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The Neuropsychology of Depression and Its Implications for Cognitive Therapy

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The past few years have witnessed a growing interest in the specialty of neuropsychology as well as continued support for cognitive therapy of depression. The purpose of this paper is the examination of the neuropsychology of depression and its implications for A. T. Beck's cognitive theory and therapy of depression ([1963] "Thinking and Depression: Idiosyncratic Content and Cognitive Distortions," Archives of General Psychiatry, *Vol. 9, pp. 324–333; [1964] "Thinking and Depression,"* Archives of General Psychiatry, *Vol. 10, pp. 561–571; [1967]* Depression: Clinical, Experimental, and Theoretical Aspects, *New York: Harper & Row). Specifically, the neuropsychological and cognitive theory and therapy literatures related to depression are reviewed followed by an integration of these areas. Neuropsychological evidence is presented that both supports cognitive theory and therapy of depression and helps explain why such therapy may prove ineffective in treating depression. Implications for clinical practice, including neuropsychological assessment of depressives, and potential future research directions are also provided.*

KEY WORDS: neuropsychology; depression; cognitive therapy.

INTRODUCTION

Over the past two decades there has been a gradual evolution within the field of psychology, from an emphasis on behavioral theories in the

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1970s to a focus on cognitive processes in the 1980s. In recent years, the emphasis has shifted toward a neuropsychological perspective as the National Institute of Mental Health (NIMH; 1989) has designated the 1990s as the decade of the brain.

The purpose of this paper is the examination of the neuropsychology of depression and its implications for cognitive theory and therapy of depression as proposed by Aaron Beck (Beck, 1963, 1964, 1967). The article is divided into three primary sections. After a brief overview of fundamental neuropsychological concepts, the neuropsychology of depression literature is examined via diverse methodological perspectives that include clinical lesion, sensory-perceptual, electrodermal, electroencephalographic, single positron emission computed tomography, positron emission tomography, and psychometric studies. Relevant sections on induced emotions and hemispheric priming are also included.

This review is followed by a comprehensive examination of Beck's cognitive theory and therapy of depression. Specifically, a review of the principal concepts of Beck's cognitive theory of depression is first provided, which includes cognitive schemata, the negative cognitive triad, automatic thoughts, and cognitive errors. An overview of Beck's cognitive therapy of depression follows, which focuses on the proposed mechanisms of change, mode-specific changes, and the predictors of response.

An integration of the neuropsychology of depression and Beck's cognitive theory and therapy of depression follows. Based on neuropsychological evidence, implications are proposed that provide support for Beck's cognitive theory and therapy of depression, offer explanations as to why cognitive therapy may be ineffective, and examine the response to cognitive therapy. Implications for clinical practice, assessment, and future research are provided.

Additionally, it should be noted that the model presented herewith remains speculative and is intended only as an exploration of the possible, vs. a discussion of the definitive, contributions that neuropsychology may provide to understanding cognitive theory and therapy of depression in terms of brain/behavior relationships and cerebral hemispheric functioning. While others have discussed the use of cognitive therapy in the treatment of poststroke depression (e.g., Rosenthal, 1984), it is hoped that this model will stimulate future research that provides direct evidence for the hypothesized links between the neuropsychology of depression and Beck's cognitive theory/therapy of depression in both brain-lesioned and nonlesioned patients. In turn, such associations may not only be valuable in furthering knowledge of the relationships between neuropsychology and cognitive theory/therapy and refining a rationale for differential depression treatment,

but these links may also contribute to a more consolidated and unified psychological science.

THE NEUROPSYCHOLOGY OF DEPRESSION

Fundamental Concepts

In general, neuropsychological investigations are based on several fundamental assumptions and principles. An underlying assumption of neuropsychology states that all behavior is mediated by changes in the physical state of the brain (Heilman and Valenstein, 1985). Hence, changes in behavior are associated with physical changes in the brain and, likewise, changes in the brain's physical state (i.e., brain lesions) can modify behavior. A second principle concerns the concept of cerebral laterality. This concept refers to the anatomical and functional differences between the two cerebral hemispheres (Kolb and Whishaw, 1990; Wexler, 1980). It has been found that many sensory and motor connections (and functions) on one side of the body are projected to, and controlled by, the contralateral hemisphere of the brain (Kolb and Whishaw, 1990; Wexler, 1980). For example, motor responses of the right leg are predominantly controlled by the left cerebral hemisphere.

The data on cerebral lateralization of right-handed persons has revealed an array of functional asymmetries. The left hemisphere (LH) appears to play a greater role in language and verbal abilities, logical reasoning, verbal memory, arithmetic calculations, and complex voluntary movements (Bryden, 1982; Dimond and Beaumont, 1974; Eccles, 1977). Conversely, the right hemisphere (RH) appears more specialized for visuospatial abilities, complex geometric patterns, prosody, and musical abilities, as well as nonverbal memory (Bryden, 1982; Dimond and Beaumont, 1974).

