more depression on the post experimental mood questionnaire than EAF group,  $t(16) = 2.47$ ,  $p < .025$ . Further comparisons showed that subjects induced to externalize failure (EAF) experienced less depression than their counterparts in control (F0) condition ( $M = 14.28$  and  $18.28$  respectively). The difference between the two means reached significance,  $t(16) = 1.87$ ,  $p < .05$ . No significant differences were found between IAF and F0 groups. Failure to obtain differences on this measure may be due to the tendency of depressed persons to make internal attributions for bad outcomes (see Table 1.3).

### 5.2.3 DISCUSSION

The present investigation examined the effects of causal attributions for success and failure on mood of mildly depressed students. In addition, the study attempted to replicate previous findings (Kuiper, 1978; Rizley, 1978; Seligman et al. 1979) that depressed persons have a tendency to attribute failure but not success to internal or personal dispositions.

The major hypothesis of this study was derived from Rizley's (1978) theoretical position that depression reflects distortion in the attribution of causality. To some extent, the current results corroborate this attributional viewpoint. It was demonstrated that depression (as measured by the BDI and MAACL) was substantially and consistently influenced by attribution



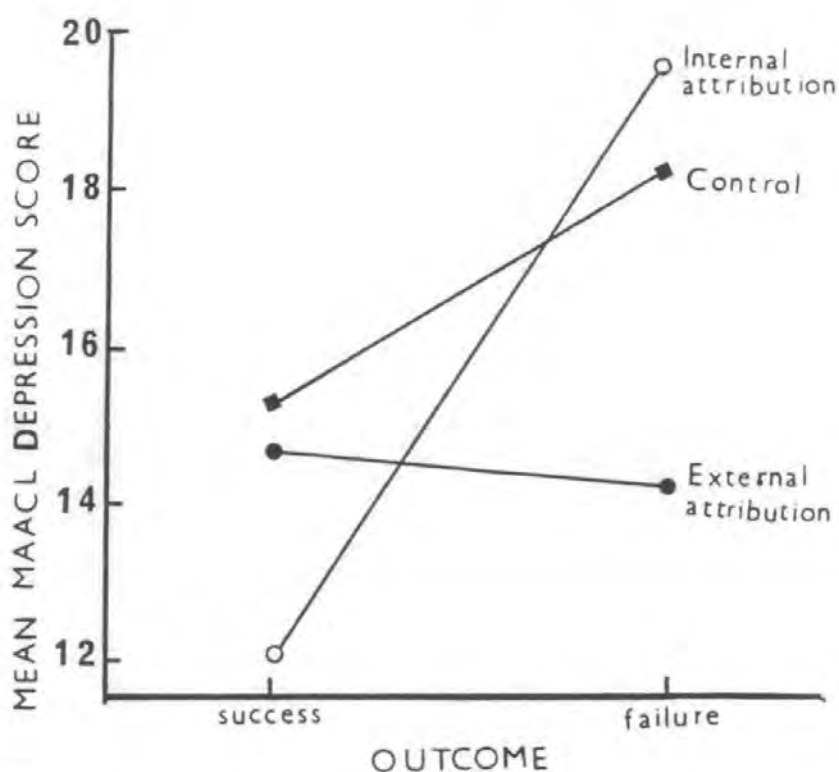


Fig. 1.1. Mean affect adjective check-List(MAACL) depression score as a function of attribution(Internal-External) and outcome(Success-Failure).

instructions. For instance, it was found that depressed subjects were more depressed and anxious after making internal attributions for failure than when failure was attributed to external or situational constraints.

The pattern of results that emerged from this study seems to suggest that a combination of failure experience and self attribution, is sufficient for a depressive episode .

Although it may seem premature to claim etiological implication for negative self-attributions, it is important to note that only subjects exhibiting these types of attributions did

experience changes in the dysphoric direction.

Why were subjects dysphoric but not so much anxious or hostile following internal attributions for failure treatment?

It is possible that this negative change in depression reflected nothing but a general tendency of depressed subjects to endorse more items that are indicative of depression than of hostility or anxiety on the MAACL. This explanation, however, becomes unlikely when considered in the light of the remaining results. For instance, it was found that following external attribution for failure treatment subjects were no more depressed than hostile or anxious. In fact they reported significantly less depression than their counterparts in failure only condition (control).

Another and probably more plausible explanation for this increase in depression could be deduced from one of Beck's clinical observations:

"When the person attributes the cause of loss to himself, the rift in his domain becomes a chasm: he suffers not only the loss itself but he discovers a deficiency in himself" (p. 10, 1974).

Based on this observation and on the results obtained in this study, negative self-attributions seem to constitute a major antecedent of a depressive experience. One may speculate that depressed persons adopt this attributional strategy to maintain and/or exacerbate their depressive state.

In line with the above proposition, a number of researchers

(e.g., Beck, 1967; Nelson & Craighead, 1977) found that depressed patients attend to information that may confirm their negative attributes (e.g., negative self concept, inadequacy, unworthiness ...).

The finding that internal attributions for failure lead to a dysphoric reaction is compatible with the view espoused by learned helplessness theorists (Abramson, Seligman and Teasdale, 1978). According to the new helplessness model, depression and its cognitive and affective symptoms result from one's firm belief that he is incompetent in exerting control over important life events.

Although the present experiment was not specifically designed to test this hypothesis, it provides data which supports the attributional account of depressive manifestations.

Consistent with the attributional explanation of depressive reactions, is the finding that only depression and to a lesser extent anxiety were affected by the attribution manipulations. Both hostility and anxiety affects tended to vary as a function of outcome (success-failure) per se. For instance, subjects displayed more anxiety and reported to have been feeling more hostile in failure conditions than in success ones. To the extent that subjects' hostile reaction was engendered by the situation it could be adaptive in nature. Since it could be argued that they were attempting to cope with a rather embarrassing situation in which they were exposed to a public (experimenter) evaluation. It is also possible that subjects'

increased hostility following unsuccessful attempts to solve anagrams, was associated with a motive to restore control over outcomes (Wortman and Brehm, 1975). Viewed from this perspective hostility and to some extent anxiety, are reactive in nature. According to Wortman and Brehm's reactance theory (1975), people who expect to have control react with hostility and anger when "freedom" of exerting it (control) is threatened. It will be recalled that instructions suggesting the ease of the task (e.g., internal attribution of failure) were likely to raise subjects' expectations of success. It follows that increased hostility in failure conditions may have resulted from the discrepancy that has been created between expectations and actual outcome.

The results obtained from the post experimental mood questionnaire indicated as expected, that subjects in failure only condition (control) felt as depressed as those in internal attribution of failure condition. This finding is not surprising, since both groups provided similar personal accounts (internal attributions) for their failure to solve most of the anagrams making the task. This tendency of depressed persons to hold themselves responsible for bad outcomes may play a crucial role in the exacerbation process referred to earlier. If this attributional explanation is correct, one would expect to prevent the damaging effects of failure experience by reducing subjects' responsibility for the undesired outcome. This hypothesis was tested and was strongly supported by the data from the present experiment. It was found that subjects

induced to attribute failure to external or situational causes, reported to have been feeling less depressed and relatively less anxious than those ascribing causality to themselves for failure. Apparently the external attribution instructions were successful in initiating cognitions by which further dysphoria was prevented.

This prophylactic effect of external attributions bears some resemblance to what Rippere (1979) described as anti-depressive behaviour. According to this author people possess a repertoire or a constellation of behaviours that they presumably display to avoid sinking into depression. A deduction from this proposition is that the external attribution manipulation equipped subjects with 'constructs' capable of tackling the situation.

Another possibility is that the manipulation corrected depressives' tendency to self blame, thereby reducing the risk of further dysphoria. Viewed from this perspective the finding may have some therapeutic implications. Teaching depressed patients to adopt self-protective attributions may be beneficial since such procedure could reduce subjects' feelings of inadequacy, worthlessness and other features of depression. For instance, Beck's cognitive therapy advocates almost similar procedures for the treatment of clinically depressed patients. Briefly, this therapy is aimed at modifying patients' negative cognitions and "silent assumptions".

Data from the attribution questionnaires showed that the

failure only (F0) group had higher internality scores than the success only (S0) group. Thus, lending support to the consistently replicated finding (e.g. Kuiper, 1978; Rizley, 1978) that depressed persons tend to make internal attributions for unsuccessful outcomes but not for successful ones. This result also lends support to Seligman et al.'s (1979) suggestion that depressives are characterized with a specific attributional style.

Finally, the contention that the helplessness deficit and depressive symptoms could be alleviated by mastery experiences was not particularly supported by the current data. In this experiment, subjects receiving success only (S0) treatment showed little or no improvements in mood. One explanation for this discrepancy is that subjects in the present experiment may not have perceived success. But the manipulation checks argue somewhat against this possibility. Another and maybe more plausible explanation lies in the depressives' 'Maladaptive' perception of causality, that is their reluctance to take credit for success. Based on the latter explanation, a procedure that makes success more attributable to personal dispositions should produce positive changes in mood. Data from the mood questionnaire tends to support this proposal. It was found that internal attributions of success group reported less depression (although not statistically significant) than success only group. Unlike success only treatment internal attribution instructions appeared to have given subjects a means for internalizing success and engaging in

positive self-evaluations.

### 5.3 EXPERIMENT 2

The results obtained in Experiment 1 strongly indicated that depressed subjects' mood was affected or even determined by the type of attributions they displayed to account for their success or failure on the anagram task.

Although Experiment 1 revealed a substantial degree of association between attribution and depression as measured by the MAACL, it was possible that some of the results supporting this finding reflected experimental artifacts rather than actual treatment effects. First, the previous experiment used not only an artificial laboratory task but also a 'single' person situation, thus the results may be seriously limited in terms of external validity. Second, the observed effects could be prone to criticism on the grounds that they were entirely based on verbal or self reports. It will be recalled that the major independent variable (depression) was in fact assessed by an inventory relying solely on subjects' judgements concerning their subjective or internal state. Although great care was taken to minimize the demand effects (Orne, 1962), the results could still qualify for alternative interpretations. Therefore Experiment 2 was conducted to remedy to the methodological inadequacies inherent to the initial investigation.

Accordingly, a dyadic situation was used and performance aspects were assessed in addition to mood ratings. In brief, Experiment 2 further examined the effects of attributions on some features

of depression (including mood and psychomotor speed) in a dyadic situation. Based on the results from Experiment 1, it was anticipated:

1. That subjects would feel more depressed and exhibit more performance deficits following internal attributions for failure than following external attributions of similar outcome.
2. That subjects would also report less depression and less performance deficits after internal attributions for success than after externalization of success.

As was found in Experiment 1, both anxiety and hostility affects were expected to vary as a function of success-failure per se.

### 5.3.1 METHOD

Subjects and Design. Forty undergraduates, 18 males and 22 females, from Plymouth Polytechnic participated in this experiment. Subjects were selected on the basis of their scores on the Zung Self-Rating Depression Scale (SDS)<sup>1</sup> (Zung, 1965). Subjects scoring 25 or more on the SDS were selected and randomly assigned to one of the following experimental conditions:

1. Internal attribution of success (IAS).
2. External attribution of success (EAS).
3. Internal attribution of failure (IAF).
4. External attribution of failure (EAF).

The SDS is an instrument widely used in research with depressed



persons (see chapter 2). Studies using the SDS to identify depressed college students (e.g., Janoff-Bulman, 1979) recommended a cutting line of 22, thus those scoring more than 22 were usually classified as depressed.

As in Experiment 1, the MAACL was administered as a further index of depression. Table 2.1 presents the means and standard deviations of the SDS and MAACL for each experimental group. The correlation between SDS and MAACL scores was .57 ( $p < .005$ ).

TABLE 2.1 - MEANS AND STANDARD DEVIATIONS OF SDS AND MAACL SCORES

OUTCOME	INTERNAL ATTRIBUTION		EXTERNAL ATTRIBUTION	
	M	SD	M	SD
SUCCESS				
SDS	34.10	6.45	33.20	6.53
MAACL	14.70	4.01	13.50	4.58
FAILURE				
SDS	31.55	6.62	33.10	6.68
MAACL	13.00	5.45	13.70	6.13

NOTE: SDS = Self Rating Depression Scale.

MAACL = Multiple Affect Adjective Checklist.

Procedure. This experiment consisted of three different phases:

- (a) Selection phase - in which subjects completed both the SDS and MAACL.

- (b) Treatment phase - in which subjects were induced to attribute success or failure on a problem solving task to either internal or external causes.
- (c) Assessment phase - in which subjects' mood as well as performance were assessed.

When a subject entered the room, he or she was seated at a table facing another subject of the same sex (confederate). Upon completion of the SDS and MAACL, both subjects (the subject and the confederate) were each given a booklet containing 20 matrices obtained from Raven's Advanced Progressive Matrices set II (1962). Subjects were then given the following instructions for the task (adapted from Prindaville and Stein, 1978):

The present experiment is designed to examine the relationships between some personality variables and problem solving strategies. The task that you will be given consists of a series of problems. The task itself is known as a pattern completion test. There are, as you can see, eight patterns at the top of the page which are arranged in a given order according to some logical principle. Your task is to select from among eight other patterns, at the bottom of the page, the pattern which goes next in the sequence according to that principle. I will be telling you whether you are correct or incorrect on every problem. You will have 25 seconds for each problem, after which I will ask for your choice.

After receiving the standard instructions, all subjects were given additional information concerning the nature of the task (easy or difficult). The procedure used to manipulate subjects'

attributions of success or failure on the problem solving task, was similar to that used in Experiment 1. The confederate's success or failure at the problem solving task was used to strengthen the attribution manipulations (this procedure has been successfully used by Wortman et al., 1976). According to Kelley (1971) an individual's attributions are influenced not only by his behaviour but also by the behaviour of other people with whom he interacts.

Following the problem solving task, all subjects were asked to estimate the number of problems they had solved; they were then told that they had succeeded or failed depending on the experimental condition to which they were assigned. Following success or failure feedback, subjects were asked to make attributions to the following internal and external factors: ability, effort, task, and luck. The procedure used to assess subjects' attributions of success or failure in the present experiment is identical to that used in Experiment 1.

Upon completion of the attribution questionnaire, subjects' mood was again assessed.

Test task. Following the administration of the MAACL (post assessment of mood), all subjects were asked to participate in the second but different problem solving experiment. The task used to assess subjects' performance consisted of 20 anagrams frequently used in human helplessness studies. Two measures of anagram performance were obtained (a) number of failures to solve within 100 seconds; (b) mean response latency for 20 anagrams.

After completing the anagram task, all subjects were adequately debriefed, paid, and thanked for their participation.

### 5.3.2 RESULTS

Preliminary analyses of the data showed that the effect for sex was not significant. Therefore this variable will be dropped in subsequent analyses.

#### Mood results

Anxiety. A 2(success-failure) x 2(internal-external attribution) analysis of anxiety change scores showed that the effect for outcome category (success-failure) was not significant,  $F(1,36) = 2.80$ . Neither the other main effect nor the interaction were significant. All  $F_s < 1.0$  (overall mean for success 6.95 failure 8.40).

Hostility. A 2 x 2 analysis of variance of hostility change scores revealed that the main effect for success and failure was significant,  $F(1,36) = 5.08$ ,  $p < .05$  (overall M for success = 7.75 and failure 10.25). As can be seen in Table 2.2, subjects reported more hostility following failure than following success.

Depression. Inspection of depression data from the MAACL showed that there was a trend for subjects to become dysphoric following negative self-attributions and to become slightly euphoric following internal attributions for success. An analysis of covariance of the depression scores indicated that the effect for success-failure reached statistical significance,  $F(1,35) =$

TABLE 2.2 - MAACL MEANS AS A FUNCTION OF SUCCESS-FAILURE  
AND INTERNAL-EXTERNAL ATTRIBUTION

OUTCOME	INTERNAL ATTRIBUTION		EXTERNAL ATTRIBUTION	
	DEPRESSION			
	Pre	Post	Pre	Post
SUCCESS	14.70	13.20	13.50	13.40
FAILURE	13.00	16.50	13.70	14.90
	ANXIETY			
	Pre	Post	Pre	Post
SUCCESS	8.50	7.00	6.30	6.90
FAILURE	7.40	8.10	8.30	8.70
	HOSTILITY			
	Pre	Post	Pre	Post
SUCCESS	9.00	8.30	7.30	7.20
FAILURE	7.90	9.80	9.00	10.70

5.75,  $p < .05$ . However, the interaction was not significant,  $F(1,35) = 1.37$  ns, although the tendency was in that direction.

Subsequent comparisons of adjusted means showed that IAF had higher depression mean than IAS group ( $M = 16.90$  and  $12.66$  respectively), the difference approached significance,  $t(18) = 1.55$ ,  $p > .05 < .10$ . Further comparisons indicated that IAF group reported more depression than EAF group ( $M = 16.90$  and  $14.91$  respectively). Using a median split, subjects were

subdivided into high depressed (scores ranging from 16 to 24 on the MAACL) and low depressed (7 to 15 on the MAACL) groups. This internal analysis showed that 'high' depressed tended to improve following IAS treatment (Pre M = 17.50 and Post 13.17). In contrast 'low' depressed ones tended to become dysphoric after IAF treatment (Pre M = 8.60 and Post 13.20).

### Behavioural measures

Anagram performance. An analysis of variance number of failures to solve anagrams indicated that neither the main effects nor the interaction were significant. As can be seen in Table 2.3, IAF group solved less anagrams than any of the remaining groups. Further analysis showed that IAF group performed worse than IAS group,  $t(17) = 2.18, p < .025$ .

TABLE 2.3 - MEAN ANAGRAM PERFORMANCE AS A FUNCTION OF  
SUCCESS-FAILURE AND INTERNAL-EXTERNAL ATTRIBUTION

OUTCOME	INTERNAL ATTRIBUTION	EXTERNAL ATTRIBUTION
NO. OF FAILURES TO SOLVE		
SUCCESS	2.67	3.90
FAILURE	5.30	4.20
MEAN RESPONSE LATENCY (in sec)		
SUCCESS	24.50	38.46
FAILURE	42.79	31.49

NOTE: This table shows the raw data; maximum response latency is 100 sec.

Psychomotor Speed. The latency data shown in Table 2.3 was subjected to logarithmic transformation before statistical analysis. A 2 x 2 analysis of variance of transformed data showed that neither of the main effects were significant. However, the interaction was very significant,  $F(1,31) = 7.93$ ,  $p < .001$ . Table 2.4 presents the results of a 2 x 2 analysis of variance. As anticipated, subjects receiving IAF treatment were slower in the subsequent test task than those given EAF treatment,  $t(17) = 1.75$ ,  $p < .05$ . Further comparison showed that IAS group were faster than EAS group,  $t(17) = 2.20$ ,  $p < .025$ .

TABLE 2.4 - RESULTS OF THE 2 x 2 ANALYSIS OF VARIANCE

SOURCE OF VARIANCE	SS	DF	MS	F	F
OUTCOME (O)	0.02	1	0.02	1	ns.
ATTRIBUTION (A)	0.02	1	0.02	1	ns.
O x A	0.46	1	0.46	7.93	<.001
ERROR	1.8	31	0.06		

### 5.3.3 DISCUSSION

This second experiment was an attempt to evaluate further the effects of manipulating attributions on depressed subjects' mood and performance. The results obtained offered mixed support for the hypotheses previously advanced. Consistent with the findings in Experiment 1, the mood results indicated, although not strongly, that subjects induced to ascribe causality to themselves for failure experience felt dysphoric

but not anxious or hostile. In contrast, those induced to attribute previous failure to external causes rated themselves as feeling only slightly depressed.

Based on these results, it does seem that the cognitive device (attribution) that subjects presumably employed to explain or interpret their unsuccessful attempts to establish control over the outcome of the initial task, had a substantial impact on their subsequent mood or reaction. As in Experiment 1, increased depression was more associated with instructions advocating incompetence or inability as an ultimate cause of failure than with instructions suggesting the 'harshness' of the environment as a causal determinant of failure. These results are generally consistent with the abundant literature that emphasises the role of negative self-inferences and other negative cognitions in the depressive process.

As observed in the initial experiment, depression tended to increase following internal attributions for failure than after external attributions of similar outcome. Apparently, this change in the depressive effect is directly related to the tendency of depressives to associate their ineffective actions with personal defects. To the extent that this interpretation is correct, it is in disagreement with Seligman's proposition (1975) that depression is the end product of an associative deficit. In other words, the failure to perceive the relationships between responses and outcomes is supposed to be the sine qua non of depression.

Although the present experiment was not specifically designed



to test Seligman's proposition, it nevertheless provides data which suggest that it is the 'associative' belief rather than the lack of it which seems to be closely linked to the depressive condition. Recent research on judgement of objective contingency (Alloy and Abramson, 1979) in depressed and non-depressed college students has also refuted the associative deficit hypothesis. In a series of experiments, Alloy and Abramson demonstrated that given a degree of objective contingency between subjects' responses and outcomes, depressed subjects were surprisingly 'realistic' and accurate in their judgements. Non-depressed subjects, in contrast, showed an 'illusion' of control over desired outcomes, even in the absence of any objective contingency between their responses and outcomes. However, when outcomes (contingent) are undesired, non-depressed subjects unlike depressed ones, tended to underestimate the degree of contingency or relationship between their actions and outcomes.

As anticipated, the behavioural data indicated that subjects' subsequent performance at the test task was influenced by the type of attributions they displayed to account for their initial success or failure on the pre-treatment task. Here again, failure experience and self-attributions seemed to have combined to undermine performance on the anagram task, or at least one aspect of the performance. Inspection of the relevant data revealed that EAF group reported lower latency scores than IAF group. Although faster at solving anagrams, the former group (EAF) solved relatively no more than their

counterparts in IAF condition. This result is not quite consistent with the Klein et al.'s finding (1976), that external attribution instructions improved depressed subjects' performance. If anything, the present data suggest that the external attribution manipulation prevented subjects' performance from deteriorating. A possible explanation for this apparent discrepancy is that the designs of the two experiments differed. Unlike the Klein et al.'s study, the present one failed to control for the effect of attribution or instructions. Failure to include a control group (Failure Only group) in the present experiment made it difficult to tell whether subjects' performance did in fact improve following the external attribution instructions.

Another explanation is that Klein and his colleagues did in fact observe, like here, a prophylactic effect of external attribution instructions, but interpreted it differently. This is quite possible inasmuch as their work is based on the 'erroneous' assumption that depression reflects a deficit in performance; and according to their rationale any manipulation aimed at correcting the helplessness symptom would automatically improve depressed subjects' performance. While laboratory produced helplessness may reflect a deficit in performance, there is little evidence suggesting that such deficit is characteristic of clinical depression. Studies that so far dealt with this issue (Lobitz and Deepost, 1979; Loeb et al. 1971; Rozensky et al. 1977), failed to obtain any significant performance differences between clinically depressed and non-depressed patients.

Although some of the mood results did not quite achieve the conventional statistical significance, there was nevertheless a trend for the data to suggest that subjects experienced more depression following internal attributions for failure than following external attributions for a similar outcome. Further, the results indicated as in Experiment 1, that attribution instructions affected depression but not hostility or anxiety.

One question should be raised at this point. Why were the present mood results less significant than those obtained in the previous experiment? A possible explanation for this lack of congruency could be deduced from the results of an experiment carried out by Golin and his colleagues (1980). In their study, they instructed a group of depressed students that they would win a prize if they were successful at solving some anagrams. Another group was informed that they would be given a 'second chance' if they failed the anagram task. The data obtained clearly showed that the 'second chance' group reported less depression, anxiety and hostility than the former group ('one chance' group).

