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ATTRIBUTIONAL STYLE: THE RELATIONSHIP BETWEEN PARANOID SCHIZOPHRENIA/DISORDER AND DEPRESSION

By

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "Attributional Style: The Relationship Between Paranoid Schizophrenia/Disorder and Depression", submitted by Carmie Lester Candido in partial fulfillment of the requirements for the degree of Master of Science.

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ABSTRACT

The existence of a specific attributional style has been well documented for depressed subjects, but so far no particular attributional style has been established for paranoid individuals. There is evidence that has demonstrated that paranoid schizophrenia is different from the other subtypes of schizophrenia and has more in common with the paranoid disorders. Theorists have further postulated that paranoid schizophrenia may be a mechanism used to mask depressive illness. The present study was an exploratory study designed to examine the following research questions: (1) How are attributional style and self-esteem affected by individuals who are depressed, paranoid, or both? (2) What is the clinical relationship between paranoia and depression? and (3) Does paranoia substitute for depression in some cases?

Forty-five patients (22 female, 23 male) over the age of 18 with no recent history of substance abuse were selected from the psychiatric units of two city hospitals as well as a psychiatric day hospital. Subjects were divided into three equal groups: one group of nonpsychotic depressed patients and two groups of paranoid patients (diagnosis of paranoid schizophrenia or paranoid disorder). One of the two paranoid groups also manifested depressive symptoms whereas the other did not.

A psychiatric history was obtained from each patient and then patients were rated on the Hamilton Rating Scale for Depression and the Maine scale for paranoid and schizophrenic symptoms. All

subjects completed the Beck Depression Inventory, Attributional Style Questionnaire, Coopersmith Self-Esteem Inventory and the paranoid scale of the Minnesota Multiphasic Personality Inventory. Comparisons among the groups on all psychological and sociodemographic variables were made using a number of different statistical techniques.

The results indicated that the attributional dimensions for good events were better at discriminating among the three groups than were the attributional dimensions for bad events. Generally, paranoid individuals demonstrated an attributional style which was opposite to that of depressed patients. In addition, individuals who were both paranoid and depressed demonstrated an attributional style often in between individuals who were either only paranoid or only depressed. Correlational analysis suggested an inverse relationship between paranoia and depression; an inverse relationship between self-esteem and depression and a direct relationship between paranoia and self-esteem. These results are discussed in relation to previous studies of attributional style in depressed and paranoid individuals. In addition, theoretical explanations are offered for the differences in degree of paranoia and depression, delusional content and suicidal ideation found between the two paranoid groups which was reflected in their respective attributional styles. The issues of depression in schizophrenia, schizoaffective illness and negative symptoms are addressed in light of the current findings, along with other theoretical and clinical implications. Finally, recommendations are offered for future research.

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CHAPTER ONE

INTRODUCTION

Modern psychiatry has made great progress in the diagnosis and treatment of mental disorders over the past few decades. Much time has been invested in an attempt to develop diagnostic systems which will provide clinicians with highly specific and discrete criteria through which more accurate diagnoses can be made. One of the problems with diagnostic systems is that the research in which these systems are based attempts to further the philosophy of placing all forms of psychopathology into discrete categories. This philosophy may lead scientists, who are in search of very specific information, down blind alleys which eventually become dead ends. Under these circumstances, one is often left with a vast amount of information which functions to not only entice future researchers, but also cloud the facts sufficiently to prevent a full understanding of various disease processes. This is particularly true of schizophrenia, for in spite of decades of research, scientists continue to be puzzled by its etiology and generally poor prognosis. Moreover, clinicians are further puzzled when individual do not fit into established categories, but rather possess symptoms of two ostensibly different illnesses. Examples would include cases where paranoia and depression or paranoid schizophrenia and depression occur together respectively. Such circumstances can make the diagnostic process both difficult and confusing and consequently may hamper or lead to inadequate treatment regimens.

With respect to depression and paranoia, there is not only evidence to suggest that depressive and paranoid symptomatology may both be conceptualized as lying on continua of severity (cf. Gotlib, 1984; Romney, 1987, respectively), but also that they may actually lie on the same continuum (Schwartz, 1963). In their discussion of paranoid schizophrenia and mania, Brockington, Wainwright and Kendall (1980) have suggested that one should "consider these patients as part of a clinical spectrum or continuum, thus avoiding the fruitless search for imperceptible boundaries" (p. 81). This same philosophy may also hold true for paranoid and depressive illness.

On the surface, paranoia and depression appear to be composed of diametrically opposed symptoms. For example, while depressed individual tend to blame themselves for their problems and engage in self-devaluative thinking, paranoids through their feelings of victimization and persecution, tend to blame others or outside circumstances for their failures and difficulties (Aaronson, 1977; Heilbrum & Bronson, 1975). As the disease progresses, however, paranoia and depression have been shown to have a number of similarities which may bring them closer together phenomenologically. Some investigators (Zigler & Glick, 1984, 1986, 1988) have even suggested that paranoid schizophrenia may be another manifestation of an underlying depression. These authors have agreed with Meissner (1978) who stated that "paranoid patients can only relinquish their paranoid stance at the risk of encountering a severe depression" (p. 125). Meissner (1981) further argued that

the schizophrenic and paranoid dynamics are two separate and discriminable processes that operate independently from one another. In support of this notion is a relatively large and growing body of literature which suggests that "paranoid schizophrenia reflects the paranoid process but has little to do with schizophrenia" (Meissner, 1981, p. 156). At the centre of this body of literature exist issues concerned with the present nosology of paranoid disorders and paranoid schizophrenia. The primary focus of this line of research has been to establish the paranoid/nonparanoid dichotomy of schizophrenia based on the evidence from biological, cognitive, genetic and epidemiological investigations.

In this study, attributional theory has been invoked in order to evaluate the relationship between paranoia and depression. This is of special importance in view of Zigler's and Glick's contention that paranoid defenses mask underlying depression. While a particular attributional style has been relatively well established for depressives, not much work along these lines has been completed for paranoid individuals. Through the comparison of attributional style, self-esteem, and the levels of depression and paranoid symptomatology in individuals who are either depressed, paranoid, or paranoid and depressed, one would be able to address the hypothesized existence of the paranoid depressive continuum, as well as the study of depression in schizophrenia. Although a vast amount of research into the study of depression in schizophrenia has been generated, the majority of these investigations have focused on either identifying pharmaceutical causes for the depression or

explaining the depressive symptoms as being representative of yet another syndrome or subtype of schizophrenia or schizoaffective illness. Of particular interest is that much of this research has not examined schizophrenia samples in terms of diagnostic subtype. In the research that has studied schizophrenia subtypes (i.e., paranoid schizophrenia), the results have a tendency to coincide with Zigler and Glick's contention that depression should be found more often in paranoid schizophrenia than any other subtype. Additional support for these authors' claims come from some of the literature which has examined suicide in schizophrenia.

This very brief introduction has served to not only outline the major areas which will be examined in detail in the literature review which follows, but also to instill the idea that, through research, arbitrary diagnostic boundaries may become less well defined.

CHAPTER TWO

LITERATURE REVIEW

ATTRIBUTIONAL MODEL OF DEPRESSION

The Theory

It was through the use of psychotropic medications and electroconvulsive therapy that the conceptual explanation of depression shifted from analytic to biological models. As a result, there has been an enormous increase in research seeking to identify biological factors associated with etiology and treatment of the disorder. Consequently, there was an accompanying decline in the influence of psychoanalytic thinking and the virtual absence of alternative psychological theories of depression (Craighead, Kennedy, Raczynski, & Dow, 1984). In the 1970s the rapidly developing fields of behavior therapy and cognitive-behavior therapy offered several overlapping, but independent, models which attempted to explain the development of depression. It has been proposed by cognitive theorists that depression is the result of particular patterns of negative cognitions and interpretations (Crocker, Alloy, & Kayne, 1988). Although Beck (1967, 1976) has contributed much to the understanding of depression from a cognitive framework, perhaps the most successful application of the non-clinically derived explanations of depression which has been put forward is based on animal experiments on learned helplessness and attribution theory to explain depression (Abramson, Seligman, & Teasdale, 1978). The animal experiments (Overmier & Seligman, 1967; Seligman & Maier,

1967) used to formulate the initial description of the learned helplessness phenomenon involved examining the behavior of dogs who had been repeatedly exposed to inescapable and uncontrollable electric shock. These dogs exhibited "...few attempts to escape the shock (motivational deficit); they were not likely to follow an occasionally successful response with another (learning or cognitive deficit) and they did not evidence much overt emotionality while being shocked (emotional deficit)" (Peterson & Seligman, 1984, p. 347). Later, the concept of learned helplessness was re-examined in more cognitive terms by Seligman (1975) who believed that learned helplessness may model depression with respect to symptoms, causes, preventions and cures. Originally, Seligman (1975) contended that the cognitive, motivational, and affective symptoms of depression all stem from an individual's experience of positive or negative events which are not dependent or contingent on his or her behavior. Consequently, the individual begins to adopt the idea that future events will also be noncontingent on his or her behavior (i.e., beyond the individual's control). Although this theory generated a vast amount of research which demonstrated the similarities between helplessness induced in the laboratory and naturally occurring depression, several inadequacies became apparent. Since there are many circumstances in life which are uncontrollable, but do not necessarily induce sadness, an expectation of uncontrollability per se is not sufficient to produce a depressed affect. Second, a decrease or loss in self-esteem, as a symptom of depression was not explained. Third, the tendency of depressed individuals to blame

themselves for events over which there is no perceived control was not explained. Finally, variations in the generality, chronicity, and intensity of helplessness and depression were not explained (Peterson & Seligman, 1984). Abramson et al. (1978) addressed these criticisms of the theory and reformulated the learned helplessness model of depression along more elaborate attributional lines. In this reformulation, they acknowledged that the occurrence of positive events was unlikely to cause depressed affect even if their occurrence was beyond the individual's control. This same line of thought would hold true for very negative events if their occurrence was deemed to be quite improbable (e.g., natural disaster). Therefore, the type of cognition that would likely produce depressed affect is not only an expectation that very negative events are uncontrollable, but also an expectation that they are highly probable and likely to occur. Consequently, Abramson et al. (1978) attributed depressed affect to feelings of hopelessness rather than helplessness. In essence, individuals can have the cognitive and motivational deficits of helplessness without developing a depressed affect, the presence of which would depend on the degree of hopelessness present (Garber, Miller, & Abramson, 1980). According to the reformulated model, there are at least two types of depressive syndromes which may occur, namely, depression with and without sad affect. Although this seems to be somewhat paradoxical, it is a plausible occurrence given the fact that depression may be composed of cognitive, motivational, affective and somatic deficits, and it is not necessary for all these components to be present in

order to receive a diagnosis of depression. For example, DSM IIIR (1987) has noted that for a diagnosis of a major depressive syndrome one of the symptoms must either be a depressed mood, or a loss of interest or pleasure.

With respect to the second criticism or issue of self-esteem loss, it has frequently been observed by other cognitive theorists that depressives suffer from cognitive deficits such as low self-evaluation, negative expectations, self-blame, self-criticism, and particularly a low self-esteem (Beck, 1967). Abramson et al. (1978) have noted that people who believe that they cannot control the occurrence of expected negative events because they lack the necessary controlling responses, but who also believe that other people can perform these responses, tend to blame themselves for their own helplessness and simultaneously experience a sense of guilt. Thus, there is a paradox whereby depressed individuals have a tendency to feel both helpless and guilty about the same event. The existence of this paradox has continued to be supported (Benassi, Dufour, & Sweeney, 1988). Abramson et al. (1978) contended that the source of this paradox is the distinction between personal and universal helplessness. When an individual cannot control or prevent the occurrence of negative events in his or her life but believes other people are able to do so, the individual will experience personal helplessness. The individual then explains or attributes this personal helplessness to internal causes rather than external causes that would render everyone else as helpless as he or she is (universal helplessness). It is this tendency to

attribute failure to oneself which leads to the loss in self-esteem and sense of guilt about one's own inadequacies (Craighead et al., 1984).

Abramson et al. (1978) have also explained that the intensity of depressed affect is proportional to: (1) the subjective importance the individual has placed on the negative event; and (2) how certain the individual is that an event will occur (i.e., the level of subjective hopelessness). Conversely, the intensity of the depressive cognitive and motivational deficits are determined by the subjective level of helplessness (i.e., having no control over the expected negative event).

Apart from the intensity of depressive affect Abramson et al. (1978) have further contended that the chronicity of depressive affect will vary directly with the degree of stability of the expectations of future helplessness. That is, helplessness which is attributed to relatively stable causes (e.g., intelligence, genetic makeup) will produce longer-lasting expectations of future helplessness and consequently depression than attributions to less stable more transient factors (e.g., fatigue, having a bad day). In contrast, a less stable cause is likely to change and become ineffective sooner, thereby alleviating sooner the helplessness and the associated depressive symptomatology. Finally, attributions to causes that are relatively general or global lead to more generalized motivational and cognitive deficits and future feelings of helplessness on a wider variety of situations. It is the stability and globality dimensions which will define the degree of

motivational and cognitive deficits which will accompany a depressed affect. Conversely, as was mentioned, it is the degree of hopelessness associated with the level of helplessness which will define whether a depressed affect will accompany the motivational and cognitive deficits. Very recently, Alloy, Abramson, Metalsky, and Hartlage (1988) have emphasized the importance of the concept of hopelessness. Crocker, Alloy, and Kayne (1988) have referred to this new emphasis as a revision of the reformulated learned helplessness model of depression. Essentially, the hopelessness theory of depression serves to review and clarify the model and also to stress that attributional style may be a vulnerability factor for depression. These authors further emphasize that depression "...may be a heterogeneous disorder ... which allows for ... such factors as genetic vulnerability, norepinephrine depletion, loss of interest in reinforcers, etc., ..." (Alloy et al., 1988, p. 9).

Wortman and Dintzer (1978) have criticized the learned helplessness model for lacking in predictive power and being unable to explain how a given attribution will be made (i.e., will an attribution precede or be in consequence of depression). Nevertheless, several succinct statements can be made regarding this theory's relationship to true depression. In summary then, depressed individuals have a tendency to: (1) attribute failure to themselves (internal factors) and success to others or to luck (external factors); (2) view factors for success and factors for failure as unstable and stable over time, respectively; (3) attribute failure to global and pervasive factors and attribute

success to specific conditions; and (4) evaluate the severity of deficits in affect and self-esteem as functions of the degree of importance placed on the specific life events in question (Abramson et al. 1978). Seligman, Abramson, Semmel, and von Baeyer (1979) have also suggested that a tendency to make external, unstable, and specific attributions for positive events would increase vulnerability to depression and be found more frequently among depressed than among nondepressed individuals. Although this finding has been supported (Golin, Sweeney, & Shaeffer, 1981) more recent evidence has suggested this attributional profile to be more highly predictive of depression in children than adults (Seligman et al., 1984). Nevertheless, and consistent with the theory, depressed individuals seem to have a characteristic depressive attributional or explanatory style (Abramson et al., 1978; Miller & Norman, 1979).

Although other similar attributional models exist (e.g., Miller & Norman, 1979), the reformulated learned helplessness theory or model has received the most attention in the literature. The popularity of the attributional style hypotheses may stem from the fact that (1) the model is consistent with other cognitive models (Beck, 1967, 1976; Rehm, 1977) and depressive symptomatology; (2) it is possible to measure attributional dimensions (Peterson et al., 1982); and (3) researchers in the area believe that attributional constructs are mediators of depressive affect (Sweeney, Anderson, & Bailey, 1986). Of the many predictions produced by these attributional models of depression, the contention that there may exist characteristic attributional styles that are correlated with

and may predispose to depression is by far the most tested hypothesis (Sweeney, Anderson, & Bailey, 1986).

Empirical Evidence

In their review of the literature Coyne and Gotlib (1983) reported that the earlier studies prior to the 1978 reformulation examined depressed - nondepressed differences in attributions for laboratory controlled tasks of success and failure. The cumulative results of these investigations were that depressed individuals attribute failure more internally than nondepressed individuals. However, the other claims of the model were not supported. Later studies have examined the attributions of depressed and nondepressed individuals using the Attributional Style Questionnaire (ASQ) which is composed of hypothetical good and bad events and was developed by Seligman et al. (1979). Some of these studies (Raps, Peterson, Reinhard, Abramson, & Seligman, 1982; Seligman et al., 1979) have found that depressed individuals, whether drawn from patient or student populations, made more internal stable and global attributions for bad outcomes than did nondepressed individuals. Similar results using a student sample have been found by Golin et al. (1981), but the correlations between depression and the ASQ dimensions were small in magnitude. Others, who also used student subjects, have found that only internal and global attributions for negative events were significantly correlated with an increase in depressed mood, but not with the stability score (Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982). Still others (Manly, McMahon, Bradley, & Davidson, 1982) have failed to find the

hypothesized differences between depressed and nondepressed individuals (i.e., a sample of pregnant women) on any dimensions of the ASQ. Similarly, Miller, Klee, and Norman (1982) also found that there were no differences in attributional style on the ASQ between depressed and nondepressed psychiatric inpatients. However, their results may have been compromised by using only one-half of the total number of hypothetical situations typically found in the ASQ (Segal & Shaw, 1986), which would have reduced the questionnaire's reliability. Nevertheless, Miller and Norman (1981) were able to alleviate learned helplessness and depression in clinically depressed inpatients through the use of experimentally-manipulated attributions for success. These authors interpreted the results as being "supportive of an attribution theory model of learned helplessness and depression" (p. 113).

Some investigators have suggested that inconsistencies in the results may stem from the possibility of there being <u>two types</u> of internal attributions, namely, characterological and behavioral self-blame. While characterological self-blame refers to attributions of blame directed at one's character or stable personality characteristics (e.g., stupidity) behavioral self-blame refers to attributions of blame directed at one's own behavior (e.g., laziness) (Coyne & Gotlib, 1983). Janoff-Bulman (1979) in a study comparing depressed and nondepressed attributions of female students for negative events found that the depressed students exhibited more characterological self-blame. Moreover, Peterson, Schwartz, and Seligman (1981) and Peterson (1988) have found a significant

positive correlation between characterological self-blame and level of depression as measured by the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), and that depression was negatively correlated with behavioral self-blame. Gotlib and Beatty (1985) have noted that depressed individuals do not necessarily display overt depressive behavior on a day-to-day basis; that is, the severity of the symptomatology exhibited by an individual may be highly variable over time. Attributional style, on the other hand, is postulated to be a relatively stable characteristic of the individual (Abramson, Seligman, & Teasdale, 1978; Janoff-Bulman, 1979). From an interactional perspective, therefore, characterological self-blame may be conceptualized as a predisposing vulnerability factor in the development and maintenance of overt depressive symptomatology. According to this conceptualization, an individual with a characterological attributional style would not necessarily be symptomatically depressed at any given time. Under the stress of an aversive life event, however, he or she would tend to exhibit characterological self-blame. This would coincide with Peterson and Seligman (1984) in that attributional style, although trait-like is not static and unaffected by life events. Although the concepts of characterological versus behavioral self-blame is unresolved, Space and Cromwell (1980) have perhaps offered the best explanation: Depression proneness (i.e., vulnerability to depression) is not the viewing of oneself (consistently) negatively but instead the viewing of oneself inconsistently, both negatively and positively. This

inconsistency allows for a swing toward a more depressed affect and greater negative self-evaluation on an episodic basis. Furthermore, an accumulation of negative views of oneself as opposed to positive views can lead to a sustained or chronic depressed mood state.

With respect to attributions for negative life events, Gong-Guy and Hammen (1980) in a comparison of depressed and nondepressed outpatients' attributions for recent life events found that when the single most upsetting event was considered, the depressed group reported more internal attributions and a nonsignificant trend towards more stable and global attributions. Similarly, Hammen and Cochran (1981) found that their depressed group attributed the causes of their most stressful life event to stable but not to more internal or global factors, but the depressed and nondepressed groups in the study did not differ in their overall attributional pattern. Finally, Harvey (1981) found the predicted difference for internality but not for the other dimensions. Although the results are inconsistent, the most common finding for studies of attributions for stressful life events is that depressed individuals, relative to nondepressed individuals tend to make more internal attributions for negative life events (Coyne & Gotlib, 1983). This has been more recently supported by Van den Bout, Cohen, Groen, and Kramer (1987). While the attributional patterns found in this line of research support the reformulated model, the results do not shed any light on the contention that (causal) attributions precede the onset of depression.

In a recent prospective study of stress and well-being in

adolescence, internal, stable and global attributions for negative events attributed to uncontrollable causes were found to be positively related to increases in depression (as predicted by the reformulated learned helplessness model), but internal and global attributions for negative events attributed to controllable causes were found to be inversely related to increases in depression (Brown & Siegel, 1988). These investigators go on to state that much of the research that has shown a temporal relation between attributions and depression have used younger subjects such as children. It is suggested that uncontrollability may be a more important factor for children than adults, since children are very likely to have less control over the events in their lives. Nevertheless, Coyne and Gotlib (1983) and Brown and Siegel (1988) agree that the paucity of research in this area precludes drawing any firm conclusions regarding the etiological role of attributions in depression. Rather, the bulk of research to date appears to be more consistent with the contention that depressive attributional patterns may be concomitants or consequences of depression, particularly with adults.

From their review of the evidence, Coyne and Gotlib (1983) have noted that depressed persons (students or patients) "tend to make more negative and self-deprecating responses to laboratory tasks and to hypothetical and actual life situations" (p. 495). In addition, depressives are more negative in their recall of feedback given on laboratory tasks, make more negative self-evaluations, and make more internal attributions for failure (Coyne & Gotlib, 1983).

Nevertheless, these authors have also noted that the relation between the predicted attributional style and depression is not as strong or consistent as proponents of the revised learned helplessness model would predict. Moreover, even when expected group differences are found, investigators have expressed disappointment concerning their magnitude, and the causal relationship of attributions to depression have yielded mixed results and thus remains unresolved (Coyne & Gotlib, 1983).

Reaffirmation of the Model

Many of the criticisms expressed by Coyne and Gotlib (1983) were addressed by Peterson and Seligman (1984) in their reaffirmation of the revised learned helplessness model and attributional style questionnaire. In support of their claims these authors have summarized five lines of research namely: (1) cross-sectional correlational studies; (2) longitudinal studies; (3) experiments of nature (i.e., studies using subjects who are currently facing naturally occurring bad events like receiving a diagnosis of cancer); (4) laboratory experiments; and (5) case studies. The cross-sectional studies (e.g., Seligman, Abramson, Semmel, & von Baeyer, 1979; Seligman et al., 1984) confirm the conclusion of Coyne and Gotlib (1983) that a depressive explanatory style for bad events co-occurs with depressive symptoms and serves to show that depressive attributions are a symptom of the clinical state of depression and have no causal impact on the onset or course of the disorder. The evidence, however, is conflicting. While some studies (e.g., Hamilton & Abramson, 1983) have shown that a

depressive attributional style co-occurs with depression and remits along with the depression, Eaves and Rush (1984) have shown that a depressive attributional style continues to be manifested following the remission of depressive symptoms. Brewin (1985) has concluded that the idea that attributional style is a symptom of depression, therefore, has only succeeded in receiving partial support.