The RH has also been theorized to play an important role in the perception and expression of emotion (Bloom *et al.*, 1990; 1992; Borod *et al.*, 1983, 1986, 1988, 1992; Moreno *et al.*, 1990). For example, Borod and her colleagues have found that in comparison to left brain-damaged subjects, individuals with RH lesions tend to exhibit significant impairments in their abilities to express and perceive lexically based emotion (Bloom *et al.*, 1990; Borod *et al.*, 1992) and facial affect (Borod *et al.*, 1986; Borod *et al.*, 1988). Tucker (1981) and Davidson and Fox (1988) have also suggested that once emotion is perceived, differential lateralization of positive and negative emotions to left and right hemispheres respectively may occur.

For a thorough review and discussion of hemispheric specialization, the reader is referred to Berent (1981) and Bryden (1982).

Clinical Lesion Studies

Some of the earliest studies to shed light on the neuropsychology of depression involved patients with unilateral brain lesions. These clinical lesion studies have generally revealed consistent differences in the affective behavior of subjects with unilateral left vs. right lesions (Coffey, 1987). In 1939, Goldstein first reported a high incidence of a "catastrophic reaction" in patients with lesions to the LH. Since then, this depressive catastrophic reaction, which is characterized by negative affect, fear, pessimism, hopelessness, and crying (Davidson, 1984), has frequently been observed in other studies, whereas an opposite reaction characterized by indifference, anosognosia, mania, and pathological laughing has been associated with right-sided lesions (Gainotti, 1972, Robinson and Starkstein, 1989; Sackeim *et al.*, 1982; Starkstein *et al.*, 1987). For example, Gainotti (1972) examined 160 cases of unilateral cerebral lesions and found that the catastrophic reaction occurred significantly more frequently among left brain-damaged patients as compared to right-sided lesioned patients who exhibited an opposite reaction characterized by indifference, anosognosia, and joking. Gasparrini *et al.* (1978) administered the Minnesota Multiphasic Personality Inventory to patients with unilateral brain lesion and found that almost 50% of the LH lesioned group had a depression score of $T > 70$ while none of the RH group exhibited this profile. As left and right lesioned patients had been matched for both severity of cognitive and motor dysfunction, this study's findings cannot be totally attributed to differences in such abilities.

Other studies have indicated that patients with left anterior cortical or subcortical brain lesions are significantly more depressed as compared to patients with any other lesion site (Parikh *et al.*, 1988; Robinson *et al.*, 1983, 1984; Robinson, Lipsey *et al.*, 1985; Starkstein *et al.*, 1987). For example, Robinson *et al.* (1983) reported that almost two-thirds of their left anterior lesioned group had a cluster of symptoms associated with major depressive disorder. Robinson and associates also found a strong positive correlation between the severity of the depression and proximity of the lesion to the left frontal pole for both cortical and subcortical lesions (Robinson *et al.*, 1985; Robinson, Starr *et al.*, 1985; Robinson and Szetela, 1981; Starkstein *et al.*, 1987). Recently, Starkstein *et al.* (1991) found evidence suggesting that the association between poststroke depression and lesion

location is restricted to patients with typical occipital asymmetry and not found in patients with reversed occipital asymmetry.

It should also be noted that intellectual and physical impairment have been found to correlate with the severity of depression in poststroke patients (Parikh *et al.*, 1988; Robinson, Starr *et al.*, 1985). However, Robinson and Lipsey (1985) have indicated that the loss of cognitive function is a poorer guide to the severity of depression than the site of the lesion while other studies have suggested that impairment does not initially produce depression (Robinsons *et al.*, 1986).

While many unilateral lesion studies have indicated the importance of the LH, especially the anterior region, in depressive syndromes, depression has also been observed in poststroke patients with RH lesions (Folstein *et al.*, 1977; Grafman *et al.*, 1986; Robinson *et al.*, 1984; Starkstein *et al.*, 1989). For example, Folstein *et al.* (1977) studied 20 patients with either left- or right-sided strokes and found that 70% of the RH group displayed depressive symptomatology while none of the LH patients exhibited such signs. Similarly, Grafman *et al.*'s (1986) study of unilateral orbitofrontal lesions also found that right-sided lesions were prone to depression. RH patients with major depression have been also shown to more frequently have parietal lobe lesions and a familial history of psychiatric disorder than patients with either no mood changes or major depression following LH lesions (Starkstein *et al.*, 1989). Furthermore, Jorge *et al.* (1993) recently found that anxious-depressions were more strongly associated with RH damage whereas major depressions were related to left anterior lesions.