It is possible that the test task (anagram task) in the present experiment was perceived by subjects as a 'second chance' or as an opportunity for them to regain control; it follows that subjects' perception of the second experiment (test task) may have interfered with attribution instructions, thereby attenuating their effects on mood.

In summary, the present study showed that inducing depressed students to adopt one attributional strategy rather than another affected, although not strongly, both their subsequent mood and performance.

#### 5.4 GENERAL DISCUSSION AND IMPLICATIONS

The major purpose of the two investigations was to examine and evaluate the effects of manipulating depressed students' attributions for success and failure on their subsequent mood and performance. Based on the previous research reviewed here (Kuiper, 1978; Rizley, 1978; Seligman et al. 1979), it was predicted that subjects' depressed mood will be influenced not only by their initial success or failure but also by the kind of attributions they display to account for their performance. The results obtained clearly supported this attributional account of the depressive reaction. It was found that subjects' subsequent mood was a function of both outcome (success-failure) and attributions made about the outcome. Overall, the results were congruent with Weiner et al (1971) proposition that the consequences of a performance are mediated by attributions that subjects make about the causes of their performance.

The finding in the present studies that increased depression was more associated with internal than external attributions for failure, provides further confirmation that negative self-attributions play an important role in the development of depressive episodes. Such finding also suggests that the intensity of depressive feelings may be related to these

depressogenic or maladaptive attributions.

The prediction that the 'damaging' effects of failure experience could be attenuated or even prevented by directing subjects' attributions towards external causes was supported. As argued earlier, this result may be relevant to therapy of depressed patients. Considering the fact that depressed persons are characterized by a tendency to engage in depressogenic attributions or in Beck's terms:

"to blame themselves for everything that goes wrong around them" (p. 115, 1974),

a procedure that corrects their depressogenic tendency to explain and interpret things that happen to them may have beneficial effects. Attempts at loosening the grip of such depressogenic attributions and beliefs may also, as demonstrated in Experiment 1, prevent further dysphoria and provide means for engaging in antidepressive or protective attributions.

The fact that subjects' subsequent reaction was influenced by attributions they displayed regarding the causes of their success or failure, demonstrates the importance of causal beliefs (attributions) in shaping affective or emotional responses. A question that should be asked at this point, is whether such attributions equally influence behaviour? The second experiment was partially designed to answer this question. The behavioural data offered mixed support for the hypothesized relationship between attribution and behaviour. Additional research using less sophisticated behavioural measures is

required to clarify and elaborate on this issue.

The mood results from both experiments indicated quite clearly that depression as reflected in both the BDI and MAACL was more affected by attribution instructions than either hostility or anxiety. Although this result may seem to suggest the specificity of certain types of attributions to depression, replication of such finding with clinical populations is needed before its relevance to theory can be seriously considered. A need also exists for further research to investigate whether, as the mood data of the present investigation tend to suggest, negative self-attributions (internal attributions for negative outcomes) induce dysphoria and other symptoms of depression.

#### FOOTNOTES

1. Because of its nature, this experiment required a relatively shorter and easier scale to administer than the Beck Depression Inventory (BDI). The Zung Self-Rating Depression Scale (SDS) appeared to fulfil these requirements.

## CHAPTER SIX

### THE CAUSAL ROLE OF ATTRIBUTIONS IN DEPRESSION

- 6.1 INTRODUCTION
- 6.2 EXPERIMENT 3
- 6.3 EXPERIMENT 4
- 6.4 GENERAL DISCUSSION



## 6.1 INTRODUCTION

The two studies to be reported in the present chapter have been carried out in an attempt to examine further the relationship between causal attribution and depression. Specifically, these studies were designed to examine the possibility, suggested by Experiments 1 and 2, that certain attributions play a causal role in depression. Although, as found in Experiments 1 and 2, mood changes occurred as a result of the induction of failure attributions, causality between internal attributions for failure (negative self-attributions) and depressive symptoms cannot be inferred without evidence showing that depression or its correlates can be induced or alleviated by manipulating attributions. In fact, the designs of previous experiments do not allow claims to be made about the aetiological role of attributions in depression, since neither of them included non-depressives.

That causal attributions are closely associated with depression has been suggested and documented by the data of several studies (e.g., Barthe and Hammen, 1981; Klein et al., 1976; Kuiper, 1978; Rizley, 1978), but little has been done since to try to determine the nature of this link. Although, in their attributional formulation of learned helplessness and depression, Abramson and her colleagues (1978) have written extensively on this issue, little evidence has been forthcoming to substantiate their claim that helplessness and depression are caused by attributions. A recent study that has directly

attempted to address the question of causality between attributions and depression is that of Seligman and his co-workers (1979). They reported data which showed positive correlations between internal, stable, and global attributions for negative outcomes and depression scores. They also found that subjects' level of depression as reflected on the BDI was negatively correlated with their tendency to make internal, and stable attributions for positive outcomes. In their discussion of the results, Seligman and his colleagues argued in accordance with Abramson et al.'s attributional formulation of depression (1978), that:

"the depressive attributional style ... followed by negative life events, actually causes depression" (p.247).

But they also conceded that their correlational data:

"do not rule out the alternative hypothesis that depression causes people to attribute bad outcomes to internal, stable, and global causes" (p.247).

Another, but more recent study that also examined the question of causality between attributions and depression is that of Golin et al. (1981). In their study, they assessed subjects' attributions of positive and negative outcomes as well as their level of depression on two separate occasions. To overcome some of the problems known to be associated with conventional correlational analyses (e.g., the problem of direction of causality), Golin and his colleagues analysed their data with a sophisticated statistical technique known

as a cross-lagged panel correlational analysis. They found evidence which lends support to Abramson et al. (1978) claim that depression is caused by a combination of a depressive attributional style and failure experiences. But they too cautioned that their results:

"should be viewed as an indicator of temporal precedence and not as a positive proof of causation" (pp. 20-21).

Although, as pointed out, there is a correlational evidence that depression is closely associated with certain <sup>Prevision</sup> attributions, there is no experimental evidence to show that these attributions actually induce depression or its correlates. The present investigation attempted to remedy to this situation by assessing the effects of reversing depressives and non-depressives' attributional style for failure - that is, inducing depressives to adopt a non-depressive attributional style (external attributions for failure), and inducing non-depressives to adopt a depressive attributional style (internal attributions for failure). Based on the attributional formulation of learned helplessness and depression (Abramson et al., 1978; Miller and Norman, 1979) and its elaboration by Seligman et al. (1979), it was predicted that these attributional changes will lead to corresponding changes in level of depression as reflected in mood, expectations, and psychomotor performance.

Another way of evaluating the aetiological significance of (certain) attributions will be to determine the degree of

specificity, if any, in the relationship between these attributions and depression. A clinical study was carried out to directly examine this specificity hypothesis. A specific relationship between attribution and depression was hypothesised. A demonstration of a specific relationship between these two variables is required before attribution can acquire an aetiological status in depression.

To summarise, the present investigation of the relationship between attributions and depression assessed, in an experiment, the effects of reversing depressives and non-depressives' attributional style for failure; and tested in a second but related study, the specificity hypothesis - that is, the hypothesis that certain types of attributions are associated with depression but not with other known psychopathological disorders.

## 6.2 EXPERIMENT 3

In the present study, the effects of inducing failure attributions on depressed and non-depressed subjects' mood, expectations, and psychomotor performance were assessed. The results of Experiment 1, and to some extent those of Experiment 2, indicated that changes in depression (increase or decrease in MAACL scores) were due to certain attributions. Specifically, it was found that changes in depression, as measured by the MAACL and some objective measures, occurred as a result of the experimental induction of failure attributions. This finding led to the speculation, already entertained by Abramson et al.

(1978) and Seligman et al. (1979), that some of these attributions are depressogenic - that is, they may play a substantial role in the aetiology or development of depressive symptoms.

Experiment 3 was designed to address, although only partly, this complex but equally important question about the causal role of attributions in depression. If certain attributions are to be granted an aetiological status in depression, then their induction (i.e., if they are made) should result in deficits similar to those commonly associated with naturally occurring depression (e.g., low mood, reduced expectations of future success or pessimism, psychomotor deficit and so on). Conversely, if such attributions are to be allocated a causal role in depression, then their modification or correction should be reflected (positively) in subsequent mood, expectations, and performance. Experiment 3 was an attempt to test, although only partly, these possibilities.

Although Experiments 1 and 2 involved attribution manipulations, their primary concern was to determine the extent of the relationship between attribution and depression. Experiment 3, however, was more directed towards evaluating the possible aetiological or causal effects of some of the attributions that have been shown to be closely linked to depression. Accordingly, both depressed and non-depressed subjects were included in the design of this experiment. It was expected that the induction of internal attributions for failure (IAF) will have more impact (negative) on non-depressed subjects' mood, expectations, and psychomotor performance than on those of depressed.

Because unlike the former ones, the latter ones (depressed) display their usual attributions (negative self-attributions). The induction of external attributions of failure, in contrast, is predicted to have more effect (relatively positive) on depressed than on non-depressed subjects. This differential effect of external attribution for failure (EAF) could also be explained by the fact that non-depressives adopt their usual attributional style for failure, whereas depressives acquire a new but a non-depressive attributional style for failure. It was also predicted that depressed subjects will report less depression following external attributions for failure (EAF) than following internal attributions for failure (IAF) or failure only (FO) (control). For non-depressed subjects, it was predicted that they will feel more depressed following internal attributions for failure (IAF) than following external attributions for failure (EAF) or failure only (FO) (control).

To summarise, the purpose of Experiment 3 was to assess the effects of failure attributions on mood, expectations, and psychomotor speed of depressed and non-depressed college students (high and low BDI's).

#### 6.2.1 METHOD

Subjects and Design. Forty-eight undergraduates, 37 females and 11 males, from Plymouth Polytechnic served as subjects in the present experiment. Subjects were selected and assigned to a depressed or non-depressed group on the basis of their

scores on the Beck Depression Inventory (BDI)<sup>1</sup> scores (Beck et al., 1961). Subjects scoring 9 or more on the BDI were assigned to the depressed group, and those with BDI scores of 8 or less were assigned to the non-depressed group. Subjects also completed the Multiple Affect Adjective Check List (MAACL) today form (Zuckerman and Lubin, 1965) before and after the experimental manipulations. The MAACL is more sensitive than the BDI to changes in depressed mood. Table 3.1 presents the means and standard deviations of BDI and MAACL scores for each of the following experimental conditions of the experiment:

1. Depressed/internal attribution for failure (D/IAF).
2. Depressed/external attribution for failure (D/EAF).
3. Depressed/failure only (D/FO).
4. Non-depressed/internal attribution for failure (ND/IAF).
5. Non-depressed/external attribution for failure (ND/EAF).
6. Non-depressed/failure only (ND/FO).

As indicated above, this experiment was a 2 (Mood-depressed/non-depressed) x 3 (Internal attribution for failure, External attribution for failure, failure only) factorial design.

The procedure used to manipulate subjects' attributions about their failure at the empathy task, was similar to that used in Experiments 1 and 2.

Empathy task. The task was described as a test of 'social intelligence', and was similar to that used by Kuiper (1978).

TABLE 3.1 - MEANS AND STANDARD DEVIATIONS OF BDI AND MAACL  
BY MOOD AND ATTRIBUTION

MOOD	IAF		EAF		FO	
	M	SD	M	SD	M	SD
<u>DEPRESSED</u>						
BDI	10.37	1.44	12.12	2.39	12.62	3.10
MAACL	13.37	5.11	15.00	7.48	16.50	2.91
<u>NON-DEPRESSED</u>						
BDI	3.50	1.65	4.87	1.51	3.62	2.18
MAACL	9.87	4.05	8.87	3.96	9.75	5.78

NOTE: BDI = Beck Depression Inventory;

MAACL = Multiple Affect Adjective Check List;

IAF = Internal Attribution for Failure;

EAF = External Attribution for Failure;

FO = Failure only.

The empathy task consisted of 50 words, extracted from a 'word association test' devised by Kent and Rosanoff (1970), each printed on a separate card and was briefly displayed on a tachistoscope. Subjects were given the following instructions (adapted from Kuiper, 1978):

The task that you will be given provides an index of social intelligence. Specifically, it measures with certain accuracy people's ability to know what other people are thinking and feeling. Briefly, the task consists of 50 words, each printed



on a separate card. You will be presented with one word at a time, your task will be to say aloud the word which most people would associate with the word shown to you. It is important to remember that the correct association (answer) is not necessarily the one which you would make, but the one most people tend to make. If your answer is correct you will hear 'correct', if your answer is not correct we will go on to the next word. Are there any questions before we begin?

Dependent measures. Three dependent measures, 2 subjective and 1 objective were obtained, as a measure of level of depression, following the experimental induction of failure and attributions. Subjects first completed the MAACL depression scale, and then they were asked to rate on a 9-point scale how well they think they would perform if given another problem solving task. This scale served as a measure of subjects' expectations of future success. Following the completion of both the inventory and the expectancy scale, subjects were asked to write numbers backwards from 100 on a blank sheet and were timed for 60 secs. This number-writing speed test has been shown to be a good and reliable measure of psychomotor speed (e.g., Coleman, 1975; Velten, 1967).

It was hoped that the combined use of both subjective and objective measures would enable a more reliable and relatively more objective assessment of the dependent variable (depression).

Procedure. Table 3.2 presents a flow chart of the experimental procedure. When the subject entered the room, he or she was seated at a table and then given the BDI and MAACL. Upon completion of the depression inventories, the subject was given information about the purpose of the empathy task, and about how a sample of Polytechnic students performed on this task (attribution manipulations). After receiving the instructions (see empathy task), the subject was led to another table on which the tachistoscope was placed. Before commencing the task, the subject was given an example, the aim being the explanation of the procedure to follow when performing on the empathy task (word association task). Following the demonstration, the subject was presented with the first word from the empathy task. Each word was typed on a separate card and displayed one at a time on the tachistoscope.

Following performance at the empathy task, the subject was asked to estimate as accurately as he/she could the number of correct answers he/she gave during performance at the empathy task. The subject was then told that he/she performed badly (failure), and was then asked to write down the major cause of his/her failure and to indicate, on a 9-point scale, whether his/her outcome was due to personal causes or external causes. The stability and globality dimension were also assessed). Immediately after assessing the subject's attributions about his/her failure on the empathy task, the subject was asked to complete the MAACL. Following completion of the MAACL, the subject was asked to indicate, on a 9-point

TABLE 3.2 - A FLOW CHART OF THE EXPERIMENTAL PROCEDURE

SELECTION PHASE	EXPERIMENTAL PHASE	ASSESSMENT PHASE
Ss with BDI scores of 9 or greater were assigned to a depressed group, and those with BDI scores of 8 or lower were assigned to a non-depressed group.	Ss were randomly assigned to one of the six experimental conditions of the experiment. Four groups 2(depressed and 2 non-depressed) were induced either to make internal (IAF) or external attributions (EAF) for their failure on the empathy task. The two remaining groups (1 depressed and 1 non-depressed) received no attribution instructions, they were given failure only (FO).	Ss' attributions about their failure on the empathy task, their mood, their expectations of future success, and their psychomotor performance were assessed.

NOTE: BDI = Beck Depression Inventory;

IAF = Internal attribution for failure;

EAF = External attribution for failure;

FO = Failure only.

scale, how well he/she thinks he/she would perform if given another problem solving task (expectancy measure). As a measure of his/her psychomotor speed, the subject was given the number-writing speed test. Following the psychomotor performance, the subject was debriefed, paid, and thanked.

### 6.2.2 RESULTS

Manipulation checks. Preliminary analyses of the data from the question that asked subjects to estimate the number of correct answers they had given during performance on the empathy task showed, as expected, that all subjects experienced failure, overall  $M = 10.52$  (21.04% correct).

Similar analyses showed that the attribution manipulation was also successful. Subjects assigned to internal attribution of failure (IAF) condition explained their failure in terms of internal causes (e.g., my nature; inability to understand others), overall  $M = 6.12$ , and those assigned to external attribution of failure (EAF) condition made external attributions for their failure on the empathy task (e.g., not enough time, the situation), overall  $M = 4.56$ . Table 3.3 displays the means and standard deviations of attribution scores and number of correct answers.

As can be seen in Table 3.3, in failure only (F0) condition (control) depressed subjects made internal attributions  $M = 7.24$ , whereas non-depressed ones made external attributions  $M = 4.37$ . This result is in line with the learned helplessness thinking that depressives and non-depressives display

TABLE 3.3 - MEANS AND STANDARD DEVIATIONS OF ATTRIBUTION  
SCORES BY MOOD AND ATTRIBUTION INSTRUCTIONS

MOOD	INTERNALITY		STABILITY		GLOBALITY	
	M	SD	M	SD	M	SD
<u>DÉPRESSED</u>						
IAF	6.25	1.64	6.25	1.39	4.62	1.94
EAF	4.62	1.59	6.00	1.80	6.12	1.85
FO	7.25	0.96	6.87	1.29	6.25	1.71
<u>NON-DEPRESSED</u>						
IAF	6.00	1.87	6.62	1.59	4.87	2.32
EAF	4.50	2.17	6.12	1.38	6.00	1.80
FO	4.37	1.97	5.50	2.69	4.37	2.24

NOTE: IAF = Internal Attribution for Failure;

EAF = External Attribution for Failure;

FO = Failure only.

Ratings are on 9-point scale; higher scores indicate that attributions are more internal; lower scores indicate that attributions are more external.

divergent attributions for negative outcomes (Abramson et al., 1978; Miller and Norman, 1979; Seligman et al., 1979).

The finding is also consistent with the results reported by both Kuiper (1978) and Rizley (1978) that depressed students explained their failure in terms of personal or internal

causes (e.g., inability, incompetence), whereas non-depressed students explained the same outcome in terms of external or situational causes (e.g., task difficulty, bad luck).

TABLE 3.4 - RESULTS OF THE 2 x 3 ANALYSIS OF VARIANCE OF MAACL SCORES

SOURCE OF VARIATION	SS	DF	MS	F	P
MOOD (A)	102.08	1	102.08	5.72	<0.05
ATTRIBUTION (B)	137.37	2	68.68	3.85	<0.05
A x B	50.55	2	25.27	1.42	ns.
ERROR	749	42	17.83		

MAACL Depression Scale. The data from the MAACL depression scale were subjected to an analysis of variance (see Table 3.4). A depression change score was obtained for all subjects (by comparing the pre and post scores on the MAACL), before performing the ANOVA. A 2 (mood-depressed/non-depressed) x 3 (IAF, EAF, FO) analysis of variance of depression change scores revealed that the effect for mood (depressed/non-depressed) was, as expected, significant,  $F(1,42) = 5.72$ ,  $p < 0.05$ . A test on the simple effects of mood (Winer, 1962), showed, as predicted, that non-depressed subjects reported more change in depression in the dysphoric direction than depressed ones following internal attribution for failure (IAF) treatment,  $F(1,42) = 5.60$ ,  $p < 0.05$ . Further, the prediction that depressed subjects will respond more positively to the external attribution for failure (EAF) treatment

was also supported by the data from the MAACL depression scale. It was found that depressed subjects reported less change in depression (in fact, they reported no change at all) in the dysphoric direction than non-depressed subjects after EAF induction,  $F(1,42) = 2.95$ ,  $p < 0.10 > 0.05$ .

The ANOVA of depression change scores also showed that the effect for attribution was significant,  $F(2,42) = 3.85$ ,  $p < 0.05$ . A test on the simple effects of attribution revealed that non-depressed subjects felt more dysphoric following IAF than following EAF or FO treatment ( $p < 0.05$ ). Depressed subjects, however, reported no more depression following IAF treatment than following other treatments ( $F < 1.0$ ). Because, as argued earlier, non-depressed subjects were adopting the depressive attributional style, whereas the depressed ones were adopting their usual attributional style for failure. As can be seen in Figure 1, the effects of reversing non-depressives and depressives' attributional style for failure were in the predicted direction. Non-depressed subjects felt more dysphoric pre  $M = 9.87$  and post  $= 17.50$ , depressed subjects, however, reported no more depression pre  $M = 15.00$  and post  $M = 15.00$ . Table 3.4 displays all other means of MAACL change scores.

Expectancy scale. An analysis of variance of the data from the question that asked subjects to indicate how well they expect to perform if given another problem solving task showed that only the effect for attribution was significant,  $F(2,42) =$

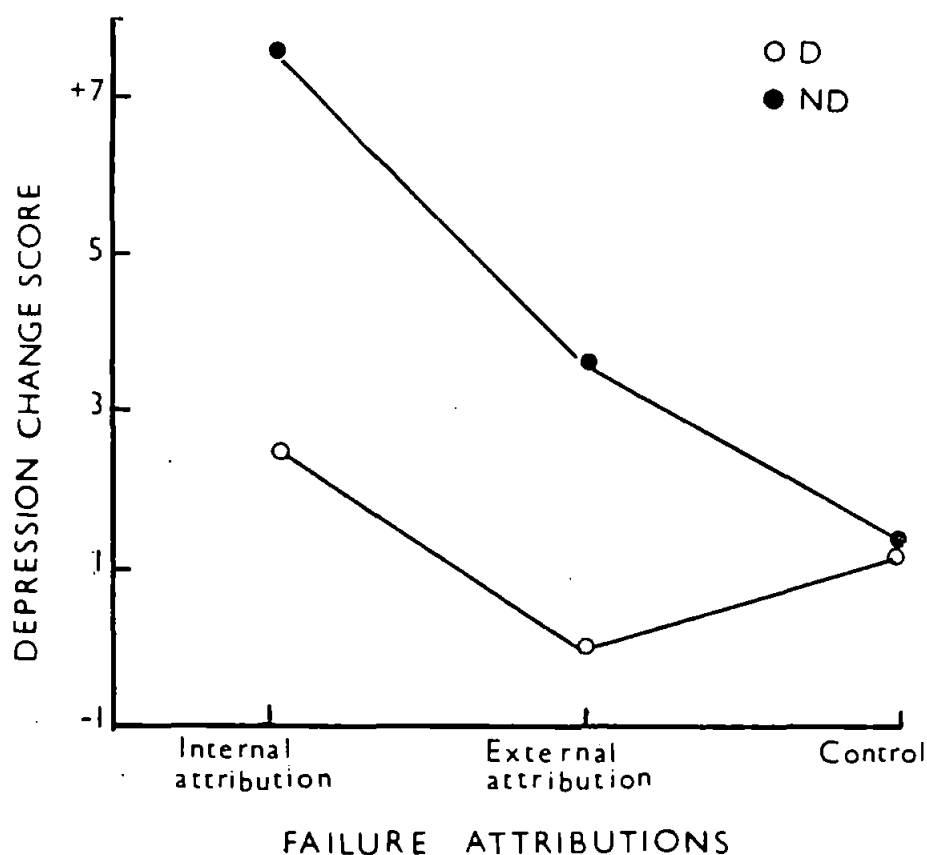


Fig. 3.1. Depression change score for depressed(D) and non-depressed(ND) subjects as a function of type of attribution.

7.45,  $p < 0.005$ . A test on the simple effects of attribution revealed that non-depressed subjects reported less expectations of success following IAF than following EAF or FO treatment,  $F(2,42) = 5.74$ ,  $p < 0.01$ . Figure 2 displays this effect. Neither the other main effect nor the interaction were significant.