The longitudinal studies (e.g., Golin, Sweeney, & Shaeffer, 1981; Gong-Guy & Hammen, 1980) have shown that a depressive explanatory style precedes the development of depressive symptoms. Similarly, the experiments of nature (e.g., Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982) have shown that depression results from the experience of bad events in individuals predisposed to a depressive explanatory style. Although these studies have perhaps come closest to verifying Peterson and Seligman's claim that a depressive explanatory style for bad events represents a characteristic vulnerability which, when present, is crucial for the onset of a subsequent depression when bad events occur, controversy continues to abound, in that there are also studies (e.g., Manly, McMahon, Bradley, & Davidson, 1982) which fail to corroborate these findings. Brewin (1985) in response to this research has suggested that there is not overwhelming support for the contention that cognitive or motivational deficits follow uncontrollable events or that individuals with a depressive explanatory style are more prone to developing depression.

Laboratory experiments (e.g., Alloy, Peterson, Abramson, & Seligman, 1984) have shown that imposing uncontrollable bad events

on individuals who made internal, stable or global attributions had the predicted effects on helplessness deficits. Still other studies which examine particular case studies (e.g., Peterson, Luborsky, & Seligman, 1983) function to illustrate that the reformulation applies predictively to the depressive symptoms of specific individuals (Peterson & Seligman, 1984). Still other studies (e.g., Miller & Norman, 1981; Lewinsohn, Steinmetz, Larson, & Franklin, 1981) are able to show that depressive symptomatology can be altered by effecting changes in attributional styles so that an individual's perceived control over his or her life (which is analogous to uncontrollability and related to attributional style) can effect recovery from depression. In spite of the conflicting evidence regarding the contention that attributional style is a symptom of depression, and/or predisposes individuals to depression in the face of bad events, Brewin (1985) has also concluded that because there is evidence to the effect that attributional style affects recovery from depression the onset and vulnerability models hold little credibility. Nevertheless, Peterson and Seligman (1984) have argued that when the evidence from the five lines of research previously mentioned are considered together, there is a convergence across these various strategies which further elucidates and strengthens the validity of the reformulated learned helplessness theory. Nevertheless, Peterson and Seligman (1984) have noted that further empirical scrutiny of the reformulated theory should focus on: (1) the role of depression in the causal attributions of good events (some of these have been discussed previously), (2) the origins of

explanatory style, and (3) explanatory style as a trait.

With respect to the latter point, although explanatory or attributional style is traitlike, it has not been viewed as a static characteristic, but rather it has been viewed as dynamic and changeable (i.e., explanatory style affects depression and depression affects explanatory style (Peterson & Seligman, 1984). The reciprocity of this relationship has been further elucidated by Brewin (1985) when he stated: "There are good reasons to believe that level of depression influences the intensity or certainty with which depressive beliefs are held ..., but causation does not appear to be one-way" (p. 305).

In summary, then, although there is support for the reformulated attributional theory, the evidence is inconsistent, in that the studies do not support all aspects of the theory. That is, all of the studies do not show all of the attributional dimensions (internality, stability and globality) to be in the predicted direction for depressed individuals. The difficulty in the interpretation of these studies is compounded by the fact that there is variation with respect to the sample being studied (e.g., pregnant women, university students or psychiatric patients), degree of depression, and the attribution measure used (e.g., real life events, laboratory tasks or hypothetical events (ASQ)). Some of these issues have been addressed in more recent studies.

Recent Studies

Since the time of Peterson and Seligman's (1984) defence of their reformulated learned helplessness theory, there have been two

other important literature reviews which have attempted to discover the cause for the inconsistencies in the literature while contributing to the fund of knowledge of attributional style and depression. In their review, Peterson, Villanova, and Raps (1985) analyzed 61 published tests of the attributional reformulation to determine the characteristics that might distinguish studies which corroborated the reformulated theory's predictions from those that did not. They found that studies which used a large sample, a large number of events about which attributions were made, and hypothetical events (i.e., like those found in the ASQ) tended to support the reformulation with respect to stable and global attributions. Independent effects were impossible to isolate due to the characteristics high intercorrelations with one another. None of the factors examined consistently distinguished supporting from nonsupporting studies with respect to internal attributions. More recently, Sweeney, Anderson, and Bailey (1986) in their meta-analyses of the relation of attributional styles to depression using 104 studies found that for negative events, attributions to internal, stable, and global causes had a reliable and significant association with depression. Similarly, for positive events, attributions to external, unstable, and specific causes were associated with depression. In addition, the composite attribution scores (the combination of internal, stable, and global scales) were positively and negatively associated with depression for negative and positive events, respectively. The effect sizes were larger for negative than for positive events and the effect sizes for positive

outcomes on the globality dimension were the smallest. Therefore, these results further corroborate the finding that "...as attributions for negative outcomes become more internal, stable, and global, depression increases" (Sweeney et al., 1986, p. 984). Sweeney et al. (1986) also reported that in addition to the researcher who measured attribution dimensions, a number of researchers tested attributional models of depression by using attribution factors such as ability and luck. The meta-analysis showed that for negative outcomes, as attributions to lack of ability increase so does depression since ability is seen as an internal and stable causal factor. Conversely, the results for the luck attribution factor which reflects an external, unstable cause, showed that for negative outcomes as attributions to bad luck increase, depression decreases. Finally, the conclusions of this study were independent of a number of mediator variables such as the type of subject studied (psychiatric vs. student vs. nonstudent depressive), the type of event about which the attribution is made (real vs. hypothetical), the depression measure used, publication status of the paper (published paper, convention presentation, dissertation), and number of subjects. In general, the effects of these mediator variables were weak and inconsistent. One exception to this pattern was that for negative outcomes the internality, stability, and globality effect sizes were larger for psychiatric depressives than for student and nonstudent depressives, but only for the globality effect size was the difference between groups significant (Sweeney et al., 1986). Eaves and Rush (1984) reported

that generalizations of the reformulated theory to clinical samples are limited given that most of the investigations of attributional biases and depression have used mildly depressed student samples. In a similar vein, Peterson et al. (1985) reported that the type of sample does not distinguish corroborating studies of the reformulated theory. Moreover, Segal and Shaw (1986) reported that the theory is better supported with studies using clinical samples, and thereby support the findings of Sweeney et al. (1986). This is of particular importance given that the type or severity of depression as it relates to the model has never really been explicitly stated (Depue & Monroe, 1978).

In contrast to Sweeney et al. (1986), Robins (1988) noted, in his attempt to explain the inconsistency of the literature, by examining only published tests, found that depression was fairly consistently related only to the composite of internal, stable, and global attributions. Those few studies with fairly high power all reported significant relations of depression to stable and global attributions as well as to the composite for positive and negative outcomes in the predicted direction. Robins (1988) further concluded that there is still little information regarding whether causal attributions are related only specifically to depression. The implication would be to study other clinical samples other than depressed individuals. This is an important implication given that some investigators (Gong-Guy & Hammen, 1980; Gotlib, 1984; Nezu, Nezu, & Nezu, 1986) have suggested that the depressive attributional style may not only be a characteristic of depression, but rather of

general psychopathology. This viewpoint, however, has not gained much support (Johnson, Petzel, & Munic, 1986; Raps et al., 1982; Robins, 1988; Sweeney et al., 1986). More recently, in a study examining the attributional styles of depressed patients, anxiety disorder patients, and normals, Heinberg, Vermilyea, Dodge, Becker, and Barlow (1987) found that attributions for negative outcomes are both sensitive and specific to depression. Attributions for positive outcomes followed a less consistent pattern and, in fact, separated depressed and nondepressed anxious patients more successfully than they separated depressed from normal subjects. Conversely, Ganellen (1988) found that attributional style could not distinguish between subjects which were depressed and those that were diagnosed with panic disorder or agoraphobia with panic attacks. However, these results may have been compromised by the fact that over half of the subjects in the anxious group also had secondary diagnoses of major affective disorder, dysthymic disorder or a previous history of an affective disorder.

Attributional Style and Self-Esteem

Both Beck (1967) and Abramson et al. (1978) have contended that depressed individuals have a low self-esteem. This fact has been shown empirically in the past and more recently by Axford and Jerrom (1986) who measured and compared self-esteem in depressed inpatients, nondepressed inpatients and a control group. It was found that depressives had significantly lower self-esteem, more negative social perception and negative feelings associated with self than either of the other two groups. In addition, a low

self-esteem can influence recovery from depression. For example, Dent and Teasdale (1988) found that women who had more global self-devaluative thinking recovered more slowly than equally depressed women showing less self-devaluative thinking. Although Brewin (1986) has noted "...that there is little evidence to support the theory that low self-esteem in depression is primarily a function of judgments about the causes of negative outcomes" (p. 474), there are studies which link self-esteem to attributional style. For example, Fitch (1970) reported that both high and low self-esteem subjects took credit for success, but low self-esteem subjects made more internal causal attributions for their failure. Ickes and Layden (1978) also found that high self-esteem subjects showed a strong preference for attributing positive outcomes to internal causes. Conversely, for negative outcomes, high self-esteem subjects made more attributions to external causes. In another investigation, Brewin and Furnham (cited in Brewin, 1985) found self-esteem to be correlated not only with internality judgments, but also with global attributions for negative outcomes. Although there is evidence for the relationships between depression and low self-esteem, depression and attributional style and, attributional style and low self-esteem, Coyne and Gotlib (1983) and Zautra, Guenther, and Chartier (1985) have noted that there are very few studies which relate depression and attributional style to self-esteem. In explanation, Brewin (1985, 1986) reported that the vast majority of studies have eschewed examination of the role of individual attributional dimensions in favor of trying to predict

depressed mood. He has contended that studies almost invariably correlate internality with a general measure of depression rather than with a measure of the self-esteem component as the theory suggests (e.g., Raps et al., 1982; Seligman et al., 1979). Furthermore, it has been reported that most studies of self-esteem do not assess level of depression and, conversely, studies of depression do not assess or control for self-esteem (Tennen & Herzberger, 1987).

In a recent study, however, Wenzlaff and Grozier (1988) examined how depressed individuals view success and failure in relation to the self. It was found that depressed individuals evaluate failure experiences in ways that are biased against the self. Individuals inflate the importance of the failed endeavor and over time, overgeneralize (analogous to the globality dimension) from their failed experiences to novel situations and thereby contribute to the development of a negative self-concept (Wenzlaff & Grozier, 1988). In a similar study, Zautra et al. (1985) found that internal and stable attributions for negative outcomes were associated with low self-esteem and depression. Conversely, internal attributions for positive outcomes was associated with high self-esteem, and inversely related to depression. Finally, Tennen and Herzberger (1987) found that high self-esteem subjects attributed positive, but not negative outcomes to internal and stable causal factors. They also rated positive outcomes as being more important than negative outcomes and believed that they had more control over positive outcomes. In addition, the tendency to

make internal attributions for failure and external, unstable and specific attributions for success is characteristic of individuals with low self-esteem independent of depression status. These authors explain the latter finding as perhaps resulting from the fact that an individual can be depressed without necessarily exhibiting a negative view of the self or self-blame (see Diagnostic and Statistical Manual of Mental Disorders - Revised DSM III-R, 1987). In addition, these authors concluded that once again a student sample was used and that perhaps these results cannot be generalizable to clinically depressed individuals or to a homogeneous group of depressed individuals.

The importance of having homogeneous diagnostic groups in the study of attributional style, along with studies dealing with paranoid patients, will be presented in the section which explains the rationale for the present study.

PARANOIA

Although some feelings or thoughts of suspiciousness and/or mistrust are common among most individuals, it is when these thoughts are elevated to an extreme, inflexible and sustained level that a diagnosis of paranoid disorder or paranoid schizophrenia may be warranted. Even with discrete diagnoses, the range of paranoid symptomatology may be variable. Furthermore, as will be presented, paranoid symptomatology can be found with a variety of other illnesses besides paranoid disorder or paranoid schizophrenia. In addition, there is also evidence which has suggested that paranoid schizophrenia is more similar to the paranoid disorders and should

be considered nosologically distinct from the other subtypes of schizophrenia. Therefore, it is important for the reader to note that unless specifically stated, the term paranoia will be used to encompass paranoid symptomatology regardless of diagnostic category (i.e., paranoid disorder or paranoid schizophrenia).

Etiology

Irrespective of the constrictiveness of formal diagnostic labels, there are several etiological theories which have been postulated to account for the existence of the syndrome of paranoia. While some theorists have argued causation through a developmental framework (Magaro, 1981; Meissner, 1978, 1981), others have emphasized abusive backgrounds in childhood (Klein & Horwitz, 1949) as being etiologically paramount. Still others have proposed a behavioral model which stresses that early prompting, modeling and reinforcement is the primary cause of paranoid thinking (Haynes, 1986). The most popular theories of paranoia follow a psychodynamic framework. Cameron (1963, 1975) has proposed that when feelings of frustration become intolerable, it is through self-imposed isolation, lowered self-esteem and an elevation of the importance of negative events, that hostility and the causes of distress and anxiety are projected to those in the environment. Thus for Cameron, the individual's environment or what Cameron coined the paranoid pseudo-community functions to: (1) give the paranoid individual an explanation for his feelings of anger, resentment and anxiety, and (2) encapsulate the disordered thinking so that overall functioning can be maintained outside the delusional system (Walker

& Brodie, 1980). Freud, on the other hand, viewed paranoia as a defense against self-reproach (Meissner, 1978). Freud contended that there was a gradual weakening of defences against self-reproaches, which were projected onto others, and later returned to consciousness in a delusional form (Cameron, 1975). Freud also believed that paranoia was the result of a failure to repress homosexual desires or tendencies and that delusions of jealousy resulted from unconscious homosexual tendencies which are defended against through denial and projection (Cameron, 1975). For example, a man who is attracted to another man, eventually suspects his wife of loving this man. Although little credence is given to this latter aspect of Freud's theory, a common theme embedded in the psychodynamic framework is that paranoia serves to protect the individual from negative feelings about the self. Through the defence mechanism of projection, individuals are able to project away from themselves feelings and beliefs which would give rise to low self-esteem and depression.

The psychodynamic theory of paranoia may also be viewed from a more sociological perspective. For example, Mirowsky and Ross (1983) have proposed that paranoia is a profound form of social alienation which stems from a sense of personal powerlessness developed through a belief in an external locus of control. According to Rotter (1966), an individual with an external locus of control has the belief that "outcomes of situations are determined by forces external to one's self, such as powerful others, luck, fate, or chance" (Mirowsky & Ross, 1983, p. 229). In contrast, a

belief in an internal locus of control is a generalized expectation that outcomes are contingent on one's own behavior. It is this sense of powerlessness and the experience of failure in the face of effort that eventually leads individuals to mistrust others. This mistrust develops particularly when the individual has experienced some form of victimization or negative life event, which in turn leads to a sense of alienation. Similarly, Aaronson (1977) has posited that paranoid individuals "frequently place all power and control outside themselves ... and have no expectation that their behavior can effect outcomes..." (p. 28). Further, "paranoia is an attempt, albeit unsuccessful, to avoid the experience of alienation, for although it is the ultimate expression of powerlessness, the individual is constantly seeking to gain mastery through the paranoid delusional system" (Aaronson, 1977, p. 28).

It is possible to identify similarities between this alienation model of paranoia and the reformulated learned helplessness model of depression. For instance, the concept of personal powerlessness developed through a belief in an external locus of control, is very similar if not identical to the concept of personal helplessness or being unable to control events (uncontrollability) or effect positive outcomes put forth in the reformulated model. In addition, both viewpoints acknowledge the importance of negative or unpleasant experiences (bad events) in the development of paranoia or depression, regardless of the temporal relationship between the occurrence of events and illness onset. Benassi et al. (1988) have noted that the greater the external locus of control, the greater

the depression. Mirowsky and Ross (1983) have acknowledged that a belief in external control (i.e., lack of personal control) is an important factor in the development of depression, whereas emotional and social support discourages the development of depression. They also acknowledge that paranoia may be instrumental in preventing adequate emotional and social support networks. Although they believe that there is a link between paranoia, a belief in external control and depression, they were unable to expand on this relationship. Thus, there are important similarities in the theories used to explain the two ostensibly different illnesses of depression and paranoia, and it may be that there are similarities in depression and paranoia which parallel the similarities in the theories used to explain their existence.

Continuum of Severity

According to the DSM-III-R (1987), the most common delusion found in the delusional disorders (i.e., paranoid disorders) are those of persecution. These individuals typically feel victimized and as a result, often rebel with anger and resentment. In contrast to depressives who blame themselves for their failures and/or difficulties, paranoids tend to blame others or outside circumstances for their failures and/or difficulties, and credit themselves with their successes and accomplishments (Aaronson, 1977; Heilbrun & Bronson, 1975). This form of thinking is often taken to the pathological extreme in paranoid schizophrenia in the form of grandiose delusions, where they have an exalted opinion of themselves.

Although paranoid schizophrenia may represent the pathological extreme, paranoid symptomatology is not peculiar to schizophrenia but rather can be observed in a wide variety of clinical contexts and with varying degrees of intensity in nearly all diagnostic categories (Meissner, 1978). Conditions in which paranoid features may be found include psychiatric, neurological, sex chromosome, metabolic, and endocrine disorders as well as drug abuse and pharmacological toxicity (Manschreck & Petri, 1978). Like paranoia, depression may also cross diagnostic boundaries and can be found to accompany a variety of other illnesses (Craighead et al., 1984; Winokur, Black, & Nasrallah, 1988). Although the level of severity of depression or paranoia may vary when they co-occur with other illnesses, the level of severity of both depression and paranoia can vary in the absence of other diagnoses. That is, depression and paranoia can be viewed along a continuum of severity. For example, feelings of sadness and/or mild depressive states have been "...postulated to represent the low end of a continuum of severity with clinical depression at the opposite pole..." (Gotlib, 1984, p. 19). Similarly, paranoid symptomatology may be thought of as being on a continuum of severity ranging from feelings of suspiciousness and inferiority through paranoid personality and paranoid disorders to the pathological extreme of paranoid schizophrenia (Magaro, 1981). This view has been recently supported by Romney (1987). In this study the theory that the development of the paranoid process is gradual and insidious was quantitatively tested by covariance-structure analysis using a computer program (LISREL).

The results supported the contention that paranoia and paranoid schizophrenia are manifestations of the same disorder and "that they differ in degree (of severity) rather than in kind" (Romney, 1987, p. 654). In other words, the respective differences in the manifestations of both depression and paranoia may be more quantitative than qualitative.

The concept of a continuum for paranoid symptomatology particularly for the delusional disorders and schizophrenia has been dismissed largely on the grounds that genetic studies usually show no excess of schizophrenia in the relatives of paranoid psychosis patients (Kendler, Masterson, & Davis, 1985; Watt, 1985). Nevertheless, as Munro (1982a, 1988) has noted, there has been a longstanding view that paranoia, paraphrenia and paranoid schizophrenia belong on a continuum, "the so-called Paranoid Spectrum" (Munro, 1988, p. 171), based on the fact that a significant number of cases of paranoia (about 10%) (Munro, 1982a), may deteriorate to paraphrenia or to schizophrenia although such deterioration is not inevitable. Further evidence comes from the fact that it is often very difficult to diagnostically distinguish between paranoid disorders and paranoid schizophrenia (Kendler & Tsuang, 1981). Often, much of this nosological confusion stems from the fact that there is overlap in symptomatology between the paranoid disorders and paranoid schizophrenia (Cromwell & Pithers, 1981). Additional difficulty in understanding the relationship between paranoid disorders and paranoid schizophrenia emerges from the use of both longitudinal and cross-sectional approaches to

psychiatric diagnosis (Kendler & Tsuang, 1981). The traditional nosologists, for example Emil Kraepelin, focused on the course of illness as the main criterion by which to categorize psychiatric syndromes, while Eugen Bleuler emphasized cross-sectional symptomatology and underlying psychological mechanisms in his classification scheme (Kendler & Tsuang, 1981). These authors have noted further that a "significant proportion of some forms of paranoid psychotic disorders are not symptomatically stable over time..." (Kendler & Tsuang, 1981, p. 608) suggesting that appreciable differences in the classification of illnesses would exist. This would hold true for the DSM-III (1980) and DSM-III-R (1987) which resemble a cross-sectional approach as opposed to the International Classification of Diseases - revision 9 (ICD-9) which follows more of a longitudinal perspective. Further controversy over the diagnostic status of paranoid disorders and paranoid schizophrenia stems from the ambiguity of the term paranoid, which is used in the literature in three ways: (1) to denote suspiciousness; (2) to denote a specific type of delusion, that is, persecutory delusion; and (3) to describe a psychiatric syndrome (Kendler, 1980). This difficulty has been somewhat rectified in DSM-III-R (1987) with the replacement of paranoid disorder with delusional disorder, a term introduced by Winokur (1977). Finally, it may be that some of the controversy is derived from the fact that there are numerous classification systems used throughout the world with some researchers like Tsuang and Winokur developing their own criteria for research purposes (Kendler & Tsuang, 1981). Malt

(1986) has suggested that "the goal for psychiatry should be to have a classification system which is internationally accepted" (p. 9). No doubt this would help to alleviate some of the confusion in diagnosis found in the literature. Still others, however, have suggested that the underlying mechanisms of paranoid symptomatology such as performance on cognitive and perceptual tasks are a better means of classification than just the symptoms themselves (Magaro, 1981).

Paranoid-Nonparanoid Dichotomy

It was Kraepelin who formulated the traditional view that paranoia was a separate illness from schizophrenia. In addition, he viewed the paranoid subtype of schizophrenia to be the most common form of the paranoid illnesses. Consequently, paranoid schizophrenia was classified under the schizophrenic illnesses. In contrast, Meissner (1981) argued that the schizophrenic and paranoid dynamics are two separate and discriminable processes that operate independently. For Meissner, the schizophrenic process is thought to have a disorganizing and disruptive effect on the organization of cognitive processes and information processing. In contrast, the paranoid process is thought to function in facilitating personality organization, and it is the intermingling of these two separate processes which is responsible for the psychopathology commonly seen in paranoid schizophrenia.

Much of Meissner's work is in keeping with Magaro's contention that paranoid schizophrenia not be included within the general category of schizophrenia, but rather be reclassified along with the

paranoid disorders (Magaro, 1981). This stance has also be reiterated by Zigler and Glick (1986) who contend that "the diagnosis of paranoid schizophrenia reflects the paranoid process but has little to do with schizophrenia" (p. 156). There is considerable evidence to support these viewpoints. For example, Zigler and Glick (1984) have noted that paranoid schizophrenics tend to be older on first admission to hospital and have a more favorable prognosis, and be generally intact and less disturbed. In addition, Magaro (1981) in his review of the literature has noted that paranoid schizophrenics are more similar in cognitive style to the paranoid disorders than nonparanoid schizophrenics in that paranoid schizophrenics are less distractible, show greater rigidity in concept formation, exhibit perceptual overinclusiveness, can process complex information more quickly, typically score higher on measures of intelligence, have faster reaction times on cognitive tasks, and generally have better premorbid adjustment. This latter point has also been supported by Ritzler (1981). Finally, Magaro (1981) has concluded that overall, the conceptual capacity of paranoids is more intact and the personality is better integrated. Conversely, nonparanoids react to stimuli in a more global fashion and have less well-developed conceptual abilities. Moreover, paranoid schizophrenics can classify or conceptualize information, but have a tendency for underreliance on perceptual input. Therefore, paranoids have a tendency to fit new perceptions into their already rigidly established schemata (i.e., conceptual rigidity). Conversely, nonparanoids rely on their perceptions but have

difficulty conceptualizing new information. Consequently, they typically exhibit a tendency toward loose associations and disorganized cognitive processes (Magaro, 1984). It is important to note that this paranoid-nonparanoid dichotomy is strengthened by the fact the performance of normal subjects on these various cognitive tasks tends to fall between that of paranoids and nonparanoids (Magaro, 1981, 1984; Zigler & Glick, 1988).