Although patients with RH lesions may appear indifferent or apathetic, Ross and associates have demonstrated that, in fact, they may, be very concerned about their deficits, may be depressed, or even may be suicidal (Ross, 1981; Ross and Rush, 1981; Ross and Stewart, 1987). Alternatively, patients with unilateral RH lesions who are not actually depressed but who appear affectively flat may be misdiagnosed as depressed (Ross, 1981, 1993; Ross and Rush, 1981). Ross and his colleagues have also proposed that indifference reactions may actually reflect deficits in the comprehension and expression of affect (i.e., aprosodias) that result from RH damage rather than patients' true affective states (Ross, 1981, 1993; Ross and Rush, 1981). Thus, while depressive symptomatology has widely been associated with unilateral LH lesions, other studies have indicated that patients with RH lesions may also suffer from depression and possess affective states that are in contrast to their indifference appearance.

The catastrophic and indifference reactions seen in clinical lesion studies have been hypothesized to result from a breakdown (due to hemisphere damage) of the reciprocal interaction between the controlling systems of the left and right hemispheres. This is thought to lead to disinhibition of

the undamaged hemisphere (Flor-Henry, 1979; Otto *et al.*, 1987; Swartzburg, 1983). For instance, when the RH is no longer "under" LH control due to damage to the left side, the catastrophic reaction may be released (Flor-Henry, 1979; Swartzburg, 1983). For a comprehensive review and discussion of the emotional reactions that have been associated with brain damage, the reader is referred to Ruckdeschel-Hibbard *et al.* (1986).

Sensory-Perceptual Studies

A diversity of neuropsychological research has demonstrated hemispheric asymmetries in depressed individuals who have no history of brain lesions. Several studies have involved the use of a dichotic listening paradigm that presents auditory stimuli simultaneously to both ears (see Lezak, 1976). One major finding from such investigations has been that depressives, as compared to controls, exhibit evidence of RH dysfunction that may even resemble the performance of right temporal lobe damaged patients (Bruder *et al.*, 1989; Yozawitz *et al.*, 1979). For example, Bruder *et al.* (1989) found that major depressives showed abnormally large right ear advantages (REA) for consonant-vowel syllables (primarily due to poor left ear performance) and also displayed REA for complex tones, which is opposite of that typically seen in normal controls. In addition to RH dysfunction, the authors noted that these findings possibly indicate RH overactivation. Other dichotic listening studies have found that depressives, in contrast to controls, did not exhibit the expected REA for verbal material (Johnson and Crockett, 1982; Moscovitch *et al.*, 1981). Moscovitch *et al.* (1981) suggested such findings may indicate that the RH is strongly primed in depressed subjects. However, the absence of the REA for verbal stimuli may also indicate LH dysfunction. Furthermore, in addition to an absence of a REA for verbal material, Johnson and Crockett (1982) failed to find a left ear superiority for musical chord processing. This may suggest both right and left hemisphere dysfunction in depressives on dichotic listening tasks.

Studies involving the use of a tachistoscope that presents stimuli to the left and right visual fields (and corresponding right and left hemispheres) have also found hemispheric asymmetries in depressives. In a study examining the processing of tachistoscopically presented verbal stimuli, depressed women displayed a trend toward RH superiority as opposed to the LH superiority of normal controls (Silberman *et al.*, 1983). It was hypothesized that the RH of depressives may share functions normally performed by the LH in nondepressives. Hence, the RH may be compensating for a relative deactivation of the LH. Crews and Harrison (1992) investigated the hemispheric processing of tachistoscopically presented happy,

sad, and neutral faces in depressed and nondepressed women. Depressed, as compared to nondepressed, women displayed significantly faster reaction times to sad faces presented their right visual fields (LH) and happy faces presented their left visual fields (RH). In light of arousal theory, these results were suggested to reflect differential arousal of both the left and right hemisphere with a relatively greater arousal of the RH. However, the depressed women also acknowledged elevated levels of anxiety. As it is unknown how anxiety and depression differentially impacted reaction times, these results should be interpreted with caution. In contrast, Bruder *et al.* (1989), in his tachistoscopic study of depressives and nondepressives, failed to demonstrate differences between groups on verbal and dot enumeration tasks. Thus, while there is some conflicting data across tachistoscopic studies of depression, a number of studies have suggested a heightened RH activation relative to that at the LH.

Electrodermal Studies

Other neuropsychological research has involved recordings of depressives' electrodermal activity (EDA). Myslobodsky and Horesh (1978) examined bilateral skin conductance in endogenous and reactive depressives during visual and verbal tasks and tone habituation conditions. Results indicated that EDA was higher at the left, as compared to the right hand, during all conditions for the endogenous group and higher on the left side during the verbal and tone habituation conditions for the reactive depressives. These findings may indicate a hyperactivity of the RH in depression if it is presumed that hemispheric influences are largely contralaterally controlled. Likewise, in a study of high-risk subsyndromal depressives and normal controls, high-risk subjects displayed larger left than right-hand skin conductance amplitudes in response to neutral tones (Lenhart and Katkin, 1986). These data are also suggestive of RH activation in affective disorders.