#### PSYCHOMOTOR SPEED DATA

A  $2 \times 3$  analysis of variance of psychomotor speed data (number-writing speed test) showed that neither the effect for mood nor



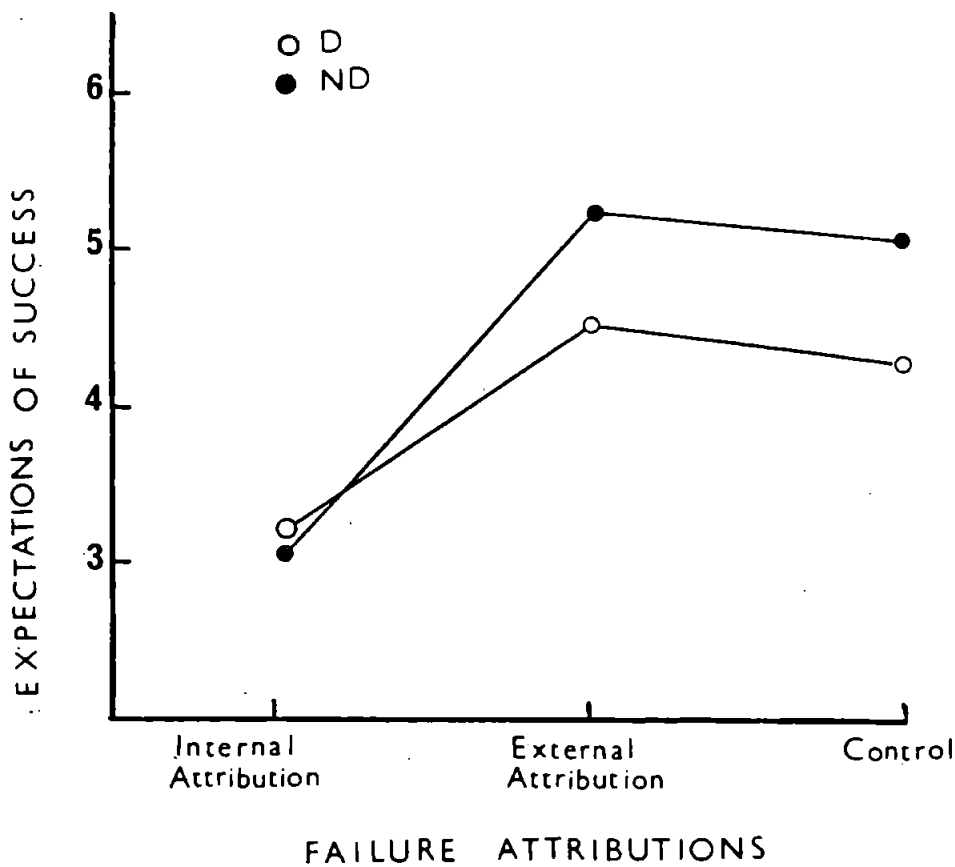


Fig. 3.2. Expectations of success for depressed(D) and non-depressed(ND) subjects as a function of type of attribution.

the interaction were significant, ( $F < 1.0$  and  $F = 2.30$  respectively). However, the effect for attribution was significant,  $F(2,42) = 3.27$ ,  $p < 0.05$ . As predicted, non-depressed subjects induced to adopt the depressive attributional style for failure (IAF) wrote fewer numbers (were slower) than those in EAF or FO condition ( $p < 0.01$ ). Table 3.5 shows all the means and standard deviations of psychomotor speed scores for all conditions.

TABLE 3.5 - MEANS OF MAACL DEPRESSION CHANGE SCORES, PSYCHOMOTOR  
SPEED SCORES, AND SELF-EXPECTANCY SCORES BY MOOD  
AND ATTRIBUTION

MOOD	IAF	EAF	FO
<u>DEPRESSED</u>			
MAACL DEPRESSION	2.63	0	1.25
SELF-EXPECTANCY	3.12	4.50	4.25
PSYCHOMOTOR SPEED	50.75	50.62	48.62
<u>NON-DEPRESSED</u>			
MAACL DEPRESSION	7.63	3.63	1.37
SELF-EXPECTANCY	3.00	5.12	5.00
PSYCHOMOTOR SPEED	48.12	59.87	46.75

NOTE: IAF = Internal Attribution for failure;

EAF = External Attribution for failure;

FO = Failure only;

MAACL = Multiple Affect Adjective Check List.

### 6.2.3 DISCUSSION

The present experiment was designed to examine further the relationship between attributions and depression. Specifically, it was an attempt to investigate whether depression (as measured by the BDI and MAACL) was causally related to certain types of attributions. Based on the results of previous experiments and on the Abramson et al.'s attribu-

tional account of learned helplessness and depression (1978) and its extension by Seligman et al. (1979), it was predicted that depression or its correlates will be manifested following internal attributions for failure (IAF), but not following external attributions for the same outcome (EAF). The data of the present experiment tend to support this hypothesis. The results of the present experiment revealed, as predicted, that inducing non-depressed subjects to make internal<sup>2</sup> attributions for failure on a problem solving task made them dysphoric. This finding that the occurrence of depression (as reflected on the MAACL) is causally related to the depressive attributional style (IAF) is consistent with the learned helplessness viewpoint that personal helplessness and depression are precipitated by a combination of stressful life events or failure and internal attributions. This finding is also consistent with the results reported by several recent studies that depression is closely associated with internal attributions of failure but not with external attributions of failure (e.g., Kuiper, 1978; Rizley, 1978; Seligman et al., 1979).

Although the results of the present study do not allow any speculation as to why internal attributions for bad outcomes have depressogenic effects, there is a possibility, nonetheless, that these kinds of attributions (negative self-attributions) induce the very negative or depressing cognitions i.e., self-blame, self-criticism, pessimism (Beck, 1976) which facilitate the manifestation of the depressive syndrome. Beck appears to

have reached the same conclusion when he stated that the depressed person:

"regards himself as deficient, inadequate, unworthy, and is prone to attribute unpleasant occurrences to a deficiency in himself. Since he attributes his difficulties to his own defects, he blames himself and becomes increasingly self-critical" (p. 129, 1976 - emphasis added).

The finding, in this study, that the tendency to attribute failure to external causes (EAF) is inconsistent with depression, is in line with the prediction that the adoption of the non-depressive attributional style for failure will prevent the dysphoric reactions that depressives usually exhibit following failure experiences. This finding is also consistent with numerous studies (e.g., Kuiper, 1978; Rizley, 1978; Seligman et al., 1979) suggesting that the tendency to externalize failure is not depressing in its effects. Further, this second finding lends some support to Abramson et al.'s view (1978) that the ascription of bad outcomes to external difficulties or causes is incongruent with depressive affect and personal helplessness.

In sum, the results of the present experiment show that depression or at least depressive affect tend to be exhibited following the adoption of the depressive attributional style but not following the adoption of the attributional style typical of non-depressives. So the present results suggest, in line with the studies cited above, that depressive affect is causally related to internal attributions for failure but not to external attributions for failure.

Although there is ample evidence, in this study, that depression in college populations is causally related to certain attributions, there is no evidence that such attributions are also involved in the aetiology of clinical depression. In fact, the extent to which clinical depression is related to these attributions can only be determined by further research involving clinical populations. Additional research, involving both clinically depressed and non-depressed patients, is necessary to determine whether these attributions are specific to depression or whether they are a common feature of all psychopathologies. It is this issue that the next study has attempted to resolve.

### 6.3 EXPERIMENT 4

The present study of the relationship between causal attribution and depression was designed to determine further whether the kind of attributions, thought to be depressogenic (e.g., Barthé and Hammen, 1981; Gong-Guy and Hammen, 1980; Rizley, 1978; Seligman et al., 1979), are involved in the aetiology of the depressive syndrome. One way of assessing the aetiological significance of these attributions is to determine whether they are specific to clinical depression or whether they are a common feature of general psychopathology. It should be pointed out that the demonstration of a specific relationship between these two variables is of great importance, since it will provide additional but stronger evidence that attributions and depression are causally associated. While the presence of a specificity in their relationship may add

support to the hypothesised causal connection, the absence of a specificity in this relationship, however, will cast serious doubt on the aetiological status of attributions in depression.

Although there is evidence to suggest that certain attributions are closely associated with depression, such evidence should not be viewed as a sufficient proof of their aetiological relevance. Because, as already pointed out, a specific relationship between attributions and clinical depression had not been established. Studies that linked attributions and non-clinical depression (as measured by different depression inventories) (e.g., Barthé and Hammen, 1981; Kuiper, 1978; Rizley, 1978; Seligman et al. 1979) failed to consider the alternative hypothesis that these attributions may also be associated with other psychopathologies. The present study was an attempt to bridge this gap. Particularly, this study addressed the question of specificity by assessing clinically depressed and non-depressed patients' attributions of negative as well as positive outcomes. It was hypothesised that depressed and non-depressed patients' attributions for negative and positive outcomes would be divergent; on the basis of the findings reported earlier and in accordance with the Abramson et al.'s attributional account of helplessness and depression (1978), that depressed compared to non-depressed patients should display internal attributions for negative outcomes and external attributions for positive outcomes.

To summarize, this study examined the question of specificity, in the relationship between attributions and depression, by comparing clinically depressed and non-depressed patients'

attributions of positive and negative outcomes. An interaction between depression (depressed/non-depressed) and outcome (positive/negative) was predicted.

#### 6.3.1 METHOD

Description of the sample. The sample consisted of 19 patients, 11 depressed and 8 non-depressed, hospitalized either because of a major depressive disorder or because of an important personality disorder. The patients included in this study had all been given a diagnosis. Those assigned to the depressed group fulfilled the research diagnostic criteria for a major depressive disorder (Feighner et al., 1972). Other criteria for inclusion in the depressed group included (a) score on the Beck Depression Inventory (BDI) (Beck et al., 1961) greater than 15; (b) no signs of organic brain damage; (c) no evidence for a history of mania. The patients assigned to the non-depressed group were selected according to the following criteria: (a) no signs of organic brain damage; (b) score on the BDI lower than 10; (c) diagnosis other than depression. As can be seen in Table 4.1, the final sample consisted of 11 depressed patients, who met the research diagnostic criteria for a primary depression, and 8 non-depressed<sup>3</sup> patients, who fulfilled the criteria for inclusion in the non-depressed group.

Assessment measures. After the initial interview, all patients included in this study completed the Beck Depression Inventory (BDI) and a short attribution questionnaire adapted from Seligman

TABLE 4.1 - CHARACTERISTICS OF THE SAMPLE

CHARACTERISTIC	DEPRESSED	NON-DEPRESSED
MALE	2	6
FEMALE	9	2
AGE (years)		
M	40.63	26.25
SD	13.30	8.12
BDI		
M	24.00	7.75
SD	7.56	3.63

NOTE: BDI = Beck Depression Inventory.

et al. (1979). The attribution questionnaire consisted of 4 hypothetical events (2 positive and 2 negative) and questions enquiring about the occurrence and the perceived causes of these events. The two positive events involved a professional achievement event: 'Imagine that you have just been promoted in your job', and a positive interpersonal sequence. The two negative events involved: 'a failure to obtain a steady employment', and a serious interpersonal difficulty. Following a detailed description of each event, subjects were asked to write, on the space provided, the major cause of the event, and then they were asked to indicate, on a 9-point scale, the extent to which the event is due to their personal qualities (internality dimension of attribution), the extent to which the event is due to other people or circumstances (the externality



dimension of attribution). Because of the difficulties, evidenced by many patients, in understanding both the stability and globality dimensions of attribution, it was decided to drop the questions related to these two attributional dimensions.

Procedure. The study took place in the psychology department at St. Lawrence's Hospital. All patients were seen individually. A semi-structured interview, that lasted approximately 25 minutes, was administered to obtain anamnestic relevant information. In addition to supplying the clinical data, the interview served as a basis for deciding on the patient's suitability to take part in the study. Following this initial interview, the patient was asked to complete the BDI. Following the administration of the BDI, the patient, if selected, was given the attribution questionnaire. All patients received the following instructions before the administration of the attribution questionnaire:

The present study is concerned with the way people explain or interpret things that happen to them in every day life. The questionnaire that you will be given was designed to examine, although only partly, this issue. The questionnaire itself consists of 4 hypothetical situations, followed by some questions about the perceived cause of each situation. Your task will be first to try to imagine yourself, as vividly as you can, in each situation, and then try to answer some questions about the cause of the situation. Please remember that we want to know your own belief about the occurrence of each event.

Following the administration of both the Beck Depression Inventory (BDI) and the attribution questionnaire, all patients were debriefed and thanked for their co-operation.

TABLE 4.2 - MEANS AND STANDARD DEVIATIONS OF ATTRIBUTION  
SCORES AS A FUNCTION OF MOOD AND NATURE OF  
EVENT

MOOD	POSITIVE EVENTS		NEGATIVE EVENTS	
	M	SD	M	SD
<u>DEPRESSED</u>				
INTERNALITY	4.95	2.33	6.13	3.36
EXTERNALITY	4.59	0.85	3.31	1.99
<u>NON-DEPRESSED</u>				
INTERNALITY	6.25	1.39	4.00	0.93
EXTERNALITY	4.06	1.34	5.50	1.50

NOTE: Ratings are on 9-point scale; higher scores on the internality scale indicate that the attribution is more internal; higher scores on the externality scale indicate that the attribution is more external.

### 6.3.2 RESULTS AND DISCUSSION

The purpose of this study was to determine whether, as suggested by several investigations (e.g. Barthé and Hammen, 1981; Kuiper, 1978; Rizley, 1978; Seligman et al., 1979), certain types of attributions are specific to depression. It was argued

that one way of testing this specificity hypothesis will be to establish whether, as predicted by Abramson et al.'s attributional formulation of learned helplessness and depression (1978), clinically depressed and non-depressed patients differ systematically in their attributions about the causes of negative as well as positive events. The results of this study are, as can be seen in Table 4.2, in line with the main prediction.

TABLE 4.3 - RESULTS OF 2 x 2 ANALYSIS OF VARIANCE OF  
INTERNALITY SCORES

SOURCE OF VARIATION	SS	DF	MS	F	P
<u>BETWEEN SUBJECTS</u>					
MOOD (A)	1.58	1	1.58	<1.0	
SUB. WITHIN GROUPS	95.71	17	5.63		
<u>WITHIN SUBJECTS</u>					
EVENT (B)	2.70	1	2.70	1.17	
A X B	19.90	1	19.90	8.65	<0.01
B X SUB. WITHIN GROUPS	39.07	17	2.30		

The internality scores were first averaged over the two negative and two positive events, and then were subjected to an analysis of variance (see Table 4.3) with depressed/non-depressed as the between subjects factor and negative/positive event as a repeated measure. A 2 x 2 analysis of variance of internality scores revealed, as expected, that the interaction between level

of depression and the nature of event (negative/positive) was significant,  $F(1,17) = 8.65, p < 0.01$ . Figure 4.1 displays this interaction. Tests on the simple main effects were conducted to interpret this interaction. A test on the simple main effect of level of depression (depressed/non-depressed) within the negative events showed, as predicted, that depressed patients made more internal attributions for negative events than non-depressed patients,  $F(1,17) = 19.45, P < 0.001$ . This finding that clinically depressed patients have an exaggerated tendency to display negative self-attributions is consistent not only with the attributional account of depression (Abramson et al., 1978; Seligman et al., 1979), but also with Beck's observation that these depressogenic attributions dominate the clinical picture of depression:

"The depressed patients, in common with other people, attempts to determine the cause of his problems - In his notion of causality, the depressed patient is prone to regard himself as the cause of his difficulties - He may carry this notion of self causality to absurd extremes. When it is pointed out that self-blame is maladaptive, he then blames himself for blaming himself" (pp. 292-293, 1976).

This finding regarding the relatively high degree of specificity in the relationship between negative self-attributions and depression was further corroborated by the clinical material collected both before and after the administration of the questionnaires. The use of the retrospective anamnesis method during the clinical interview offered a possibility to examine patients' attributions as they naturally occurred.

Specifically, a retrospective elicitation of (patients')

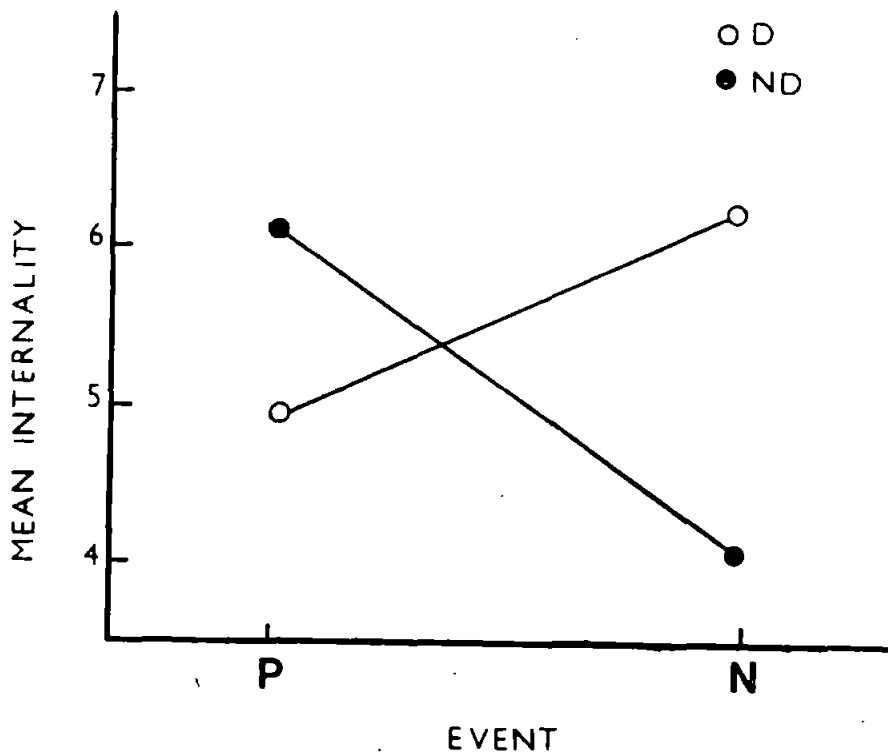


Fig. 4.1. Mean internality scores of depressed(D) and non-depressed(ND) for positive(P) and negative(N) events.

personal negative events provided an opportunity to examine the kind of attributions they make to account for their unpleasant experiences. As expected, most depressed patients were inclined to relate personal negative events to perceived personal defects. A typical depressogenic attribution, entertained by a depressed patient, was readily made by a female patient whose son had apparently been convicted for a minor offence. The patient showed an exaggerated sense of responsibility for the incident. A similar depressogenic account was provided by a patient whose wife was suffering from a chronic medical condition. Another patient, whose boyfriend had discontinued their relationship, concluded that she was

to blame for her personal disappointment: 'I am not a nice person to live with...'. Clearly, depressed patients' accounts of their misfortunes appeared to be different both in content and in form from those of non-depressed patients.

Taken together, the data from the questionnaire and the clinical observations confirm that negative self-attributions are, as hypothesised by Abramson et al. (1978), specific to the state of depression.

The second prediction that a high degree of specificity exists in the relationship between external attributions for positive events and depression, was not strongly supported by the data from the attribution questionnaire. The predicted interaction between the level of depression (depressed/non-depressed) and the nature of event (positive/negative) achieved only the conventional statistical significance,  $F(1,17) = 6.47, p < 0.05$ . A test on the simple main effects of level of depression within positive events showed that depressed patients displayed more external attributions for positive events than non-depressed patients,  $F(1,17) = 7.10, p < 0.05$ . Contrary to the prediction, this result indicates that there is only a limited specificity in the relationship between external attributions for positive outcome and depression.

This finding that external attributions for good outcomes and depression may have a limited degree of specificity in their relationship was confirmed by the observations made during the clinical interviews. Specifically, it was found that only

severely depressed patients (a score of 35 or more on the BDI) showed a tendency to account for positive personal events in terms of external causes. For instance, a severely depressed young woman who had apparently been promoted just before her hospitalization accounted for the event (promotion) more in terms of external circumstances: 'I was promoted... because there was a vacancy in the office'. An interesting possibility is that external attributions for positive outcomes may be more symptomatic of depression (i.e., a consequence of being depressed) than a part of its aetiology (i.e., an antecedent of depression).

To summarise, the present study tested the specificity hypothesis - that is, the possibility that certain attributions are specific to depression - by comparing clinically depressed and non-depressed patients' attributions for both negative and positive events. The results obtained provided only a partial support for this hypothesis. Specifically, the data from the internality question revealed, as proposed by Abramson et al. (1978) and Seligman et al. (1979), that there may be a high degree of specificity in the relationship between negative self-attributions and the state of depression. The data from the externality question, however, indicated that there is only a limited degree of specificity in the relationship between external attributions for positive outcomes and depression.

Although, as suggested by the data of the present study, negative self-attributions and to some extent external attribu-

tions of good outcomes, are specific to depression, a conclusion with regard to their aetiological significance cannot easily be drawn without further work. Causal attribution is only one among other factors that are believed to play a role in the aetiology of depression (cf. Akiskal & McKinney, 1975).

#### 6.4 GENERAL DISCUSSION

The two studies just reviewed were concerned with the issue of whether attributions play an aetiological role in depression. Experiment 3 was partially designed to clarify this issue. It was reasoned, on the basis of both the results of Experiments 1 and 2 and the attributional formulation of learned helplessness and depression (Abramson et al., 1978), that one way of determining whether attributions play a causal role in depression would be to reverse non-depressives and depressives' attributional style for failure (cf. Seligman et al., 1979) and assess the effects on mood, psychomotor speed, and expectations. The results obtained revealed, as expected, that inducing non-depressives to adopt a depressive attributional style for failure (i.e., internal attributions for failure) resulted in depressed mood, and to some extent in reduced expectations of success, and low psychomotor performance. In contrast, the adoption by depressives of a non-depressive attributional style for failure (i.e., external attributions for failure) resulted in no such effects. These results were interpreted as consistent with Abramson et al. (1978) and Seligman et al. (1979) contention that internal attributions for negative outcomes have an aetiological status in depression.



While there is ample evidence, from both the present work and that of Golin et al. (1981), that depression in its mild form (non-clinical) is causally related to negative self-attributions (i.e., the tendency to make internal attributions for negative outcomes), it is not clear whether such attributions are also important in the aetiology of clinical depression. It was argued that one way of assessing their aetiological importance or relevance would be to determine whether they are specific to depression or whether they are a general characteristic of psychopathology. The results from this clinical study indicated, as anticipated, that there is a high degree of specificity in the relationship between negative self-attributions and depression. These results add substance to the claims, by learned helplessness and attribution theorists (e.g., Miller & Norman, 1979; Seligman et al., 1979), that these type of attributions (i.e., negative self-attributions) play an aetiological role in depression.

In conclusion, there is both experimental and clinical evidence that attributions are involved in the aetiology and/or development of depressive symptoms and disorders. In particular, there is an indication from the present work that the depressives' tendency to make negative self-attributions may be at the basis of their typical symptomatology and complaints. Whether their 'depressogenic' tendency to account for personal negative events reflects the influence of their mood (i.e., depressed mood) is a question that cannot be answered at this point. Although there is ample evidence, from both the

present investigation and others (e.g., Golin et al., 1981), that attributions induce depressive symptoms, the possibility that the depressive condition may also lead people to display these depressogenic attributions cannot be ruled out without further work. In particular, additional research is needed to examine more closely the relationship between mood states (e.g., depressed mood) and causal attributions. Only by examining every aspect of this relationship (between causal attribution and depression) that its true nature will be fully known.