Since concept formation is considered to be a left hemisphere function while perceptual integration is considered to be a right hemisphere function, it may be that paranoids and nonparanoids exhibit some dysfunctioning in the right and left hemispheres, respectively (Magaro, 1984). Although Liddle (1987a) has corroborated Magaro's cognitive profile of paranoids, he has noted from neuropsychological evidence that correlates of paranoid symptomatology seem to be associated with left temporal lobe dysfunction, whereas correlates of positive symptoms (e.g., shallow, euphoric or silly affect, thought derailment or tangentiality) and negative symptoms (e.g., apathy, slowness or flat affect) may be associated with dysfunction in two different sites within the frontal lobes. In contrast, Stevens (1988) has cited evidence to suggest that patients with temporal lobe epilepsy in conjunction with paranoid illness (paranoid psychosis or paranoid schizophrenia) more commonly have right sided cerebral hemisphere lesions. Moreover, Singh, Kay, and Opler (1988), based on their observation of pharmacogenic response to anticholinergic agents, have suggested that paranoid schizophrenia is a distinct category and that

nonparanoids may be subdivided into positive and negative subtypes.

Further evidence for the paranoid-nonparanoid dichotomy has emerged from genetic studies. Although Tsuang, Winokur, and Crowe (1980) have contended that such a dichotomy based on genetic studies is unfounded, in their review of the literature, Kendler and Davis (1981) have noted that paranoid schizophrenia tends to run in families at least to a moderate degree and that the evidence of schizophrenia is less common among the relatives of paranoid schizophrenics as compared to nonparanoid schizophrenics.

Additional biological evidence comes from a comparison of biochemical activity in both chronic paranoid and nonparanoid schizophrenics. It has been reported that paranoid schizophrenics have a significantly lower platelet monoamine oxidase activity as compared to the nonparanoid group (Potkin, Cannon, Murphy, & Wyatt, 1978; Walker & Levine, 1988). Although these results have generally not been confirmed, there is also evidence to suggest that brain norepinephrine levels are higher in paranoid than in nonparanoid schizophrenics (Kendler & Davis, 1981).

Much of the biological evidence is controversial, particularly with respect to emotion and cerebral laterality (Coffey, 1987). Nevertheless, evidence from a variety of different types of research including cognitive style of paranoids, information processing, familial heritability of paranoid schizophrenia and neurotransmitter assays all contribute to the contention that paranoid schizophrenia may be a separate disorder from the other subtypes of schizophrenia.

Paranoia and Depression

Although there is evidence to suggest both a continuum of paranoid symptomatology and a valid differentiation between paranoid schizophrenia and the nonparanoid subtypes of schizophrenia, there is also evidence to suggest that there is a relationship between paranoid symtomatology and affective illness (i.e., depression) apart from the similarities in the etiological theories mentioned previously.

For Zigler and Glick (1984, 1986, 1988) the parallels between paranoia and depression are three-fold. These authors have noted that both paranoia and depression involve a preoccupation with the self, and that feelings of guilt and inadequacy are of primary importance in both depression and paranoia in that such feelings underly paranoid persecutory delusions. Second, both depression and paranoia can be conceptualized along a continuum of severity and both states can manifest themselves as symptoms or as circumscribed illnesses. Third, both depression and paranoia as symptoms can accompany a variety of other illnesses. For example, it was found that 40% of all psychiatric admissions to a hospital had paranoid symptoms of which less than one-third were diagnosed as paranoid schizophrenic. In addition, 70% of psychotic depressives, 50% of affective disorders and 63% of those with organic brain syndromes had paranoid symptoms (Freedman & Schwab, 1978).

In his review of the evidence Flor-Henry (1976, 1988 in press) has noted that there is much support for the notion that schizophrenia involves a temporal-limbic dysfunction in the left

hemisphere and the schizophreniform psychoses are associated with left hemisphere lesions, while affective disorders are associated with right hemisphere lesions. As previously mentioned, paranoid schizophrenics are thought to have right hemisphere dysfunction (Magaro, 1984). Gruzelier (1981) contends that paranoids may not have a dysfunctional right hemisphere, but rather exhibit an overactivation of the left hemisphere in comparison to the right hemisphere. Whether dysfunctional or relatively underactive, the right hemisphere has been implicated in both paranoid and affective symptomatology. The foregone evidence serves to strengthen the paranoid-nonparanoid dichotomy while providing evidence for more than a phenomenological relationship between paranoid and depressive illness. Further evidence comes from studies which examine thought processes. As previously mentioned, Magaro (1981) identified several cognitive patterns unique to paranoid individuals, in particular overinclusive thinking. However, this form of thought process has also been found in depressed individuals (Payne & Hirst, 1957; Carter, 1986) and manic individuals (Andreasen & Powers, 1974; Harrow, Grossman, Silverstein, & Meltzer, 1982). More recently, Carter (1986) found that psychotically depressed individuals exhibit similar thought deficits to those shown by chronic paranoid schizophrenics. For example, both groups exhibit more idiosyncratic (i.e., looseness, incoherence, and illogicality), overinclusive and concrete thinking than normal subjects. Carter (1986) has interpreted her findings as being partly supportive of the speculation by Harrow et al. (1982) that there exists a general

psychosis factor which underlies various psychotic illnesses. Carter (1986) also entertains the suggestion that these results "may be a function of the depressive affect shared by both schizophrenics and depressives" (p. 340), and only a comparison of nonpsychotic depressed and nonpsychotic nondepressed to paranoid schizophrenics would help to clarify this issue. Still further evidence of the relationship between depression and paranoia comes from several diagnostic cases.

Case Studies

Ward et al. (1982) have described a woman who was initially diagnosed with late onset paraphrenia and treated with a variety of medications to no avail. Subsequent to an escalation in depressive symptomatology she was found to respond abnormally to a dexamethasone challenge (Dexamethasone Suppression Test), i.e., her plasma cortisol level was not suppressed, and treated with electroconvulsive therapy which alleviated both her depressive and paranoid symptoms. Similarly, de St. Croix, Dry, and Webster (1988) have described a case where a man was diagnosed with paranoid schizophrenia with concomitant flat affect and depressive features. On the day of his suicide, evidence of paranoid and depressive symptomatology was not apparent, although one would expect that his depression might have escalated given his successful suicide. Still further, Freedman and Schwab (1978) have described three cases where individuals who were treated unsuccessfully for paranoid schizophrenia or paranoid disorder, were later discovered to actually be suffering from depression. These authors concluded that

paranoid delusions are "a reliable indicator of such diagnoses as organic brain syndrome, schizophrenia, affective disorder, paranoid state, and psychotic depressive reaction, but not of schizophrenia in particular" (Freedman & Schwab, 1978, p. 390). Similar diagnostic difficulties have been encountered with individuals who experience an isolated delusion of a hypochondriacal nature (i.e., monosymptomatic hypochondriacal psychosis or MHP) the diagnostic criteria of which has been defined by Munro (1982b). Although the diagnosis of MHP is difficult and attempts may range from paranoia, obsessive-compulsive disorder to an atypical affective disorder, in at least one case, it was found that with various drug treatments either the patient's paranoid or depressive symptomatology was at the forefront with both being resolved when treated with antidepressants (Ross, Siddiqui, & Matas, 1987). There have also been some longitudinal studies which have shown depression giving way to paranoid symptoms over the course of time (Carlson & Goodwin, 1973; Kovacs & Beck, 1978). Finally, Freud documented his theoretical formulations of paranoia based on the autobiographical memoirs of Daniel Paul Schreber. Recent evidence, however, would suggest that Schreber's illness was primarily affective rather than paranoid (Rinsley, 1984-85).

Thus there is an accumulation of evidence which suggests that paranoid schizophrenia is most similar to the paranoid disorders and different from nonparanoid schizophrenia. In addition, there is mounting evidence from cognitive studies, some from genetic studies and especially case histories illustrating diagnostic conundrums

which suggest that paranoids are also similar to depressives. The case studies previously outlined which show paranoid symptomatology overshadowing depression and paranoid symptomatology later yielding to depression to subsequently allow for a "correct" diagnosis and treatment, lend support for what may be considered a paranoid depressive continuum.

Paranoid-Depressive Continuum

Schwartz (1963) published a paper which focused on the paranoid-depressive continuum where he argued that personal responsibility is central to this continuum. As previously mentioned, depressives often have low self-esteem and tend to blame themselves while paranoids tend to project blame onto others. Moreover, Kolb and Brodie (1982) have noted that paranoids blame others for dissatisfactions with the self and thereby enhance self-esteem and "prevent a realistic but intolerable self-evaluation" (p. 447). Consistent with this view is Meissner (1978, 1981) who contended that a lowered self-esteem, feelings of inadequacy and depression are central psychodynamic issues in paranoid illness. Similarly, Heilbrun and Bronson (1975) asserted on the basis of their experimental findings that the onset of paranoid mechanisms follow feelings of inadequacy and low self-esteem. Moreover, "paranoid patients can only relinquish their paranoid stance at the risk of encountering a severe depression" (Meissner, 1978, p. 125). Further, Allen (1967) contended that depression is often found to underly paranoia and that depression and paranoia may substitute for one another.

This so-called paranoid-depression continuum can be given credence through a closer examination of DSM-III-R (1987) particularly by scrutinizing the types of delusion associated with each disorder. The delusions which occur in both paranoid illness and delusional depression are categorized according to their content; that is, depressive delusions range from mood congruent to mood-incongruent (i.e., thought insertion, thought broadcasting, persecution and of being controlled), the latter being more common in paranoid illness than in depression. The fact that Kettering, Harrow, Grossman, and Meltzer (1987) have noted depressives with mood-incongruent delusions are less depressed than depressives with mood congruent delusions, may partially support the contention that depression can be overshadowed by delusions more typically found in paranoids. Presumably then, with such a continuum, individuals with more mood congruent delusions would be more depressed and less paranoid than those with more mood incongruent delusions who would be more paranoid and less depressed. Furthermore, with the possible trend in both directions and the fact that most disease entities are seldom static, one can understand how paranoia may change into depression and vice versa.

Zigler's and Glick's Unorthodox View

Zigler and Glick's (1984, 1986, 1988) elaboration of the basic psychodynamic premise of paranoia perhaps provides the best explanation for the evidence concerning the relationship between paranoid illness and depression. These authors contend that depressives, in their need to escape the psychic pain of a depressed

mood, often employ maladaptive coping mechanisms such as the overuse of alcohol or drugs or mania. For them, paranoia simply represents another mechanism by which depressed individuals ward off a breakthrough into consciousness of depressive thoughts, thereby avoiding the painful experience of depression. Therefore, delusions of persecution protect an individual against a sense of personal inadequacy by projecting responsibility for the inadequacy on to others or circumstances. Persecutory delusions and ideas of reference, by drawing attention to the self, also help to provide a sense of self-importance which in turn facilitates enhancing the self. Grandiose delusions epitomize the enhancement of self-esteem.

Zigler and Glick (1984, 1986, 1988) have conceptualized the relationship of paranoid schizophrenia to schizophrenia and depression by drawing a metaphorical parallel between gene expression and the manifestation of mental illness. Essentially, a genotype (i.e., the underlying condition) can be overtly manifested in a number of different phenotypes (i.e., outward manifestations of the genotype). Zigler and Glick (1984, 1986, 1988) contend that depression and schizophrenia represent genotypes which can give rise to a number of different phenotypes. Conversely, paranoia is essentially a phenotype that can be found in conjunction with a number of differing genotypes. Generally, they assert that unipolar depression, mania, alcoholism and paranoia are the phenotypic expressions of the depression genotype, while the phenotypic paranoid illness could be the manifestations of several genotypes, namely, depression, substance abuse, and organic brain dysfunctions

associated with aging (Zigler & Glick, 1988). Further, although these genotypes all involve feelings of inadequacy, guilt and depression, the phenotype that paranoia most resembles is that of mania, since in both instances, the depression is turned upside-down which results in the assertion of the individual's self-worth or well-being (Zigler & Glick, 1988). Apart from the cognitive and epidemiological similarities between mania and paranoia already mentioned, Pope and Lipinski (1978) have noted that because of the similarities in delusions and temperament (i.e., excitement, irritability), it is often difficult to distinguish between the two groups. In addition to this finding Brockington, Wainwright, and Kendell (1980) have noted that paranoid schizophrenic and manic patients have a number of similar features, including heritability, response to neuroleptics, and the tendency to post-psychotic depression.

Zigler and Glick (1984, 1986, 1988) further contend that depression occurs either before the paranoid delusions are formed or after the latter have abated. Therefore, they expect depressive symptomatology "to be manifested either in the early stages of the formation of delusions or at the time when paranoid symptomatology is abating" (Zigler & Glick, 1986, p. 161-162). By contrast, as the delusion is solidified and well-organized, evidence of depression should be minimal. Depression, then, should appear in paranoia to the extent that the paranoid defenses fail to operate. Consistent with the paranoid-depressive continuum and Meissner's (1981) view of paranoid schizophrenia, Zigler and Glick (1988) have developed the

"unorthodox view" that "paranoid schizophrenia is not a true schizophrenia but is best conceptualized as one of several possible responses to an underlying depressive mode" (p. 289). An alternative, but weaker form of this hypothesis would be that there are two types of paranoid schizophrenics: (1) depressives and (2) true schizophrenics who respond with paranoia to the schizophrenic process (Zigler & Glick, 1988). According to these theorists, depression should be more predominantly found in paranoid schizophrenics than in nonparanoid schizophrenics. Before this can be explored the issue of depression in schizophrenia must be more fully addressed.

DEPRESSION IN SCHIZOPHRENIA

Although many schizophrenics experience delusions, hallucinations, incoherence, and associated social and behavioral impairments, these people are generally not schizophrenic 24 hours a day (Strauss, Bowers, & Keith, 1982). Rather, many must cope with varying degrees of cognitive distortions, psychophysiological arousal, information overload, and thought disorder. Because the vulnerability to, and symptoms of schizophrenia are more variable than constant over time and across patients, the basic premise to be concluded is that for most patients, schizophrenia is an illness characterized by exacerbations, remissions and perhaps most importantly, variability in symptomatology (Herz, 1985). Moreover, "the concept of schizophrenia as a unitary disease is diminishing" (Haier, 1980, p. 417). The wide spectrum of schizophrenic symptomatology seems to be no longer compatible with the concept of

a unilateral, uniform, inexorably progressing organic illness process. Rather, schizophrenia appears to be better hypothesized as a multi-conditioned life process, occurring in people with a particular vulnerability, interacting with complex life events and circumstances (Ciompi, 1984). Although biological, social and environmental factors are important, they should not be viewed as being mechanically deterministic in the pathogenesis of schizophrenia; rather, schizophrenic individuals through making choices can "actively contribute to the development and course of their own disorders" (Strauss et al., 1982, p. 435). The question of the etiology of schizophrenia is complicated and further compounded when symptoms of other illnesses are combined within the schizophrenic framework. This is particularly true of depressive symptoms which have been found to be the most common symptom experienced by the schizophrenic patient living in the community (Cheadle, Freeman, & Korrer, 1978).

Becker, Singh, Meisler, and Shilcutt (1985) have estimated that the average incidence of depression in schizophrenia is approximately fifty percent. Similar rates have been reported by McGlashan and Carpenter (1976a) and Johnson (1981). Moreover, Martin, Cloninger, Guze, and Clayton (1985) have noted that consistently high rates of depression have been found in studies of schizophrenic patients. There is controversy in the literature as to whether depression in schizophrenia represents a primary depression, a mixture of depressive and schizophrenic symptoms (Hirsch, 1982; Knights & Hirsch, 1981), or is secondary to the

schizophrenic illness and emerges only after an acute episode has subsided (McGlashan & Carpenter, 1976a, 1976b). There is also no consensus regarding the prognosis, drug treatment, or clinical presentation of depressive symptoms in schizophrenia. Investigators in the field contend that a major depressive syndrome per se perhaps differs from the depressive symptoms found in schizophrenia. To date, there is still much controversy as to what constitutes depression in schizophrenia, and investigators are not sure how negative symptoms, affective flattening, anhedonia, avolitional apathy and neuroleptic induced symptoms are related to depression in schizophrenia. For the following discussion, the term depression will encompass a wide range of symptom severity. This stance will aid in the understanding of the various etiological theories for depression in schizophrenia.

Etiology

Depression in schizophrenia is not well understood despite the recent upsurge in its study worldwide. The confusion regarding the clinical significance and treatment of depression in schizophrenia may be due to several factors including, "underdiagnosis, and failure to appreciate the impact of depression on the clinical course of schizophrenia (e.g., relapse and poor outcome)" (Becker et al., 1985, p. 26). There have been several explanations postulated to account for depressive symptoms in schizophrenia.

One theory is that depressive symptoms are merely a side effect of medication, the so-called neuroleptic-induced or pharmacogenic depression (Galdi, 1983). Knights and Hirsch (1981) have reported

that although some investigators have observed schizophrenics with depressive symptoms following treatment with neuroleptic medications, the incidence was low and only a small number of patients were subsequently treated with antidepressants. Other investigators (Johnson, 1981; Moller & von Zerssen, 1981; Siris, Strahan, Mandeli, Cooper, & Casey, 1988) have generally concluded that neuroleptics are unlikely to be the major cause of depression in schizophrenia, since depressive symptoms can be observed at various stages of the illness and not only as a consequence of neuroleptic treatment.

A variant of pharmacogenic depression is the concept of "akinetic" depression coined by Van Putten and May (1978). They contend that because some schizophrenic patients were slow and lethargic and seemingly depressed, they may be suffering from a drug-induced parkinsonism (i.e., an atypical form of extrapyramidal symptom of akinesia) or akinetic depression both of which respond to anti-cholinergics (i.e., anti-parkinsonism agents). Rifkin, Quitkin, and Klein (1975), Siris (1987) and Van Putten and May (1978) all agree that the distinction between akinetic depression and other perhaps more legitimate depressive symptoms is difficult. Moller and von Zerssen (1981) have shown that depressive symptoms are as common in schizophrenics on anti-cholinergics as in those who are not. Furthermore, Johnson (1981) has found that anti-cholinergics are no more effective than placebo for the treatment of depression in schizophrenia. Hirsch (1982) contends that there is little evidence to "regard the akinetic syndrome as a

form of depression (other than) to remind us that apparently anergic depressed schizophrenics may have a drug-induced Parkinsonism" (p. 421). Moreover, Rifkin (1987) has concluded that "the relationship of akinesia to depression as well as the nature of that depression continues to be incompletely understood" (p. 5). Rifkin (1987) has suggested that in order to make the differential diagnosis, akinesia must be ruled out either by eliciting a response with anti-cholinergics or through a discontinuation of the neuroleptic. "Only after akinesia is ruled out should treatment with antidepressants be considered" (Rifkin, 1987, p. 6).

A third concept suggests that depression in schizophrenia may be the result of the schizophrenic gaining insight into the illness itself and the detrimental effects the illness has had on his or her life. In this sense, this post-psychotic depression may be a reaction (i.e., a reactive depression) to being ill and not pharmacogenically induced (Knights & Hirsch, 1981). Moreover, the depression experienced by schizophrenics may be a normal reaction to a serious illness. Winokur, Black, and Nasrallah (1988) have, however, found that reactive depression is a more typical response to medical illnesses than psychiatric illnesses. But these investigators did not include depressed schizophrenics in their sample. McGlashan and Carpenter (1976a, 1976b) and Siris, Harmon, and Endicott (1981) have noted that the post-psychotic phenomenon can be observed in recovering schizophrenics in 25% to 40%, respectively. Siris, et al. (1981) have concluded that the post-psychotic depressive symptoms are an "important source of

morbidity in some patients with schizophrenia, but the full syndromal manifestation of depression occurs less frequently than do prominent isolated symptoms" (p. 1023). Moreover, McGlashan (1982) has concluded that "the post-psychotic depression in schizophrenia probably represents a heterogeneous collection of syndromes" (p. 119). McGlashan (1982) has suggested that because of the heterogeneity of the post-psychotic depressive symptoms in schizophrenia, it is often confused with what he labeled "aphanisis" or syndrome of pseudodepression which is characterized by psychic blankness whereby the patient often presents a wooden and empty exterior and is supposedly similar to the "burnout case" often seen in chronic schizophrenia. However, Becker et al. (1985) has identified this so-called wooden and empty exterior as being typical of depressed schizophrenic patients, although these authors did not specify when such symptoms would appear in the course of the schizophrenic illness.

An additional problem with the concept of post-psychotic depression is due to its vagueness. Leff, Tress, and Edwards (1988a) have suggested that much of the conflicting evidence with respect to course of depression in schizophrenia and pharmacogenic response can be attributed to the post-psychotic depression being applied to three distinct groups of patients: (1) those with depression present at the beginning and end of a schizophrenic breakdown, (2) those when depression is only present shortly after the schizophrenic symptoms have been resolved and, (3) those where depression occurs several months after the schizophrenic symptoms

have diminished.

A fourth notion is that depressive illness may coexist with schizophrenia as a matter of chance (Hirsch, 1982). To validate this proposition, Hirsch (1982) has suggested that the depression would have to occur with an equal frequency at each phase of the schizophrenic illness and be equally common in patients who are receiving antipsychotic medications and those who are not. In contrast to the evidence establishing the existence of postpsychotic depression, there is evidence to suggest that those two illnesses do coexist. There is also evidence to suggest that depressive symptoms are more prevalent in the prodromal phase of an acute schizophrenic episode: Herz (1985) has noted that for the week prior to full schizophrenic relapse, most of the symptoms which occur are consistent with depression.

Finally, Hirsch (1982) has suggested that depression may be "an integral part of the schizophrenic process, commonly present, but unnoticed during the acute phase when the florid psychosis is most evident and other symptoms are understandably ignored" (p. 421-422). Furthermore, he has suggested that one would expect depressive symptoms to be more prevalent in the acute phase when the psychosis is more active and decrease rather than increase with neuroleptic treatment. However, as already mentioned, evidence exists for the occurrence of depressive symptoms in the prodromal phase and the post-psychotic period. Further support for Hirsch's argument comes from Knights and Hirsch (1981) who found that depressive symptoms are not only more prevalent in the acute phase of the schizophrenic

illness, but that these depressive symptoms decrease rather than increase in severity with the commencement of neuroleptic medication. Similarly Knights, Okasha, Salih, and Hirsch (1979) found that although depressive symptoms are part of the developing phase of an acute exacerbation, they are most prevalent after admission, but decrease with neuroleptics, and wax and wane spontaneously with lower prevalence during the recovery phase independent of treatment with antidepressants. In fact, Bowers and Astrachan (1967) have found depressed schizophrenics to be unresponsive to antidepressants. Lerner and Moscovich (1985) have confirmed a predominance of depressive symptoms in the acute phase, in spite of their existence following the acute psychotic episode and also noted that they remit more slowly than the psychotic symptoms. Johnson (1985) found in a number of schizophrenics who relapsed while drug-free that 30% exhibited symptoms of depression while drug-free. Similar results were obtained by Strian, Heger, and Klicpera (1982) who like Hirsch (1986) noted that depression in schizophrenia is predominantly an experience or consequence of the underlying illness and that pharmacological factors may not be of such great importance.