Electroencephalographic Studies

The brain's electrical activity (electroencephalograms [EEG]) has also been used to investigate hemispheric asymmetries in depressives. Two earlier studies conducted by d'Elia and Perris (1973, 1974) examined quantitative EEG asymmetries in patients with psychotic depression. Results indicated significantly lower within patient variability on the left side vs. the RH in depressives. Further, the investigators found that left-sided involvement in depressives was proportional to the degree of depression.

These data suggest pronounced LH involvement in depression and that this hemisphere may be "overaroused" relative to the RH. It should be noted, however, that psychotic, as opposed to nonpsychotic, depressives served as subjects in these studies with no control groups used for comparisons. Just as psychotic vs. nonpsychotic depressives differ in their clinical presentations, the two diagnostic groups are also likely to differ in their EEG presentations. Hence, caution is advised in extrapolating these data to nonpsychotic depressives.

Several investigators have examined resting EEG asymmetries in nonpsychotic depressed subjects and found evidence for heightened right frontal activation in depressives, as compared to their LH and nondepressed controls (Kano *et al.*, 1992; Schaffer *et al.*, 1983). These studies lend support to the hypothesis of increased RH activation in the frontal lobes of depressives. Flor-Henry (1978) has also reported an increased RH activation in depression, especially in the right temporal lobe. In contrast, other research of resting EEG activity has indicated a decreased activation of the left frontal region in depressives as compared to controls (Davidson, 1992; Henriques and Davidson, 1991).

Other studies, however, have indicated alternative patterns of activation asymmetries in depressives. For example, Knott and Lapierre (1987) found EEG evidence for deactivation of the right side in depression whereas Davidson *et al.* (1987) found that depressives exhibit decreased right-parietal activation. Hence, at present, the EEG results are suggestive of a heightened right-sided activation relative to that at the LH in depression, although such EEG findings have not been consistently replicated across all studies (Swartzburg, 1983).

SPECT Studies

Measures of regional cerebral blood flow (rCBF) have been used to examine hemispheric asymmetries in depressives. For example, Delvenne *et al.* (1990) examined rCBF of major depressives and controls using single positron emission computed tomography (SPECT). Endogenous depressives evidenced significantly lower cortical blood flow in the LH as compared to controls. Similarly, Mathew *et al.* (1980) examined the rCBF of 13 patients with major depression and 13 controls. Depressives exhibited significantly lower cerebral blood flow values for the LH as compared to controls. Flow values were also negatively correlated with the depth of depression. Together, these results are indicative of hypoperfusion of the LH in depression, which suggests neural hypoactivity (Mathew *et al.*, 1980).

PET Studies

Positron emission tomography (PET) has also been employed to examine the glucose metabolic rates in various cerebral regions (Schwartz *et al.*, 1987). In their study of cerebral glucose metabolism, Baxter *et al.* (1989) found that the rate for the left dorsal anterolateral prefrontal cortex divided by the rate for the whole ipsilateral hemisphere in major depressives was significantly lower as compared to controls. There was also a negative correlation between this ratio and depression ratings. Other PET studies have found evidence of decreased rCBF rates in the left anterior cingulate and left dorsolateral prefrontal cortex of major depressives (Bench *et al.*, 1992; Dolan *et al.*, 1992). Dolan *et al.* (1992) also found that a cognitively impaired depressed group vs. a nonimpaired depressed group displayed decreases of rCBF in the left anterior medial prefrontal cortex. This finding suggests that cognitively impaired depressives may have somewhat different cortical abnormalities than nonimpaired depressives. Additionally, Baxter *et al.* (1985) found no significant differences in left–right asymmetries in metabolic rates for unipolar depressives as compared to controls, but there was a subgroup of depressives with lower left frontal cortical rates. This subgroup could not be distinguished, however, from other depressives on clinical grounds. In contrast, other studies (e.g., Hurwitz *et al.*, 1990) have found significant reductions for both anterior left and right cortical regions, with metabolic reductions occurring more frequently in the right vs. the left regions. Thus, while most PET studies lend support to the hypothesis of left frontal dysfunction/hypoactivation in depression, other abnormal metabolic patterns have also been found that may possibly be due to differences between samples of depressives tested, specific procedural techniques, and/or accuracy of PET measurement (Phelps *et al.*, 1984).

Interim Summary

It must be cautioned that the preceding review may represent an oversimplification of the importance (role) of the level (cortical, subcortical) and type of brain pathology in depression. For example, Gilley (1990) found differences in the severity of depressive symptomatology across three neurodegenerative disorders. Specifically, depression was most severe in Parkinson's disease (subcortical), intermediate in subcortical vascular disease, and least severe in Alzheimer's disease (cortical). Thus, depression may also be a function of the anatomical level and type of brain involvement.