## FOOTNOTES

1. The validity and reliability of the Beck Depression Inventory (BDI) as a measure of depression in both clinical and non-clinical populations are well documented and established (see chapter two).
2. Although in this experiment only the internality dimension (internal-external attribution) of attribution was manipulated, there is a reason to believe that subjects assigned to IAF condition displayed the depressive attributional style described by Abramson et al (1978) and Seligman et al (1979) - that is, they made internal, stable, and global attributions for failure (see Table 3.3).
3. The non-depressed group consisted of 3 first-admission schizophrenics, 3 alcoholics, and 2 patients with hysterical features.

## CHAPTER SEVEN

### INDUCED MOOD STATES AND CAUSAL ATTRIBUTIONS

- 7.1 INTRODUCTION
- 7.2 EXPERIMENT 5
- 7.3 EXPERIMENT 6
- 7.4 SUMMARY AND CONCLUSIONS

## 7.1 INTRODUCTION

An issue not resolved by the previous studies is whether the depressives' tendency to display internal attributions for bad outcomes and external attributions for good outcome is influenced or perhaps promoted by their characteristic mood state (i.e., depressed mood). Although there was no indication, from previous studies or any other study reviewed so far, that these depressogenic attributions are induced by depressed mood, there is a possibility, however, that they may be reinforced or strengthened by such a mood state or condition. That is, there is a possibility that a reciprocal relationship may exist between depressogenic attributions and depressive condition. The present study attempted to examine this proposition by assessing the effects of induced mood states on causal attributions for positive and negative outcomes.

While learned helplessness and attribution theorists (e.g., Abramson, Seligman, and Teasdale, 1978; Miller and Norman, 1979; Seligman et al., 1979) may have emphasised the primacy of attributions in the depressive experience, they did not exclude the possibility that these attributions may be reinforced or even shaped by the depressive condition. In fact, the possibility that mood states may have a substantial

impact on cognitions has been thoroughly considered by Teasdale and Fogarty (1979) and Teasdale, Taylor, and Fogarty (1980). They reported evidence consistent with the reciprocity view of the relationship between negative cognitions and pathological mood states. This is how Teasdale and Fogarty (1979) concluded their report:

"Cognitive models of depression... need to be extended to include a reciprocal relationship between cognitions and the state of depression" (p.256).

From the clinical point of view, Beck, a leading authority in this field of research, also appeared to favour the reciprocity view of the aetiology and development of the depressive syndrome:

"Essentially, we believe that depressive illness involves a vicious cycle in which cognitive distortions, negative affective experience, and maladaptive behaviour become mutually reinforcing, resulting in self-perpetuating closed system" (Beck and Burns, 1978, p. 203).

Accordingly, the following two experiments were designed to determine whether attributions for positive and negative outcomes vary as a function of mood states.

## 7.2 EXPERIMENT 5

The present investigation of the relationship between mood states and causal attributions was an attempt to determine whether there is a biasing effect of mood on attribution.

While no attempts have been made to assess the effects of differential mood states on attributions, evidence regarding the effects of different moods on other types of cognitions

(e.g., retrieval processes) is available (e.g., Lloyd, and Lishman, 1975; Teasdale, and Fogarty, 1979; Teasdale, Taylor, and Fogarty, 1980). In an important study, Teasdale and his co-workers (1980) reported results which clearly showed that even higher cognitive processes such as memory are affected by mood states; they found that memory for happy and unhappy experiences is facilitated by mood. Using the Velten mood induction procedure (Velten, 1968), Teasdale and his colleagues found, that following the experimental induction of depressed mood, subjects were more likely to retrieve unhappy memories than happy ones. By contrast, elated subjects retrieved significantly more happy memories than unhappy memories. The Teasdale et al.'s finding that mood states facilitate the accessibility of certain cognitions, points to the possibility that attributions about the causes of different outcomes may also be promoted or influenced by mood states. The present experiment tested this proposition by inducing non-depressed college students into either depressed or elated mood, and then assessing their attributions for positive and negative events. It was anticipated that elated subjects (elation condition) would make more internal, stable, and global attributions for positive events than depressed subjects (depression condition). As a test of the reciprocity hypothesis, it was predicted that depressed subjects (depression induction) would display more internal, stable, and global attributions for negative events than elated subjects (elation induction).

To summarise, the present experiment was carried out to assess the effects of induced mood states on attributions for positive and negative events.

#### 7.2.1 METHOD

Overview. 20 subjects selected on the basis of their Beck Depression Inventory (BDI) (Beck et al., 1961) scores, received depressed mood induction or elated mood induction (Velten, 1968), and their attributions about positive and negative events were then assessed.

Subjects and Design. 20 undergraduates scoring less than 9 on the BDI were selected and used as subjects in this experiment. The mean BDI was 5.60 and age 23.90 yr. The 20 subjects (14 females and 6 males) were drawn from a sample of 36 students and were randomly assigned to one of the 2 conditions of the experiment.

A 2(elated-depressed) x 2(positive-negative event), with repeated measures on the second factor, design was used.

#### Materials

Mood Induction. The mood induction technique used in the present experiment was the one devised and developed by Velten (1968). Briefly, this verbal mood induction procedure consists of positive or elating statements (e.g., 'I have a sense of power and vigor') or depressing self-referent statements like 'I am discouraged and unhappy about myself'. In



each condition, subjects read, first silently and then aloud, 40 cards containing either positive (elation condition) or negative (depression condition) self-referent statements. Subjects were instructed to read each card first to themselves and then out loud. The cards were presented in the standard sequence, i.e., progressing from neutral statements like 'Today is neither better nor worse than any other day' to depressing statements like 'Everything seems utterly futile and empty' or elating statements such as 'I'm really feeling sharp now' or 'I'm full of energy'. Prior to commencing the mood task, all subjects received 7 cards containing the instructions. Briefly, the instructions reminded the subject that he/she should try to feel the mood suggested by each of 40 mood statements. The instructions emphasised that this could be done either by repeating the statements over and over, imagining a situation dominated by such mood, or by a combination of both techniques.

Mood Checks. The Multiple Affect Adjective Check List (MAACL) (Zuckerman and Lubin, 1965) was used to assess the effectiveness of this verbal mood induction procedure. Briefly, the MAACL provides two measures, one of depression and the other of anxiety. To ensure a more objective assessment of mood states, a measure of psychomotor speed was also obtained, this number-writing speed measure, also used by Velten in his original study (1968), was obtained by instructing subjects to write numbers from 100 backwards for 1 min period.

Dependent Measures. An attribution style questionnaire (see Appendix A4) was used, to assess subjects' attributions about 3 positive events (e.g., 'You have been promoted in your job') and 3 negative life events (e.g., 'You go out on a date, and it goes badly'), following the mood induction. This attributional style questionnaire, adapted from Seligman et al. (1979), provides measures about the internality, stability, and globality dimensions of attributions (see chapter 4 for a detailed discussion about these attributional dimensions). All measures of these three attributional dimensions were on a 9-point scale. (High scores on these scales indicate that the causal attribution is internal, stable, and global).

Procedure. A flow chart of the experimental procedure can be seen in Table 5.1. As can be seen in this table, only subjects scoring less than 9 on the BDI were used in this experiment. Following the completion of the BDI, all subjects were given the 7 cards containing the standard instructions of the Velten verbal mood induction task. Subjects were instructed to read each mood card (first to themselves and then aloud) and to try to imagine themselves in the mood state suggested by the statement typed on the card. They were told that an electronic device will signal them when to start reading each card. The auditory signals were spaced 18 sec. apart. Subjects were instructed to pick up a new card whenever they hear the tone signal. Following a brief

TABLE 5.1 - EXPERIMENTAL PROCEDURE

SELECTION	MOOD INDUCTION	MANIPULATION CHECKS	ATTRIBUTION MEASURES
BDI <9 was the main selection requirement. All Ss scored less than 9 on this depression scale.	10 Ss received depression induction 10 Ss received elation induction.	All Ss completed the MAACL (depression and anxiety scale). A psychomotor measure was also obtained.	Subjects' attributions (including the internality, stability, and globality dimensions) about pleasant and unpleasant life events were recorded.

demonstration of the procedure, a deck of cards was placed in front of the subject, and a signal was then given to him/her to start reading the first card.

After the mood induction task, subjects were asked to complete the MAACL and to write numbers, on a sheet of paper, from 100 backwards for 60 sec. period. Following this psychomotor exercise subjects were asked to complete the attribution style questionnaire. This questionnaire consisted of 3 positive and 3 negative situations. Subjects were first instructed

to imagine themselves, as vividly as they can, in each situation and then write down the major cause of each situation or event in the blank provided. Further questions inquiring about the internality, the stability, and the globality of the cause were also included in the questionnaire. Upon completion of the attribution style questionnaire, all subjects were debriefed, paid, and thanked for their co-operation.

TABLE 5.2 - MANIPULATION CHECKS

MEASURE	DEPRESSION M	ELATION M	STATISTICAL t	ANALYSIS P
DEPRESSION	15.70	7.20	3.54	<.005
ANXIETY	8.00	5.00	2.71	<.01
PSYCHOMOTOR SPEED	49.30	53.80	2.04	ns.

Manipulation Checks. As can be seen in Table 5.2, the mood task was relatively effective in inducing the mood states of depression and elation. As expected, depressed subjects reported to have been feeling more depressed than elated subjects,  $M = 15.70$  and  $7.20$  ( $p < .005$ ) respectively. Subjects in the depression condition reported also more anxiety than their counterparts in the elation condition,  $M = 8.00$  and  $5.00$  ( $p < .01$ ) respectively. However, the two groups did not differ very significantly in their psychomotor speed performance, for depressed  $M = 49.30$  and elated  $M = 53.80$  ( $p < .05$ ).

Attribution Measures. The attribution ratings were first averaged over the three positive and three negative events and then were subjected to analyses of variance, with depressed/elated as the between-subjects factor and positive/negative event as a repeated measure.

Internality Dimension. In order to determine whether attributions for positive and negative events vary as a function of induced mood states (depression/elation) the internality ratings (the extent to which the event is due to personal factors) were subjected to an analysis of variance.

A 2(depression/elation) x 2(positive/negative) analysis of variance, with repeated measures on the second factor, of internality ratings revealed that neither the effect for mood nor the interaction were significant ( $F_s < 1.0$ ).

However, the effect for event was significant; such that subjects regardless of the mood induction they received (elation or depression) made more internal attributions for positive than negative events,  $F(1,18) = 11.18$ ,  $p < .005$ . This pattern of attributions is similar to the one non-depressives display to account for success and failure in laboratory situations (cf. Rizley, 1978).

Stability Dimension. A 2 x 2 analysis of variance of stability ratings also showed that the effect for event (positive/negative) was very significant,  $F(1,18) = 17.45$ ,  $p < .001$ . Regardless of their mood, subjects made more stable

attributions for positive than negative events, overall mean for positive events was 7.03 and negative events 5.36. Neither the other main effect (mood) nor the mood X event interaction were significant ( $F_s < 1.0$ ).

Globality Dimension. As can be seen in Table 5.3, the globality ratings were also inconsistent with the main prediction of this study. A 2 x 2 analysis of variance of globality ratings showed that, contrary to the prediction, neither the effect of mood nor the interaction were significant. The other main effect, however, was significant,  $F(1,18) = 11.43, p < .005$ . Subjects made more global attributions for positive than negative events regardless of their mood state, overall mean for positive events 6.17 and negative events 4.27.

TABLE 5.3 - MEANS OF INTERNALITY, STABILITY, AND GLOBALITY RATINGS

MOOD	POSITIVE EVENTS	NEGATIVE EVENTS
<u>DEPRESSION</u>		
INTERNALITY	6.56	5.10
STABILITY	7.09	5.67
GLOBALITY	5.98	4.30
<u>ELATION</u>		
INTERNALITY	6.50	4.85
STABILITY	6.97	5.05
GLOBALITY	6.37	4.25

NOTE: Ratings are on 9-point scale; Higher ratings indicate that attributions are more internal, stable, and global.

### 7.2.3 DISCUSSION

The purpose of this study was to determine whether there is a reciprocal relationship between depressogenic attributions and the state of depression. It was argued that one way of clarifying this issue would be to examine the relationship between mood states and attributions for positive and negative events. The Velten mood induction technique provided the opportunity to study the relationship between these two variables. It was predicted, on the basis of Teasdale et al.'s results (1980) and in accordance with the reciprocity view of the aetiology of depression, that attributions for positive and negative events would vary as a function of mood states. Specifically, it was expected, that elated subjects will make more internal, stable, and global attributions for positive events than depressed subjects. For negative events, however, it was predicted that depressed subjects will make more internal, stable, and global attributions than their counterparts in the elation condition. The results of the present experiment failed to support these predictions.

The failure of this study to provide evidence for the biasing effects of mood states on attributions may be due to one of the following reasons:

- (a) It is possible that the verbal mood induction procedure used in the present experiment failed to produce significant mood changes. Indeed, the pattern of attributions displayed by the subjects, in the present

experiment, was typical of non-depressives. That is, subjects made more internal, stable, and global attributions for positive than negative events. It was as if they had not experienced any mood change. Although the manipulation checks argue for the effectiveness of the mood manipulations, one should not underestimate the power of demand characteristics (Orne, 1962) in creating such impression.

- (b) Another possibility is that attributions are more fundamental than mood states and therefore cannot easily be influenced or modified by such a transient mood as elation or depression. However, this possibility appears without basis when considered in the light of recent experimental findings. Indeed, as already pointed out, several studies found evidence that even higher cognitive processes, such as memory, are subjected to the influence of mood states (e.g., Teasdale, Taylor, and Fogarty, 1980).
- (c) Another but more plausible explanation for the failure of the results to show any effect for mood may lie in the instrument used to assess the dependent variable (attribution). Although described as an adequate measure of attributions (see Seligman et al., 1979), the attribution scale used in the present study may have lacked the necessary sensitivity to register the various degrees of internality, stability, or globality of the cause. In fact, a recent study (Blaney, Behar, and Head, 1980) reported results which suggested that



the Seligman et al.'s attributional style scale (1979) may have more serious drawbacks than just lack of sensitivity.

To summarise, the present experiment was carried out in an attempt to determine whether attributions for positive and negative outcomes vary as a function of mood states. The results obtained failed to provide any evidence for the biasing effects of mood states on attributions. The failure of the results to show an effect for mood was attributed to some defects in the instrument employed, in this study, to assess the main dependent variable (attribution). This methodological consideration prompted another experiment in which the relationship between mood states and causal attributions was further examined.

### 7.3 EXPERIMENT 6

The present experiment was designed to examine further the relationship between mood and attribution - it was carried out in an attempt to investigate whether mood states have biasing effects on attributions of causality. It was reasoned earlier that if mood has any influence at all on attribution then the induction of different mood states should produce corresponding differences on attributions. The previous experiment tested this specific proposition and found no evidence to support it. This failure to show any effect for mood was attributed to the inadequacy of the scale used to assess the dependent variable (i.e., attribution). The

present experiment attempted to remedy to this by using the attribution scale commonly used in the studies linking attributions to depression (e.g., Kuiper, 1978; Rizley, 1978).

In addition to using a different attribution scale, the following changes were introduced: (a) Subjects' attributions were not about hypothetical events but about success or failure on a problem solving task; (b) the attribution questionnaire used in the present experiment did not ask about attributional dimensions (e.g., internality, stability, globality) but about causal determinants of success or failure; (c) the design of this experiment was a 2(elation/depression) x 2(success/failure) with no repeated measures.

The purpose of this experiment was to examine the effects of induced elation and depression on causal attributions for success and failure on a problem solving task.

### 7.3.1 METHOD

Overview. 32 subjects selected on the basis of their Beck Depression Inventory (BDI) (Beck et al., 1961) scores, received elation induction or depression induction following performance on a problem solving task, and their attributions about success and failure were then assessed.

Subjects and Design. 32 undergraduates scoring less than 9 on the BDI were selected and used as subjects in the present experiment. The mean BDI was 3.62 and age 20.52 yr. The

subjects were randomly assigned to one of the following four conditions of the experiment:

1. Elation success (ES).
2. Elation failure (EF).
3. Depression success (DS).
4. Depression failure (DF).

A 2(elation/depression) x 2(success/failure) factorial design was used, as before.

### Materials

Mood Induction. The mood induction procedure was the same as that used and described in the previous experiment.

Briefly, subjects were instructed to read 40 cards containing mood statements. The subjects assigned to the depression condition read depressing statement, and those assigned to the elation condition read elating or euphoric statements. As in the previous experiment, the subjects were encouraged to feel the mood suggested by each of the 40 cards making up the verbal mood induction task.

Mood Measures. As in the previous experiment, the Multiple Affect Adjective Check List (MAACL) (Zuckerman & Lubin, 1965) was used to check on the effectiveness of the mood induction procedures or manipulations. In addition to measuring the subjects' level of anxiety and depression, a measure of their psychomotor speed (number-writing speed) was also obtained.

Attribution Measures. As pointed out in the introduction, subjects' attributions about their success or failure on the

problem solving task (see below for the description of the task) were assessed with the attribution questionnaire described and used in Experiments 1 and 2 (see Appendix A2). Unlike the scale used in the previous experiment, the present questionnaire lists the causal determinants of success or failure, i.e., ability, effort, task, and luck, and asks the subjects to indicate (on a 9-point scale) the extent to which each of these factors contributed to his/her performance.

Treatment Task. The task used to manipulate subjects' performance was the same as that used in Experiment 2. This task was described as a pattern completion test. It consisted of 20 matrices (or problems) obtained from Raven's Advanced Progressive Matrices set II (1962). Subjects were given 20 sec. for each problem. After each problem, subjects were told either they were right (success condition) or wrong (failure condition). Following performance on this problem solving task, subjects were told either they had succeeded or failed.

Procedure. Prior to performing on the problem solving task, all subjects were asked to complete the BDI. Upon completion of the BDI, subjects were given the first problem from the problem solving task. Following performance on the pattern completion task, subjects were led to another table on which a deck of cards was displayed. The procedure used to induce mood states was the same as that used in the previous experiment. Following the mood induction task, subjects' mood (anxiety, depression), number-writing speed, and attributions about their

success or failure on the pattern completion task were assessed. Following the administration of the attribution questionnaire, all subjects were debriefed, paid, and thanked for their co-operation.

### 7.3.2 RESULTS

Table 6.1 shows the effects of induced elation and depression on mood (as measured by the MAACL) and on number-writing speed. As can be seen in this table, the mood manipulations had been effective. As expected, subjects assigned to the depression condition reported to have been feeling more depressed and anxious than their counterparts in the elation condition,  $F(1,28) = 55.19, p < .0001$  and  $F(1,28) = 16.48, p < .001$  respectively. The writing-speed measure also showed that depressed subjects (depression induction) wrote fewer numbers than elated subjects, overall mean 52.00 and 56.74 respectively. However, this difference did not achieve the conventional statistical significance,  $F(1,28) = 2.86, ns$ .

Induced Mood and Attributions. The purpose of this study was to assess the effects of induced elation and depression on attributions for success and failure. It was predicted that elated and depressed subjects will differ in their attributions for success and failure. As can be seen in Table 6.2, three of the four measures of attributions (i.e., ability, effort, and luck) support this prediction.

Ability Attribution. The hypothesis that attributions for success and failure will be biased by mood was supported. A

TABLE 6.1 - MEANS AND STANDARD DEVIATIONS OF MAACL SCORES AND  
PSYCHOMOTOR SPEED (NUMBER-WRITING SPEED) SCORES

MOOD	SUCCESS		FAILURE	
	M	SD	M	SD
<u>ELATION</u>				
MAACL ANXIETY	4.62	1.81	4.87	1.74
MAACL DEPRESSION	6.25	2.05	6.87	2.53
PSY. SPEED	58.37	6.06	55.12	6.81
<u>DEPRESSION</u>				
MAACL ANXIETY	9.25	2.63	9.75	2.64
MAACL DEPRESSION	16.50	2.00	17.50	5.31
PSY. SPEED	51.00	7.41	53.00	6.18

NOTE: MAACL = Multiple Affect Adjective Check List.

2(elation/depression) x 2(success/failure) analysis of variance of ability ratings revealed, as expected, that the mood X task outcome interaction was significant,  $F(1,28) = 15.01, p < 0.001$ . Figure 6.1 displays this interaction. Tests for simple main effects of mood revealed that ability attributions (internal attributions) for success and failure varied as a function of induced elation and depression. The analyses showed that depressed (depression induction) compared to elated subjects (elation induction) made more ability attributions for failure,  $F(1,28) = 11.72, p < 0.001$ , and rated ability as a less

important causal determinant of success,  $F(1,28) = 4.22$ ,  $p < 0.05$ . Further evidence that subjects' attributions of success and failure are biased by mood was provided by the finding that elated subjects displayed more ability attributions for success than for failure,  $F(1,28) = 12.68$ ,  $P < 0.001$ . This finding that mood leads to differential attributions for success and failure suggests the possibility that the depressives and non-depressives divergent attributions for positive and negative outcomes may be partly due to their characteristic mood.

TABLE 6.2 - MEANS AND STANDARD DEVIATIONS OF ATTRIBUTION SCORES  
AS A FUNCTION OF MOOD AND OUTCOME

MOOD	ABILITY		EFFORT		TASK		LUCK	
	M	SD	M	SD	M	SD	M	SD
<u>ELATION</u>								
SUCCESS	6.50	1.73	5.62	1.81	5.87	1.63	4.50	2.12
FAILURE	3.25	2.04	2.37	0.50	5.87	2.58	3.75	1.78
<u>DEPRESSION</u>								
SUCCESS	4.62	1.78	5.87	1.63	5.75	0.66	2.00	1.65
FAILURE	6.37	1.24	5.25	1.98	5.37	1.67	4.62	1.87

NOTE: Ratings are on a 9-point scale.

Effort Attribution. The data from this second attribution measure also support the mood explanation of the differential attributions for success and failure. A  $2 \times 2$  analysis of

variance of effort ratings revealed that the effect for mood was significant,  $F(1,28) = 5.93, p < 0.05$ . As Table 6.2 indicates, subjects made more effort attributions for failure (internal attributions for failure) following depression induction than following elation induction,  $F(1,28) = 10.04, p < 0.005$ . The other main effect was also significant,  $F(1,28) = 9.31, p < 0.005$ . This effect reflects the tendency of elated subjects to attribute more success than failure to personal effort ( $P < 0.001$ ). Note the strong parallel between elated subjects' attributions and those typical of non-depressives.

Further analyses showed that mood and task outcome (success/failure) interacted, although not very significantly, to affect effort attributions,  $F(1,28) = 4.00, p < 0.10 > 0.05$ . This mood X outcome interaction is displayed in Figure 6.2.

Task Attribution. A 2 x 2 analysis of variance of task ratings showed that neither the main effects nor the mood X outcome interaction were significant ( $F < 1.0$ ). Therefore the data from this attributional measure will not be reported.