Koh, Grinker, Marusarz, and Forman (1981) have also lended support to the notion that depression is an integral part of schizophrenia by citing evidence which suggests that anhedonia (i.e., the absence of pleasure) is an integral part of schizophrenia and may be similar to feelings experienced by depressed patients. Evidence has also been cited for a dopamine-beta-hydroxylase

deficiency in anhedonia (Koh et al., 1981), which could lead to a neurotransmitter depletion (i.e., norepinephrine) also implicated in depression. Harrow, Grinker, Holzman, and Kayton (1977) have also noted that anhedonia is not necessary or unique to schizophrenia, but is less common in acute and is both more common and prominent in chronic and paranoid schizophrenics. Further, Walker and Lewine (1988) have cited evidence to suggest that genetic factors play a larger role in the etiology of negative symptoms than of positive symptoms and that the heritability of anhedonia is partially independent of either a diagnosis of schizophrenia or depression.

Knights and Hirsch (1981) have contended that the causation of depression in schizophrenia "may well prove to be heterogeneous, but [for them] the most economical main hypothesis is a shared pathophysiological mechanism accounting in part for schizophrenic and depressive symptoms, and not a drug induced one" (p. 625).

Thus there is evidence to suggest that depression in schizophrenia may be the result of neuroleptic treatment (Galdi, 1983), pharmacogenic side effects (Van Putten & May, 1978), a gain in insight of the detrimental effects of the schizophrenic illness (Knights & Hirsch, 1981), post-psychotic phenomenon following the resolution of schizophrenic symptoms (Siris et al., 1981), and a coexistence of depressive and schizophrenic symptoms (Hirsch, 1982). Apart from the confusion in the literature regarding the use of the term post-psychotic depression, there is also confusion with the terms acute and prodromal since researchers sometimes have a tendency to use them interchangeably. Nevertheless, although there

is evidence which support and negate all of these theories of causation, it is unlikely that any one explanation will account for all of the data.

Negative Symptoms

Another controversy surrounds the relationship between negative symptoms (i.e., flat affect, poverty of speech and expressive gesture, psychomotor retardation, anhedonia and attentional deficits) and depressive symptoms (Pogue-Geile & Harrow, 1984; Prosser et al., 1987; Walker & Lewine, 1988). Much of the controversy surrounds the fact that negative symptoms are not necessarily unique to schizophrenics, but can also be seen in nonschizophrenic patients with depression such as individuals with Parkinson's disease (Prosser et al., 1987). Crow (1980, 1985) proposed two types of schizophrenia representing two dimensions of pathology: type I and type II. The former is characterized by positive symptoms (i.e., hallucinations, delusions, thought disorder and bizarre behavior) which usually occur during an acute illness, while the latter is characterized by negative symptoms, chronicity, and correlates of brain damage. Similarly, in their review of the evidence, Walker and Lewine (1988) have noted that patients with more negative symptoms show a stronger positive relationship with premorbid dysfunction and poorer outcomes, slower processing of visual information and motor deficits, and brain abnormalities. In addition, Crow viewed these two types of schizophrenic illnesses as being independent syndromes reflecting different underlying pathological processes but not as being mutually exclusive (Liddle,

1987a).

Andreasen and Olsen (1982) have adopted a different view and have regarded positive and negative symptoms as being characteristic of two distinct types of illness, although their conclusions have been questioned on methodological grounds (Liddle, 1987b). In a study which compared anticholinergic response in groups of catatonic, hebephrenic and paranoid schizophrenics, Singh, Kay, and Opler (1987) have suggested that only the nonparanoid schizophrenics can be divided into positive and negative subtypes, while the paranoid group should be considered as being nosologically distinct. This evidence not only supports the contention that paranoid schizophrenics should be nosologically separated from the other subtypes of schizophrenia, but also suggest that depression in paranoid schizophrenia may be different from negative symptoms.

Walker and Lewine (1988) have concluded that the presence of negative symptoms may (1) represent a more severe form of schizophrenia with greater genetic heritability periodically manifested through positive symptoms, or (2) be an enduring trait of the individual that is partly influenced genetically but independently from schizophrenia and increases the probability of the occurrence of schizophrenia in vulnerable individuals. Although rating scales can tap into negative symptoms "it is likely that they are tapping enduring, trait-like characteristics as well as transient state-like phenomena" (Walker & Lewine, 1988, p. 326).

It is also very difficult to distinguish between negative

symptoms and Parkinsonian side effects like akinesia and sedation, and it may be that negative symptoms reflect the "coexistence of inadequately treated side effect ... during neuroleptic treatment" (Prosser et al., 1987, p. 104). The fact that negative symptoms can occur in patients with Parkinson's disease and in schizophrenics with neuroleptic-induced parkinsonian syndromes would make their differential diagnosis quite difficult, if they are different at all. It has been suggested that parkinsonism and negative symptoms might both represent clinical manifestations of a common neurochemical pathway characterized by dopaminergic hypoactivity (Prosser et al., 1987). If this is a valid hypothesis, it may mean that parkinsonian side effects and negative symptoms are indistinguishable since some investigators have been "unable to identify any negative symptom that is not also attributable to parkinsonian akinesia (Prosser et al., 1987, p. 104). Bleich, Brown, Kahn, and van Praag (1988) have also noted that some authors contend that there are two kinds of negative symptoms, those which are core features of the schizophrenic illness and those which are considered to be secondary and "derived from depression, or emotional and social withdrawal due to psychotic decompensation" (p. 298).

There are also unresolved issues in the literature with respect to what constitutes a negative or positive symptom. Liddle (1987b) noted that Andreasen (1982) viewed inappropriate affect as a negative symptom whereas Crow (1980) viewed it as being a positive symptom. Similarly, while Andreasen and Crow viewed derailment and

incoherence of thought as positive symptoms, Lewine, Fogg, and Meltzer (1983) viewed them as negative symptoms (Liddle, 1987b).

Apart from performing pharmacological manipulations in the hope of detecting extrapyramidal symptoms, parkinsonian side effects or other drug effects the distinction between negative and depressive symptoms may continue to be difficult, at least from a pharmacological perspective. This may prove to be even more difficult with paranoid schizophrenics who have been found to have an increase of negative symptoms with anticholinergic medications (Singh, Kay, & Opler, 1987). Prosser et al., (1987) found that there are no significant correlations between negative symptoms and the cognitive features of depression or neuroleptic and anticholinergic plasma activity. In view of this finding, the conclusions of Martin, Cloninger, Guze, and Clayton (1985) are worth noting. They suggested that to distinguish depression from other syndromes, like the presence of negative symptoms, clinicians must continue to rely on the presence or absence of marked dysphoria, suicidal ideation, eating and sleeping problems, loss of interest and feelings of guilt and self-reproach.

Schizoaffective Illness

The fact that the diagnostic category of schizoaffective psychosis exists in DSM-III-R (1987) lends credence to the possibility that a third psychosis (i.e., schizoaffective psychosis) exists which encompasses and perhaps explains the coexistence of both schizophrenic and depressive symptomatology. Lieberman (1979) has stated that "schizoaffective illness is held to be the example,

par excellence, that defies the validity of Kraepelin's original dichotomy" (p. 436). Some investigators (Goodnick & Meltzer, 1984) have reported that successful treatment studies support the hypothesis that schizoaffective illness is similar to primary affective disorders. Moreover, schizoaffective patients have been shown to have a better response to medications than individuals with pure schizophrenia (Andreasen, 1987). With respect to outcome, in their review of the evidence, Harrow and Grossman (1984) have noted that the outcome of schizoaffective disorders is poorer than that of affective disorders, but better than that of schizophrenia. These authors, however, were no closer to solving the classification conundrum of schizoaffective disorders. More recently, however, in a long-term comparative outcome study Williams and McGlashan (1987) found that recent onset schizoaffective patients resembled unipolar depressives in a variety of demographic variables and in their premorbid functioning profile, but that among long-term inpatients, schizoaffectives more closely resembled schizophrenics than affective disorder patients. Family history studies seem to indicate that patients with schizoaffective disorders have higher rates of schizophrenia familialy than do individuals with pure schizophrenia (Andreasen, 1987). McGuffin (1988, in press) has found that genetically, schizoaffectives have an excess number of relatives with either schizophrenia or affective disorders, not with schizoaffective illness. In addition, schizoaffectives with manic features (i.e., schizo-bipolars or schizo-manics) may be more closely related to affective disorders than schizoaffectives who

only have depressive features (Clayton, 1984). Moreover, Shenton, Soloway, and Holzman (1987) found that the thought disorders of schizoaffective and schizophrenics are very similar to one another.

The fact that there is so much variability in the clinical evidence in support of the category of schizoaffective disorders may lead one to conclude that many clinicians may be misdiagnosing or making premature diagnoses and that in fact diagnoses of schizophrenia or affective disorder should be made. Some have reported that schizoaffective disorder may represent a group of atypical patients with schizophrenia and with affective disorder (Andreasen, 1987). This viewpoint would coincide with the coexistence theory which was discussed previously. Others have contended that the conflicting evidence should be viewed as evidence for the heterogeneity of schizoaffective disorder and that researchers should concentrate on identifying homogenous subgroups of schizoaffective patients (Tsuang & Simpson, 1984). Johnson (1985) has noted that there is "no consensus on the definition, natural history or prognosis of this condition. It remains a useful research hypothesis, but at the present time is of unproven clinical value" (p. 34). Similarly, Brockington (1986) has noted "there is no consensus as to what schizoaffective psychosis is, and this concept is not to be compared with schizophrenia in its clarity and usefulness" (p. 197). Moreover, Andreasen (1987) has suggested that schizoaffective disorder probably refers to a "mixed bag" and that "the boundary between schizophrenia and affective disorders must remain flexible..." (p. 13). A notion consistent with a flexible

approach is that schizoaffective disorder may represent "an intermediate region on a unidimensional or hierarchical continuum of psychotic illness" (Tsuang & Simpson, 1984, p. 22). This notion was put forward earlier by Foulds and Bedford (1975). These authors proposed that the psychiatric symptoms range from nonspecific psychological symptoms through neurotic and affective psychoses to schizophrenia and finally organic brain disease (Leff et al., 1988b). According to this scheme "a patient with schizophrenia should always exhibit symptoms of affective psychoses such as grandiose delusions, specific neurotic symptoms, and non-specific neurotic symptoms" (Leff et al., 1988b, p. 29). This particular scheme is consistent with Hirsch's (1982, 1986) contention that depressive symptomatology is an integral part of the schizophrenia illness. The fact that depression in schizophrenia is a relatively common phenomenon, together with Hirsch's viewpoint and the Foulds and Bedford schema would seem to make the diagnostic category of schizoaffective disorder rather redundant (Leff et al., 1988). It is this flexible approach to the understanding of depression in schizophrenia which seems to be most able to account for the diverse data which has been generated to date.

Depression in Paranoid Schizophrenia

As previously outlined, there is uncertainty as to whether depression occurs more often during the early, acute phase of schizophrenia (Hirsch, 1982), during the post-psychotic phase (Siris et al., 1984), or during both the acute phase and after acute psychotic schizophrenic episodes (Lerner & Moscovich, 1985). All

three viewpoints are consistent with Zigler's and Glick's contention that in paranoid schizophrenia, there is the expectation for "depressive symptomatology to be manifested either in the early stages of the formation of delusions or at the time when paranoid symptomatology is abating" (Zigler & Glick, 1986, p. 161-162). In contrast, when the delusion is well-formed and intact, evidence of depression should be minimal. In addition, there should be an inverse relationship between depressive and paranoid symptomatology during the course of the disorder, and depression should be found more in paranoid schizophrenia than in any other subtype (Zigler & Glick, 1984, 1986, 1988).

The vast majority of studies examining depression in schizophrenia included in McGlashan and Carpenter's (1976a) review, did not differentiate schizophrenia by subtype. This trend has not been altered much to date. In the Siris et al. (1984) study of course-related depressive syndromes in schizophrenia, although not directly stated, the results showed that paranoid subtypes were among the highest to exhibit depressive symptomatology when compared to nonparanoid and chronic schizophrenics. Peterson and Seligman (1984) have noted that early parental loss has been implicated in the development of depression later in life. This may have direct bearing on the Roy (1980) finding that of the 100 chronic paranoid schizophrenics, 30% became depressed and had suffered an early parental loss.

Suicide in Schizophrenia

Unsurprisingly, depression secondary to schizophrenia has been

linked to suicidal behavior in schizophrenics (Black, Winokur, & Warrack, 1985). Although many risk factors for suicide have been identified, attempts to predict which schizophrenics will suicide have been unsuccessful because too many false positive and false negative patients are being identified with the various combinations of risk factors (Roy, 1986). Studies on suicide in schizophrenics add further support for Zigler's and Glick's contention that more paranoid schizophrenics should be depressed than any other subtypes. For example, Drake, Gates, Whitaker, and Cotton (1985) reported that schizophrenics who suicide tend to be more educated. From the evidence cited by Magaro (1981) on the cognitive capabilities of paranoid schizophrenics, they would likely be more educated than the other subtypes. Although Drake et al. (1985) have cited several studies which suggest that paranoid schizophrenics have a higher suicide risk than other diagnostic groups, these authors also contend that there are no controlled studies to support this claim. However, Roy (1982) showed that of 24 male schizophrenics who suicided 16 were undifferentiated while eight were paranoid, and of six female schizophrenics who suicided one was undifferentiated while five were paranoid. More recently, Harrow and Westermeyer (in press) noted that paranoid schizophrenics are more likely to commit suicide than other subtypes and that low self-esteem is a risk factor for suicide in schizophrenics. This latter finding has also been noted by Roy, Thompson, and Kennedy (1983). In addition, Drake and Cotton (1986) noted that depressed schizophrenics with feelings of hopelessness are at a greater risk for suicide than non-depressed

patients with schizophrenia.

One might expect that suicidal behavior in paranoid schizophrenics may be a function of their delusions. In his review of the evidence Roy (1986) suggested that command auditory hallucinations or persecutory delusions are probably infrequent causes of suicidal behavior in schizophrenics. Drake, Gates, Cotton, and Whitaker (1984) agree that "suicidal behavior in schizophrenics seems to be motivated by a nondelusional assessment of the future ... [but is] expressed in terms of which are neither mood-distorted nor thought-disordered" (p. 616). Rather, "suicide occurs more frequently during periods of depression and hopelessness than during episodes of intense psychosis" (Drake et al., 1985, p. 90). As discussed previously, feelings of alienation and personal powerlessness have been implicated in the development of paranoid thinking. Analogous to this is the relationship of personal helplessness in the development of depression. Gotlib and Beatty (1985) noted that depressive behavior "only serves to further alienate others from the depressive ... and in the extreme can result in complete withdrawal ..." (p. 101). In his summary remarks at the Depression in Schizophrenia conference (July 7, 1988) Dr. Bryan Tanney concluded that in order to help prevent suicide in schizophrenics efforts must be made to prevent their alienation. These parallels which have been drawn not only serve to elucidate the relationship between depression and paranoia, but also serve to explain some of the evidence for depression in schizophrenia.

RATIONALE OF THE STUDY

As previously mentioned Coyne and Gotlib (1983) noted that many of the preliminary investigations of attributional style used student samples which may have compromised the validity of a depressive attributional or explanatory style. Since that time there has been a widespread acknowledgement that clinical samples of depressed individuals need to be evaluated (Eaves & Rush, 1984; Peterson et al., 1981; Seligman et al., 1979) in addition to other psychiatric populations (Brodbeck & Michelson, 1986; Love, 1988; Peterson & Seligman, 1984), particularly in view of the controversy which surrounds the reformulated learned helplessness model. Although the existence of a depressive explanatory style appears to be well established (Coyne & Gotlib, 1983; Peterson & Seligman, 1984; Peterson, Villanova, & Raps, 1985; Sweeney et al., 1986), it has also been recognized that there is a need for samples to be somewhat more homogeneous with respect to diagnosis (Ganellen, 1988). Depue and Monroe (1978) noted that Abramson, Seligman, and Teasdale (1978) did not specify what kind or degree of severity of depression upon which their theory was built. Depue and Monroe (1978) have also noted that the clinical manifestations of depressive disorders are enormously heterogeneous and that distinctions should be made between unipolar and bipolar depressive illness. Hamilton and Abramson (1983) have also noted that the cognitive models (Beck, 1967, 1976; Abramson, Seligman, & Teasdale, 1978) have addressed depression alone and not mania. Recently, however, Seligman et al.(1988) in an investigation comparing

attributional style in unipolar and bipolar depressives, found that both groups had a depressive explanatory style with no difference between unipolar versus bipolar depressives and no differences for endogenous versus nonendogenous unipolar depressives. In addition, it is important to distinguish between psychotic and nonpsychotic depression since there is often an indistinct boundary between psychotic depression and schizophrenia (Hamilton & Abramson, 1983), with respect to potential similarities in symptoms such as delusions, hallucinations, withdrawal and apathy to name a few.

Although there have been studies which have looked at the attributional style of subjects other than depressives such as chronic pain patients (Love, 1988), agoraphobics (Brodbeck & Michelson, 1987), anxiety disorders (Ganellen, 1988) to name three, there have been very few investigations of the attributional style in paranoia (i.e., paranoid disorders or paranoid schizophrenia). Shean (1978) has noted that the attributional theory has not been applied to schizophrenia because attributions rely on cognitive processes, which are considered to be disturbed in schizophrenics. Nevertheless some investigations, albeit a few, have examined attributional patterns in such patients. Rossler and Lackus (1986) noted that schizophrenics tend to make more uncommon attributions and tend to make more external causal attributions "...even when interpreting situations that should usually be explained by their special circumstances" (p. 386). This study did not however distinguish between the various subtypes of schizophrenia.

In a series of studies by Shaver et al. (1984) attributions of

causality were examined in paranoid, nonparanoid and undifferentiated schizophrenics. In several laboratory tasks it was found that there were no differences between paranoids and nonparanoids in how they attributed blameworthiness. Paranoids showed more sophistication in their attributions than nonparanoids indicating cognitive processes which are perhaps more intact in the paranoids. It is important to note that the tasks used in these studies are not analogous to the attributional measures typically used (i.e., the ASQ) and reported in the literature. Consequently, the relevance and applicability to the reformulated learned helplessness model of depression and to depression itself may be quite limited. However, Zimmerman, Coryell, Corenthal, and Wilson (1986) have compared the attributional style of healthy controls, schizophrenics, psychotic depressives, and nonpsychotic depressions using the ASQ. The results showed the expected differences between depressed and healthy controls, but there were no differences between psychotic versus nonpsychotic depressives. The schizophrenics scored higher than the healthy controls on the globality dimension, but there was no difference between schizophrenics and healthy controls on the internality, stability and composite scores. In addition, there were no significant differences between schizophrenic and depressed patients. Zimmerman et al. (1986) noted that many of the schizophrenic patients had significant levels of depression. Although when the data was reanalyzed with the exclusion of those depressed schizophrenics the findings remained the same. This study, however,

did not examine schizophrenic subtypes (i.e., paranoid schizophrenia) and these investigators relied exclusively on the Hamilton Rating Scale for depression without including a comparable self-report measure of depression in order to help boost their diagnostic reliability. In addition, originally there were 25 schizophrenics, 50 controls, 57 psychotic depressives and 87 nonpsychotic depressives who completed the ASQ. When the depressed schizophrenics were excluded only 15 schizophrenics remained. The fact that there were such unequal groups may have compromised the "power of the analysis" (Kraemer & Thiemann, 1987, p. 42).

In another study, Romney and McElheran (1987) compared personality attributional style in paranoid individuals (with diagnoses ranging from paranoid personality to paranoid schizophrenia) and depressives and found no significant differences in attributional style between these two groups. Nevertheless, this study, along with Shaver et al. (1984) tend to support Zigler's and Glick's contention that depression may underlie paranoid symptomatology. In the Romney and McElheran study it was suspected that the paranoid group may have been contaminated with individuals who had both paranoid and depressive symptomatology (D.M. Romney, personal communication, November 16, 1987). A similar fate may have befallen the results of the Shaver et al. (1984) study. Conversely, in a study evaluating affective impairment in young acute schizophrenics, Lindenmeyer and Kay (1987) found that paranoid schizophrenics were rated as less depressed than their nonparanoid counterparts at baseline, but after two years inappropriate affect

characterized the paranoids. The results of this study may have been compromised by the fact that these investigators used indirect indicators of depression such as speech rate, sad affective voice tone, spontaneity of thought, facial expression, eye contact and vocal emphasis, and did not examine more conventional correlates of depression such as sadness, tearfulness, suicidal ideation, hopelessness and some of the negative signs all of which are more salient concomitants of depression. The fact that these investigations found the paranoids to have more inappropriate affect by examining affective lability, and angry tone, is not surprising since paranoids are typically emotionally labile and filled with anger. This study, along with Romney and McElheran (1987) and Shaver et al. (1984), emphasizes that the relationships between schizophrenia and depression and paranoia and depression do not lend themselves to easy explanations.

It appears that no study has examined attributional style of paranoid individuals while simultaneously examining levels of depression, paranoia and self-esteem. One would expect depressed individuals to blame themselves, while paranoids blame others for bad events in their lives. However, with respect to Zigler and Glick, it is unknown whether the ASQ would measure the attributional style of the genotype (depression) or the phenotype (paranoia) or some mixture of the two. Consequently, in order to discriminate between depression and paranoia, measures of the level of severity of each of these dimensions will be employed. This will enable this researcher to account for differences in attributional style as a

function of depression or paranoia, particularly in cases where depressive and paranoid symptomatology co-exist in single individuals. That is, according to Zigler and Glick, where the depression is incompletely masked by paranoid symptomatology. This is particularly important given that past studies of attributions in paranoids did not control for the presence of depressive symptomatology. Finally, low self-esteem is associated with both depression and paranoia as reported by cognitive theorists and psychodynamic theorists, respectively. As previously mentioned, there are very few studies which relate depression and attributional style to low self-esteem in depressed patients (and paranoid patients) (Zautra et al., 1985). In the case of paranoia, however, low self-esteem is thought to be defended against through the paranoid delusion. In a single study which examined whether individuals with bipolar affective disorders have feelings of low self-esteem, when not in the depressive phase of their illness (Winters & Neale, 1985) found that remitted bipolars scored the same as normals and higher than remitted depressives on self-esteem. Conversely, these same bipolars tended to make inferences about the causes of failures in a similar fashion as depressives "thereby suggesting the presence of low self-worth schema" (p. 282). As previously mentioned, Zigler and Glick contend that paranoia and mania resemble one another. However, when depressive and paranoid symptomatology occur together it is unclear how self-esteem will be affected. Moreover, it is unclear how measures of attributional style, self-esteem, depression and paranoia will correlate with each

other. Therefore, due to the exploratory nature of this study specific hypotheses cannot be stated. However, there are several research questions which will be addressed: (1) How are attributional style and self-esteem affected by individuals who are depressed, paranoid, or both? (2) What is the clinical relationship between paranoia and depression? and (3) Does paranoia substitute for depression in some cases?

CHAPTER THREE

METHODOLOGY

Subjects

The participants in this study were 45 English speaking, patient volunteers who were either being treated for depression, paranoid disorder or paranoid schizophrenia on an inpatient or outpatient basis at two major hospitals in Calgary, Alberta, namely the Holy Cross and Bow Valley Centre (Calgary General Hospital). Inpatient subjects were obtained from the psychiatric wards of these two hospitals, and the outpatient subjects were obtained from the Psychiatric Day Hospital Program located within the Bow Valley Centre. All subjects who were seen while they were hospitalized were considered to be in the active phase of an illness whether they were depressed or paranoid. Those subjects who were collected from the outpatient service were considered to be having exacerbations of their symptomatology at the time of their participation in this study.