In sum, evidence from a diversity of neuropsychological research, including clinical lesion, perceptual, electrodermal, EEG, SPECT, and PET studies, suggests that depression is associated with heightened RH activity (especially over the frontal region) relative to that at the LH. Similar to what has been described in studies cited herewithin, this differential activation may occur via either (Otto *et al.*, 1987) an increase in the activity of the RH or by a decrease in LH activity. It should be noted that these studies do not prove that depression is caused by such hemispheric asymmetries as the temporal cause and effect relationship between this differential activation and depression is unknown, as well as the possible influence of other variables (for example, stressors, biochemical influences) on the expression of depression. Rather, these studies should be viewed as patterns of cerebral activity that correlate with the expression of depression (Baxter *et al.*, 1989). Finally, as various studies have found dissimilar patterns of activation, these conclusions are not unequivocable.

Psychometric Studies

Intimately related to the neuropsychology of depression is research that has investigated the neuropsychological test performances of depressives. Numerous studies have found impairments in depressives on tests reported to be sensitive to executive functions (frontal lobe regions). For example, depressives have been found to display deficit performances relative to controls (nondepressives) on the Trail Making (Fisher *et al.*, 1986; Shipley *et al.*, 1981) and Category tests (Savard *et al.*, 1980; Watson *et al.*, 1978) of the Halstead-Reitan Battery. These tests appear most sensitive to both left and right frontal lobe dysfunction (see Kolb and Whishaw, 1990; Lezak, 1976). Impaired performances in depressives have also been seen on the Stroop Color-Word Test (Fisher *et al.*, 1986) and the Wisconsin Card Sorting Test (Martin *et al.*, 1991) where perseverative responses were noted. These measures are also reported to be most sensitive to frontal dysfunction (see Kolb and Whishaw, 1990; Lezak, 1976). Additionally, depressives have displayed significant deficits on the Left Hemisphere Lateralization Scale as well as the Right Frontal Localization Scale of the Luria-Nebraska Neuropsychological Battery (Newman Silverstein, 1987). However, this group of depressives also displayed psychomotor retardation, which may have influenced these results. Miller (1975), and Cassens *et al.* (1990) have indicated that a number of studies have unveiled an association between motor deficits and both mild and severe levels of depression. Further, increasing severity of depression has been strongly associated with motor performance impairments, especially those requiring sustained effort

(Cohen *et al.*, 1982). Depressives have also displayed impairments on various tests of abstraction ability (Braff and Beck, 1974; Newman and Sweet, 1986; Shipley *et al.*, 1981). Although abstraction is likely associated with frontal lobe functioning (Stuss and Benson, 1984), it should be cautioned that this ability may be disrupted by dysfunction in a number of functionally discrete brain areas (for example, tertiary areas of the posterior cortex) or via diffuse cerebral dysfunction (Luria, 1973).

The posterior brain regions have also been associated with neuropsychological deficits in depressives. A number of researchers have found consistent impairments in right posterior-sensitive visuospatial processing and constructional tasks (Dean*et al.*, 1987; Fromm and Schopflocher, 1984; Kronfol *et al.*, 1978; Shipley *et al.*, 1981; Watson *et al.*, 1978). For example, in their study of moderate to severe depressives, Kronfol *et al.* (1978) found impaired performances (compared to medical patients) on the following right parietal lobe tasks: Judgment of Line Orientation, 3-dimensional Constructional Praxis, and Facial Recognition. Fromm and Schopflocher (1984) also found selective impairments in visuospatial processing on an expanded Halstead-Reitan Neuropsychological battery administered to a group of depressed patients. Furthermore, Cassens *et al.* (1990) have stated that visuospatial and visuomotor tasks are consistently impaired in individuals with different subtypes of depression (e.g., reactive, endogenous).

Depressed individuals appear to be susceptible to a diversity of memory impairments (Cassens *et al.*, 1990). Two research groups have found performance deficits in depressives on major subfunctions of the Wechsler Memory Scale, including Mental Control, Verbal Learning, and Visual Reproduction (Breslow *et al.*, 1980; Stromgren, 1977). Results of the Verbal Learning and Visual Reproduction subfunctions suggest left and right hemispheric dysfunction, respectively (see Bigler, 1988). Other studies have also found evidence of LH dysfunction on memory tests. For example, impairments have been observed in depressives' short-term verbal memory (Cassens *et al.*, 1990; Fromm and Schopflocher, 1984; Sternberg and Jarvik, 1976), sentence repetition (Kronfol *et al.*, 1978), and long-term memory (Cassens *et al.*, 1990). Alternatively, RH memory tasks have shown impairments in depressives. These deficits include short-term nonverbal/visuospatial memory (Cassens *et al.*, 1990; Fromm and Schopflocher, 1984; Sternberg and Jarvik, 1976), visual memory (Deptula and Yozawitz, 1984), and long-term nonverbal memory (Dean *et al.*, 1987). Hence, it appears that both left- and right-sided controlled memory may be impaired in depression.