Luck Attribution. A 2 x 2 analysis of variance of the data from this measure also showed that neither of the two main effects were significant. However, the interaction achieved statistical significance,  $F(1,28) = 5.73, p < 0.05$ . This interaction can be seen in Figure 6.3 which shows that the results from this attribution measure (i.e., luck attribution) parallel those obtained from ability attribution measure. It



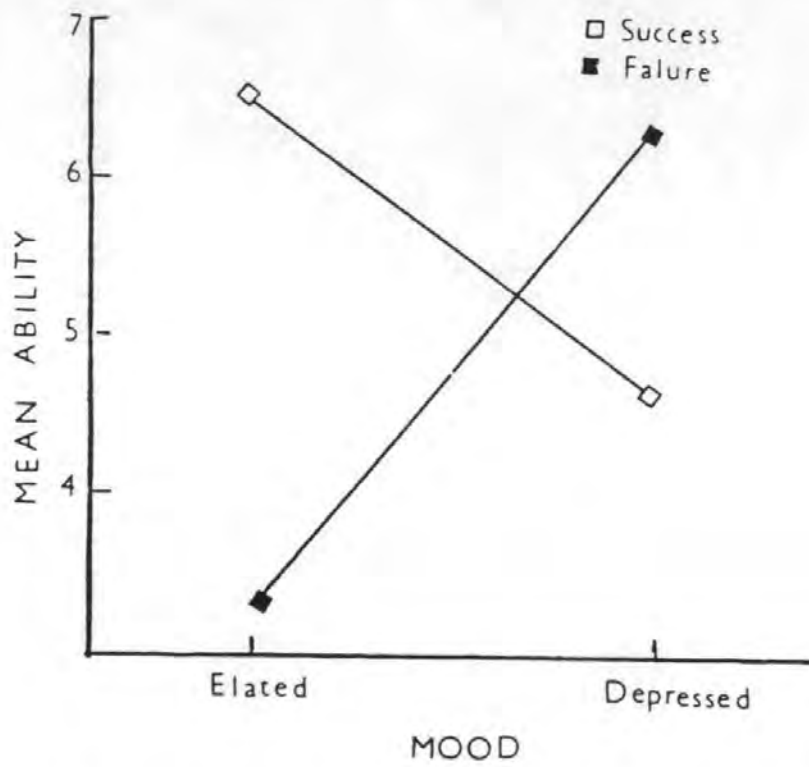


Fig. 6.1. Mean ability ratings as a function of mood (Elation-Depression) and outcome (Success-Failure).

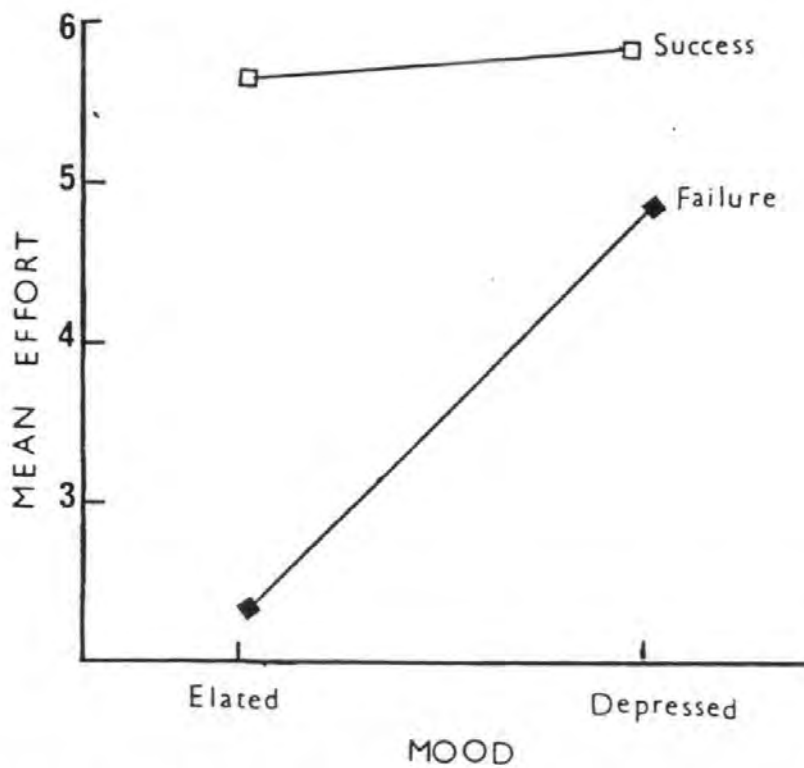


Fig. 6.2. Mean effort ratings as a function of mood (Elated-Depressed) and outcome (Success-Failure).

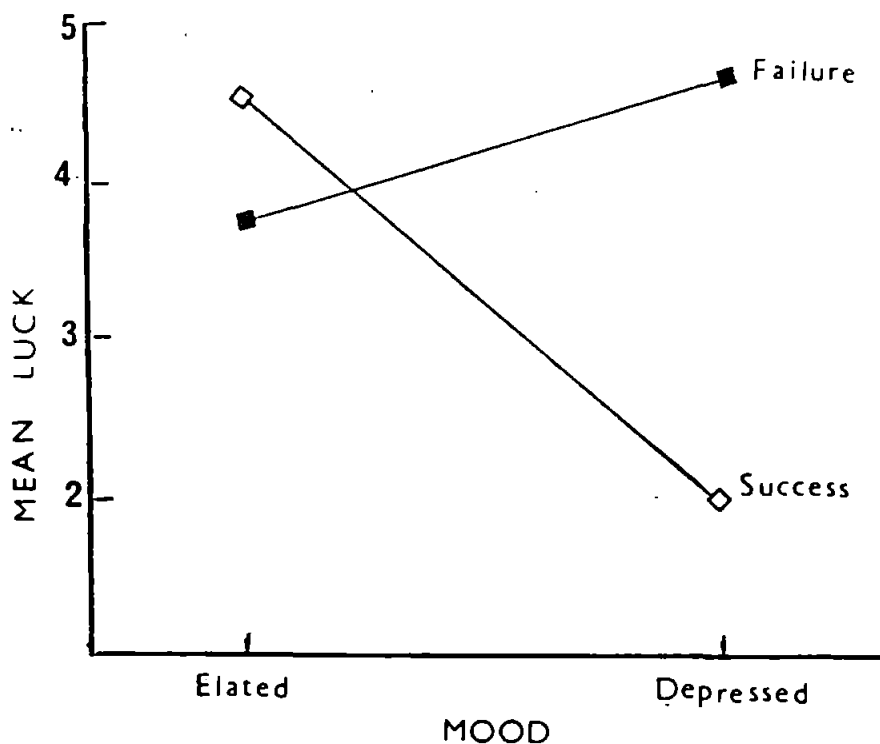


Fig. 6.3. Mean luck ratings as a function of mood (elated vs depressed) and outcome (success vs failure).

as as if subjects viewed luck as a personal factor, rather than a feature of the external world. Accordingly, they ascribed more success to luck following elation induction than following depression induction,  $F(1,28) = 6.29, p < 0.05$ . Further evidence that subjects may view luck as a personal disposition was provided by the finding that depressed subjects (depression induction made more luck attributions for failure than for success,  $F(1,28) = 6.94, p < 0.05$ ).

### 7.3.3 DISCUSSION

The present experiment assessed the effects of induced elation and depression on causal attributions for success

and failure. The results obtained showed, as predicted, that subjects' attributions about their success or failure on the problem solving task were strongly affected or biased by mood. Specifically, it was found that inducing elated mood in non-depressives led them to account more for success than failure in terms of personal or internal causes (ability, effort). In contrast, when non-depressives received a depressed mood induction, they tended to attribute causality to themselves more for failure than for success.

This finding that induced mood states differentially affected causal attributions for success and failure extends further the list of cognitive processes that are reported to be biased by mood states (e.g., Teasdale, and Fogarty, 1979; Teasdale, Taylor, and Fogarty, 1980; Alloy, Abramson, and Viscusi, 1980). Indeed, a number of recent studies reported ample evidence that mood states exert a strong influence on certain types of cognition. The results of the present study suggest that the biasing effects of mood states are not restricted to retrieval of memories (Teasdale, Taylor, and Fogarty, 1980), or to judgement of contingency (Alloy, Abramson, and Viscusi, 1981). There is a possibility that a number of other cognitions, not considered by these studies, may also reflect mood biases.

The present finding that the experimental manipulations of mood differentially affected causal attributions for success and failure suggests that attributional preferences or biases

may partly derive from mood sources, so people's differential attributions for success and failure may reflect differences in mood. In fact, this possibility that judgemental biases may be closely related to mood states has been considered in a recent study by Alloy, Abramson, and Viscusi (1981). In their study, Alloy and her colleagues attempted to determine whether the reported differences in judgement of personal control between depressives and non-depressives (Alloy and Abramson, 1979) reflect differences in mood states. Their results showed that depressives and non-depressives' judgemental differences do indeed reflect mood biases. By inducing depressed mood in non-depressives and elated mood in depressives they reversed their characteristic ways of judging personal control. That is, their data showed a strong effect of mood on judgement of contingency.

The results of the present experiment also showed that inducing depressed mood in non-depressed students reversed their judgements of causality, so that following depression induction, non-depressed subjects exhibited depressogenic attributions for success and failure. That is, they tended to attribute more causality to internal or personal causes (ability, effort) for failure than for success. This finding that the induction of depressed mood in non-depressives led them to display depressogenic attributions adds substance to the earlier proposition that depressogenic attributions may be promoted by the mood state they induce in the first place.

The finding in the present study that induced depressed mood caused non-depressives to account in a depressive fashion for their performance on the problem solving task has an important implication for the attributional model of depression (Abramson, Seligman, and Teasdale, 1978). While Abramson and her colleagues have hypothesized a causal relationship between attribution and depressive affect - that is, that certain attributions lead to depressive affect and symptoms, results from Experiments 1 - 6 tend to suggest that attribution and depressive affect may have a reciprocal relationship: certain attributions (e.g., negative self-attributions) lead to depressive affect, and the depressive affect may in turn generate the kinds of attribution that are likely to promote or maintain the depressive state. To the extent that this interpretation is correct, it requires that the attributional model (Abramson et al., 1978) and other cognitive models (e.g., Beck, 1967) should be extended to account for this and other findings (e.g., Teasdale et al., 1980; Alloy et al., 1981) advocating reciprocity in the relationship between cognitive biases and depressive affect.

#### 7.4 SUMMARY AND CONCLUSIONS

The present investigation of the relationship between mood states and causal attributions was carried out to determine whether depressogenic attributions (e.g., negative self-attributions, external attributions of positive outcomes) are promoted or indeed related to depressed mood. It was reasoned that one way of examining this question would be by

assessing the effects of induced moods on attributions for positive and negative outcomes. Accordingly, the effects of mood manipulations on attribution of causality were assessed in two related studies. Contrary to the prediction, the results of the first study failed to show any effect for mood. Regardless of the mood manipulation they received, subjects in this experiment displayed more internal, stable, and global attributions for positive than negative outcomes. It was argued that the results of this experiment reflected inadequacies of the scale used to assess attributions. As a result, a second experiment was designed in which a relatively more valid and reliable attribution scale was used. As predicted, the results showed a marked effect of mood on causal attributions of success and failure - it was found that mood differentially affected attributions of causality for success and failure. The demand characteristics (Orne, 1962) explanation for this finding was ruled out, since only three of the four measures of attributions showed the mood effect. Had all four attribution measures revealed the effect for mood, the results would have qualified for this explanation.

A question that needs to be answered at this point, is how to reconcile the results of these two experiments? As already argued, there is a strong possibility that the failure of the results of the first experiment to show an effect for induced mood may be due to the inadequacy of the method used to assess the main dependent variable (attribution). Another explanation for these divergent results, is that perhaps mood

states exert an influence on individual attributions (ability attribution, effort attribution, luck attribution - as found in the second experiment) but not on attributional styles (internality, stability, and globality - assessed in the first experiment). Because, attributional styles are, unlike attributions to individual factors or causes, generalised beliefs or attitudes (cf. Abramson et al., 1978) that can resist the influence of transient mood states. Although attractive, this explanation is unlikely since the results of the second experiment (Experiment 6) showed that even internal, stable attributions (i.e., ability attributions) were affected by induced elated and depressed mood states.

The finding, in the second experiment, that induced depressed mood led to depressogenic attributions (i.e., the tendency to attribute more failure than success to personal or internal factors) was interpreted as supporting the reciprocity view of the relationship between attribution and depressive affect. This reciprocity view of the relationship between different components of depression (i.e., cognitive, and affective) appears to be more relevant to the clinical reality of depression. It was argued on the basis of both the present results and the results reported by others (e.g., Teasdale, Taylor, and Fogarty, 1980; Alloy, Abramson, and Viscusi, 1981) that cognitive models of depression (e.g., Abramson et al., 1978; Beck et al., 1979) should be extended to capture the clinical reality of this syndrome.

An important question left unanswered, is how mood affects attributions of causality? One can only speculate at this point since mood or affect is still considered to be post-cognitive (Zajonc, 1980, has a detailed but constructive criticism of this conception). A plausible explanation, as to how mood states affect judgements of causality, is that the mood manipulations may have influenced subjects' perceptions of themselves, which in turn affected their causal explanations. Another explanation, inspired by Kelley's informational conception of the attribution process (1967) is that the mood induction procedures may have increased the salience of certain informational cues (e.g., consensual information determines ability attribution) which in turn determined subjects' attributions of their performance. The question of whether perception, information use, or any other variable, mediated the effect of mood on attribution of causality can only be solved by future research.



## CHAPTER EIGHT

### EXPLAINING ATTRIBUTIONAL PREFERENCES:

### THE ORIGIN OF DEPRESSOGENIC ATTRIBUTIONS

- 8.1 INTRODUCTION
- 8.2 EXPERIMENT 7
- 8.3 EXPERIMENT 8
- 8.4 DISCUSSION AND CONCLUSIONS

## 8.1 INTRODUCTION

An important issue left unexplored in the attribution-depression field concerns the nature or the origin of depressogenic attributions. While a great deal may have been learned about their various consequences (including their affective and behavioural consequences), understanding of their origin and antecedent conditions is still surprisingly very limited. Indeed, much of the empirical research in the attribution-human helplessness field has limited itself to demonstrating systematic attributional differences between depressives and non-depressives. As a consequence, the antecedents of their attributional tendencies are still uncertain.

A similar situation of neglect also exists on the theoretical front. Recent theories, including learned helplessness formulations (Abramson et al., 1978; Miller and Norman, 1979), have devoted relatively little attention to the factors that may be responsible for the depressives-non-depressives' attributional tendencies and preferences. In fact, Abramson and her colleagues (1980) acknowledged the limitation of their model with respect to accounting for the nature of attributional preferences and biases. Commenting on the issue, Abramson, Garber, and Seligman (1980) conceded that:

"the attributional reformulation is relatively silent with respect to specifying the determinants of what particular attribution a person happens to make for

uncontrollability" (p.18, 1980).

Clearly, there is a need not only to specify the determinants of depressogenic attributions but also to clarify why depressives and non-depressives arrive at different causal attributions when faced with uncontrollable or aversive situations.

The present investigation, carried out to clarify these issues, was designed to investigate whether depressives and non-depressives' attributional tendencies and biases are related to their characteristic ways of processing and utilizing information.

While a multitude of factors may promote depressives and non-depressives attributional preferences, clinical literature suggests that their attributional differences may be better accounted for by differences in information processing strategies (e.g., Beck, 1967; Beck et al., 1979). Indeed, there is a strong possibility, as Beck's clinical observations tend to suggest, that depressives and non-depressives' attributional differences may have an information basis. Consistent with this proposition, is the commonly reported finding that depressed persons, compared to non-depressed ones, process and organize information within characteristic depressive schemas. Thus, there is a strong possibility that depressives and non-depressives characteristic ways of processing information may constitute the basis of their attributional tendencies.

More relevant to the argument that biases or differences in attributions may have an informational origin, is Kelley's (1967, 1973) theorizing about how people arrive at causal attributions or judgements. In his 1967 paper, Kelley proposed that attributors consider three types of information before they make causal attributions: distinctiveness, consistency, and consensus (chapter four gives a detailed review of Kelley's ANOVA model of attribution processes). It is therefore possible that depressives-nondepressives' attributional differences reflect differences in the utilization of distinctiveness, consistency, and consensus information - that is, they may base their attributions on different informational cues. In that case depressives and non-depressives should exhibit differences in the ways in which they evaluate causal information (i.e., consensus, consistency etc.). Experiment 7 was designed to test this specific proposition.

## 8.2 EXPERIMENT 7

The present experiment was designed to investigate whether, as suggested by Beck's clinical observations and Kelley's theoretical statements, depressives and non-depressives' attributional tendencies reflect differences in information processing and utilization. As an initial step towards clarifying this issue, it was decided to examine depressed and non-depressed students' assumption about attribution-relevant information (i.e., causal information).

Depressed and non-depressed students were asked to indicate the importance (on a 11-point scale) of having consistency, consensus, and distinctiveness information if they were attempting to understand the causes of personal positive and negative events. The general prediction was that depressed and non-depressed students (high and low BDI scores respectively) would differ in their evaluations of causal information.

### 8.2.1 METHOD

Subjects and design. 30 undergraduates (15 depressed and 15 non-depressed) were recruited and used as subjects in the present experiment. Subjects were assigned to the depressed group if they scored more than 9 on the Beck Depression Inventory (BDI) (Beck et al., 1961), and to the non-depressed group if they scored less than 9 on the same scale. Mean BDI of depressed was 12.73 and of non-depressed subjects 3.33. The Multiple Affect Adjective Check List (MAACL) (Zuckerman & Lubin, 1965) was used as a further index of depression. Mean MAACL of depressed was 16.46 and of non-depressed subjects 7.93.

This experiment was a 2(depressed-non-depressed) x 2(positive-negative event) design, with repeated measures on the second factor.

Information Measures. A questionnaire consisting of three hypothetical positive events (e.g., 'passing a driving test') and three negative events (e.g., 'failing a job interview') was

constructed and administered to subjects following the completion of both the BDI and MAACL. Subjects were first asked to imagine that each event described in the questionnaire happened to them, and then they were asked to estimate (on a 11-point scale) the importance of acquiring or having consensus information (information concerning the performances of other people in the same achievement situation), consistency information (information concerning the subject's past performances in the same achievement situation), and distinctiveness information (information concerning the subject's performance in other achievement situations) when attempting to understand the occurrence or the causes of personal positive and negative events.

#### PROCEDURE

After completing both the BDI and MAACL, all subjects received a short questionnaire, consisting of three positive and three negative events, together with the following instructions:

The present study is concerned with the way people explain or understand things that happen to them in everyday life. More specifically, the present study is an attempt to find out how people organize and process information when they make judgments about the causes of events that happen to them. In this experiment you will be given a series of situations involving success or failure, and then you will be asked to estimate the importance of having certain informational cues (listed below each situation) if you were attempting to understand the causes of your success and failure. Please note that

your task consists mainly of (a) imagining that each event described in the questionnaire happened to you personally, and then (b) estimating on 11-point scales the importance of having certain types of information if you were attempting to understand why certain events happened to you. Please don't hesitate to ask for further clarifications if necessary.

Following the administration of this 'information' questionnaire, all subjects were paid and thanked for their co-operation.

### 8.2.2 RESULTS AND DISCUSSION

In order to see whether depressed and non-depressed subjects displayed differences in information evaluations, distinctiveness, consistency, and consensus ratings were subjected to analyses of variance, with mood (depressed-non-depressed) as the between-subjects factor and positive-negative event as a repeated measure.

#### DISTINCTIVENESS DATA

A 2(depressed-non-depressed) x 2(positive-negative event) analysis of variance of distinctiveness ratings (averaged over the three positive and the three negative events) showed that neither the effect for mood nor the interaction were significant. However, the effect for event (positive-negative) was significant,  $F(1,28) = 9.05, p < 0.01$ . Newman-Keuls (Winer, 1962) comparisons of means showed that non-depressed subjects ascribed more importance to distinctiveness information when explaining negative events than when explaining positive events,  $M = 6.86$

and 5.06 ( $p < .01$ ). The same pattern of results was also obtained for depressed subjects, although statistically not significant (see Table 7.1).

Consistency Data. A 2 x 2 ANOVA of consistency ratings (averaged over the three positive and the three negative events) showed that neither the effect for mood nor the effect for event was significant. The interaction between mood and event also failed to reach significance,  $F(1,28) = 2.49$ , ns. The Newman-Keuls test on means indicated, however, that when asked to explain the occurrence of personal negative event, depressed and non-depressed subjects differed in their evaluations of consistency information. Depressed subjects rated consistency information to be more important than did non-depressed subjects,  $M = 8.60$  and  $7.06$  ( $p < .01$ ) respectively. Further comparisons of means revealed that depressed subjects rated consistency information to be more important when attributing negative vents than when attributing positive personal events,  $M = 8.60$  and  $6.80$  ( $p < .01$ ) respectively.

Consensus Data. A 2 x 2 ANOVA of consensus ratings (averaged over the three positive and the three negative events) revealed that the main effect for mood was significant,  $F(1,28) = 4.57$ ,  $p < 0.05$ . The main effect for event (positive-negative event) was not significant. However, the mood (depressed-non-depressed) x event (positive-negative) interaction approached statistical significance,  $F(1,28) =$



TABLE 7.1 - MEANS OF DISTINCTIVENESS, CONSISTENCY, AND CONSENSUS RATINGS AS A FUNCTION OF MOOD (DEPRESSED-NON-DEPRESSED) AND TYPE OF EVENT (POSITIVE-NEGATIVE)

	<u>POSITIVE EVENTS</u>		
	Distinctiveness	Consistency	Consensus
DEPRESSED	5.86	6.80	9.13
NON-DEPRESSED	5.06	7.20	6.53
	<u>NEGATIVE EVENTS</u>		
	Distinctiveness	Consistency	Consensus
DEPRESSED	7.40	8.60	8.60
NON-DEPRESSED	6.86	7.06	7.53

NOTE: Ratings are on 11-point scales, ranging from 1 = not at all important to 11 = extremely important.

3.09,  $p < 0.10 > 0.05$ . Figure 7.1 displays this interaction. As can be seen in this figure, depressed and non-depressed subjects differed in their evaluations of consensus information when they were asked to attribute or explain positive personal events; depressed subjects rated consensus information to be more important than did non-depressed subjects,  $M = 9.13$  and  $6.53$  ( $p < 0.01$ ) respectively. As expected, non-depressed subjects ascribed more importance to consensus information when accounting for personal negative events than when accounting for positive events,  $M = 7.53$  and  $6.53$  ( $p < 0.01$ ) respectively.

The prediction that depressed and non-depressed subjects

would differ in their evaluations of causal information (i.e., consensus, consistency and distinctiveness) received some support in the present study. The results showed that depressed and non-depressed students differed in their evaluations of two of the three types of information (consistency, and consensus). These results are consistent with recent theorizing about the informational basis of attributional biases and differences (e.g., Manson and Snyder, 1977; Miller and Ross, 1975; Nisbett and Ross, 1980).

The finding that depressed subjects rated consistency information to be more important than did non-depressed subjects when they were asked to account for negative events suggests that depressives may be more inclined than non-depressives to search for and use personal history information when making causal attributions for personal negative events. Perhaps depressives' maladaptive attributions are related to their tendency to use personal information (e.g., consistency) as opposed to environmental types of information (e.g., consensus). Future research that concentrates on the types of information that depressives process and use when engaged in attributional activities should test this proposition.

The prediction that depressives and non-depressives will differ in their evaluations of causal information was relatively supported by the data from the consensus scale. It was found that depressed subjects rated consensus information to be more important than did non-depressed subjects, when they were asked to account for personal positive events. This

finding adds substance to the earlier argument that depressives and non-depressives' attributional preferences and differences may be related to biased use of causal information.

Most relevant to this argument, however, is the finding, in the present study, that subjects' evaluations of consistency, and consensus information tended to be a function not only of their mood (depressed-non-depressed) but also of the type of event (positive-negative) they were asked to account for. This is another indication that biased use of causal information may be at the basis of depressives-non-depressives' differential attributions for personal outcomes.