The 45 subjects (22 female, 23 male) were divided into three groups of 15. This number of subjects per group was sufficient for an effect size of one standard deviation, a power of 80% and a p value of .05 (Bartko, Carpenter, & McGlashan, 1988). The first group (paranoid only) consisted of 3 female and 12 male subjects with a mean age of 37.47 years and a standard deviation of 11.89. These subjects were diagnosed as having either a paranoid disorder (N=4) or paranoid schizophrenia (N=11) with no accompanying signs of

depressive symptomatology. The mean duration of illness was 9.93 years with a standard deviation of 9.15. Only one subject (a male paranoid disorder) had no previous admissions and/or treatment. With the exception of one female and one male, both diagnosed with paranoid disorder, all other subjects in this group were treated with psychotropic medication (major tranquilizers such as chlorpromazine and thioridazine), eight of whom were also treated with antiparkinsonian (anticholinergic) agents such as benztropine and trihexyphenidyl.

The second group consisted of 5 female and 10 male subjects with a mean age of 37.47 years and standard deviation of 13.65, who were diagnosed as having either a paranoid disorder (N=?) or paranoid schizophrenia (N=13). These subjects also exhibited a significant degree of depressive symptomatology in conjunction with and perhaps secondary to their primary diagnosis. As a group, these subjects were drawn from the total number of paranoid patients based on a cut-off score of greater than or equal to 10 on the Beck Depression Inventory (BDI; Beck et al., 1961) since scores of less than 10 are not considered to be depressed. The mean duration of illness for this group was 8.87 years with a standard deviation of 9.15. Only one subject (a male paranoid disorder) had had no previous admissions and/or treatment. All subjects in this group were being treated with psychotropic medication (major tranquilizers). In addition, two of these subjects were also treated with antidepressants and six also with antiparkinsonian (anticholinergic) agents.

The third group (depressed only) consisted of 14 female and 1 male subject with a mean age of 41.93 years and a standard deviation of 11.63 who were all diagnosed with having a major unipolar depression and exhibiting no paranoid symptomatology. The mean duration of illness (based on the length of time from a patient's first hospital admission to the time of this study) was 5.20 years with a standard deviation of 5.89. Only one subject in this group had no previous admissions and/or treatment. With one exception, all subjects in this group were being treated with electroconvulsive therapy (ECT) and/or psychotropic medication (antidepressants, major or minor tranquilizers, or sedatives).

With the following selection criteria, patients were included in the study if, (1) they were adults (age 18 or older); (2) they had a diagnosis of major depression (nonpsychotic), paranoid disorder or paranoid schizophrenia as outlined in DSM-IIIR (1987); (3) they had no recent history of substance abuse; and (4) substance abuse was not a precipitating factor in their presenting problem.

Although diagnoses were made by each subject's psychiatrist, in order to ensure diagnostic conformity, this researcher confirmed each diagnosis using DSM-IIIR (1987) criteria. With respect to schizophrenia, in a review of several diagnostic systems including DSM-III (1980), it was concluded that no one criteria for diagnosis is better than another because all lack construct validity and therefore to choose one over another was strictly arbitrary (Fenton, Mosher, & Matthews, 1981). However, Gruenberg, Kendler, and Tsuang (1985) have noted that with DSM-III, Research Diagnostic Criteria

(RDC) and Tsuang-Winokur systems, the diagnosis of the paranoid subtype of schizophrenia is most reliable. Moreover, the DSM-III criteria have an intermediate degree of diagnostic stringency between that of the most stringent Tsuang-Winokur system and the least stringent ICD-9. Selection criterion (3) and (4) were satisfied following a review of each patient's medical chart, a brief interview of each patient by this researcher and thorough confirmation from individual therapists, psychiatrists and/or nursing staff.

Psychological Measures

Beck Depression Inventory (BDI) - The Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961) is a 21 item self-report inventory designed to assess the severity of current depressive symptomatology in the areas of affect, cognition, behavior and negative signs. Data for the validity and reliability with a number of patient and nonpatient samples have been reported by Beck et al. (1961), Beck (1970), Beck, Steer, and Garbin (1988), Bumberry, Oliver, and McClure (1978), and Strober, Green, and Carlson (1981). The more recent version (Beck, Rush, Shaw, & Emery, 1979) in which subjects are asked to report about their feelings during the past week, was used as an independent measure of the severity of depression and to identify those paranoid individuals who were also experiencing depressive symptomatology. Scores of 0 to 9 are generally considered normal, 10 to 15 mild, 16 to 19 mild to moderate (dysphoria), 20 to 29 moderate to severe and, 30 to 63 severe (Beck, 1970).

Hamilton Rating Scale for Depression (HRSD) - The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960, 1967) is the most frequently used clinician rating scale for depression. It is a 17 item clinician-rated scale used as an objective measure of the severity of depressive symptomatology. Acceptable validity and reliability data for the scale's total score has been reported by Bech et al. (1975) and Robins (1976). The HRSD was included as a measure of concurrent validity for the severity of depressive symptomatology.

There are very few other observer-rating scales for depression in current use, and they have generally shown a lower validity than the HRSD (Hamilton, 1982). Moreover, self-assessment scales tend to have a lower concurrent validity among themselves than with clinician-rated scales (Hamilton, 1982). Steer, Beck, and Garrison (1982) have noted that there is generally a high level of agreement between the BDI and clinician-rated levels of depression, and correlations in the magnitude of 0.90 have been reported. As noted by Kendall, Hollon, Beck, Hammen, and Ingram (1987) and Snaith (1981), both clinician-rated and patient-rated scales are liable to their own sources of error, so consequently investigations using both types of measures tend to produce more soundly based conclusions. Various cutoff points have become standard for determining levels of severity with the HRSD. Total scores of less than 6 essentially mean no depression, greater than 17 mild to moderate depression, and scores greater than 24 for severe depression (Endicott, Cohen, Nee, Fleiss, & Sarantakos, 1981).

Coopersmith Self-Esteem Inventory (CSEI) - Adult Form - The Coopersmith Self-Esteem Inventory - Adult Form is a 25 item self-administering questionnaire used to evaluate attitudes toward the self in individuals aged 16 and older. The adult form was adapted from the school short form. The wording was changed in eight items to reflect adult lifestyles and experiences and was designed to evaluate the self-esteem with respect to his or her sense of capability, significance, successfulness and worthiness (Coopersmith, 1984). Ahmed, Valliant, and Swindle (1985) found with the exception of Cronbach's alpha value of .75, that the adult form is heterogeneous and congruent with Coopersmith's concept of self-esteem. Acceptable validity and reliability data has been cited for the school forms; however, correlations of total scores on the school forms and the adult form have been found to exceed .80 (Coopersmith, 1984). Although no discrete cutoff scores are given, Coopersmith has noted the upper quartile as being indicative of high self-esteem; lower as being indicative of low self-esteem, and the interquartile range as being indicative of medium self-esteem (Coopersmith, 1984). Although there do not appear to be many investigations using the adult form, specific scores were not required in this study. Rather, this inventory was included as a dependent measure used to gauge relative differences in self-esteem in the three groups. The inventory itself, was chosen for its simplicity and the relatively short amount of time required by the patient for its completion.

MMPI Clinical Scale Pa (#6) - In order to have a self-report

measure of the degree of paranoid symptomatology or paranoid thinking the 40 items which comprise the paranoia clinical scale of the Minnesota Multiphasic Personality Inventory Form-R (MMPI; Hathaway & McKinley, 1964, Appendix A) were isolated from the entire questionnaire in order to make a 40 item true or false questionnaire. Although each scale of the MMPI has its own reliability and validity, there has been only limited study of the individual items on scale 6. Reliability coefficients for scale 6 using psychiatric patients have been cited as .65 and .75 (Hathaway & McKinley, 1983). Greene (1980) has noted that "regardless of the method used to develop subscales within scale 6, three factors are identified" (p. 95), they are Persecutory Ideas, Poignancy and Naivete. Dahlstrom, Welsch, and Dahlstrom (1972a) have noted that the paranoia scale is the most easily faked perhaps because more items are obvious (23) than subtle (17) with respect to what is being measured, and the scale is subject to all true response sets since there are more true (24) than false (16) deviant responses to the paranoia scale. While there is controversy with respect to which items, obvious or subtle, best discriminate level of paranoids from nonparanoids, Hovanitz, Gynther, and Green (1985) have noted that the full scale appears to possess more discriminant validity than the obvious or subtle subscales. Their results did not support the response set interpretation, but did offer additional support for the construct validity of the full scale. Dahlstrom et al. (1972b) noted that this scale is quite sensitive to fluctuations in the degree and intensity of delusional material in psychiatric

cases, albeit, the research evidence is limited. This scale was used as an independent measure of paranoid ideation. Once each subject's scale was scored, the results for each were converted to a T score, which was calculated using a profile sheet. T scores in the 45 to 59 range were considered normal, while elevations in the 60 to 69 range and 70 and above range are considered to include moderate and marked levels of paranoid symptomatology, respectively.

Maine Scale - The Maine Scale (Magaro, Abrams, & Cantrell, 1981, Appendix B) is a brief clinician-rated scale composed of two 5 item scales for paranoid and nonparanoid schizophrenia. Each item requires rating one symptom on a 5-point, anchored, Likert-type scale. The ratings on each 5-item scale are summed to yield scores for paranoid and schizophrenic symptomatology. The information for the scale can be obtained from both direct interview and medical records (Magaro, 1981). For this reason, the Maine Scale is constructed to measure current and long term state. However, when the scale is completed with interview information, a relationship to degree of pathology has been found (Magaro et al., 1981). Acceptable reliability and validity data have been reported by Magaro et al. (1981). This scale was included in this study as an objective independent measure of paranoid symptomatology and was completed for each subject using information gained from the psychiatric interview.

Attributional Style Questionnaire (ASQ) - The Attributional Style Questionnaire (ASQ; Peterson et al., 1982, Appendix C; Peterson & Villanova, 1988) is a 60 item scale designed to measure

an individual's causal attributions for negative and positive outcomes along the dimensions of internal versus external, stable versus unstable and global versus specific as well as the degree of importance people attach to negative and positive outcomes. For the positive and negative events, three attributional style scores corresponding to the three dimensions are calculated by summing over the appropriate items and dividing by 6. In addition positive and negative event composite scores can be calculated by averaging the appropriate dimension scores for the 6 positive and 6 negative events. As previously mentioned, Peterson and Seligman (1984) in their review found that depressive deficits were associated with a pessimistic explanatory style in students, depressed patients, prisoners, and children. Moreover, in their meta-analytic review of 104 studies, Sweeney, Anderson, and Bailey (1986) found strong evidence of a relationship between attributional style and depression. Peterson et al. (1982) recommended using composite scores to obtain higher internal consistencies than are possible with the scores for a single dimension. These authors reported alpha coefficients of .75 and .72 for the composite scale scores for positive and negative outcomes, respectively, while obtaining a mean coefficient alpha of only .54 (range from .44 to .69) for individual dimensions. The test-retest correlations for the composite scale scores over a 5-week period for positive and negative outcomes over .70 and .64 respectively, in a non-clinic sample (Peterson et al., 1982). Major criticisms of the validity and reliability of the ASQ (Arntz, Gerlsma, & Albersnagel, 1985) must be considered with

caution, given that the sample used in their validation study included young adolescents who were not clinically depressed, but rather were subjected to minor artificial laboratory manipulations of mood. This is of particular importance given the fact a Dutch-language version (DASQ) of the ASQ was found to be related to depression in ways that the ASQ has been shown to be and that differences between Dutch and American samples may reflect cultural differences (Cohen, vanden Bout, Kramer, & van Vliet, 1986). More recently, in a study examining whether test-takers can fake the test, Schulman, Seligman, and Amsterdam (1987) found that when individuals were given financial incentive and/or explanation on how to beat the test, there were no significant differences between these two groups or when measured against a control group; thereby demonstrating the validity of the test. The ASQ was included as the dependent measure of attributional style and was predicted to vary with respect to degree of depressive symptomatology.

Procedure

All subjects for this investigation were obtained by monitoring the appropriate wards through contact with several key people including ward supervisors and psychiatrists who were cooperating with this research. Written consent to see individual patients was obtained from the appropriate psychiatrist as the situation arose, or in the case of the Holy Cross Hospital, global consent was obtained from all but one psychiatrist, which allowed this researcher to see subjects at his own discretion. All subjects were presented with an information sheet concerning the study, prior to

the request to sign a consent form (see Appendix D for information sheet and consent forms).

They were then interviewed briefly to obtain a psychiatric history. More specifically, each patient was asked about sociodemographic information such as, age, date of birth, duration of present illness, history of substance abuse (alcohol and drugs), level of education achieved, and present use of medication. Use of medication and dosage was confirmed through medical chart examination. Patients were also asked about whether they were experiencing delusions and/or hallucinations and if so, to elaborate on them. In depth questions were asked about depressive symptoms as outlined in the clinician-rated HRSD. For example, patients were asked about sleeping or eating difficulties, somatic symptoms, feelings of depression, suicidal ideation, and social activities. Similarly, symptoms of paranoia and schizophrenia were explored within the context of the clinician-rated Maine scale which pursues information about delusions, ideas of reference, grandiosity, hostility, hallucinations, thought processes and emotionality. In addition, symptoms of depression, paranoia and schizophrenia were evaluated using the diagnostic criteria for mood disorders, schizophrenic disorders (paranoid schizophrenia) and delusional disorders found in the DSM IIIR (1987). Subsequently, four questionnaires were administered in a random fashion and on an individual basis: Beck Depression Inventory (BDI), Attributional Style Questionnaire (ASQ), Coopersmith Self-Esteem Inventory (CSEI), and 40 true and false questions which constitute the paranoia

clinical scale of the Minnesota Multiphasic Personality Inventory (MMPI). All questionnaires were completed in the privacy of an interview room while in the presence of this researcher so as to curtail any problems or misunderstandings that may have arisen. The scores on all questionnaires for all subjects were collected and compiled into the three subject groups for data analyses. All individuals participating in the study were guaranteed anonymity and that their results would be used for research purposes only. No follow-up participation was solicited. Patient's individual results were provided to their psychiatrist and/or therapists if the patient gave his or her approval.

Data Analyses

Descriptive statistics (means and standard deviations) for the subject sample both in total and subdivided by group were calculated for all psychological measures (BDI, HRSD, COOP, MAINE, MMPI, and ASQ). Analyses of variance were performed to examine between group differences on the sociodemographic variables of age and duration of illness. Chi^2 tests were used to evaluate differences in the current use of psychotropic medication by group, sex distribution by group, differences in level of education, past history of substance abuse, and differences in suicidal ideation. Differences between proportions was used to evaluate differences in delusional content. Further analyses of variance were performed to determine any between group differences on the psychological measures. All significant findings were scrutinized post hoc using the Tukey (Honestly Significant Difference) test. Pearson product-moment correlations

were computed among the sociodemographic variables of age and duration of illness and for all the psychological variables.

CHAPTER FOUR

RESULTS

Sociodemographic Variables

The three experimental groups (paranoid, paranoid/depressed, depressed) were compared on the following sociodemographic variables: diagnosis, age, duration of illness, sex, use of psychotropic medication, education and history of substance abuse.

Although a diagnosis of paranoid schizophrenia predominated in both the paranoid and paranoid/depressed groups, proportionally there was no significant difference with respect to diagnosis in these two groups [$chi^2(1)=.83$, $p > .05$]. Furthermore, and as expected, only patients with the diagnosis of a major depression were included within the depressed group (see Table 1). A summary of the means and standard deviations for age and duration of illness for all subjects by group are presented in Table 2. Analyses of variance revealed that there was no significant between group differences with respect to age [$F(2,42)=0.65$, $p=.53$] or duration of illness [$F(2,42)=1.37$, $p=.26$] (see Table 2). However, there was a significant difference in the number of males and females in each group [$chi^2(2)=18.32$, $p < .05$]. Overall, there were more females in the depressed group and more males in the paranoid and paranoid/depressed groups (see Table 3). In addition, there was no significant difference among the groups with respect to the use of psychotropic medication [$chi^2(2)=2.14$, $p > .05$]. Almost all

Table 1

Breakdown of Diagnosis by Group

Group	Diagnosis		
	Paranoid Schizophrenia	Paranoid Disorder	Major Depression
Paranoid	73.3% n=11	26.7% n=4	0% n=0
Paranoid/Depressed	86.7% n=13	13.3% n=2	0% n=0
Depressed	0% n=0	0% n=0	100% n=15

Table 2

Means and Standard Deviations for Age and Duration of Illness

	Group						Total	
	Paranoid n=15		Paranoid/ Depressed n=15		Depressed n=15		N=45	
Variable	M	SD	M	SD	M	SD	M	SD
Age	37.47	11.89	37.47	13.65	41.93	11.63	38.96	12.32
Duration of Illness	9.93	9.15	8.87	9.15	5.20	5.89	8.00	8.28

Table 3

Frequency of Males and Females for the Groups

Group	Male	Female
Paranoid	80% (n=12)	20% (n=3)
Paranoid/Depressed	66.7% (n=10)	33.3% (n=5)
Depressed	6.7% (n=1)	93.3% (n=14)

subjects in all three groups were receiving some form of medication during their participation in this study (see Table 4). Finally, there were no significant differences among the three groups in educational level achieved [$chi^2(4)=8.74$, $p > .05$] (see Table 5) or past history of substance abuse (illicit drugs and/or alcohol) [$chi^2(2)=3.15$, $p > .05$] (see Table 6).

Psychological Measures

A summary of the means and standard deviations of all psychological measures including the sociodemographic variables of age and duration of illness for all subjects by group are presented in Table 7.

Analyses of Variance

ANOVA's were conducted on each of the nonattributional psychological measures in order to identify those measures which were able to successfully discriminate among the three groups (paranoid, paranoid/depressed, depressed). The ANOVA's indicated that there were significant differences among the three groups on the following measures: PARA [$F(2,42)=29.64$, $p < .0001$], BDI [$F(2,42)=108.41$, $p < .0001$], HRSD [$F(2,42)=159.99$, $p < .0001$], PA [$F(2,42)=78.92$, $p < .0001$], MMPI6 [$F(2,42)=7.54$, $p < .0016$], COOP [$F(2,42)=42.75$, $p < .0001$], and NONPA [$F(2,42)=9.59$, $p < .0004$). These ANOVA's are presented in Table 8.

ANOVA's were also performed on the attributional psychological dimensions. These ANOVA's indicated that there were significant differences among the three groups on the following dimensions: INTG [$F(2,42)=38.32$, $p < .0001$], STABG [$F(2,42)=19.44$, $p < .0001$],

Table 4

Use of Psychotropic Medication within each Group

Group	Currently Taking Medication	Not Taking Medication
Paranoid	86.7% (n=13)	13.3% (n=2)
Paranoid/Depressed	100% (n=15)	0% (n=0)
Depressed	93.3% (n=14)	6.7% (n=1)

Table 5

Educational Level Achieved for Each Group

	Education		
Group	Junior High	Senior High	University
Paranoid	6.7% (n=1)	33.3% (n=5)	60% (n=9)
Paranoid/Depressed	13.3% (n=2)	60% (n=9)	26.7% (n=4)
Depressed	0% (n=0)	80% (n=12)	20% (n=3)

Table 6

Past History of Substance Abuse for the Groups

Group	Substance Abuse Yes	No
Paranoid	13.3% (n=2)	86.7% (n=13)
Paranoid/Depressed	20% (n=3)	80% (n=12)
Depressed	0% (n=0)	100% (n=15)

Table 7

Means and Standard Deviations on all Psychological Measures

Variable *	Group: Paranoid n=15 M	SD	Paranoid/Depressed n=15 M	SD	Depressed n=15 M	SD	Total N=45 M	SD
BDI	5.87	2.97	19.53	6.58	33.87	5.41	19.76	12.63
HRSD	4.60	2.35	13.47	3.98	26.80	3.71	14.96	9.81
COOP	77.33	11.97	45.07	16.39	27.20	16.37	49.87	25.62
PA	15.40	2.44	11.93	2.66	5.73	0.80	11.02	4.55
NONPA	7.40	1.76	7.67	1.80	5.47	0.64	6.84	1.77
PARA	8.00	3.42	4.27	3.10	0.27	1.16	4.17	2.56
MMPI6	74.60	9.63	73.00	9.85	63.67	4.29	70.42	9.49
INTG	6.42	0.66	5.31	0.78	3.97	0.85	5.23	1.26
STABG	6.07	0.83	4.99	0.88	4.23	0.73	5.10	1.10
GLOBG	5.85	0.94	4.47	1.16	4.61	0.70	4.98	1.12
IMPG	6.17	0.96	5.50	1.14	5.27	0.93	5.64	1.06
GOOD	6.09	0.71	4.92	0.64	4.27	0.58	5.09	0.99
INTB	3.95	1.12	4.59	1.35	5.91	0.57	4.81	1.33
STABB	4.53	1.15	4.33	0.92	5.18	0.64	4.68	0.98
GLOBB	4.21	1.31	3.89	1.05	5.32	0.71	4.47	1.20
IMPB	5.14	1.35	5.38	1.37	5.64	0.84	5.39	1.20
BAD	4.22	0.95	4.27	0.98	5.48	0.56	4.66	1.02

* Key on next page

Table 7 (continued) KEY

BDI	Beck Depression Inventory
HRSD	Hamilton Rating Scale for Depression
COOP	Coopersmith Self-Esteem Inventory (Adult Form)
PA	Paranoid subscale of the Maine Scale
NONPA	Nonparanoid subscale of the Maine Scale
PARA	PA minus NONPA
MMPI6	Paranoid scale of the MMPI
INTG	Internality dimension (good events) of the ASQ
STABG	Stability dimension (good events) of the ASQ
GLOBB	Globality dimension (good events) of the ASQ
IMPG	Importance dimension (good events) of the ASQ
GOOD	Composite for good events of the ASQ
INTB	Internality dimension (bad events) of the ASQ
STABB	Stability dimension (bad events) of the ASQ
GLOBB	Globality dimension (bad events) of the ASQ
IMPB	Importance dimension (bad events) of the ASQ
BAD	Composite for bad events of the ASQ

Table 8

Analyses of Variance for the Nonattributional Psychological Measures PARA, BDI, HRSD, PA, MMPI6, COOP, NONPA

	Source of Variation	SS	df	MS	F	p
PARA	Between Groups	448.71	2	224.36	29.64	.00001
	Within Groups	317.87	42	7.57		
	Total	766.58	44	231.93		
BDI	Between Groups	588.11	2	2940.56	108.41	.00001
	Within Groups	1139.20	42	27.12		
	Total	7020.31	44	2967.68		
HRSD	Between Groups	3746.18	2	1873.09	159.99	.00001
	Within Groups	491.73	42	11.71		
	Total	4237.91	44	1884.80		
PA	Between Groups	719.51	2	359.76	78.92	.00001
	Within Groups	191.47	42	4.56		
	Total	910.98	44	364.32		
MMPI6	Between Groups	1046.04	2	523.02	7.54	.0016
	Within Groups	2914.93	42	69.40		
	Total	3960.97	44	592.42		
COOP	Between Groups	19368.53	2	9684.27	42.75	.00001
	Within Groups	9514.67	42	226.54		
	Total	28883.20	44	9910.81		
NONPA	Between Groups	43.24	2	21.62	9.59	.0004
	Within Groups	94.68	42	2.26		
	Total	137.92	44	23.88		

GLOBG [$F(2,42)=9.66$, $p < .0003$], GOOD [$F(2,42)=30.88$, $p < .0001$], INTB [$F(2,42)=13.22$, $p < .0001$], STABB [$F(2,42)=3.40$, $p < .0428$]. GLOBB [$F(2,42)=7.69$, $p < .0014$], and BAD [$F(2,42)=10.57$, $p < .0002$). These ANOVA's are presented in Table 9. There were no significant between group differences on the IMPG dimension [$F(2,42)=3.19$, $p > .05$] (although this was almost significant at $p=.05$) or the IMPB dimension [$F(2,42)=.64$, $p=.53$], (See Table 9).