In sum, depressed individuals may exhibit a wide diversity of neuropsychological deficits suggestive of both left and right hemisphere involvement. However, due to the inclusion of depressives from different settings

(inpatient/outpatient), ages, groups, genders, and subtypes of depression as well as the failure of many studies to adequately identify their samples (Miller, 1975), it remains unclear if there are specific patterns of cognitive deficits associated with different subgroups of depressives (McAllister, 1981). Miller (1975) has indicated that different subtypes of depression (reactive/psychotic; mild/severe) tend to display similar deficits that appear to differ only as regards the degree of impairment rather than the type of deficit. He also notes that severe depressions are likely to be associated with more impaired performances on neuropsychological tests.

Based on the findings across a diversity of neuropsychological studies (for example, EEG and PET) of the association between the frontal cortical regions and depression, it seems plausible that many of the memory/posterior brain region test deficits that have been associated with depression may at least be partly due, to frontal lobe dysfunction. For instance, studies that have found decrements in depressives on memory and posterior cortical region tests have also found deficits in sustained attention/motivation (Cassens *et al.*, 1990; Cohen *et al.*, 1982), vigilance (Frith *et al.*, 1983), and concentration (Abrams and Taylor, 1987; Newman and Sweet, 1986)—abilities that have been hypothesized to play important roles in the impairment of memory test performances. Although such attentional and concentration abilities may arise from dysfunction in myriad brain areas (Lezak, 1976; Luria, 1973), the frontal lobes have been suggested to play an important role in vigilance levels (Luria, 1973), attentional processes (Stuss and Benson, 1984), as well as maintaining active effort/motives for voluntary recall (Luria, 1973). Further, the psychomotor retardation reported to influence depressives' posterior region test performances (Miller, 1975; Weckowicz *et al.*, 1978) may likely be attributable to frontal lobe dysfunction (Stuss and Benson, 1984). Various studies have also indicated that the frontal lobes are involved in a diversity of other posterior region tasks—for example, spatial orientation (see Kolb and Whishaw, 1990). Hence, the abnormal frontal lobe activation asymmetries related to depression may promote impairments on tasks that have been associated with other cortical regions. While it seems probable that many of the test impairments seen in depressives may be at least partly due to frontal lobe dysfunction, this hypothesis should be regarded as speculative until it can be verified empirically.

Induced Emotion Studies

A number of neuropsychological studies have examined the relationship between induction of positive and negative affects and the differential

activation of the cerebral hemispheres. This research has induced positive and negative emotions via affect-promoting questions/cognitions or film clips and simultaneously recorded EEG activity (Ahern and Swartz, 1985; Davidson *et al.*, 1979, 1990; Wheeler *et al.*, 1993). In general, results of these investigations reveal induced positive affect is associated with increased left frontal activation, whereas heightened right frontal activation is associated with more intense reports of negative affect. These findings suggest that the left and right frontal regions may be differentially specialized for positive and negative affects respectively. Thus, it appears that heightened RH activation relative to that at the LH is associated with not only depression, as noted previously, but also increased negative affect.

It has been hypothesized that this left–right anterior activation asymmetry for positive and negative affects is also associated with approach and withdrawal/avoidance behaviors respectively (Davidson, 1984; Davidson *et al.*, 1990). For example, Sobotka *et al.* (1992) manipulated reward and punishment contingencies to produce approach and withdrawal emotional states while recording EEG activity. Results indicated heightened left frontal activation during reward trials and increased right frontal activation during punishment. These data support the hypothesis that the left and right frontal regions are differentially specialized for approach and avoidance behavior respectively.

Hemispheric Priming Studies

As will become apparent later in this paper's integration of the neuropsychology of depression and cognitive theory/therapy, it is important to note that the cerebral hemispheres may be differentially primed/activated by nonemotional stimuli. A diversity of studies examining EEG (Davidson *et al.*, 1987), rCBF (Gur *et al.*, 1982; Gur and Reivich, 1980), and EDA (Myslobodsky and Horesh, 1978) activity have found increased LH activation during verbal tasks whereas heightened RH activity has been demonstrated during spatial tasks. These data suggest that verbal and spatial tasks may differentially prime the left and right hemispheres.

BECK'S COGNITIVE THEORY AND THERAPY OF DEPRESSION

Beck's Cognitive Theory

While a diversity of cognitive theories of depression have been proposed, debatably one of the most well-known and widely investigated is

Beck's cognitive theory of depression (Beck, 1963, 1964, 1967, 1991; Beck *et al.*, 1979). Thus, this paper will focus exclusively on his model of depression.