While the present study may have answered the question about the origin of attributional preferences, it left unanswered a further question - the reason as to why depressives and non-depressives may be biased in their information evaluations and perhaps utilization is not clear. One can only speculate at this point. Perhaps both depressives and non-depressives hold different causal hypotheses about the occurrence of personal positive and negative events, and then they may apply the principle of cognitive economy (cf. Hansen, 1980) in the process of confirming such hypotheses (i.e., searching for relevant causal information). It is also possible that their biased evaluations of causal information may be motivationally based. The search for information may be motivated or guided by the need to maintain a stable view of the self-concept (stable self-conception) (Heider, 1958). Only future research could determine whether a cognitive or a

motivational explanation accounts better for these informational biases.

Although it may have provided some insight into the nature of attributional biases, the present study is limited in many ways. An obvious limitation of this study is its assumption that people's attributions are based on distinctiveness, consistency, and consensus information. Even Kelley conceded that the making of causal attributions may involve the use of informational cues other than the ones considered in the present study. A further limitation of this study is its tendency to assimilate information evaluation to information utilisation. Although differences in information evaluation may actually reflect differences in information utilisation, the hypothesis that depressives and non-depressives use information differently requires stronger evidence than that provided by the present study. A third limitation of this study is its inability to reveal whether depressives and non-depressives actually differ in the amount and/or types of causal information they use to arrive at their causal attributions. Because of these limitations, another investigation was conducted in which differences between depressives and non-depressives in information processing and utilisation were further examined.

### 8.3 EXPERIMENT 8

The present study further investigated whether, as the results of the previous investigation tend to suggest, depressives and non-depressives' attributional biases and differences are

related to differences in the amount and/or types of information they use to make causal attributions. As pointed out in the previous section, Experiment 7 looked at information processing and utilization in a rather restricted context. So in the present study the amount as well as the types of information that subjects themselves search for and use when making causal judgements about the occurrence of important events was assessed. Depressed and non-depressed subjects (high and low BDI scores respectively) were asked to list the types and amount of information they would require to account for the occurrence of an hypothetical event. It was hoped that by allowing subjects to seek and use freely the amount of information they require, a better insight may be gained into how depressives and non-depressives process, organize, and use information to generate causal attributions about important life events.

To summarise, the purpose of this study was to assess the types and amount of information that depressed and non-depressed subjects search for and use in making causal attributions about important events. Based on the results of Experiment 7, it was predicted that depressed and non-depressed subjects would evidence or produce differences in both the amount and types of causal information they require to arrive at their causal explanations and attributions.

#### 8.3.1 METHOD

Subjects. 20 undergraduates (10 depressed and 10 non-depressed

selected on the basis of their Beck Depression Inventory (BDI) (Beck et al., 1961) were used as subjects in the present study. Subjects scoring more than 9 on the BDI were assigned to the depressed group, and those scoring less than 9 on the same scale were identified as non-depressed and were assigned to the non-depressed group. The mean BDI of depressed was 12.70 and of non-depressed subjects 2.70.

Information Measures. To ensure a reasonable assessment of information processing and utilisation, both quantitative and qualitative measures were employed. As a measure of the amount of information searched for and used by subjects to make judgements about causality, the number of questions (information requested) that subjects asked was counted. The second measure, concerning the types of information requested, was obtained by content-analysing the information needed by subjects for their causal explanations.

Procedure. Following the administration of the BDI, both depressed and non-depressed subjects were provided with a brief description of an hypothetical event. The event described concerned a first-year Polytechnic student who failed his/her exams. Subjects were asked to list, on a sheet of paper, all the information they thought they would need to determine the cause of this first year student's failure. Before commencing the task, all subjects received the following instructions:

The present study is concerned with the way people process, organize, and utilise information when they try to understand important things that happen to them in life. Previous studies in this line of research revealed that before achieving a reasonable understanding of things that happen to them or to others, people search for and gather a certain amount of information. The present study attempts to determine whether people differ in the amount and types of information they seek and use when making judgements about the causes of important events. Your major task here consists of listing, on the sheet of paper provided, what information you would need to determine the major cause of a first-year student's failure at his/her exams.

Following the information search task, all subjects were paid and thanked.

### 8.3.2 RESULTS

As predicted depressed and non-depressed subjects differed significantly in the amount (number of questions asked) of information they needed to make causal attributions about the occurrence of the negative event,  $F(1,19) = 13.19$   $p < 0.005$ .

As Figure 8.1 indicates, non-depressed subjects requested significantly more information than their counterparts in the depressed group,  $M = 7.60$  and  $4.00$  respectively.

The second set of data (concerning the types of information requested) was subjected to content analysis using a set of

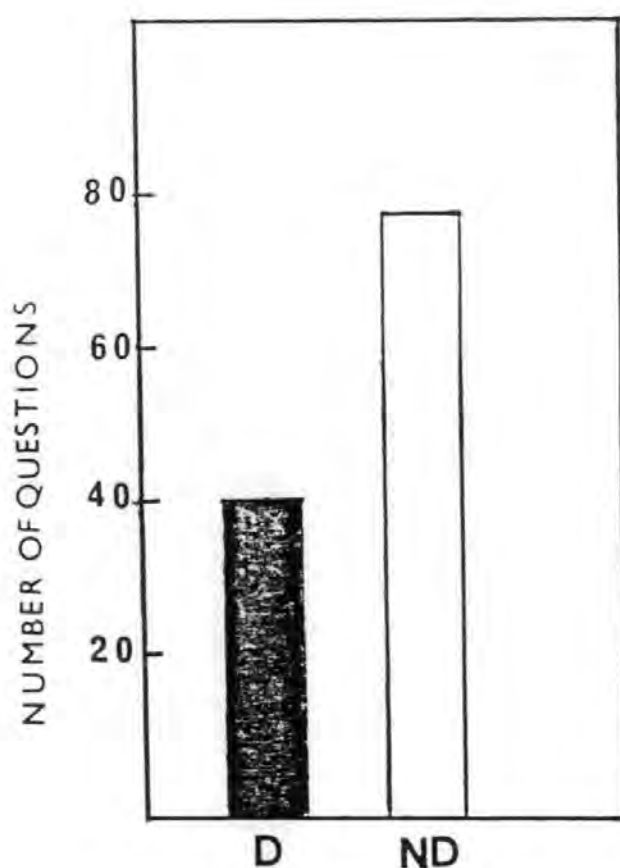


Fig. 8.1. Number of questions asked by depressed(D) and non-depressed(ND) subjects.

constructed categories and then to frequency counts.

In the analysis six information categories were identified (including 'Uncodable') - This was achieved by categorising information according to whether it referred to (a) the actor's (student) characteristics (e.g., 'his/her basic ability before commencing the course'), (b) the actor's state (e.g., 'has the student any health problem?'), (c) the actor's past exam. records (e.g., 'the student's performance at previous exams'), (d) the actor's preparation (e.g., 'amount of time spent attending lectures and revision'), or to circumstance/people/environment (e.g., 'is the student living in a noisy student flat where it would be difficult to concentrate or even to decide that



he/she wanted to do some work'). As can be seen in Table 8.1, depressed and non-depressed subjects differed in the types of information they needed to account for the student's failure at his/her exams. Depressed subjects tended to ask more questions (requested more information) about the actor's characteristics than did non-depressed subjects,  $t(18) = 1.80, p < 0.10 > 0.05$ . This tendency of depressed subjects to favour the search for and use of personal characteristics information (e.g., the student's mental capacity') when making causal attributions was clearly reflected in the frequency counts data - As Table 8.1 indicates, the number of questions they asked about the actor's characteristics accounted for 30% of the total number of the questions asked. Non-depressed subjects, in contrast, preferred information about the actor's preparation (e.g., 'how much time and effort the student put into revision?'). The results revealed that non-depressed subjects requested significantly more of this type of information (actor's preparation) than did depressed subjects,  $t(18) = 3.56, p < 0.01$ .

Subsequent analyses also revealed that non-depressed subjects tended to search for more information about the actor's state (e.g., 'was anything particularly wrong on the day of the exam?') and about the environment/people (e.g., 'what kind of friends he/she had made and whether he/she had problems with housing') than did depressed subjects (see Table 8.1).

TABLE 8.1 - NUMBER OF QUESTIONS ASKED BY DEPRESSED AND NON-  
DEPRESSED SUBJECTS FOR EACH INFORMATION CATEGORY

INFORMATION CATEGORY	DEPRESSED	NON-DEPRESSED
Actor's characteristics	12 (30%)	6 (7.89%)
Actor's state	5 (12.5%)	21 (27.63%)
Actor's past exam. record	4 (10%)	6 (7.89%)
Actor's preparation	7 (17.50%)	17 (22.36%)
Environment/people	9 (22.50%)	18 (23.68%)
Uncodable	3 (7.50%)	8 (10.52%)

#### 8.4 DISCUSSION AND CONCLUSIONS

The hypothesis that depressives and non-depressives differ in the amount and types of information they search for and use when making causal attributions was supported by the data from the present study. The results obtained showed that depressed and non-depressed subjects differed in the amount and to some extent in the types of information they seek when attempting to account for a negative event. This finding that subjects' information search was a function of their characteristic mood (depressed-non-depressed) suggests that depressives and non-depressives attributional tendencies may be related to their characteristic ways of processing and utilizing information when making causal attributions.

At present there appears to be no satisfactory theoretical explanation for this finding. One reason as to why depressed

subjects, in the present study, requested less information than did non-depressed ones, is that perhaps depressives, unlike non-depressives, possess a well-developed schema about failure (the hypothetical event in the present study) and therefore may need less information to account for the occurrence of such outcome. Another possibility is that depressives, relative to non-depressives, hold fewer causal hypotheses about the occurrence of the event, and therefore may require less information to evaluate them (hypotheses). Both explanations are of course speculative, and should be treated as such until proved otherwise.

The finding, of this study, that depressed subjects, relative to non-depressed ones, tended to request characterological information (information concerning the actor's characteristics) may account better for the depressives' maladaptive attributions (i.e., depressogenic attributions). Depressogenic or negative self-attributions may be the result of the tendency to relate personal misfortunes to one's character. However, what leads depressives to concentrate on personal information (as opposed to environmental types of information) for their causal inferences is not yet clear. There is a possibility that their biased search for and use of causal information is guided by their preconceptions or intuitive theories about causality. They may, as has been suggested in the previous discussion, search for information that confirm their implicit hypotheses about the occurrence of events. There is also the possibility that their biased use of information is related

to their need to maintain a stable self-conception (e.g., Heider, 1958). Regardless of what causes depressives to concentrate on (or use) certain types of information and neglect others, it is increasingly apparent that their typical attributions may be promoted, if not caused, by their particular ways of using information.

The present finding that depressogenic attributions may develop as a result of the tendency to use characterological information has interesting implications for therapy. If, as suggested by Experiments 1 - 4, these kinds of attributions are important in the causation of depression, then therapy of depression could also be directed at them. The present results suggest that therapy aimed at correcting or loosening the grip of depressogenic attributions can do so by challenging their informational basis.

It should be pointed out that the results of the present study do not rule out motivational or affective explanations of depressives' maladaptive attributions. Indeed, the results of Experiment 6 dictate that the role of affective processes in promoting and/or maintaining such attributions should not be ignored.

In conclusion, depressed subjects were found to differ from non-depressed subjects in the amount and to some extent in the types of information they seek and utilise when making causal attributions about failure. These results were interpreted

as consistent with the informational hypothesis about the origin of depressogenic attributions. It should be pointed out, however, that the present study shares the limitations of the previous one. For instance, it was implicitly assumed that actors and observers operate in the same way with regard to seeking and utilising information in making causal attributions. And finally, since the present study used non-clinically depressed persons, its results would not necessarily hold for clinically depressed persons.

PART 3

SUMMARY AND CONCLUSIONS

CHAPTER NINE  
THE AETIOLOGICAL IMPORTANCE  
OF  
CAUSAL ATTRIBUTIONS

The present research programme was inspired by the consistent finding, of human helplessness studies, that depressed college students, relative to non-depressed ones, make internal stable attributions for negative outcomes (e.g., failure) and tend to make external attributions for positive outcomes (e.g., success) (e.g., Barthé and Hammen, 1981; Kuiper, 1978; Rizley, 1978; Seligman et al., 1979). The main goal of this research was to clarify the relationship between causal attribution and depression. Within this field, the learned helplessness theory of depression (Abramson, Seligman, and Teasdale, 1978; Miller and Norman, 1979) offers a comprehensive framework for examining such a relationship. As such, the learned helplessness model served as a frame of reference for most of the experimental and clinical work reported in previous chapters.

Given the recent speculations about the importance of attributions in the genesis and development of depressive symptoms and disorders (e.g., Miller and Norman, 1979; Seligman et al., 1979), a detailed investigation of the relationship between causal attribution and depression became warranted. Although there has been considerable research documenting the link between certain attributions and depression (e.g., Kuiper, 1978; Rizley, 1978; Seligman et al., 1979), there is surprisingly little evidence bearing on the aetiological importance or significance of such attributions. Accordingly, a series of experiments, which addressed this issue and other related issues, were



carried out. These experiments have produced a number of findings concerning both the status of causal attributions and the nature of their relationship to depression. To some extent, the studies endorse the learned helplessness view that certain attributions assume an aetiological role in depression.

Reasonably good evidence was found for the depressogenic effects of negative self-attributions (i.e., internal attributions for bad outcomes). Consistent with the recent theorising about the attributional basis of helplessness and depressive symptoms or deficits (e.g., loss of motivation, loss of self-esteem, self-blame, etc.) (e.g., Abramson et al., 1978; Seligman et al., 1979), the results of Experiments 1 - 4 clearly showed that depression, as measured by the BDI and MAACL, was causally linked to the tendency to account for personal negative events in terms of personal shortcomings. Specifically, there was an unambiguous experimental evidence that depression or rather its correlates (e.g., dysphoria, low psychomotor performance, reduced expectations of future success etc.) are induced by negative self-attributions. From these results, there is a clear indication that the attributional strategy that depressives tend to adopt when faced with uncontrollable or aversive situations (i.e., depressive attributional style for failure) is at the origin of their typical symptomatology and disturbances.

More relevant perhaps to the issue concerning the aetiological significance of attributions in depression, are the results

from the clinical study (i.e., Experiment 4). The results revealed, as hypothesised by Seligman and his associates (1979), that there is a high degree of specificity in the relationship between the tendency to make internal attributions for personal negative events (i.e., negative self-attributions) and depression of clinical proportions. This finding regarding the specificity of effect of negative self-attributions lends credence to the claim by learned helplessness theorists and investigators (e.g., Miller and Norman, 1979; Kuiper, 1978) that attribution processes operate in the genesis and development of depressive symptoms. More importantly, the finding provides strong evidence for the aetiological importance of negative self-attributions (in depression).

There is, however, a theoretical ambiguity that remains regarding exactly how these kind of attributions (i.e., negative self-attributions) induce depression or its symptoms. The learned helplessness theorists (Abramson, Seligman, and Teasdale, 1978) speculated that the depressogenic effects of these attributions are mediated by expectancy. That is, attributions affect expectancy which in turn undermines motivation and hence helplessness, hopelessness, and other symptoms of depression. Another explanation, derived from Beck's cognitive formulation of the aetiology of depressive phenomena (Beck, 1967; Beck et al., 1979), proposes that negative self-attributions produce depressive symptoms (e.g., loss of self-esteem, self-blame, hopelessness, loss of motivation,

etc.) by activating the latent negative attitudes towards the self, the environment, and the future (the so-called cognitive triad). Therefore an additional work for future research in this area should be to evaluate the relative strength of these competing explanations of the depressogenic effects of causal attributions.

A good deal of recent research (e.g., Golin et al., 1981; Seligman et al., 1979) has assumed that the tendency to attribute personal positive outcomes to external or situational causes, is as depressogenic in its effects as the tendency to assume responsibility for personal negative events (i.e., the tendency to make internal attributions for negative outcomes). The present work failed to provide any support for such an assumption. The results obtained seemed to suggest that, although external attributions for positive outcomes may be associated with depression, they are of little or no aetiological significance to depression. At best, they could be described as symptomatic (as opposed to more aetiological type of attributions). Of course, future studies that assess depressed patients' attributions of personal events both before and after treatment (i.e., longitudinal studies) will be methodologically more appropriate to clarify whether external attributions for positive outcomes are a consequence (symptomatic) or an antecedent of depression. Until further research has elaborated on this issue, the notion by Seligman and his associates (1979) that the tendency to externalise positive outcomes enjoys an aetiological status in depression

should be treated with caution.

With respect to the question of whether the depressives' tendency to make internal attributions for negative outcomes but not for positive outcomes reflect the influence of their characteristic mood (i.e., depressed mood), the results of Experiment 6 offered a relatively clear answer - depressives' typical attributions are promoted if not induced by their characteristic mood. The results obtained indicated that depressogenic attributions are closely related to depressed mood. Specifically, the results showed that the experimental induction of depressed mood in non-depressives led them to display the type of attributions that are typical of depressives - that is, they tended to account more for failure than for success in terms of internal or personal causes (e.g., ability attributions, effort attributions).

This finding that inducing transient depressive mood state in non-depressives led them to exhibit depressogenic attributions may have profound implications for the theoretical understanding of both the relationship between causal attribution and depression and of the cognitive functioning of depressed persons. Whereas the recent attributional formulations of helplessness and depressive phenomena (Abramson et al., 1978; Miller and Norman, 1979; Seligman et al., 1979) treat depressive affect merely as an epiphenomenon of maladaptive or depressogenic attributions, the results from

the present work suggest that depressive affect may be a critical variable in reinforcing or promoting such attributions and developing the depressive state. One possibility, already considered in chapter 7, is that there may be a reciprocal relationship between causal attribution and depression - that is, depressogenic attributions induce depressive affect which in turn generates and reinforces the types of attributions that are likely to maintain and/or develop the state of depression. A more interesting possibility is that the relationship between attribution and depression may take the form of a positive feedback model. Indeed, this view appears to be more congruent with the results that emerged from the present work (Experiments 1 - 6) and that of other investigators (e.g., Golin et al., 1981). There was suggestive evidence from these investigations that causal attribution and depression reinforce each other in a continuous manner. And in so doing they perpetuate the state of depression. To the extent that this interpretation of the results is correct, it requires that the Abramson-Seligman-Teasdale attributional formulation of helplessness and depression should be extended to capture this new but complex dimension of the relationship between attribution and depression. Failure to incorporate affective variables in this model could undermine the value of its account of helplessness and depressive deficits.

Another possible implication of the findings concerning the biasing effects of depressed mood on attributions is that the

influence of this mood state may not be limited to attribution processes. In fact, there is a possibility that the depressed persons' cognitive functioning as a whole may be coloured by their pathological mood state (i.e., depressed mood).

Teasdale, Taylor, and Fogarty (1980) seemed to have reached a similar conclusion, in a study assessing the effects of experimentally induced elation and depression on retrieval of happy and unhappy experiences. They reported results which suggested a strong effect of induced mood state on these types of cognition. Specifically, they found that induced depressed mood facilitated the retrieval of unhappy but not happy memories. In contrast, the retrieval of happy experiences was found to occur more in the elated mood state than in the depressed mood state. The Teasdale et al.'s finding concerning the biasing effect of mood state on retrieval processes further attests to the importance of affect in influencing cognitive activity or functioning in both its adaptive and maladaptive forms.

Using a similar experimental paradigm, Alloy, Abramson, and Viscusi (1981) also reported ample evidence for the biasing influence of mood state on cognition. In a recent study assessing the effects of different mood states on judgement of contingency (i.e., response-outcome relationship), Alloy and her colleagues found that by making depressives elated (experimental induction of elated mood) and non-depressives depressed (depression induction) they reversed their typical ways of judging personal control and contingency. Their

data clearly showed that the depressives' tendency to accurately judge their personal control (see Alloy and Abramson, 1979, for a detailed account of this finding) was reversed (i.e., they showed an illusion of control) following the elation induction. Similarly, the non-depressives' tendency to show an illusion of control was 'corrected' following the depression induction - that is, they tended to give accurate judgements of contingency between their responses and outcomes. This finding is yet another indication that affective factors may exert a biasing influence on a number of cognitive processes. While further studies will be required to elaborate on this issue (e.g., specification of how mood affects cognition), the Alloy et al. results (1981) together with the results of the present work and those reported by Teasdale and Fogarty (1979) and Teasdale et al. (1980) and many others strongly suggest that affect may be at the basis of many cognitive biases that characterize both depressed and non-depressed persons.

In summary: substantial evidence emerged from the present work that negative self-attributions have depressogenic effects - that is, they induce depressive symptoms. This was taken as a reasonable indication that these kind of cognitions have an aetiological status in depression (i.e., depressogenic attributions are important in the causation of depression). The results concerning the biasing influence of mood state on attributions was taken as an indication that causal attribution and depression entertain at least a reciprocal relationship.

So far the discussion has been concerned with theoretical status of attributions and the nature of the relationship they may entertain with depression. A further issue addressed by the present research concerns how these so-called depressogenic attributions (i.e., internal attributions for bad outcomes) develop. In line with Kelley's theorizing about the informational basis of attributions (Kelley, 1967, 1973), it was found that these type of attributions are formed as a result of selective use of information. The results of Experiment 8 showed that depressives, compared to non-depressives, favoured the use of personal or characterological information when making attributions about bad events. Thus, depressogenic attributions may develop as a result of the utilization of characterological information. An important question that should be asked at this point is what leads depressed people to favour the search for and the use of this type of information when they engage in attributional activities? Two important factors were delineated that may lead depressives to use characterological information and then display depressogenic attributions. The first factor concerns the frequency of exposure to aversive situations or events. The second factor concerns the person's ongoing self-esteem.

There is an indication from recent work that repeated exposure to stressful life events often leads people to display maladaptive or depressogenic attributions for their difficulties. A



recent study by Peterson, Schwartz, and Seligman (1981) confirmed this. They found that the tendency to blame one's character (characterological self-blame) was positively associated with frequent experience with negative life events: suggesting that people who display characterological or depressogenic attributions may arrive at them by covariation analyses. While further research may be required to specify the cognitive operation involved in the making of these kind of attributions, the findings of Peterson and his colleagues (1981) do provide support for the possibility that these attributions (i.e., depressogenic attributions) may be caused by repeated exposure to life stressors.

A further factor believed to determine whether characterological information will be used and depressogenic attributions displayed concerns the person's ongoing self-esteem. There is suggestive evidence from recent studies that lowered self-esteem may be an important antecedent of depressives' maladaptive attributions (e.g., Ickes and Layden, 1978). In a series of studies assessing the relationship between levels of self-esteem and attributional styles for failure experience, Ickes and Layden (1978) obtained data which clearly indicated that the tendency to make negative self-attributions (i.e., internal attributions for failure) was closely related to low self-esteem. While situational factors and other latent negative attitudes (i.e., Beck's cognitive triad) may also constitute a source of influence, lowered self-esteem appears to be the most important antecedent of these type of attributions.

In sum, there is emerging evidence that characterological information is used and then depressogenic attributions are made following repeated experience with stressful life events. Such frequent exposure to bad events is believed to lead the person to infer, on the basis of a covariation analysis (cf. Kelley, 1967, 1973), that something about himself caused the events. Similarly, the level of self-esteem is believed to be important in determining whether a person accounts for personal negative events in terms of personal causes. Evidence in support of this observation has been provided by a series of important studies carried out by Ickes and Layden (1978). They reported results which clearly suggested that low self-esteem may be at the origin of depressives' maladaptive attributions. Although other personality and situational factors may also influence when depressogenic attributions are made, lowered self-esteem and frequent experience with bad events emerge as the most important determinant of these type of attributions.