Tukey HDS post hoc analyses were completed on all the ANOVA's with significant findings (see Table 10). On the PA measure the paranoid group was found to be significantly more paranoid than the paranoid/depressed group which was, in turn, significantly more paranoid than the depressed group. On the MMPI6, the paranoid and the paranoid/depressed groups were found to be significantly more paranoid than the depressed group, but there was no significant difference between the paranoid and paranoid/depressed groups. Similar results were obtained for the NONPA measure. However, when the NONPA measure was subtracted from the PA measure to yield PARA, the paranoid group was found to be significantly more paranoid than the paranoid/depressed group which was, in turn, more paranoid than the depressed group. On the BDI and HRSD the depressed group was significantly more depressed than the paranoid/depressed group which was in turn significantly more depressed than the paranoid group. With the self-esteem measure (COOP), the paranoid group had a significantly higher level of self-esteem than the paranoid/depressed group which in turn had significantly higher self-esteem than the depressed group.

Table 9

Analyses of Variance for the Attributional Psychological Measures: INTG, STABG, GLOBG, IMPG, GOOD, INTB, STABB, GLOBB, IMPB, BAD

	Source of Variation	SS	df	MS	F	p
INTG	Between Groups	45.33	2	22.66	38.32	.00001
	Within Groups	24.84	42	0.59		
	Total	70.17	44	23.25		
STABG	Between Groups	25.80	2	12.90	19.44	.00001
	Within Groups	27.87	42	.66		
	Total	53.67	44	13.56		
GLOBG	Between Groups	17.49	2	8.75	9.66	.0003
	Within Groups	38.03	42	.91		
	Total	55.52	44	9.66		
IMPG	Between Groups	6.52	2	3.26	3.19	.0515
	Within Groups	43.02	42	1.02		
	Total	49.54	44	4.28		
GOOD	Between Groups	25.59	2	12.79	30.88	.00001
	Within Groups	17.40	42	.41		
	Total	42.99	44	13.20		
INTB	Between Groups	29.90	2	14.95	13.22	.00001
	Within Groups	47.52	42	1.13		
	Total	77.42	44	16.08		
STABB	Between Groups	5.83	2	2.92	3.40	.0428
	Within Groups	36.03	42	.86		
	Total	41.86	44	4.78		
GLOBB	Between Groups	16.97	2	8.48	7.69	.0014
	Within Groups	46.33	42	1.10		
	Total	63.30	44	9.58		

Table 9 (continued)

Analyses of Variance for the Attributional Psychological Measures: INTG, STABG, GLOBG, IMPG, GOOD, INTB, STABB, GLOBB, IMPB, BAD

	Source of Variation	SS	df	MS	F	p
IMPB	Between Groups	1.87	2	.93	.64	.5332
	Within Groups	61.42	42	1.46		
	Total	63.29	44	2.39		
BAD	Between Groups	15.32	2	7.66	10.57	.0002
	Within Groups	30.43	42	.73		
	Total	45.75	44	8.39		

Table 10

Tukey Post Hoc Q Values of Group Pairs for the Attributional and Nonattributional Measures

	Q Values		
Measure	Paranoid vs. Paranoid/Depressed	Paranoid vs. Depressed	Paranoid/Depressed vs. Depressed
PA	10.83*	30.21*	19.38*
MMPI6	1.29	7.53*	8.82*
NONPA	1.19	8.67*	9.87*
PARA	9.11*	18.86*	9.76*
BDI	17.61*	36.08*	18.47*
HRSD	17.39*	45.53*	26.14*
COOP	14.40*	22.38*	7.98*
INTG	10.09*	22.32*	12.22*
STABG	9.08*	15.37*	6.29*
GLOBG	9.78*	8.77*	1.01
GOOD	12.19*	18.99*	6.79*
INTB	3.01	12.38*	8.38*
STABB	1.45	2.66	6.11*
GLOBB	2.05	7.08*	9.13*
BAD	0.39	9.94*	9.54*

Note: $Q_{cv}(44)=3.43$, $p=.05$

* denotes significant difference

With respect to the attributional dimensions for good events, post hoc analyses indicated that on internality and stability the paranoid group attributed good events (INTG, STABG) to internal and stable causes significantly more than the paranoid/depressed group which in turn did so significantly more than the depressed groups. Although the paranoid group was found to attribute good events significantly more to global causes than the paranoid/depressed and depressed groups, there was no significant difference between the paranoid/depressed and depressed groups on the globality dimension for good events. Nevertheless, on the composite for good events (GOOD) (average of INTG, STABG, GLOBG) the paranoid group was found to attribute good events to significantly more internal, stable and global causes than the paranoid/depressed group which, in turn, did so significantly more than the depressed group.

With respect to attributions for bad events, post hoc analyses indicated that on internality and globality the depressed group attributed bad events (INTB, GLOBB) to internal and global causes significantly more than the paranoid and paranoid/depressed groups. However, there was no significant difference between the paranoid and paranoid/depressed groups on internality or globality for bad events. On the stability dimension (STABB), the depressed group attributed bad events to stable causes significantly more than the paranoid/depressed group. But there was no significant difference between the depressed and paranoid groups or between the paranoid and paranoid/depressed groups on the stability dimension (STABB). Finally, on the composite for bad events (BAD) the depressed group

was found to attribute bad events significantly more to internal, stable and global causes than the paranoid/depressed and paranoid groups. There was no significant difference between the paranoid and paranoid/depressed group on the BAD dimension.

Correlations Between Measures

Both the self-report (BDI) and clinician rated (HRSD) measures of depression were found to be very highly correlated (r=.94) with one another. Similarly, the Maine paranoid subscale (PA) and the MMPI6 paranoid subscale were correlated (r=.66) with one another, but to a lesser degree than were the depression scales. In addition, PARA was significantly correlated very highly with the PA (r=.92) and moderately highly with the MMPI6 (r=.67). Conversely, there was no significant relationship between NONPA and either of the measures of paranoia. As expected self-esteem (COOP) was negatively correlated with the measures of depression (BDI, r=-.83; HRSD, r=-.78). Conversely, self-esteem (COOP) was positively correlated with the measures of paranoia (PA, r=.69; MMPI6, r=.37). See Table 11 for the correlations for the nonattributional psychological measures.

The Pearson product-moment correlations for the attributional dimensions of the ASQ are shown in Table 12. These results showed that for good events internality (INTG) was significantly correlated with the dimensions of stability (STABG) (r=.70), globality (GLOBG) (r=.47), and importance (IMPG) (r=.28). Stability (STABG) was correlated with globality (GLOBG) (r=.63), and with importance (IMPG) (r=.42). Similarly, globality (GLOBG) was correlated with

Table 11

Pearson Product-Moment Correlational Coefficients
for the Nonattributional Psychological Measures

	HRSD	COOP	PA	NONPA	PARA	MMPI6
BDI	.94**	-.83**	-.79***	-.44**	-.67***	-.33*
HRSD		-.78***	-.82***	-.44***	-.69***	-.37**
COOP			.69***	.26*	.64***	.37**
PA				.23	.92***	.66***
NONPA					.01	.10
PARA						.67***

Note: For a key to these psychological measures see Table 7.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 12

Pearson Product-Moment Correlation Coefficients for the Attributional Dimensions of the ASQ

	STABG	GLOBG	IMPG	GOOD	INTB	STABB	GLOBB	IMPB	BAD
INTG	.70***	.47**	.28*	.86***	-.43**	-.22	-.27**	-.13	-.37**
STABG		.63***	.42**	.90***	-.48***	-.16	.13	-.04	-.33*
GLOBG			.61***	.81***	-.15	-.13	.23	.28*	.06
IMPG				.51***	-.17	-.09	.02	.47**	-.09
GOOD					-.41**	-.10	-.08	-.04	-.25*
INTB						.64***	.61***	.35**	.88***
STABB							.70***	.24	.86***
GLOBB								.37**	.88***
IMPB									.37**

Note: For a key to these attributional dimensions see Table 7.

* $p < .05$, ** $p < .01$, *** $p < .001$.

importance (IMPG) (r=.61). With respect to the correlations with the composite for good events (GOOD), the highest correlation was with stability (STABG) (r=.90) followed by internality (INTG) (r=.86), globality (GLOBG) (r=.81) and finally importance (IMPG) (r=.51).

The results similarly showed that for bad events internality (INTB) was significantly correlated with the dimensions of stability (STABB) (r=.64), globality (GLOBB) (r=.61) and importance (IMPB) (r=.35). Stability (STABB) was correlated with globality (GLOBB) (r=.70), but not with importance (IMPB). However, globality (GLOBB) was correlated with importance (IMPB) (r=.37). With respect to the correlations with the composite for bad events (BAD), the highest correlations were with internality (INTB) (r=.88) and globality (GLOBB) (r=.88) followed by stability (STABB) (r=.86) and finally importance (IMPB) (r=.37).

Through a comparison between the various dimensions for good and bad events, it was found that the internality dimensions for good (INTG) and bad (INTB) events were negatively correlated (r=-.43). Conversely, the importance dimensions for good (IMPG) and bad (IMPB) events were positively correlated (r=.47). However, no significant correlations were found between good and bad events on the stability and globality dimensions.

Shown in Table 13 are the correlations between the attributional and nonattributional psychological measures. The measures of depression (BDI, HRSD) were found to be significantly negatively correlated with the attributional dimensions for good

Table 13

Pearson Product-Moment Correlation Coefficients Between the Attributional and Nonattributional Measures

	INTG	STABG	GLOBG	IMPG	GOOD	INTB	STABB	GLOBB	IMPB	BAD
BDI	-.69***	-.61***	-.42**	-.33*	-.67***	.58***	.29*	.43**	.26*	.52***
HRSD	-.66***	-.61***	-.35**	-.35**	-.63***	.63***	.34*	.48***	.24	.58***
COOP	.65***	.65***	.41**	.19	.66***	-.65***	-.34*	-.48***	-.34**	-.59***
PA	.67***	.55***	.36**	.24	.62***	-.65***	-.41**	-.38**	-.23	-.57***
NONPA	.45**	.38**	.08	.07	.36**	-.18	-.10	-.23	-.10	-.21
PARA	.54***	.44**	.36**	.23	.52***	-.63***	-.40**	-.32*	-.21	-.53***
MMPI6	.47***	.24	.23	.11	.37**	-.51***	-.35**	-.19	-.06	.41**

Note: For a key to these measures see Table 7.

* $p < .05$, ** $p < .01$, *** $p < 001$.

events (INTG, GOOD, STABG, GLOBG, and IMPG) and significantly positively correlated with the attributional dimensions for bad events (INTB, BAD, GLOBB, STABB, and IMPB). Overall for the attributional dimensions for good events, the BDI and HRSD were negatively correlated most highly with internality (INTG) (r=-.69 and r=-.66, respectively) followed by the composite for good events (GOOD) (r=-.67 and r=-.63 respectively), stability (STABG) (r=-.61 and r=-.61, respectively), globality (GLOBG) (r=-.42 and r=-.35, respectively) and finally importance (IMPG) (r=-.33 and r=-.35, respectively). Similarly, with the attributional dimensions for bad events, the BDI and HRSD positively correlated most highly with internality (INTB) (r=.58 and r=.63, respectively), followed by the composite for bad events (BAD) (r=.52 and r=.58, respectively), globality (GLOBB) (r=.43 and r=.48, respectively) and stability (STABB) (r=.29 and r=.34, respectively). For the importance dimension (IMPB) only the BDI was found to correlate significantly (r=.26).

Generally, these correlations suggest that as the level of depression increases, individuals tend to attribute (1) bad events to more internal, stable and global causes, and (2) good events to more external, unstable and specific causes. Conversely, as the level of depression decreases, individuals tend to attribute (1) bad events to more external, unstable and specific causes, and (2) good events to more internal, stable and global causes.

With respect to degree of paranoia, the measures of paranoia (PA, MMPI6) were found to be significantly positively correlated

with the attributional dimensions for good events (INTG, GOOD, STABG, GLOBG) and significantly negatively correlated with the attributional dimensions for bad events (INTB, BAD, STABB, GLOBB). Overall, for the attributional dimensions for good events, the PA and MMPI6 positively correlated most highly with internality (INTG) (r=.67 and r=.47, respectively), followed by the composite for good events (GOOD) (r=.62 and r=.37, respectively). The stability (STABG) and globality (GLOBG) dimensions only correlated with the PA (r=.55 and r=.36, respectively). There were no significant relationships for the dimensions of STABG and GLOBG with the MMPI6. Similarly, there was no significant relationship between the importance dimension for good events (IMPG) and either of the measures of paranoia (PA or MMPI6).

With the attributional dimensions for bad events, the PA and MMPI6 negatively correlated most highly with internality (INTB) (r=-.65 and r=-.51, respectively), followed by the composite for bad events (BAD) (r=-.57 and r=-.41, respectively), and stability (STABB) (r=-.41 and r=-.35, respectively). For the globality dimension (GLOBB), only the PA was found to correlate significantly (r=-.38). As with the importance for good events (IMPG), the importance for bad events dimension (IMPB), was found to have no significant relationship with either of the measures of paranoia (PA, MMPI6).

Generally, these correlations suggest that as the degree of paranoia increases, individuals tend to attribute (1) bad events to more external, unstable and specific causes, and (2) good events to more internal, stable and global causes. Conversely, as the degree

of paranoia decreases, individuals tend to attribute (1) bad events to internal, stable and global causes, and (2) good events to more external, unstable and specific causes.

With respect to the measure of self-esteem (COOP), it was found that self-esteem was significantly correlated positively with the attributional dimensions for good events (GOOD, INTG, STABG, and GLOBG), but negatively correlated with the attributional dimensions for bad events (INTB, BAD, GLOBB, STABB, IMPG). For the attributional dimensions for good events, the self-esteem (COOP) correlated most highly with the composite (GOOD) (r=.65), followed by internality (INTG) (r=.65), stability (STABG) (r=.65), and globality (GLOBG) (r=.41). There was found to be no significant relationship between self-esteem (COOP) and importance for good events (IMPG).

For the attributional dimensions for bad events, the COOP correlated most highly with internality (INTB) (r=-.65), followed by the composite (BAD) (r=-.59), globality (GLOBB) (r=-.48), stability (STABB) (r=-.34), and finally importance (IMPB) (r=-.34). Generally, these correlations suggest that as the level of self-esteem increases, these individuals tend to attribute (1) bad events to more external, unstable and specific causes, and (2) good events to more internal, stable and global causes. Conversely, as self-esteem decreases, these individuals attribute (1) bad events to internal, stable and global causes, and (2) good events to more external, unstable and specific causes.

The nonparanoid subscale (NONPA) was found to correlate with only a few attributional dimensions, namely INTG (r=.45), STABG

(r=.38) and GOOD (r=.36). In contrast PARA (i.e., the paranoid subscale minus the nonparanoid subscale) was found to correlate positively and negatively with most of the attributional dimensions for good and bad events, respectively. For good events, PARA was found to correlate most highly with internality (INTG) (r=.54), followed by the composite (GOOD) (r=.52), stability (STABG) (r=.44) and globality (GLOB) (r=.36). Similarly, for the attributional dimensions for bad events, PARA was found to correlate most highly with internality (INTB) (r=-.63), followed by the composite (BAD) (r=-.53), stability (STABB) (r=-.40), and globality (GLOBB) (r=-.32). Neither of the importance dimensions for good (IMPG) or BAD (IMPB) events was significantly correlated with PARA.

Clinical Observations

Individuals in the depressed only group were very often tearful and looked tired and sad. Many of their responses to questions were filled with sighs, and much of their conversation was lacking in continuity owing either to thought blocking or paucity of thought. This group exhibited the most psychomotor retardation and consequently required the most encouragement in order to complete the tasks at hand. At times, this researcher had to read some of the questions to some of the subjects when they grew tired of reading the questions themselves. Although most subjects in the paranoid and paranoid depressed groups were able to complete the assessment in approximately two hours, the depressed group on average took approximately 20 to 30 minutes longer to complete the assessment. Delusional thought processes were absent from patients in the

depressed group.

The patients in the paranoid/depressed group often appeared to be worried and apprehensive and exhibited more psychomotor retardation than the paranoid group, but less than that exhibited by the depressed group. As a group, the paranoid/depressed, like the depressed, was at times difficult to engage in the assessment process. While the depressed group's hesitancy to participate in the research was likely due to the behavioral, cognitive and motivational concomitants of their depressed affect, the paranoid/depressed groups hesitancy was likely due to a combination of a depressed affect and a general suspiciousness or uneasiness with the assessment/research situation.

In contrast to the depressed and paranoid/depressed groups, the paranoid group seemed to be a little more lively and active. Individuals in the paranoid group were also, at times, reluctant to participate with the research. While the hesitancies and concerns about testing for the paranoid/depressed group were generally more passively concealed and less overtly expressed, the paranoid group generally voiced, quite openly and assertively, their protestations about testing. It is important to note that all subjects, once engaged in the assessment, followed through to completion with their required participation in the study.

With respect to the delusional thought processes of subjects in the paranoid and paranoid/depressed groups, it was possible to compare delusional content through examination of the 5-point ratings for all 5-items which comprise the Maine paranoid subscale (PA).

There were no significant differences between these two groups with respect to ideas of being controlled ($Z=.83$, $p > .05$), delusions of persecution ($Z=.34$, $p > .05$], ideas of reference, being punished or conspired against ($Z=1.15$, $p > .05$]. The trend for the paranoid group to express more hostility than the paranoid/depressed group approached significance ($Z=2.45$, $p > .05$). Finally, the paranoid group was found to be significantly more grandiose than the paranoid/depressed group ($Z=3.78$, $p < .05$). (See Table 14).

Apart from the types of delusions present, frequency of current suicidal ideation was also examined for the three groups. Overall, there were significant differences among the three groups with respect to the presence or absence of suicidal ideation [$chi^2(2)=20.9$, $p < .05$] (See Table 15). More specifically, although there was no significant difference between the paranoid/depressed and depressed groups [$chi^2(1)=.391$, $p=.53$], the paranoid group was found to have significantly less suicidal ideation when compared to both the paranoid/depressed group [$chi^2(1)=7.36$, $p < .007$] and the depressed group [$chi^2(1)=10.29$, $p < .001$].

Table 14

Sum of PA subscale items for the Paranoid and Paranoid/Depressed Groups

Group	PA Item Totals P_1	P_2	P_3	P_4	P_5
Paranoid	46	50	44	47	44
Paranoid/Depressed	41	48	21	40	29

Note: P_1 = being controlled

P_2 = persecution, conspiring, punishing

P_3 = grandiosity

P_4 = ideas of reference, being watched or talked about

P_5 = expression of hostility

Table 15

Presence of Suicidal Ideation by Group

Group	Suicidal Ideation Yes	No
Paranoid	6.7% n=1	93.3% n=14
Paranoid/Depressed	66.7% n=10	33.3% n=5
Depressed	86.7% n=13	13.3% n=2

CHAPTER FIVE

DISCUSSION

The purpose of this study was to examine the relationship between paranoia and depression with respect to attributional style and level of self-esteem, particularly in individuals where both paranoia and depression are present simultaneously. The results of this study will be summarized and interpreted with respect to the significant and nonsignificant differences among the three experimental groups on all of the psychological measures followed by some comments on the results of the correlational analysis. Under the headings of theoretical implications and clinical implications, the issues of attributional style, depression in schizophrenia, suicide in schizophrenia, schizoaffective illness and negative symptoms will be addressed in the light of the current findings. Following this, the limitations of the study and the recommendations for future research will be discussed.

Sociodemographic Variables

Sociodemographic data obtained for this study revealed a significant difference in the number of males and females among the three groups. There were more females in the depressed group and more males in the paranoid and paranoid/depressed groups. This finding is consistent with the fact that there are generally more females afflicted with depression and more males who are schizophrenic (Maxmen, 1986).

Nevertheless, unequal numbers of males and females in each group in no way adversely affected the methodology of this study. The fact that there was no significant between group differences with respect to the duration of illness, age, use of psychotropic medication, level of education or history of substance abuse has eliminated these variables as potential confounding variables whose effect on the results would have had to be explored. Rather, with the exception of sex, these nonsignificant findings demonstrate that these groups, are sociodemographically homogeneous, and at least in this study, any psychological differences among the three groups would more likely be due to differences in their respective psychopathology.

Nonattributional Psychological Measures

With respect to the level of paranoia the PA and the PARA measures were better at discriminating among the three experimental groups than was the MMPI6. The MMPI6, like the NONPA measure could not discriminate between the paranoid only and paranoid/depressed groups. Given that the NONPA subscale is a measure of schizophrenic symptoms, it was not surprising that the two paranoid groups could not be distinguished from one another, since both of these groups are primarily made up of schizophrenic patients. It seems that the self-report measure of paranoia (MMPI6) is less discriminating than the clinician-rated measure (PA). This may be due to the psychometric properties of the scale in that the MMPI6 may not be as sensitive to differences in paranoid symptomatology as the PA measure which assesses more severe types of paranoid symptoms, for

example, different types of delusions (i.e., persecution and grandiose). In addition, the MMPI6 has been noted for being the least reliable subscale of the MMPI (Dahlstrom et al., 1972a). The fact that the paranoid only group were significantly more grandiose than the paranoid/depressed group may account for the PA measure being able to identify significantly more paranoid symptomatology in the paranoid only group than the paranoid/depressed group. Overall, at least for the PA and PARA measures, the paranoid only group had the highest degree of paranoia, while the depressed only group had the least and the paranoid/depressed group fell between these two extremes, but remained significantly different from the other two groups.

Both the measures of depression (BDI and HRSD) and the measure of self-esteem (COOP) were found to be able to discriminate significantly among the three groups. While the depressed only group exhibited the most depression and lowest self-esteem, the paranoid only group exhibited the least depression and the highest self-esteem and the paranoid/depressed group exhibited a level of depression and self-esteem between and significantly different from these two extremes. Although these findings are important in themselves, they will be discussed in relation to the attributional findings and within the context of theoretical and clinical implications.

Attributional Psychological Measures

With respect to the attributional dimensions of the ASQ, it would seem that the attributional dimensions for good events

including the composite (GOOD) were better able to discriminate the three experimental groups from one another than were the attributional dimensions for bad events including the composite (BAD). Moreover, the composite scores for good and bad events were better at discriminating among the three groups than were the individual attributional dimensions. This tends to support the contention of Peterson and Seligman (1984) and Peterson et al. (1982) that the reliability of the ASQ is improved by using the combination of the individual dimensions (composite scores) rather than the individual dimensions separately. Although the results of the individual attributional dimensions will be addressed separately, for most of the following discussion, the composite scores will be primarily used for the interpretation of results.

Based on the composite scores for good and bad events, the depressed group relative to the paranoid and paranoid/depressed groups made more internal, stable and global attributions for bad events and more external, unstable and specific attributions for good events. Consequently, the findings of Coyne and Gotlib (1983), Peterson and Seligman (1984), Peterson et al. (1985), and Sweeney et al. (1986) have been supported and have further confirmed the existence of a depressive attributional style. In contrast to the depressives, paranoid individuals (i.e., those in the paranoid and paranoid/depressed groups) attributed good events and bad events in an opposite manner to the depressives. That is, while depressed individuals attributed bad events to internal, stable and global causes, and good events to external, unstable and specific causes,

the paranoids attributed bad events to more external, unstable and specific causes and good events to more internal, stable and global causes. Reflected in the attributional style of paranoid individuals are the results of Aaronson (1977) and Heilbrun and Bronson (1975) in that in contrast to depressives who blame themselves for their failures and/or difficulties and credit others for the positive (good) events in their lives, the paranoids credit themselves for their successes and accomplishments (good events) and blame others for their failures and/or problems (bad events).