Fundamental Assumptions

A fundamental assumption of Beck's theory is the primary importance of cognitions in mediating depression as well as emotional and behavioral responses (Beck and Clark, 1988; Wright and Beck, 1983). By definition, Beck defines cognition as the processes involved in the perception, interpretation, and recall of information (information processing; Beck, 1991). Beck and his associates state that cognitions are generally automatic thoughts that are within a person's awareness, fairly labile, and capable of being elicited by a variety of stimuli (Beck *et al.*, 1983). Further, cognitions are thought to be a necessary, but not sufficient, component involved in producing depression as other factors such as environmental stressors, biological/genetic processes, and developmental factors may interact with an individual's cognitions to result in depression (Beck, 1967; Beck, 1987; Beck and Clark, 1988). For an in-depth review of such integrative models, the reader is referred to Beck (1987).

Beck has proposed several major concepts to help explain and clarify the psychological substrates of depression (Beck, 1963, 1964, 1967, 1976; Beck *et al.*, 1979). These principal components include cognitive schemata, the negative cognitive triad, cognitive errors (errors in information processing), and automatic thoughts.

Cognitive Schemata

According to cognitive theory, distorted, negative thinking (which may result in depression) occurs when an underlying maladaptive schema is activated by external/environmental events (Wright and Beck, 1983). By definition, schemata are a major and relatively stable component of cognitive organization used for screening, coding, and evaluating stimuli that impinge upon an individual (Beck, 1964, 1967). Schemata are also known as the idiosyncratic attitudes, beliefs, and assumptions that influence how a person conceptualizes and interprets experiences (Beck, 1964). These constructs are hypothesized to arise from past experiences and conclusions derived from previously observed cause and effect relationships (Beck *et al.*, 1983; Wright and Beck, 1983).

In depression, schemata are hypothesized to be maladaptive/negative conceptions of such things as one's personal characteristics, worth, perform-

ances, and self-expectations (Beck, 1964). These maladaptive schemata may remain dormant for extended periods of time until they are reactivated by external stimuli or negative events (for example, environmental stress (Beck *et al.*, 1979; Kovacs and Beck, 1978). Once activated, maladaptive schemata tend to become rigid and judgmental and serve to disrupt and replace adaptive schemata that are involved in objective reality testing (Beck, 1964; Beck and Clark, 1988). Maladaptive schemata generally lead to increased negative affect, guilt, low self-worth, and eventually depression (Beck, 1964). These schemata also tend to filter and screen out data that would support alternative explanations (Wright and Beck, 1983). Beck (1964) has suggested that the negative affect produced by the idiosyncratic schemata may also facilitate depressive schemata activity and "enhance the downward spiral in depression." Hence, as the schemata become more and more active, an individual's conceptualization of events becomes increasingly distorted/negative, which may result in increased levels of depression and negative affect (Beck *et al.*, 1979; Kovacs and Beck, 1978).

Beck and his associates have suggested that different psychological disorders, for example depression vs. anxiety, have relatively distinct maladaptive cognitions and schemata associated with them (Beck, 1967; Beck and Clark, 1988). These cognitions are proposed to differ significantly from individuals with no disorders. Based on this content-specificity hypothesis (Beck and Clark, 1988), a number of investigations have sought to provide support for the maladaptive schemata thought to underlie depression specifically. Studies have examined the differences between depressed, anxious, and/or control subjects regarding the specific content of their cognitions (Clark *et al.* 1989, 1990), processing of information (Ingram *et al.*, 1987), and their recall of content-specific adjectives (Derry and Kuiper, 1981; Greenberg and Beck, 1989). Overall, results indicated that depressives, as opposed to other groups, acknowledged more thoughts of loss, failure (Clark *et al.*, 1989), hopelessness, and low self-worth (Clark *et al.*, 1990), as well as selective processing of depressive information and production of less self-serving attributions (Ingram *et al.*, 1987). Depressives, as opposed to other groups, have also displayed enhanced recall for depression-relevant, negative stimuli (Derry and Kuiper, 1981; Greenberg and Beck, 1989). Furthermore, Miller and Norman (1986), in their investigation of the persistence of negative cognitions, found that a significantly greater percentage of high distorting depressives displayed elevated levels of depressive cognitions after clinical improvement as compared to controls. Together these studies appear to support the occurrence of underlying negative schemata in depressives that guide their cognitive processes and information processing and that may likely persist over time.

The Dysfunctional Attitude Scale (DAS) has also been developed to assess assumptions and attitudes that may serve as maladaptive schemata (see Beck *et al.*, 1983, for an overview of this scale). In general, depressives have been found to score higher on this scale relative to controls (Dobson and Shaw, 1986). Studies have also indicated that the severity of depression is positively associated with higher DAS scores (Dobson and Shaw, 1986; Hollon *et al.*, 1986). Hence, these self-report results also appear to support the occurrence of negative attitudes/schemata in depressives.