In conclusion, the present research produced a number of findings concerning the theoretical status of negative self-attributions, the nature of the relationship they entertain with depression, and their antecedent conditions. The results obtained endorse the learned helplessness claim that internal attributions for personal negative outcomes are important in the aetiology and development of depressive and helplessness phenomena (e.g., Abramson et al., 1978; Seligman

et al., 1979). In particular, there was a clear indication that the attributional strategies that depressives tend to adopt when faced with bad outcomes may well be at the cause of their typical symptomatology and complaints. Overall, the present research showed that negative self-attributions elicit depressive symptoms, and these attributions are based on personal or characterological information made salient by such factors as low self-esteem, frequent experience with negative life events, and depressed mood.

CHAPTER TEN

A MODEL FOR THE DEVELOPMENT  
OF A DEPRESSIVE DISORDER

In Figure 10 a proposed positive feedback model of the development of a depressive disorder is outlined. The model encapsulates results that emerged from the present research programme - it outlines the factors that have been shown to play a critical role in the genesis and development of depressive disorders.

Like the reformulated learned helplessness model (Abramson, Seligman, and Teasdale, 1978; Miller and Norman, 1979), the present one emphasises the importance of attributions in generating and shaping depressive symptoms and disorders. In particular, the present model assumes that the types of attributions displayed to account for personal negative outcomes are crucial in determining whether depression develops. However, unlike the learned helplessness model, the present one gives explicit attention to the factors that influence the sort of attributions people make for unpleasant experiences. In addition to specifying the determinants of depressogenic attributions, the model provides a basis for understanding how depressive symptoms unfold. This, according to the present attributional framework, occurs as a result of a continuous interplay among the factors outlined in Figure 10. Complex though it is, this multifactorial view of the development and maintenance of depression is more in keeping with the complex reality of the phenomenon of depression (e.g., Akiskal and McKinney, 1975).

But the central claim of the present model is that level of

self-esteem and prior experience with stressful life events (i.e., frequency of exposure to life stressors) are critical in determining whether depressogenic attributions will be displayed and whether depressive symptoms will be elicited. The evidence in support of this claim is briefly examined below.

Determinants of depressogenic attributions. Perhaps a major weakness of the Abramson et al. reformulation of the learned helplessness model lies in its failure to specify the antecedent conditions of attributions for failure or uncontrollability. As Wortman and Dintzer (1978) pointed out, this problem could seriously undermine the predictive power of the model. The present research does not claim to resolve completely the issue, but it may offer a lead in that direction.

As was discussed earlier, there is a possibility that depressogenic attributions are formulated as a result of repeated experience with life stressors. A recent study by Peterson, Schwartz, and Seligman (1981) reported data that confirmed this possibility. They found evidence that frequent exposure to negative life events often leads people to use characterological information and then display depressogenic attributions for personal negative events. Of course this notion that negative self-attributions may be based on prior experience with life stressors is not new. Indeed, learned helplessness theorists and investigators (e.g., Klein et al., 1976; Miller

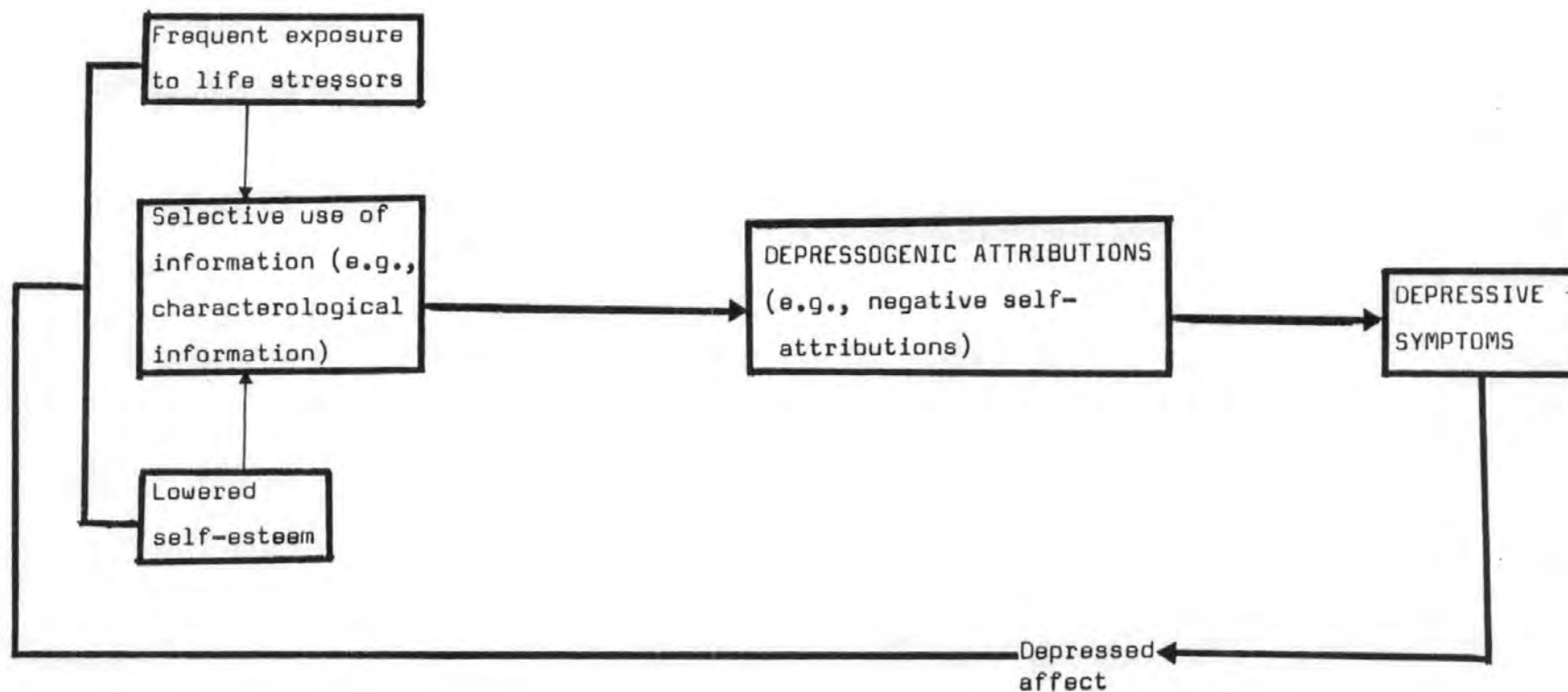


Figure 10. Proposed positive feedback model of the development of a depressive disorder.

and Norman, 1979) have argued that helplessness training can change one's attributions for uncontrollability or helplessness from external to internal or personal factors. In fact, such attributional shift is regarded as necessary for the generalization of helplessness and hopelessness.

Similarly, the notion that low self-esteem may be an important antecedent of negative self-attributions is not new.

Theorists of depression such as Beck (1967) and Bibring (1953) have for some time argued that low self-esteem provides a fertile soil for the manifestation of all sorts of depressive cognitions including causal cognitions. Empirical evidence for this notion has been provided by a series of studies recently carried out by Ickes and Layden (1978). There was a clear indication from their work that lowered self-esteem may be a contributory factor to the development and the formulation of depressogenic attributions.

Overall, there is emerging evidence that frequent experience with bad life events leads people to display negative self-attributions. Empirical evidence that such attributions may also be related to low self-esteem is also accumulating.

Negative self-attributions and depressive symptoms. Ample evidence was obtained from the present work that negative self-attributions have an aetiological status in depression - that is, there was a clear indication from the evidence presented in earlier chapters that these type of attributions are important in the causation of depression. Consistent



with Seligman et al. (1979) attributional analysis of depressive symptoms, it was found that only negative self-attributions (i.e., the tendency to attribute failure internally - to one's character) elicited depressive correlates or symptoms. External attributions of failure, in contrast, appeared to exert a prophylactic effect. And as such they could be said to be inconsistent with depression.

Further support for the aetiological importance of negative self-attributions has been provided by a recent correlational study. In an important study, Golin and his colleagues (1981) obtained data which clearly showed that these type of attributions are closely associated with depression. More importantly, however, is their finding that these attributions predate depression. The Golin et al. finding that the typical symptomatology of depressives may be related to their exaggerated tendency to make negative self-attributions further attests to the aetiological relevance and importance of these attributions. In short, clear evidence emerged from both the present work and that of others that negative self-attributions have an aetiological status in depression, and these attributions may be at the basis of many symptoms and deficits that characterise depressed people.

Overall then, the model presented here proposes evidence for the type of attributions that induce depressive symptoms, and the factors that lead people to display these kind of attributions. This model should be seen as a first step towards a

conceptual framework for understanding how a depressive disorder develops.

As noted earlier, this model represents more of a summary of what emerged from the present work about the relationship between causal attribution and depression, than a new theory about the genesis and development of depressive phenomena. Nevertheless, limited though it is the model could make a contribution to the literature on depression. Indeed, unlike the Abramson et al. attributional model, the present one is relatively precise about the antecedent conditions of depressogenic attributions. In doing so the present model allows a specific prediction: Given a typical helpless situation (i.e., uncontrollable situation), only a person with lowered self-esteem and/or with prior experience with life stressors will exhibit depressogenic attributions and thus develop depression. In contrast, a person enjoying a high level of self-esteem and/or a history of success or control will account for failure (loss of control) more in terms of external or behavioural causes than in terms of stable personal causes.

In addition to providing a means for identifying people who are likely to make depressogenic attributions for their difficulties, the present model could have a role in structuring and explaining the results from the present research programme. Indeed, another benefit of the proposed model is that it can serve as a guide for analysing and explaining the results per-

taining to the relationship between causal attribution and depression. In addition to accounting for the results concerning the depressogenic effects of attributions, the model may account for the biasing influence of depressed mood or affect on attributions of causality.

Finally, the model could also explain why negative life events often lead to depression (e.g., Brown and Harris, 1978; Paykel, 1974). The present model suggests that it is by inducing depressogenic attributions in people (i.e., changing attributions from external causes to stable personal causes) that negative life events generate depressive disorders. Although personality or vulnerability factors may also mediate the effects of stressful events, causal cognitions appear to be the mechanism through which these types of events cause depression.

In conclusion, the results that emerged from the present research suggest that the typical symptomatology of depressives could be understood in terms of the attributions they tend to make for their personal difficulties and deficiencies. This research supports the idea that negative self-attributions are important in the causation of depression. To some extent, the results endorse the Abramson et al.'s view that helplessness and depressive deficits are shaped by attributions. In particular there was a clear indication from the evidence presented in earlier chapters that depression develops in individuals who have an exaggerated tendency to make negative

self-attributions. While future research will be required to elaborate and refine the ideas presented here, it is clear that the present work together with the model which summarises it provide a basis for a theoretical understanding of the genesis and development of depressive disorders.

APPENDIX A

ASSESSMENT SCALES

## APPENDIX A1

### DEPRESSION SCALES

1. BECK DEPRESSION INVENTORY (BDI)
2. Multiple Affect Adjective Check List (MAACL)

B D I

CONFIDENTIAL

Name ..... Age ..... Sex .....

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY. Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all statements in each group before making your choice.

1.    0 I do not feel sad.  
      1 I feel sad.  
      2 I am sad all the time and I can't snap out of it.  
      3 I am so sad or unhappy that I can't stand it.
2.    0 I am not particularly discouraged about the future.  
      1 I feel discouraged about the future.  
      2 I feel I have nothing to look forward to.  
      3 I feel that the future is hopeless and that things cannot improve.
3.    0 I do not feel like a failure.  
      1 I feel I have failed more than the average person.  
      2 As I look on my life, all I can see is a lot of failures.  
      3 I feel I am a complete failure as a person.
4.    0 I get as much satisfaction out of things as I used to.  
      1 I don't enjoy things the way I used to.  
      2 I don't get real satisfaction out of anything any more.  
      3 I am dissatisfied or bored with everything.
5.    0 I don't feel particularly guilty.  
      1 I feel guilty a good part of the time.  
      2 I feel quite guilty most of the time.  
      3 I feel guilty all the time.
6.    0 I don't feel I am being punished.  
      1 I feel I may be punished.  
      2 I expect to be punished.  
      3 I feel I am being punished.
7.    0 I don't feel disappointed in myself.  
      1 I am disappointed in myself.  
      2 I am disgusted with myself.  
      3 I hate myself.
8.    0 I don't feel I am any worse than anybody else.  
      1 I am critical of myself for my weaknesses or mistakes.  
      2 I blame myself all the time for my faults.  
      3 I blame myself for everything bad that happens.

9. 0 I don't have any thoughts of killing myself.  
1 I have thoughts of killing myself, but I would not carry them out.  
2 I would like to kill myself.  
3 I would kill myself if I had the chance.
10. 0 I don't cry any more than usual.  
1 I cry more than I used to.  
2 I cry all the time now.  
3 I used to be able to cry, but now I can't cry even though I want to.
11. 0 I am no more irritated now than I ever am.  
1 I get annoyed or irritated more easily than I used to.  
2 I feel irritated all the time now.  
3 I don't get irritated at all by the things that used to irritate me.
12. 0 I have not lost interest in other people.  
1 I am less interested in other people than I used to be.  
2 I have lost most of my interest in other people.  
3 I have lost all of my interest in other people.
13. 0 I make decisions about as well as I ever could.  
1 I put off making decisions more than I used to.  
2 I have greater difficulty in making decisions than before.  
3 I can't make decisions at all any more.
14. 0 I don't feel I look any worse than I used to.  
1 I am worried that I am looking old or unattractive.  
2 I feel that there are permanent changes in my appearance that make me look unattractive  
3 I believe that I look ugly.
15. 0 I can work about as well as before.  
1 It takes an extra effort to get started at doing something.  
2 I have to push myself very hard to do anything.  
3 I can't do any work at all.
16. 0 I can sleep as well as usual.  
1 I don't sleep as well as I used to.  
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.  
3 I wake up several hours earlier than I used to and cannot get back to sleep.
17. 0 I don't get more tired than usual.  
1 I get tired more easily than I used to.  
2 I get tired from doing almost anything.  
3 I am too tired to do anything.
18. 0 My appetite is no worse than usual.  
1 My appetite is not as good as it used to be.  
2 My appetite is much worse now.  
3 I have no appetite at all any more.
19. 0 I haven't lost much weight, if any, lately.  
1 I have lost more than 5 pounds.  
2 I have lost more than 10 pounds.  
3 I have lost more than 15 pounds.  
I am purposely trying to lose weight by eating less. Yes.... No ....



20. 0 I am no more worried about my health than usual.  
1 I am worried about physical problems such as aches and pains;  
or upset stomach; or constipation.  
2 I am very worried about physical problems and it's hard to think  
of much else.  
3 I am so worried about my physical problems, that I can't think  
about anything else.
21. 0 I have not noticed any recent change in my interest in sex.  
1 I am less interested in sex than I used to be.  
2 I am much less interested in sex now.  
3 I have lost interest in sex completely.

MACCL

INSTRUCTIONS

On this sheet you will find words which describe different kinds of moods and feelings. Mark an X in the box beside the words which describe how you feel now - at this moment. Some of the words may sound alike, but we want you to check all the words that describe your feelings. Work rapidly.

1.	<input type="checkbox"/>	Active	27.	<input type="checkbox"/>	Co-operative	53.	<input type="checkbox"/>	Gentle
2.	<input type="checkbox"/>	Adventurous	28.	<input type="checkbox"/>	Critical	54.	<input type="checkbox"/>	Glad
3.	<input type="checkbox"/>	Affectionate	29.	<input type="checkbox"/>	Cross	55.	<input type="checkbox"/>	Gloomy
4.	<input type="checkbox"/>	Afraid	30.	<input type="checkbox"/>	Cruel	56.	<input type="checkbox"/>	Good
5.	<input type="checkbox"/>	Agitated	31.	<input type="checkbox"/>	Daring	57.	<input type="checkbox"/>	Good-natured
6.	<input type="checkbox"/>	Agreeable	32.	<input type="checkbox"/>	Desperate	58.	<input type="checkbox"/>	Grim
7.	<input type="checkbox"/>	Aggressive	33.	<input type="checkbox"/>	Destroyed	59.	<input type="checkbox"/>	Happy
8.	<input type="checkbox"/>	Alive	34.	<input type="checkbox"/>	Devoted	60.	<input type="checkbox"/>	Healthy
9.	<input type="checkbox"/>	Alone	35.	<input type="checkbox"/>	Disagreeable	61.	<input type="checkbox"/>	Hopeless
10.	<input type="checkbox"/>	Amiable	36.	<input type="checkbox"/>	Discontented	62.	<input type="checkbox"/>	Hostile
11.	<input type="checkbox"/>	Amused	37.	<input type="checkbox"/>	Discouraged	63.	<input type="checkbox"/>	Impatient
12.	<input type="checkbox"/>	Angry	38.	<input type="checkbox"/>	Disgusted	64.	<input type="checkbox"/>	Incensed
13.	<input type="checkbox"/>	Annoyed	39.	<input type="checkbox"/>	Displeased	65.	<input type="checkbox"/>	Indignant
14.	<input type="checkbox"/>	Awful	40.	<input type="checkbox"/>	Energetic	66.	<input type="checkbox"/>	Inspired
15.	<input type="checkbox"/>	Bashful	41.	<input type="checkbox"/>	Enraged	67.	<input type="checkbox"/>	Interested
16.	<input type="checkbox"/>	Bitter	42.	<input type="checkbox"/>	Enthusiastic	68.	<input type="checkbox"/>	Irritated
17.	<input type="checkbox"/>	Blue	43.	<input type="checkbox"/>	Fearful	69.	<input type="checkbox"/>	Jealous
18.	<input type="checkbox"/>	Bored	44.	<input type="checkbox"/>	Fine	70.	<input type="checkbox"/>	Joyful
19.	<input type="checkbox"/>	Calm	45.	<input type="checkbox"/>	Fit	71.	<input type="checkbox"/>	Kindly
20.	<input type="checkbox"/>	Cautious	46.	<input type="checkbox"/>	Forlorn	72.	<input type="checkbox"/>	Lonely
21.	<input type="checkbox"/>	Cheerful	47.	<input type="checkbox"/>	Frank	73.	<input type="checkbox"/>	Lost
22.	<input type="checkbox"/>	Clean	48.	<input type="checkbox"/>	Free	74.	<input type="checkbox"/>	Loving
23.	<input type="checkbox"/>	Complaining	49.	<input type="checkbox"/>	Friendly	75.	<input type="checkbox"/>	Low
24.	<input type="checkbox"/>	Contented	50.	<input type="checkbox"/>	Frightened	76.	<input type="checkbox"/>	Lucky
25.	<input type="checkbox"/>	Contrary	51.	<input type="checkbox"/>	Furious	77.	<input type="checkbox"/>	Mad
26.	<input type="checkbox"/>	Cool	52.	<input type="checkbox"/>	Gay	78.	<input type="checkbox"/>	Mean

79. ☐ Meek  
 80. ☐ Merry  
 81. ☐ Mild  
 82. ☐ Miserable  
 83. ☐ Nervous  
 84. ☐ Obliging  
 85. ☐ Offended  
 86. ☐ Outraged  
 87. ☐ Panicky  
 88. ☐ Patient  
 89. ☐ Peaceful  
 90. ☐ Pleased  
 91. ☐ Pleasant  
 92. ☐ Polite  
 93. ☐ Powerful  
 94. ☐ Quiet  
 95. ☐ Reckless  
 96. ☐ Rejected

97. ☐ Rough  
 98. ☐ Sad  
 99. ☐ Safe  
 100. ☐ Satisfied  
 101. ☐ Secure  
 102. ☐ Shaky  
 103. ☐ Shy  
 104. ☐ Soothed  
 105. ☐ Steady  
 106. ☐ Stubborn  
 107. ☐ Stormy  
 108. ☐ Strong  
 109. ☐ Suffering  
 110. ☐ Sullen  
 111. ☐ Sunk  
 112. ☐ Sympathetic  
 113. ☐ Tame  
 114. ☐ Tender

115. ☐ Tense  
 116. ☐ Terrible  
 117. ☐ Terrified  
 118. ☐ Thoughtful  
 119. ☐ Timid  
 120. ☐ Tormented  
 121. ☐ Understanding  
 122. ☐ Unhappy  
 123. ☐ Unsociable  
 124. ☐ Upset  
 125. ☐ Vexed  
 126. ☐ Warm  
 127. ☐ Whole  
 128. ☐ Wild  
 129. ☐ Wilful  
 130. ☐ Wilted  
 131. ☐ Worrying  
 132. ☐ Young

APPENDIX A2

ATTRIBUTION QUESTIONNAIRE

1. ATTRIBUTION QUESTIONNAIRE (SUCCESS CONDITION)
2. ATTRIBUTION QUESTIONNAIRE (FAILURE CONDITION)

## ATTRIBUTION QUESTIONNAIRE

### INSTRUCTIONS

How a person does on tasks like the one you have just completed depends on a number of factors.

On some occasions the task is an easy one. Even people who are not very skilful, or who don't try very hard, are successful. On harder tasks these people might not do so well.

Some people succeed mainly because they apply themselves to the task and try very hard. In this way they are sometimes able to make up for any lack of skill or for bad luck. Even if the task is difficult, such people may do well. Were they to lose interest and not try so hard, they would probably not do so well.

Other people are successful because they are just lucky enough - they happen to hit upon the correct answers largely by chance. They therefore do well even if they are not particularly skilful, or don't try too hard.

Some others succeed because they have skill and ability. These people don't really have to try very hard even on fairly difficult tasks. And good luck isn't really involved for these people. Given another task they would probably do just as well because they have good ability.

Consider the result that you have just obtained on the task, and indicate

1. To what extent do you think your success was because you tried very hard (effort)?

2. To what extent do you think your success was because the task was easy?

3. To what extent do you think your success was because you were lucky?

4. To what extent do you think your success was because you have skill and ability?

definitely not  
a cause of my  
success

1 2 3 4 5 6 7 8 9

definitely a  
cause of my  
success

## ATTRIBUTION QUESTIONNAIRE

### INSTRUCTIONS

How a person does in tasks like the one you have just completed depends on a number of factors.

On some occasions the task is a difficult one. Even people who are very skilful, or who try very hard, are not successful - on easier tasks these people might do well.

Some people fail mainly because they don't try very hard. Even if the task is easy, such people may not do well. But if they try very hard they would probably do well.

Often people are unsuccessful because they are just not lucky enough. They therefore fail even if they are particularly skilful, or try too hard.

Some often fail because they lack skill and ability. These people must try very hard even on easy tasks. And bad luck isn't involved for these people. Given another task they would probably not do well because they lack ability.

Consider the result that you have just obtained on the task, and

indicate on the scale below:

1. To what extent do you think your failure was because you did not try very hard (lack of effort)?

definitely not a cause of my failure	L-----J	1	2	3	4	5	6	7	8	9	definitely a cause of my failure
--	---------	---	---	---	---	---	---	---	---	---	--

2. To what extent do you think your failure was because the task was difficult?

definitely not a cause of my failure	L-----J	1	2	3	4	5	6	7	8	9	definitely a cause of my failure
--	---------	---	---	---	---	---	---	---	---	---	--

3. To what extent do you think your failure was because you were unlucky?

definitely not a cause of my failure	L-----J	1	2	3	4	5	6	7	8	9	definitely a cause of my failure
--	---------	---	---	---	---	---	---	---	---	---	--

4. To what extent do you think your failure was because you lack skill and ability?

definitely not a cause of my failure	L-----J	1	2	3	4	5	6	7	8	9	definitely a cause of my failure
--	---------	---	---	---	---	---	---	---	---	---	--



APPENDIX A3

ATTRIBUTIONAL STYLE QUESTIONNAIRE

## ATTRIBUTION QUESTIONNAIRE

### INSTRUCTIONS

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.