The composite scores for good events showed that patients who are both paranoid and depressed tend to attribute causes for good events with a style that falls between that of the paranoid only and depressed only groups. That is, the paranoid/depressed patients attributed good events to internal, stable and global causes more than the depressed only group but less than the paranoid only group. Putting it more simply, the paranoid/depressed patients credited themselves for good events more than the depressed only group, but less than the paranoid only group. These findings may be explained in relation to the degree of depression found among the three groups. The fact that the paranoid/depressed group's level of depression was between that of the depressed only group (most depressed) and the paranoid only group (least depressed) and that the severity of depression was reflected in their respective attributional style, at least for good events, tends to support the contention of others (Johnson, Petzel, & Munic, 1986; Raps et al., 1982; Robins, 1988; Sweeney et al., 1986) that a depressive

attributional style is not a characteristic of general psychopathology, but rather is sensitive and specific to depression (Heinberg et al., 1987). Thus, it is likely the level of depression present which has determined an attributional style for good events for the paranoid/depressed group which is between the paranoid only group (least depressed) and depressed only group (most depressed). This same line of reasoning can be adopted for the attributional style for bad events for the three groups, but perhaps with lesser confidence.

For the composite scores for bad events then, although the depressed only group attributed bad events to more internal, stable and global causes than did the paranoid/depressed and paranoid only groups, the latter two groups were not significantly different from one another in how they attributed bad events in their lives. This may be explained with respect to the individual attributional dimensions of the ASQ. With respect to the individual dimensions for good events (INTG, STABG, GLOBG) all three experimental groups scored significantly different from one another on these measures with the exception of GLOBG dimension which could not distinguish between the depressed and paranoid/depressed groups. Hence, with only one nonsignificant difference, the composite was able to discriminate significantly among the three groups. In contrast, for the individual dimensions for bad events, the INTB and GLOBB could not discriminate between the paranoid only and paranoid/depressed groups and the STABB dimension could not discriminate between the paranoid only and paranoid/depressed groups or between the depressed

only and paranoid only groups. Since more bad dimensions were unable to discriminate among the groups, particularly between the paranoid/depressed and paranoid only groups, the discrimination power of the BAD composite was in turn reduced.

There may be several reasons for the discrepancy between the discriminatory power between the good and bad dimensions. First, it may be that the bad dimensions of the ASQ are more sensitive to larger differences in the level of depression like those found between the depressed only and the paranoid only group and less sensitive in discriminating groups where the differences in the level of depression are not as large, or when one group has more than one symptom complex like the paranoid/depressed group. Such a hypothesis would tend to support the results of Ganellen (1988) who found that attributional style could not distinguish between subjects who were depressed and those who had agoraphobia or panic disorders but who also had either a major affective disorder, dysthymic disorder or a history of an affective disorder. Second, the better discriminatory power of the good events may be the result of the degree of importance placed on the good or bad events. As previously mentioned, the intensity or severity of a depressed affect is partly determined by the subjective level of importance the individual has placed on the negative event. Although there was no difference among the three groups with respect to the degree of importance for bad events (IMPB) or good events (IMPG), the IMPG dimension was almost able to discriminate the three groups from one another at the $p = .05$ level. This difference may have contributed

to the better discriminatory power of the dimensions for good events. Third, the differences in discriminatory power may be the result of the similarities and differences in the delusional content between the paranoid and paranoid/depressed groups. For example, there was no difference between these two groups with respect to their delusions of persecution, but the paranoid only group had significantly more grandiose ideas and higher self-esteem than did the paranoid/depressed group. It may be that the less self-esteem enhancing more negative delusions of persecution are more instrumental in determining the attributional style for negative or bad events, and the more self-esteem enhancing, more positive delusions of grandiosity are more instrumental in determining the attributional style for positive or good events. This would account for the fewer differences between the paranoid and paranoid/depressed groups on the bad dimensions as opposed to the good dimensions and the generally better discriminatory power of the good dimensions as opposed to the bad dimensions.

With respect to the degree of importance for good or bad events among the three groups, generally all three groups found both good and bad events to be important (this is based on the fact that all three groups' mean scores on the IMPB and IMPG dimensions were above 4 which is considered to be a neutral response). This may be due to paranoids and depressives having a preoccupation with the self as suggested by Zigler and Glick (1984, 1986, 1988), and therefore they would tend to view events both good and bad as having some importance to the self. It is important to note that comparisons of

these results to those of other studies is not possible since most studies do not report their findings on these dimensions because IMPG and IMPB are not taken into account (Peterson & Seligman, 1984).

In their meta-analysis Sweeney et al. (1986) noted that for positive outcomes and negative outcomes, effect sizes ranging from largest to smallest can be ordered as follows: GOOD, STABG, INTG, GLOBG, and INTB, STABB, GLOBB and BAD. With respect to the current study, for good events the order GOOD, INTG, STABG, and GLOBG is in general agreement with the Sweeney et al. (1986) study. Similarly, but with the exception of the STABB dimension, for bad events the order INTB, BAD, GLOBB, STABB is in general agreement with the meta-analysis. As previously mentioned, helplessness which is attributed to relatively stable causes will produce longer-lasting depressions. The fact that the STABB dimension showed the least discriminatory power for bad events in that there were no significant differences between the paranoid only and paranoid/depressed groups or between the depressed only and paranoid only groups has perhaps exemplified the instability of depressive illness in relation to paranoid illness and lends more credence to the existence of a paranoid-depressive continuum (cf. Schwartz, 1963).

As for the individual attributional dimensions, few studies have shown all of the individual attributional dimensions to be in the predicted direction when comparing depressed and other samples. Therefore, it was not surprising that the current study failed to

find significant differences among the three groups on all of the attributional dimensions. If Zigler and Glick (1984, 1986, 1988) are correct in that paranoid illness masks an underlying depression then this would support the pattern of results obtained in that there were more non-significant differences between either the depressed and paranoid/depressed groups or between the paranoid/depressed and paranoid groups and fewer insignificant differences between the two extremes of the so-called paranoid-depressive continuum, the groups of depressed only and paranoid only. This combined with the findings that level of depression decreased, and self-esteem and paranoia increased from the depressed group through the paranoid/depressed to the paranoid group would tend to support Zigler's and Glick's theory (1984, 1986, 1988), and the existence of the paranoid-depressive continuum (cf. Schwartz, 1963).

Insofar as the results of the current study have shown that low self-esteem subjects (depressed individuals) made more internal attributions for failure or bad events, and high self-esteem subjects (paranoid individuals) showed a preference for attributing positive outcomes or good events to internal causes and negative outcomes or bad events to external causes have supported the results of Fitch (1970), Ickes and Layden (1978), and Tennen and Herzberger (1987). The fact that as self-esteem decreased and depression increased, a more depressive attributional style emerged which tends to support the concept of characterological self-blame and the results of Peterson et al. (1981) and Peterson (1988) who found a

significant positive correlation between characterological self-blame and level of depression and that depression was negatively correlated with behavioral self-blame. The reformulated learned helplessness theory has dictated that the internality dimension for bad events determines the level of self-esteem (Peterson & Seligman, 1984). This would tend to support the existence of characterological self-blame since a low self-esteem, high degree of depression and internal attributions for bad events were positively related to each other. These relationships will be pursued further in the section which addresses the correlational data.

The fact that the current study found significant differences between paranoid and depressed individuals is in contrast to the Romney and McElheran (1987) and Shaver et al. (1984) studies. It may be that the current study, unlike these others, not only controlled for depression but also had more homogeneous diagnostic groups which helped in the realization of significant results. In addition, in contrast to the Shaver et al. (1984) study, the current study used the ASQ, the most researched and most widely used measure of attributional style (Sweeney et al., (1986) which may also have contributed to the fact that significant results were found.

Correlational Analysis

The fact that the BDI and HRSD were found to be highly correlated with one another ($r = .94$) was not surprising given that similar correlations have also been reported by other investigators (cf. Beck, Steer, & Garbin, 1988; Steer, Beck, & Garrison, 1982).

Although Kendall et al. (1987) have noted that more soundly based conclusions can be generated by using both a self-report and clinician-rated measure of depression, the results of the current study would seem to indicate that to use both measures of depression may be somewhat redundant, particularly for individuals in the depressed group whose depression was also confirmed by psychiatric diagnosis. However, for individuals in the paranoid/depressed group whose depressive symptoms were largely not diagnosed by their psychiatrists, the use of both measures of depression proved to be useful in corroborating each other's findings.

Both the PA and the MMPI6 proved to be adequate measures of the degree of paranoia and were found to be significantly correlated with one another. As expected (cf. Magaro, Abrams, & Cantrell, 1981) the NONPA was not significantly correlated with either the PA or the MMPI6. This was not surprising, as previously mentioned, given that the NONPA subscale was designed to measure schizophrenic symptoms, while the PA was designed to measure paranoid symptoms. However, the PARA measure was found to be significantly correlated with the PA and the MMPI6. This supports the contention that the two subscales (PA and NONPA) of the Maine scale do not measure the same symptoms (Magaro et al., 1981).

The finding that self-esteem (COOP) was inversely related to depression has corroborated the findings of several studies (cf. Axford & Jerrom, 1986; Wenzlaff & Grozier, 1988; Zautra et al., 1985). Conversely, self-esteem was highly positively correlated with paranoia. In the psychodynamic framework, paranoia is

considered to be a way for individuals to protect themselves from feelings of depression and low self-esteem. The direct relationship between paranoia and self-esteem, that is, as self-esteem increases degree of paranoia increases would lend support to this contention. In relation to attributional style then, as previously mentioned, paranoids blame others for dissatisfactions with the self and thereby enhance self-esteem and "prevent a realistic but intolerable self-evaluation" (Kolb & Brodie, 1982, p. 447).

The finding that there were high intercorrelations between the various attributional dimensions on the ASQ within good and bad events has supported the conclusions of Peterson and Seligman (1984) who reported that the dimensions "are substantially intercorrelated within good and within bad events, thereby bolstering reliability to more acceptable levels..." (p. 352). It is perhaps for this very reason that most studies have not bothered to report the intercorrelations for the ASQ dimensions. Nevertheless, the range of the intercorrelations within good and bad events for the current study are similar to the ranges reported by Zautra et al. (1985) and higher than the ranges reported by Manly et al. (1982) and Peterson et al. (1982). Generally, the intercorrelations among the ASQ dimensions were higher for the current study than they were for other studies. Although the current study used relatively homogeneous diagnostic categories and that the level of depression was relatively consistent within each experimental group, the range of depressive symptoms for the entire sample across groups varied widely and this may have accounted for the larger intercorrelations

among the ASQ dimensions found in the current study.

Given that the current investigation has complied with the suggestion of Peterson and Villanova (1988) "that samples should be studied in which subjects show a sufficient range in depressive symptoms" (p. 88), it was not surprising that the attributional dimensions for good and bad events, were negatively correlated with each other, although most were not significant. The fact that the internality dimensions for good and bad events were significantly negatively correlated may be due to the diversity between the depressed only and paranoid only group in their level of depression and that depressed individuals blame themselves while paranoids blame others, which has been reflected in their respective attributional styles. The finding that the importance dimensions for good and bad events were positively correlated has exemplified the point previously discussed that paranoids and depressives may view good and bad events as being equally important because they both tend to be preoccupied with the self (Zigler & Glick, 1984, 1986, 1988).

For the correlations between the attributional and nonattributional psychological measures it was found that the measures of depression (BDI and HRSD) were significantly negatively correlated with the attributional dimensions for good events and significantly positively correlated with the attributional dimensions for bad events. These results have shown that as the level of depression increases, individuals attributed bad events to more internal, stable and global causes, and good events to more

external, unstable and specific causes. Conversely, as depression decreases, individuals attributed bad events to more external, unstable and specific causes and good events to more internal, stable and global causes. Therefore, insofar as the results have shown a significant relationship between depression and internal, stable and global attributions for bad events concurs with the findings of other studies (Coyne & Gotlib, 1983; Sweeney et al., 1986). Moreover, that depressed individuals also made more attributions to external, unstable and specific causes for good events has been supported by Golin et al. (1981) and Sweeney et al. (1986). The finding that the composite for bad events (BAD) was significantly correlated with depression has been supported by Robins (1988) and Sweeney et al. (1986).

With respect to the magnitude of the correlations between the depression scales and the ASQ dimensions, particularly the composites, similar magnitudes have been reported by Crocker, Alloy, and Kayne (1988) and Nezu, Nezu, and Nezu (1986). However, in contrast to Sweeney et al. (1986) who reported that effect sizes were greater for bad events than for good events, the current study found the opposite to be true. This has been discussed previously with respect to the better discriminating power of good events over bad events for the three experimental groups.

For the correlations between the depression measures and the importance dimensions IMPG and IMPB, the results showed that as depression increased, the importance of good events decreased while the importance of bad events increased. This finding tends to

support the reformulated attributional theory (Abramson et al., 1978) in that the intensity of depressed affect is proportional to the subjective level of importance the individual has placed on the negative event.

The correlations between measures of paranoia (PA and MMPI6) and the attributional dimensions generally showed that as the degree of paranoia increased, these individuals attributed bad events to more external, unstable and specific causes. Conversely as paranoia decreased individuals attributed bad events to internal, stable and global causes and good events to external, unstable and specific causes. Essentially, the attribution style of depressed patients was opposite to the style of paranoid patients. Moreover, as paranoia decreases and depression increases attributions for good and bad events become more similar to one another. Thus, as previously outlined, depressed individuals blame themselves for their failures and credit others for their successes, while paranoids blame others for their failures and credit themselves for their successes. Generally, the MMPI6 made fewer significant correlations with good and bad attributional dimensions than did the PA measure. Although Dahlstrom et al. (1972a) have noted that the paranoia subscale of the MMPI (MMPI6) is the most easily faked of all the clinical scales of the MMPI, this measure still proved to be a valid assessment tool for the degree of paranoia and as a correlate of attributional style. Neither of the paranoid measures was significantly correlated with either of the importance dimensions (IMPG, IMPB). Nevertheless, there was a trend for

paranoia to be positively correlated with the importance of good events (IMPG) and negatively correlated with the importance of bad events (IMPB), which is opposite to the correlations found between these dimensions and the depression measures.

As for the measure of self-esteem (COOP), the results generally showed that, as self-esteem increases, individuals attributed bad events to more external, unstable and specific causes, and good events to more internal, stable and global causes, Conversely, as self-esteem decreases attributions for bad events became more internal, stable and global, while they became more external, unstable and specific for good events. Thus, when depression is high, self-esteem and paranoia are low and when depression is low, self-esteem and paranoia are high. Overall, the relationships between attributional style, self-esteem and depression tend to support the findings of Wenzlaff and Grozier (1988) that depressed individuals evaluate failure experiences (analogous to bad events) in ways that are biased against the self. Similarly, the current study has also concurred with the results of Zautra et al. (1985) in that internal and stable attributions for negative outcomes were associated with low self-esteem and depression, and with the results of Tennen and Herzberger (1987) who found that high self-esteem subjects attributed positive, but not negative outcomes to internal and stable causal factors. Although the correlational data of the current study lend support for the reformulated learned helplessness theory of depression in that internal attributions, low self-esteem and depression are significantly correlated to each other, the

current study does not shed any light on the contention that it is the tendency to attribute failure to oneself which leads to the loss in self-esteem. Longitudinal studies and not cross-sectional or correlational data will be required to examine this issue.

Theoretical Implications

Paranoia, in the psychodynamic framework, is considered to be a way for individuals to protect themselves from feelings of depression and low self-esteem. Insofar as the results have shown paranoia and depression to vary inversely and proportionally with one another would tend to support this claim. As previously noted, Winters and Neale (1985) found that remitted bipolars report a normal self-esteem compared to depressives who report a low self-esteem. These investigators noted that a within-subjects design that tested bipolars during both phases of the disorder would address whether bipolars would report a high self-esteem during a manic phase. It should be remembered that Zigler and Glick (1984, 1986, 1988) have stated that paranoia resembles mania. Consequently, one might speculate that bipolars in the manic phase would report a higher self-esteem (like that found in the paranoid only group in the current study) than would bipolars whose mania is in remission (like the paranoid/depressed group in the current study). Such a finding would support Zigler's and Glick's contention that depression is turned upside-down which results in the assertion of the individual's self-worth or well-being. If Zigler and Glick are correct, then this would explain the fact that the paranoid/depressed group's level of self-esteem and degree of

paranoia were lower than that of the paranoid only group. The lower paranoia in the paranoid/depressed group would also account for the presence of more depression than was found in the paranoid only group, since there would be less masking of that depression. As for attributional style, Winters and Neale (1985) found that remitted bipolars had a depressive attributional style. In the current study, the paranoid/depressed group had a more depressive attributional style than did the paranoid only group where there was little evidence of the presence of depression. If paranoia does mask depression then one might speculate that as paranoia decreases or increases, depression would increase or decrease, respectively, and attributional style would become more depressive or less depressive, respectively. This is what the results have suggested. Moreover, if paranoia and depression merely coexist as separate disease entities, then in the paranoid/depressed group one might expect the severity of depression and paranoia to be equal to the severity in the depressed only and paranoid only groups, respectively. Rather, the results tend to support an inverse relationship between paranoia and depression as proposed by Zigler and Glick (1984, 1986, 1988).

With respect to the masking of depression several factors including chronicity, and delusion formation need to be discussed. Because the paranoid and paranoid/depressed groups have mean durations of illness of 9.93 and 8.87 years respectively, they would be considered to be suffering from chronic illnesses as defined as an illness duration greater than two years (DSMIII-R, 1987). Zigler

and Glick, (1984, 1986, 1988) have contended that depression should occur either before the paranoid delusions are formed or after they have abated. It has been asserted that typically, once paranoid delusions are formed, they are never really controlled or eliminated with medication (R. Williams, personal communication, June 16, 1988). Most of the subjects in the current study were considered to be chronic and having acute exacerbations of symptoms. Since virtually all paranoid only and paranoid/depressed subjects were being treated with medication one might assume that because the paranoid/depressed group were having depressive symptoms that they were in the post-psychotic phase of their illness and exhibiting post-psychotic depression. However, because they were having acute exacerbations of paranoid symptoms one might assume that they were in the acute phase of a psychotic or schizophrenic episode. This would account for the depressive and paranoid symptoms occurring simultaneously according to Zigler and Glick (1984, 1986, 1988), who contend that depressive symptoms should be more prominent when the paranoid delusions are either forming or abating. However, this would not account for the fact that the paranoid only group did not exhibit any depressive symptoms given that they too were having exacerbations of paranoid symptoms and being treated with medication. According to Zigler and Glick (1984, 1986, 1988), however one could argue that the paranoid only group's paranoid delusions were better developed and therefore evidence of depression was minimal or nonexistent. Although it is not known what would account for this difference in expression in symptomatology, it may

be related to the types of delusions differentiating these two groups.

According to psychodynamic theory, it is the grandiose delusions which epitomize the enhancement of self-esteem (Zigler & Glick, 1984, 1986, 1988) and defend against feelings of self-reproach (Cameron, 1975). Although there were no differences between the paranoid only and paranoid/depressed groups with respect to ideas of being controlled, ideas of reference, being punished or conspired against, expressions of hostility or delusions of persecution, the paranoid only group was significantly more grandiose than was the paranoid/depressed group. According to Romney (1987) who confirmed Lorr's simplex model of paranoia which depicts a progressive sequence of six stages beginning with hostile attitude and subsequently moving through verbalized hostility, resentment, blaming others, delusions of persecution and finally delusions of influence, "there are probably two further stages in the chain than those given in the model, viz., delusions of grandeur...and auditory hallucinations...but Lorr did not include these stages in his simplical analysis" (p. 654). The implication is that delusions of grandeur are formed after delusions of persecution. Although Salzman (1960) has contended that the development of persecutory delusions are a secondary response to the failure of the grandiose delusions to protect the individuals from feelings of worthlessness and low self-esteem, the conclusions of Romney (1987) would more closely coincide with the more traditional view that delusions of grandiosity are developed secondarily "in an

attempt to rationalize the (persecutory) delusion and to explain the attention of the world on the patient" (Salzman, 1960, p. 680). In view of this, it may be that the paranoid only group has more developed grandiose delusions which help to explain their delusions of persecution and protect themselves from feelings of worthlessness, low self-esteem and feelings of depression. If this is the case then this would explain why the paranoid only group had more grandiosity, less depression, a less depressive attributional style than the paranoid/depressed group. Along this line of reasoning, as the results have shown, one would expect that the paranoid/depressed group would have more depression and lower self-esteem than the paranoid only group whose paranoia is perhaps more developed and perhaps more severe and more completely masks the underlying feelings of depression. This has been supported by Arieti (1974) who has noted that the presence of depression improves the schizophrenic's prognosis. Conversely, "the more the patient projects toward others and exonerates himself, the more severe is the psychosis. If, on the other hand, he believes that he is persecuted because he is somehow guilty or responsible, the prognosis is better" (Arieti, 1974, p. 64). Thus, this responsibility is related to blameworthiness and attributional style which has reflected the level of depression and self-esteem in both the paranoid only and paranoid/depressed groups.

Given the cross-sectional nature of the current study, whether or not paranoia masks or substitutes for depression has only been partially addressed. As previously mentioned, insofar as the

results have supported an inverse relationship between the levels of depression and paranoia would tend to support this contention. Additional support for this contention and for paranoia substituting for depression comes from one of the subjects, a female paranoid disorder who not only had persecutory delusions, but also many grandiose delusions. This patient had no evidence of depression and was included in the paranoid only group. On interview this patient spontaneously stated that "I was tired of always feeling bad about myself so I guess I developed these ideas (meaning her delusions) to help myself feel better about myself." Although the process of masking depression with paranoid symptoms is supposed to be an unconscious process according to psychodynamic theory, the patient's ability to consciously voice this masking process may be taken as a sign of insight. Thus, it seems that this patient's grandiose delusions helped her to feel better about herself and presumably eliminate her depressive feelings and raise her level of self-esteem.

Related to the type of delusion present and the level of self-esteem is the presence or absence of suicidal ideation. Allen (1967) has contended that "it is an underlying depression which is always primary and that the paranoia is an attempt to deal with the accompanying suicidal impulse" (p. 435). In addition, when "suicidal ideation and grandiose or persecutory delusions exist concurrently...in these situations it is the paranoid delusions which are most heavily invested (and) if the affective investment changes serious risk of suicide is present" (Allen, 1967, p. 436). Consequently, the paranoid only patients whose delusions were more

grandiose may have expended more energy maintaining their delusions and subsequently had less suicidal ideation and depression compared to the paranoid/depressed group whose delusions were less grandiose and therefore expended less energy maintaining them and consequently had more suicidal ideation and depression. As previously mentioned, a low self-esteem is a risk factor for suicide in schizophrenia (Harrow & Westermeyer, in press; Roy, Thompson, & Kennedy, 1983) and schizophrenics with paranoid ideation may be at a higher risk for suicidal ideation (Drake et al., 1985; Harrow & Westermeyer, in press). In view of this, it may be that individuals in the paranoid/depressed group who have a lower self-esteem, more depression, a more depressive attributional style, more suicidal ideation and less grandiosity are at a greater risk for suicidal behavior than individuals in the paranoid only group. Grandiose delusions may not only function in protecting or enhancing self-esteem, but also may be instrumental in warding off suicidal thoughts and feelings of depression, as theory has suggested.