Answer some questions about the cause. 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 some questions about the cause.
5. Go on to the next situation.

YOU HAVE BEEN PROMOTED IN YOUR JOB

1. Write down one major cause .....

2. Is the cause of your promotion due to something about you or something about other people or circumstances? (Circle one number)

Totally due to  
other people or  
circumstances

Totally due  
to me

1 2 3 4 5 6 7 8 9

3. In the future when promoted in your job, will this cause again be present? (Circle one number)

Will never again  
be present

Will always  
be present

1 2 3 4 5 6 7 8 9

4. Is the cause something that just influences gaining promotion, or does it also influence other areas of your life? (Circle one number)

Influences just  
this particular  
situation

Influences  
all situations  
in my life

1 2 3 4 5 6 7 8 9

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

Not at all  
important

Extremely  
important

1 2 3 4 5 6 7 8 9

YOU GO OUT ON A DATE, AND IT GOES BADLY

1. Write down one major cause .....

2. Is the cause of your unsuccessful date due to something about you  
or something about other people or circumstances? (Circle one number)

Totally due to other people or circumstances									Totally due to me
--	--	--	--	--	--	--	--	--	----------------------

1	2	3	4	5	6	7	8	9
---	---	---	---	---	---	---	---	---

3. In the future when going out on a date, will this cause again be  
present? (Circle one number)

Will never again be present									Will always be present
--------------------------------	--	--	--	--	--	--	--	--	---------------------------

1	2	3	4	5	6	7	8	9
---	---	---	---	---	---	---	---	---

4. Is the cause something that just influences going out on a date, or does  
it also influence other areas of your life? (Circle one number)

Influences just this particular situation									Influences all situations in my life
---	--	--	--	--	--	--	--	--	--

1	2	3	4	5	6	7	8	9
---	---	---	---	---	---	---	---	---

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

Not at all important									Extremely important
-------------------------	--	--	--	--	--	--	--	--	------------------------

1	2	3	4	5	6	7	8	9
---	---	---	---	---	---	---	---	---

YOU HAVE BEEN LOOKING FOR A JOB UNSUCCESSFULLY FOR SOME TIME

1. Write down one major cause .....

2. 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

Totally due  
to me

1 2 3 4 5 6 7 8 9

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

Will never again  
be present

Will always  
be present

1 2 3 4 5 6 7 8 9

4. 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

Influences  
all situations  
in my life

1 2 3 4 5 6 7 8 9

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 8 9

YOU MEET A FRIEND WHO COMPLIMENTS YOU ON YOUR APPEARANCE

1. Write down one major cause .....

2. Is the cause of this event due to something about you or something about other people or circumstances? (Circle one number)

Totally due to  
other people or  
circumstances

Totally due  
to me

1 2 3 4 5 6 7 8 9

3. In the future when meeting friends, will this cause again be present?  
(Circle one number)

Will never again  
be present

Will always  
be present

1 2 3 4 5 6 7 8 9

4. Is the cause something that just influences this event or does it also influence other areas of your life? (Circle one number)

Influences just  
this particular  
situation

Influences all  
situations in  
my life

1 2 3 4 5 6 7 8 9

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

Not at all  
important

Extremely  
important

1 2 3 4 5 6 7 8 9

APPENDIX B

RAW DATA

## RAW DATA

### KEY

BDI	=	Beck Depression Inventory
SDS	=	Self-rating Depression Scale
MAACL	=	Multiple Affect Adjective Check List
MAACL Dep.	=	MAACL depression scale
MAACL Anx.	=	MAACL anxiety scale
MAACL Hos.	=	MAACL hostility scale
IAS	=	Internal Attribution of Success
IAF	=	Internal Attribution of Failure
EAS	=	External Attribution of Success
EAF	=	External Attribution of Failure
SO	=	Success Only
FO	=	Failure Only
A	=	Ability Attribution
E	=	Effort Attribution
T	=	Task Attribution
L	=	Luck Attribution
D	=	Depressed
ND	=	Nondepressed
Cs	=	Consensus information
Cc	=	Consistency information
Dt	=	Distinctiveness information
S	=	Success
F	=	Failure



# EXPERIMENT 1

I A S

E A S

S O

B D I	Pre. M A A C L			ATTRIBUTIONS				Post. M A A C L		
	Dep.	Anx.	Hos.	A	E	T	L	Dep.	Anx.	Hos.
8	13	4	7	4	5	5	2	10	5	6
16	13	6	12	6	5	2	2	10	7	11
13	13	6	6	4	5	3	2	11	7	8
15	15	8	10	4	5	4	3	8	8	11
10	20	8	10	5	6	5	4	18	8	9
16	19	10	7	4	7	6	4	14	9	10
12	5	4	9	5	6	3	3	4	4	9
10	17	9	8	3	4	3	5	15	8	9
6	16	9	12	5	7	3	6	19	9	11
106	131	64	81	40	50	34	31	109	65	84
11	13	9	7	5	5	7	2	12	7	9
15	17	11	13	3	4	7	2	19	11	15
16	18	14	13	3	5	5	5	18	8	12
7	9	2	3	6	6	6	4	11	3	4
9	10	9	6	3	5	5	1	12	8	5
12	20	10	13	1	4	3	1	12	8	5
14	21	9	10	2	5	3	2	19	7	10
10	17	11	10	4	7	3	5	19	7	11
10	8	3	5	6	2	1	1	7	3	8
104	133	78	80	33	43	40	19	129	62	79
12	17	4	8	4	3	4	2	16	4	7
13	17	9	9	1	1	7	2	18	9	10
16	18	8	12	5	5	4	3	18	8	11
14	16	12	10	2	2	4	1	20	11	15
8	20	10	9	3	6	4	6	20	8	10
15	15	8	9	4	5	4	4	16	6	8
7	14	9	11	5	6	3	2	15	9	10
6	14	3	7	7	2	4	1	12	4	5
8	18	8	7	4	7	1	2	18	7	4
99	149	71	82	35	37	35	23	153	66	80

EXPERIMENT 1 (continued)

B D I	Pre. M A A C L			ATTRIBUTIONS				Post. M A A C L		
	Dep.	Anx.	Hos.	A	E	T	L	Dep.	Anx.	Hos.
6	3	3	5	3	3	3	1	7	5	5
14	13	12	13	7	1	1	1	19	16	14
16	12	9	10	5	4	1	1	15	7	10
13	14	10	12	4	4	3	2	18	8	12
12	6	4	3	3	1	1	1	14	9	11
12	14	3	3	2	1	2	2	29	17	16
13	21	12	12	5	1	1	1	27	14	12
6	16	8	10	3	2	4	5	19	10	15
6	17	9	9	5	6	1	5	17	10	9
98	116	70	77	37	23	17	19	165	96	104
13	14	10	7	2	3	4	3	12	7	8
9	12	10	9	4	2	4	3	12	7	10
11	18	4	7	2	2	5	2	16	7	11
14	18	10	11	6	2	6	3	17	10	9
12	17	11	10	4	2	6	2	19	10	12
11	13	8	7	4	3	5	3	18	9	9
9	15	8	6	6	1	6	2	11	8	6
10	8	7	6	2	3	3	3	9	8	9
10	8	5	3	1	1	3	1	8	6	5
99	123	73	66	31	19	42	22	122	72	79
12	15	11	9	5	5	4	4	18	8	11
13	7	2	8	7	6	6	1	20	6	12
7	13	9	6	6	2	5	4	14	7	7
10	14	6	8	2	6	4	1	15	6	10
16	18	10	12	5	5	3	3	21	13	12
11	17	6	10	5	2	4	3	19	6	9
23	12	7	8	4	6	4	3	23	9	13
9	11	3	7	6	2	2	1	15	7	11
9	16	9	9	5	2	5	1	17	8	9
110	123	63	77	45	36	37	21	162	70	94

# EXPERIMENT 2

I A S

E A S

S D S	Pre. M A A C L			ATTRIBUTIONS				Post. M A A C L			ANAGRAMS	
	Anx.	Hos.	Dep.	A	E	T	L	Anx.	Hos.	Dep.	F S	Lat.
32	8	12	19	3	5	4	3	7	12	16	4	30.2
36	11	11	16	7	4	3	6	10	12	16	1	23.75
41	10	11	18	8	3	3	6	2	1	4	1	10.70
44	8	11	12	6	5	7	5	7	9	14	0	5.00
29	3	4	8	6	6	5	2	5	10	14	2	-
28	7	9	16	4	5	3	7	5	9	14	2	33.5
36	5	4	8	5	5	5	1	5	5	10	7	46.05
21	12	11	14	6	2	3	1	7	10	15	5	38.45
39	10	8	20	6	7	5	4	12	4	14	-	-
35	11	9	16	5	7	4	3	10	11	15	2	8.50
341	85	90	147	56	49	42	38	70	83	132	24	196.00
35	8	10	15	5	8	1	9	8	8	15	13	70.90
25	5	9	14	8	8	7	2	5	8	15	0	-
42	12	12	18	5	3	3	8	7	11	16	2	23.70
31	2	6	10	2	4	6	7	10	11	20	5	39.85
37	4	4	6	2	7	4	6	4	2	4	1	-
43	10	6	20	5	3	4	7	9	6	13	1	26.40
31	6	6	14	5	4	2	4	5	6	11	5	38.95
38	5	6	18	5	3	4	7	7	8	17	6	49.80
24	7	10	14	8	6	6	1	9	8	16	6	45.15
26	4	4	6	5	5	8	4	5	4	7	0	12.95
332	63	73	135	50	51	45	55	69	72	134	39	307.70

Note. Anx. = anxiety; Hos. = hostility; Dep. = depression; FS = failure to solve; Lat. = latency; A = ability attribution; E = effort attribution; T = task attribution; L = luck attribution.

EXPERIMENT 2 (continued)

S D S	Pre. M A A C L			ATTRIBUTIONS				Post. M A A C L			ANAGRAMS	
	Anx.	Hos.	Dep.	A	E	T	L	Anx.	Hos.	Dep.	F S	Lat.
25	2	4	2	5	3	7	5	5	9	11	3	28.90
30	9	8	17	6	3	7	2	9	11	18	1	23.65
27	7	8	18	1	8	3	1	8	10	19	0	15.85
33	7	7	8	2	7	4	3	9	6	8	10	69.55
31	6	6	15	3	3	6	1	6	9	17	7	47.35
32	4	6	7	9	9	9	9	6	9	10	3	34.90
-	12	12	16	2	6	1	1	10	13	18	5	39.75
29	9	10	11	3	3	3	6	8	9	20	9	65.55
49	8	8	16	5	9	1	1	10	11	21	5	40.65
28	10	10	20	9	1	1	1	10	11	23	10	61.80
284	74	79	130	45	52	42	30	81	98	165	53	427.95
26	4	9	15	5	6	8	4	4	10	15	3	21.90
41	7	8	14	3	7	9	4	7	10	16	0	13.85
32	5	5	4	7	3	8	3	9	7	8	1	26.05
34	13	10	16	7	3	7	2	14	15	23	10	66.25
44	9	8	17	2	2	4	1	10	10	17	0	5.45
42	12	13	24	6	7	3	5	11	15	19	7	53.75
33	5	4	7	7	5	3	1	9	11	7	2	16.80
28	14	8	15	5	3	6	1	8	9	15	8	-
25	9	10	17	9	2	9	1	9	11	15	2	22.50
26	5	15	8	3	2	3	1	6	9	14	9	56.85
331	83	90	137	54	40	60	23	87	107	149	42	283.40

Note. Anx. = anxiety; Hos. = hostility; Dep. = depression; FS = failure to solve; Lat. = latency; A = ability attribution; E = effort attribution; T = task attribution; L = luck attribution.

# EXPERIMENT 3

IAF/D

B D I	Age	MAACL 1	ATTRIBUTIONS			MAACL 2	Exp.	P S
			Int.	Sta.	Glo.			
9	19	10	6	6	4	16	2	57
10	21	11	3	5	6	12	4	51
12	19	11	6	5	3	20	3	47
9	18	10	8	8	3	17	3	54
13	18	24	9	9	9	24	1	43
10	20	20	6	6	5	15	4	47
11	19	10	6	6	4	13	3	48
9	22	11	6	5	3	11	5	59

83	156	107	50	50	37	128	25	406
----	-----	-----	----	----	----	-----	----	-----

EAF/D

13	19	11	5	6	7	22	4	50
15	20	11	4	4	7	10	6	52
11	21	5	3	9	9	7	7	52
11	24	22	7	7	6	17	1	44
9	19	17	7	5	6	15	5	54
15	19	26	3	3	6	19	5	43
10	20	18	5	7	6	14	5	53
13	21	10	3	7	2	16	3	57

97	163	120	37	48	49	120	36	405
----	-----	-----	----	----	----	-----	----	-----

F O / D

12	20	12	7	6	6	18	5	50
16	18	25	5	6	8	21	4	54
9	18	16	8	8	4	19	5	49
10	19	9	8	6	3	12	5	48
10	20	12	7	7	7	15	5	61
15	24	26	7	9	7	24	3	49
18	20	18	8	8	7	16	5	35
11	20	14	8	5	8	17	2	43

101	159	132	58	55	50	142	34	389
-----	-----	-----	----	----	----	-----	----	-----

Note. Int. = internality; Sta. = stability; Glo. = globality; PS = psychomotor speed; Exp. = expectation.

EXPERIMENT 3 (continued)

B D I	Age	MAACL 1	ATTRIBUTIONS			MAACL 2	Exp.	P S
			Int.	Sta.	Glo.			
6	18	18	8	8	1	24	1	43
3	19	4	3	5	6	16	1	42
2	18	10	7	6	5	24	4	53
3	18	7	5	6	3	19	2	50
3	20	11	8	9	9	15	3	48
4	22	6	8	7	6	8	5	56
6	21	11	5	4	3	14	5	51
1	19	12	4	8	6	20	3	42
28	155	79	48	53	39	140	24	385
3	18	13	6	5	5	16	3	60
5	19	7	3	6	2	14	5	60
4	19	8	3	6	8	16	5	66
5	28	4	1	9	6	8	5	61
4	20	14	6	4	6	12	5	59
7	29	14	6	6	6	14	6	60
4	19	7	3	7	8	13	4	49
7	50	4	8	6	7	7	8	64
39	202	71	36	49	48	100	41	479
0	21	3	6	8	6	3	4	49
3	20	9	4	7	3	18	5	49
8	18	2	6	8	7	2	4	52
2	19	17	7	9	7	16	6	35
5	28	8	3	2	1	11	6	48
4	38	17	2	2	2	15	6	47
4	18	16	3	3	6	19	4	48
3	19	6	4	5	3	5	5	46
29	181	78	35	44	35	89	40	374

Note. Int. = internality; Sta. = stability; Glo. = globality; PS = psychomotor speed; Exp. = expectation.

# EXPERIMENT 4

B D I	Age	A T T R I B U T I O N S								
		P P		I P		P N		I N		
		Int.	Ext.	Int.	Ext.	Int.	Ext.	Int.	Ext.	
D patients	18	46	1	1	1	8	9	1	9	1
	28	38	5	5	4	4	3	4	9	4
	30	42	5	4	3	3	5	5	4	1
	15	62	9	9	9	4	9	1	1	1
	18	45	2	2	2	8	9	1	9	1
	18	22	5	7	2	2	4	4	4	4
	20	31	6	5	6	5	7	5	4	5
	38	46	7	5	7	4	9	1	9	1
	16	30	7	5	7	5	7	5	1	9
	31	63	2	5	4	4	7	4	9	5
	32	22	9	1	6	5	5	6	2	4
264	447	58	49	51	52	74	37	61	36	
ND patients	6	21	7	4	7	5	2	1	5	5
	9	23	4	5	5	5	6	5	6	5
	13	45	5	5	5	5	5	5	4	5
	14	21	9	1	6	5	5	5	1	9
	4	31	5	3	7	5	5	5	1	9
	6	19	9	1	9	1	7	5	1	9
	4	21	6	5	5	5	2	5	5	5
	6	29	4	5	7	5	7	5	2	5
62	210	49	29	51	36	39	36	25	52	

Note. PP = Personal positive events; IP = Interpersonal positive events; PN = Personal negative events; IN = Interpersonal negative events; Int. = Internality; Ext. = Externality.

# EXPERIMENT 5

DEPRESSION

B D I	Age	M A A C L			P S	Negative events			Positive events		
		Dep.	Anx.			Int.	Sta.	Glo.	Int.	Sta.	Glo.
2	20	4	3	55		3.00	5.75	1.00	7.25	8.00	8.00
2	19	20	12	45		5.50	6.50	3.25	4.25	7.50	4.00
4	33	19	7	49		6.25	4.75	4.50	8.00	7.25	7.50
10	38	25	11	51		6.25	5.50	6.25	5.00	5.00	5.00
1	21	8	5	46		7.25	7.25	5.50	8.75	8.50	4.75
8	19	10	8	61		4.00	3.25	3.50	7.25	6.25	6.25
4	18	20	10	53		4.75	6.75	6.00	6.25	8.00	7.75
8	28	20	8	37		5.50	5.25	3.75	6.33	7.67	5.33
4	22	20	10	41		3.00	6.25	3.75	5.00	5.50	4.25
4	31	11	6	55		5.50	5.50	5.50	7.50	7.25	7.00
47	249	157	80	493		51.00	56.75	43.00	65.58	70.92	59.83

ELATION

5	21	6	6	64		4.00	6.75	5.00	6.75	8.25	8.75
8	18	13	8	50		6.75	3.50	2.75	4.00	6.25	4.50
5	20	12	8	50		4.50	6.00	3.50	7.75	7.00	4.75
8	21	8	3	48		4.25	5.00	6.50	9.00	7.50	8.25
8	20	4	4	59		5.50	4.50	2.50	6.75	7.50	7.25
8	26	11	4	62		5.50	3.50	2.25	7.75	6.25	5.00
6	20	7	5	55		6.00	6.25	5.50	4.75	5.25	5.50
7	19	5	4	46		2.25	3.00	2.50	6.50	6.75	5.50
7	22	3	5	57		4.25	6.75	5.50	6.25	6.75	6.00
3	28	3	3	47		5.50	5.25	6.50	5.50	8.25	8.25
65	215	72	50	538		48.50	50.50	42.50	65.00	63.75	63.75

Note. Dep. = depression; Anx. = anxiety; Int. = internality; Sta. = stability; Glo. = globality; P S = psychomotor speed.



# EXPERIMENT 6

S/DEPRESSION

B D I	M A A C L		P S	A T T R I B U T I O N S			
	Dep.	Anx.		A	E	T	L
0	13	7	55	7	8	6	1
4	17	9	42	5	7	3	3
1	24	14	60	3	4	3	1
1	19	11	63	3	7	7	1
5	13	8	45	6	7	7	6
3	15	12	42	3	7	6	2
5	20	6	51	3	3	7	1
4	11	7	50	7	4	7	1
23	132	74	408	37	47	46	16

S/ELATION

4	7	7	60	6	8	3	2
4	7	4	48	5	5	4	3
3	4	3	59	8	3	6	3
1	7	8	65	7	7	7	6
7	7	5	52	7	4	7	2
2	5	3	68	7	6	5	6
4	3	3	57	3	4	7	6
5	10	4	58	9	8	8	8
30	50	37	467	52	45	47	36

Note. Dep. = depression; Anx. = anxiety; PS = psychomotor speed; A = ability attribution; E = effort attribution; T = task attribution; L = luck attribution.

EXPERIMENT 6 (continued)

B D I	M A A C L		P S	A T T R I B U T I O N S			
	Dep.	Anx.		A	E	T	L
6	16	8	60	6	6	6	7
4	20	7	47	7	7	5	6
3	13	12	45	4	7	3	2
4	19	15	51	7	2	7	4
4	25	11	47	5	4	8	5
6	8	8	59	7	3	7	4
4	24	10	53	7	8	3	2
7	15	7	62	8	5	4	7
38	140	78	424	51	42	43	37
1	7	6	65	6	4	8	1
6	10	3	52	4	2	4	5
3	5	8	50	1	1	2	1
3	9	4	55	7	2	8	3
1	8	3	49	2	1	2	4
4	2	4	59	2	3	8	5
3	5	5	65	2	2	8	5
4	9	6	46	2	4	7	6
25	55	39	441	26	19	47	30

Note: Dep. = depression; Anx. = anxiety; PS = psychomotor speed; A = ability attribution; E = effort attribution; T = task attribution; L = luck attribution.

# EXPERIMENT 7

B D I	Age	MAACL	Positive events			Negative events			
			Cs	Cc	Dt	Cs	Cc	Dt	
DEPRESSED	13	21	18	9	9	8	9	9	10
	9	21	10	10	8.5	6.5	10	10	10
	12	22	25	11	10.5	7.0	10.5	10	10
	19	26	22	7.5	5.5	9.0	9.5	9.5	5.5
	13	19	12	8	5	5	9	6.5	7
	19	20	19	7.5	6	7	9.5	9	9.5
	9	20	17	8.5	8	4	9	8.5	8
	10	25	18	8.5	6	6.5	5	4	1.5
	9	19	14	10	3.5	3.5	11	8	8.5
	22	24	26	7	4	4	7.5	9.5	8.5
	10	20	8	11	9	3.5	7.5	9	8
	14	22	22	5.5	9.5	8.5	7	5.5	7
	9	24	6	6	6	7	7.5	8	7.5
	13	21	16	8	8.5	7.5	9	9	7
	10	20	14	8.5	7	7	11	10	9.5
191	324	247	126	106	94	127	121.5	117.5	
N. DEPRESSED	1	22	3	2	4.5	4.5	3	8.5	8
	4	18	8	8.5	8	10	10	8	7.5
	1	30	3	5.5	8.5	7	7.5	8.5	8
	3	20	9	5.5	6.5	6	4.5	10	9
	5	23	5	8.5	6	7.5	6	5.5	9.5
	1	30	7	3	6	5.5	4	6	6.5
	4	20	6	8.5	6.5	5	8	7.5	7
	6	19	14	4	4.5	4	3.5	4	4.5
	1	21	8	8.5	8	3.5	10.5	7	6.5
	6	21	4	8.5	9.5	4.5	10	3.5	4
	2	21	12	3.5	7.5	6	5.5	6	8
	6	21	12	8.5	8	7.5	10.5	10.5	8.5
	2	21	4	9	9	8	10	10	9
	2	19	16	8	9	3.5	9.5	8.5	4.5
	6	19	8	9	7	3.5	7	9	6.5
51	325	119	100.5	108.5	86	109.5	112.5	107	

Note. Cs = consensus; Cc = consistency; Dt = distinctiveness

## EXPERIMENT 8

D E P R E S S E D		N O N D E P R E S S E D	
B D I	Nb. of questions	B D I	Nb. of questions
10	4	1	6
14	3	1	4
11	7	3	5
14	5	4	6
14	5	3	10
17	3	4	9
10	4	6	13
14	2	1	7
9	4	1	10
14	3	3	6
127	40	27	76

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