Apart from providing evidence in support of the theoretical formulations of Zigler and Glick (1984, 1986, 1988), the current study can also address the issue of negative symptoms and schizoaffective illness. As previously mentioned, Martin et al. (1985) have noted that to distinguish depression from other syndromes, clinicians must continue to rely on the presence or absence of marked dysphoria, suicidal ideation, eating and sleeping problems, loss of interest and feelings of guilt and self-reproach. In view of the results of the current study, it seems apparent that

more than just negative symptoms were being tapped by the measures of depression. Although there are various controversial explanations to account for the presence of negative symptoms in schizophrenics as well as controversial explanations regarding how negative symptoms and depression are related to one another in schizophrenia, Andreasen (1980) has noted that there has been a general trend to deal with positive and negative symptoms as distinct entities and that positive and negative symptoms frequently co-occur in single patients is typically ignored. This position is similar to Hirsch's (1982) who suggested that depression in schizophrenia is often unnoticed when florid psychotic symptoms are present. Further, Andreasen (1980) suggested that positive and negative symptoms may represent syndromes at opposite ends of a continuum which may overlap in single patients, and different treatments may be needed at different places along the continuum. Still further, Andreasen (1980) has suggested that "patients with mixed positive and negative symptoms are at an intermediate stage in the course of the illness; this hypothesis assumes that some patients may eventually evolve from a positive state to a negative state, and that the negative state represents the true or underlying disorder" (p. 385). Consequently, patients with mixed symptoms "are in fact negative, but the predominantly negative syndrome has not yet developed" (Andreasen, 1980, p. 385). Andreasen's view of positive and negative symptoms can be thought of as being synonymous with paranoid and depressive symptoms, respectively, in Zigler's and Glick's theoretical stance in that both types of symptoms, whether

they are called positive or negative or paranoid or depressive are at opposite ends of a continuum with the negative or depressive state representing the true underlying disorder which has not yet developed in patients with positive and negative symptoms. For Zigler and Glick (1984, 1986, 1988), it is not that the negative or depressive symptoms have not yet developed, but rather that they have only been partially masked, as in the paranoid/depressed group, and completely masked, as in the paranoid only group. These viewpoints also seem to be consistent with schizoaffective illness and the notion that "the boundary between schizophrenia and affective disorders must remain flexible" (Andreasen, 1987, p. 13), and that schizoaffective disorder may represent "an intermediate region on a unidimensional or hierarchical continuum of psychotic illness (Tsuang & Simpson, 1984, p. 22). Just as the research into depression in schizophrenia has mostly not examined schizophrenia by subtype (i.e., paranoid schizophrenia), the research into schizoaffective illness has not reported whether the patients had paranoid symptoms. It may be that schizoaffective patients resemble some of the patients in the paranoid/depressed experimental group in which their paranoid and depressive symptoms appear to be equally prominent.

Clinical Implications

Although depressive symptoms are a ubiquitous phenomenon, the fact that the vast majority of the paranoid subjects depressive symptoms were undiagnosed at the time of their participation in this study, would suggest that more effort must be made on the part of

mental health care workers in general to look for the signs of symptoms of depression in paranoid individuals. Although this may not be an easy endeavour when florid psychotic symptoms are present (Hirsch, 1986), if depression is not sought, it will not be found. This is particularly important in that the paranoid individuals in the current study who had depressive symptoms also had significantly more suicidal ideation than did the paranoid individuals who did not have depressive symptoms. If paranoia masks depression, then individuals in the paranoid only group would also likely be at risk for suicide once their paranoid symptoms have abated and their depression is unmasked. Although Roy (1986) has noted that auditory hallucinations or persecutory delusions are probably infrequent causes of suicidal behavior in schizophrenics, it may be that individuals with relatively few grandiose delusions (i.e., the paranoid/depressed group) as compared to those with more (i.e., the paranoid only group) may be at a higher risk for suicidal behavior. Consequently, if closer attention is paid to the type of delusions present this may prove to be one way of monitoring or evaluating suicidal potential.

While some therapists have contended that to challenge a paranoid individual's delusions is an ineffective therapeutic approach, at least in the initial stages of psychotherapy (Walker & Brodie, 1985), others have contended that when a patient has a capacity for affect and relatedness, direct confrontation about the reality of the delusion would be appropriate (Rudden, Gilmore, & Frances, 1982). Nevertheless, the establishment of good rapport and

trust are important in the therapeutic relationship with paranoid individuals, before the origins of the patients' delusions can be explored (Rudden et al., 1982; Walker & Brodie, 1985). Because depressive symptoms which co-occur with other illnesses such as schizophrenia are viewed as secondary to the primary illness, they are often not given therapeutic priority. If paranoid symptomatology masks depression then perhaps therapies like cognitive therapy (Beck, Rush, Shaw, & Emery, 1979) and/or attributional therapy (Weiner, 1988) used traditionally to treat depression would be more efficacious in some cases. This rationale would coincide with previously mentioned cases in which some individuals were being unsuccessfully treated medically for paranoid schizophrenia or paranoid disorder, where treatment for depression might have been more appropriate. Although the current study has not shed any light on whether causal attributions precede or follow the onset of depression, the fact that depressive symptoms and attributional style have been shown to improve over time with cognitive therapy would support the validity of this form of intervention where depressive symptoms are concerned. Seligman et al. (1988) have also noted that because a depressive explanatory style puts people at risk for depression, then undoing this type of explanatory style may be a curative element in the cognitive therapy of depression. Similarly, for paranoid individuals these same techniques may be applied in order to help them change their particular attributional style of blaming others for their problems and taking credit for their successes. So rather than confronting

the paranoid individuals' delusions directly, which may be therapeutically ineffective, a therapist might use attribution therapy techniques such as altering the patient's perception of the outcomes of situations, modifying causal antecedents, changing causal meaning and/or behavior or directly changing causal ascriptions (Weiner, 1988) to help reduce or eliminate paranoid symptoms. The ASQ itself, may be one way of not only assessing an individual's attributional style, but it may also prove to be a way to monitor attributional style during the course of therapy.

Finally, the results of this study indicate that in the study of depression in schizophrenia, paranoid schizophrenics should be treated as a separate category apart from the other subtypes of schizophrenia, particularly in view of the evidence that more paranoid schizophrenics are depressed than other subtypes (Siris et al., 1984) and that more paranoid schizophrenics are at a higher risk for suicide than other subtypes (Harrow & Westemeyer, in press).

Limitations

There are a couple of limitations to the present study which should be mentioned. First, although this study has found some compelling evidence for the contention that paranoia and depression are inversely related to one another and that perhaps paranoia substitutes or masks depressive symptoms in some cases, the fact that this was a cross-sectional study cannot prove this contention. Longitudinal data would be required to ultimately address this issue. Second, although Bartko et al. (1988) have noted that 15 subjects in each group is a sufficient number to find statistical

significance (when it exists) 80% of the time with p=.05, conclusions based on studies which have used larger sample sizes typically carry more weight and perhaps more credibility. Third, the fact that it was not possible to control for the use of medication has made it equally impossible to control for the possibility that the medicated patients, particularly those on neuroleptics, may have experienced some drug induced side effects which may have affected their symptomatology. In other words, it is impossible to know if pharmacogenic effects contributed to the cause or severity of the symptomatology in these subjects.

Recommendations for Future Research

In view of the fact that the current study has not been carried out previously, it would be important for it to be replicated using a larger number of subjects in each group. With respect to the limitations previously mentioned, in future studies it would be helpful to control for medication use so as to help eliminate the possibility that pharmacogenic effects may be influencing the existing symptomatology. It would also be useful to carry out this study on a longitudinal basis. This would allow the investigator to measure the levels of depression, paranoia and self-esteem and attributional style at several time periods in order to evaluate the inverse relationship between depression and paranoia in single individuals and to see whether changes in symptomatology are reflected in the resulting attributional style. The current study has not shed any light on whether a depressive attributional style is a predispositional factor in the onset of depression or simply

another symptom of depression. This question will continue to remain unanswered unless attributional styles are examined in individuals before the onset of illness. Such an endeavour would require large samples to be followed over a long period of time.

The fact that more delusional grandiosity was found in the paranoid only group may have implications for their potential for violence, particularly in view of the finding that the paranoid only group had an almost significantly higher degree of expression of hostility. Walker and Brodie (1985) have noted that paranoid individuals are often angry and sometimes may pose a threat to others through the acting out of violent behavior. Although Shore, Filson, and Johnson (1988) have noted that the male gender and prior violence continue to be the best predictors of future violence in paranoid schizophrenics and that most paranoid schizophrenics are not violent, they also noted that threats, persecutory delusions, and command hallucinations may be associated with increased risk of violent crime arrest in some paranoid schizophrenics. In the current study the potential for violence may be different in the paranoid and paranoid/depressed groups. If persecutory delusions are a risk factor for violent behavior, then one would expect there to be no difference between the two groups, given that there were no differences in persecutory delusions between these two groups. However, one might expect that individuals in the paranoid/depressed group who have more depression, lower self-esteem and more suicidal ideation to be more capable of harming themselves and perhaps less capable of harming others. The Shore et al. (1988) study failed to

mention if level of depression, presence of suicidal ideation or presence of delusions of grandiosity were measured. It is possible that the combination of persecution and grandiose delusions would be more indicative of violent paranoid schizophrenics. It seems plausible that paranoid individuals like those found in the paranoid only group, who have higher self-esteem, no depression, less suicidal ideation, a trend toward more expression of hostility and a tendency to blame others for their problems would be more capable of bringing harm to others, than themselves. This would suggest that more emphasis should be placed on the delusions themselves in an attempt to fully understand paranoid illness. The study of delusions has largely been overshadowed in favour of the study of formal thought disorder which was considered to be the pathognomonic symptom of schizophrenia (Winters & Neale, 1983). The fact that there were differences in the level of grandiosity between the paranoid and paranoid/depressed groups may lend support to the notion that more research needs to be completed in order to understand the cognitive processes that underlie delusion formation and to determine why some individuals have different types of delusions. For example, it has been suggested that mood congruent and mood incongruent delusions may differ in terms of the processes related to inference making (Winters & Neale, 1983).

Summary and Conclusions

The purpose of this study was to address several research questions, namely: (1) How are attributional style and self-esteem affected by individuals who are depressed, paranoid, or both? (2)

What is the clinical relationship between paranoia and depression? and (3) Does paranoia substitute for depression on some cases? Besides confirming the existence of a depressive attributional style in depressed patients, the results also showed that paranoid patients attribute the causes of good and bad events in a manner that is opposite to depressed patients. In addition, the level of self-esteem was found to correlate positively with the degree of paranoia and negatively with the degree of depression, and this was reflected in the subject's attributional style. Moreover, empirical evidence was found to support the contention that paranoia and depression are inversely related to one another (Zigler & Glick, 1984, 1986, 1988) and that perhaps paranoia substitutes for or masks an underlying depression (cf. Zigler & Glick, 1984, 1986, 1988). Should future research replicate these results, this would have important implications for the treatment of paranoid individuals both medically and psychologically. It may be shown in future studies that psychological treatments typically used to treat depression may be beneficial in treating paranoid individuals such as cognitive therapy (Beck et al., 1979) and attributional therapy (Weiner, 1988). Apart from the therapeutic approaches in dealing with paranoid patients, this thesis also addressed the current view of depression in schizophrenia, schizoaffective illness, negative symptoms and suicide in schizophrenia. Specifically, the current study has lent support to the agrument for examining paranoid schizophrenics as a separate category from nonparanoid schizophrenics in the study of depression in schizophrenia.

Moreover, this thesis has perhaps helped to explain some of the controversial evidence regarding depression in schizophrenia and how the concepts of schizoaffective illness and negative symptoms may be viewed in a different light. Perhaps more importantly was the information generated about the relationship between delusions, depression and suicidal ideation in paranoid individuals. Insofar as individuals who are paranoid and depressed have different delusional content and more suicidal ideation than paranoid individuals who are not exhibiting depressive symptoms, there is cause for future studies to examine the processes of delusion formation and how they are related to depression and suicidal ideation. Ultimately, any knowledgeable gains in these areas would be beneficial in the understanding and treatment of paranoid individuals.

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APPENDIX A

MMPI Clinical Scale Pa (#6)

This inventory consists of numbered statements. Please read each statement and answer either TRUE or FALSE as it applies to you.

1. I am sure I get a raw deal from life. .....
2. No one seems to understand me. .....
3. Evil spirits possess me at times. .....
4. If people had not had it in for me I would have been much more successful. .....
5. I think most people would lie to get ahead. .....
6. I am happy most of the time. .....
7. Some people are so bossy that I feel like doing the opposite of what they request, even though I know they are right. .....
8. Someone has it in for me. .....
9. I have never done anything dangerous for the thrill of it. ....
10. Most people are honest chiefly through fear of being caught. .....
11. I believe I am being plotted against. .....
12. I believe I am being followed. .....
13. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it. .....
14. I know who is responsible for most of my troubles. .....
15. Someone has been trying to poison me. .....
16. I feel that I have often been punished without cause. .....
17. I cry easily. .....
18. I believe I am a condemned person. .....
19. Something exciting will almost always pull me out of it when I am feeling low. .....
20. Someone has control over my mind. .....

Appendix A (continued)

21. I do not often notice my ears ringing or buzzing. .....

22. I am sure I am being talked about. .....

23. At one or more times in my life I felt that someone was making me do things by hypnotizing me. .....

24. Someone has been trying to influence my mind. .....

25. I have never been in trouble with the law. .....

26. I think that I feel more intensely than most people do. .....

27. Even when I am with people I feel lonely much of the time. ....

28. The man who provides temptation by leaving valuable property unprotected is about as much to blame for its theft as the one who steals it. .....

29. Once in a while I think of things too bad to talk about. .....

30. I think nearly anyone would tell a lie to keep out of trouble. .....

31. I am more sensitive than most other people. .....

32. Most people inwardly dislike putting themselves out to help other people. .....

33. At time I have fits of laughing and crying that cannot control. .....

34. My mother or father often made me obey even when I thought that it was unreasonable. .....

35. I have certainly had more than my share of things to worry about. .....

36. At times I hear so well it bothers me. .....

37. I have no enemies who really wish to harm me. .....

38. I tend to be on my guard with people who are somewhat more friendly than I had expected. .....

39. People say insulting and vulgar things about. .....

40. I feel uneasy indoors. .....

APPENDIX B

Journal of Consulting and Clinical Psychology, 49(3), 438-447, 1981

P. Magaro, L. Abrams, and P. Cantrell

The Maine Scale of Paranoid and Nonparanoid Schizophrenia

Paranoid subscale

P1. Does he tend to suspect or believe on slight evidence or without good reason that people and external forces are trying to or now do influence his behavior, control his thinking?

1. No unjustified suspicions.
2. Will admit suspicion when pressed.
3. Easily admits suspicion.
4. Openly states others are trying to control him.
5. Has firm conviction that he is influenced or controlled.

P2. Does he tend to suspect or to believe on slight evidence or without good reason that some people are against him (persecuting, conspiring, cheating, depriving, punishing) in various ways?

1. No unjustified suspicions expressed.
2. When pressed expresses belief that he is conspired against.
3. Frequently inclined to suspect.
4. Frank inclination to believe in persecution.
5. Strongly expresses conviction of persecution.

P3. Does he have an exaggeratedly high opinion of himself or an unjustified belief or conviction of having unusual ability, knowledge, power, wealth or status?

1. No expressed high opinion of himself.
2. When pressed expresses a high opinion of himself.
3. Frequently expresses high opinion of himself.
4. Open conviction of unusual power, wealth, etc.
5. Strongly expresses conviction of grandiose or fantastic power, wealth, etc.

P4. Does he tend to suspect or believe on slight evidence or without good reason that some people talk about, refer to or watch him?

1. No unjustified suspicions.
2. Will admit suspicion.
3. Easily admits suspicion.
4. Openly states that he is watched.
5. Has firm conviction of being watched.

Appendix B (continued)

P5. Compared to others how openly hostile is he? Does he show hostility or a high degree of ill will, resentment, bitterness or hate?

1. No open hostility.
2. Relatively little hostility.
3. Some hostility.
4. Rather hostile.
5. Very hostile.

Nonparanoid subscale

N1. Does he have perceptions (auditory, visual) without normal external stimulus correspondence?

1. None.
2. When pressed admits hallucinations.
3. Easily admits hallucinations.
4. Openly admits frequent hallucinations.
5. Openly hallucinates.

N2. On the basis of the integration of the verbal productions of the patient, does he exhibit thought processes which are confused, disconnected or disorganized?

1. As normal.
2. Slight disorganization.
3. Mild disorganization.
4. Marked disorganization.
5. Complete disorganization.

N3. How incongruous are his emotional responses? e.g., giggling or crying for no apparent reason or not showing any emotion when emotion would be appropriately shown.

1. As normal.
2. Slightly different from normal.
3. Responses somewhat incongruous.
4. Distinctly incongruous.
5. Very markedly incongruous.

Appendix B (continued)

N4. How well oriented is he as to time? For instance, does he know (a) the season; (b) the month; (c) the calendar year; (d) the day of the week; (e) how long he has been in hospital?

1. As normal.
2. Occasional confusion.
3. Slight confusion.
4. Frequent confusion.
5. Marked continuous confusion.

N5. Does he assume or maintain peculiar, unnatural, or bizarre postures?

1. None.
2. On rare occasions.
3. For short periods.
4. Frequently.
5. All the time.

APPENDIX C

Cognitive Therapy and Research, 6, 27-37, 1982

C. Peterson, A. Semmel, C. Von Baeyer, L.Y. Abramson, G.T. Metalsky, M.E.P. Seligman

Attributional Style Questionnaire

Name _____

Date _____

Phone # _____

DIRECTIONS

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

1) Read each situation and vividly imagine it happening to you.

2) Decide what you feel would be the major cause of the situation if it happened to you.

3) Write one cause in the blank provided.

4) Answer three questions about the cause.

5) Answer one question about the situation.

6) Go on to the next situation.

Appendix C (continued)

YOU MEET A FRIEND WHO COMPLIMENTS YOU ON YOUR APPEARANCE.

1) Write down the one major cause_____

2) Is the cause of your friend's compliment due to something about you or something about the other person or circumstances? (Circle one number)

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

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

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

4) Is the cause something that just affects interacting with friends or does it also influence other areas of your life? (Circle one number)

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

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

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

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

6) Write down one major cause _____

7) Is the cause of your unsuccessful job search due to something about you or something about other people or circumstances? (Circle one number)

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

Appendix C (continued)

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

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

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

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

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

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

YOU BECOME VERY RICH.

11) Write down the one major cause _____

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

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

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

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

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

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

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

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

Appendix C (continued)

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

16) Write down the one major cause _____

17) Is the cause of your not helping your friend due to something about you or something about other people or circumstances? (Circle one number)

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

18) In the future when a friend comes to you with a problem, will this cause again be present? (Circle one number)

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

19) Is the cause something that just affects what happens when a friend comes to you with a problem or does it also influence other areas of your life? (Circle one number)

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

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

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

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

21) Write down the one major cause _____

22) Is the cause of the audience reacting negatively due to something about you or something about other people or circumstances? (Circle one number)

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

Appendix C (continued)

23) In the future when giving talks, will this cause again be present? (Circle one number)

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

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

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

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

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

YOU DO A PROJECT WHICH IS HIGHLY PRAISED.

26) Write down the one major cause _____

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

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

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

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

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

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

Appendix C (continued)

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

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

YOU MEET A FRIEND WHO ACTS HOSTILELY TOWARD YOU.

31) Write down the one major cause _____

32) Is the cause of your friend acting hostile due to something about you or something about other people or circumstances? (Circle one number)

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

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

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

34) Is the cause something that just influences interacting with friends or does it also influence other areas of your life? (Circle one number)

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

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

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

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

36) Write down the one major cause _____

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

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

Appendix C (continued)

38) In the future when doing the work that others expect, will this cause be present? (Circle one number)

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

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

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

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

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

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

41) Write down the one major cause _____

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

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

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

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

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

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

Appendix C (continued)

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

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

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

46) Write down one major cause _____

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

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

48) In the future when applying for a position, will this cause again be present? (Circle one number)

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

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

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

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

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

YOU GO OUT ON A DATE AND IT GOES BADLY.

51) Write down the one major cause _____

Appendix C (continued)

52) Is the cause of the date going badly due to something about you or something about other people or circumstances? (Circle one number)

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

53) In the future when dating, will this cause again be present? (Circle one number)

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

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

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

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

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

YOU GET A RAISE.

56) Write down the one major cause _____

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

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

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

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

Appendix C (continued)

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

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

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

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

APPENDIX D

INFORMATION SHEET (to subjects)

Dear _____,

You are invited to participate in a research project that I am currently conducting for my Master's thesis in Educational Psychology (Clinical/Community Program) at the University of Calgary. I am interested in examining how different individuals view the causes of different situations or problems in their lives. As a participant in this study you would be interviewed briefly and then asked to complete four questionnaires requiring you to respond to short answer, multiple choice and true and false questions. These questionnaires will ask you about how you view the causes of situations in your life, and about your feelings. I, Carmie Candido, will explain the instructions and stay with you while you complete the questionnaires. This should take approximately two hours depending on how fast you work.

Your participation in this study is on a voluntary basis, and your decision of whether or not to participate in this study will in no way affect your access to treatment. As a participant in this study confidentiality is assured. Once written consent is obtained by you and your psychiatrist and you have completed the questionnaires your name will be transformed into a numerical coding system. All questionnaires, consent forms and data would be locked in a cabinet and only myself and my academic supervisor would have

Appendix D (continued)

access to this information. As participants in this study you have the right to withdraw from the study at any time and you also have the right to have access to the results of this study, (should you desire, a summary of the results may be mailed to you). Once the study has been published, all questionnaires, consent forms and data would be destroyed. If you have further questions that you may wish to ask, please do not hesitate to contact me or Dr. Romney. Our names, addresses and telephone numbers are below.

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APPENDIX D

CONSENT FORM (Psychiatrist)

I, _____, psychiatrist for _____, give my consent for _____, to participate in this study which will be examining the relationship between depressed and paranoid individuals in how they attribute the causes of good and bad events in their lives. I understand that my patient will be interviewed briefly and then asked to complete four psychological questionnaires.

The total time required by my patient will be approximately two hours. I have been informed that my patient will be given assurances of confidentiality, right to withdraw or be asked to withdraw at any time, and access to results of the study if he or she so desires.

_____
Psychiatrist's Name

_____
Signature

_____
Date

APPENDIX D

CONSENT FORM (Subjects)

I, _____, consent to participate in this study examining how different individuals attribute the causes of good and bad events in their lives. I understand that I will be interviewed briefly and then asked to complete four questionnaires. Approximately two hours of my time will be required in total.

I am volunteering to participate in this study with the understanding that I may withdraw, or be asked to withdraw at any time. Furthermore, I have been informed that (1) code numbers will be submitted for my name on information collected, (2) during the study all information collected will be stored in a locked cabinet and will be accessible to only those researchers involved in this study, and (3) upon publication of the study, all collected information will be destroyed. I further understand that I will have access to the results of this study should I desire.

_____
Name

_____
Signature of Subject

_____
Name of Witness

_____
Signature of Witness

_____
Date