Negative Cognitive Triad

As a consequence of activated maladaptive/negative schemata by environmental events, distorted thinking may occur in three general areas known as the negative cognitive triad: negative views of the self, of one's experiences (world), and the future (Beck, 1967; Beck *et al.*, 1979; Wright and Beck, 1983). One component of depressed individuals is their negative view of themselves. Such persons tend to think of themselves as deficient, inadequate, undesirable, and unworthy (Beck, 1967; Beck *et al.*, 1979). Depressives are also likely to attribute unpleasant experiences and their negative views of themselves as due to psychological, moral, or physical deficits in themselves (Beck, 1967).

Negative views of the environment (world) around them (depressives) is a second component of the triad (Beck, 1967). Depressives tend to interpret their interaction with their environments as filled with obstacles, burdens, or traumatic situations. Further, depressed individuals often think their interactions with their environments represent defeats or deprivation (Beck, 1967).

A final component of the cognitive triad is negative views of the future. Depressed persons often think that current problems and suffering will continue indefinitely (Beck, 1967). They anticipate a life of hardships, frustration, deprivation, and expect to fail at any task they consider doing (Beck, 1967; Beck *et al.*, 1979). These distorted views of the cognitive triad may also feed back into depressed individuals' thinking and serve to strengthen the associated maladaptive schemata.

Various studies have been undertaken to empirically verify the negative cognitive triad. For example, Greenberg and Beck (1989) examined depressed, anxious, and control subjects' cognitions related to their self, world, and future via use of trait rating and incidental recall tasks. Results indicated depressives, as compared to other groups, affirmed more negative/depression-relevant stimuli as descriptive of their selves, world, and future. Similarly, another study conducted by Blackburn and Eunson (1989)

tended to confirm these findings. The investigators sampled 200 thoughts of depressives at the beginning of cognitive therapy and found that all three elements of the negative triad were present in these subjects; however, negative views of the self and world as compared to the future occurred with greater frequency. Together, these results lend credence for the occurrence of the negative cognitive triad in depressed persons.

Automatic Thoughts

Beck has reported, based on his clinical observations and interviews of depressives, that many negative/depressotypic thoughts tend to arise automatically and involuntarily, without any prior reflection or reasoning (Beck, 1963, 1976). Depressed subjects tend to be only partially aware of the occurrence of these automatic cognitions, which are hypothesized to result from the influence of environmental events on the depressotypic schemata (Beck *et al.*, 1979). Beck (1976) has indicated that automatic thoughts are generally regarded as plausible by depressives even though they may actually seem unreasonable to nondepressives. Depressed persons usually accept these thoughts without questioning their validity. Automatic thoughts are also theorized to be the least stable of any of Beck's hypothetical cognitive constructs as they may arise and disappear rapidly (Beck *et al.*, 1983).

The presence of negative automatic thoughts has been demonstrated in depressives via the Automatic Thoughts Questionnaire (Ingram *et al.*, 1987; Kwon and Oei, 1992). For example, Ingram *et al.* (1987) administered this questionnaire to depressed, anxious, and control subjects. Results indicated that the depressed subjects acknowledged significantly more negative automatic thoughts as compared to both anxious and control subjects. Such results appear to provide supporting evidence for the occurrence of negative automatic thoughts in depressives.

Cognitive Errors

Several major subtypes of reasoning errors, also known as cognitive errors (or systematic errors), have been identified in the automatic thoughts of depressed individuals (Beck, 1963, 1967; Beck *et al.*, 1978; Wright and Beck, 1983). These thinking errors are hypothesized to maintain depressives' negative beliefs despite evidence to the contrary (Beck *et al.*, 1979) and are typically associated with depressotypic negative affect (Wright and Beck, 1983). Cognitive errors have also been termed primitive as they result in depressives making broad global judgments about events (Beck *et al.*,

1979). Beck (1963, 1967) has classified these depressotypic cognitions into three broad categories depending on how they deviate from logical, realistic thinking. These categories include, with examples of each, paralogical (arbitrary inference, selective abstraction, and overgeneralization), stylistic (exaggeration or magnification/minimization), and semantic (inexact labeling; (Beck, 1963, 1967).

Arbitrary inference involves the inaccurate interpretation of an event in light of no factual supporting evidence or when the conclusion is opposite to the evidence (Beck, 1963). Selective abstraction is defined as the process of focusing on a single detail of an event and forming impressions based solely on this detail while ignoring other features of the situation (Beck, 1963). In overgeneralization, depressives form a broad conclusion about themselves based on an isolated incident (Beck, 1963). Similarly, exaggeration involves the overestimation or underestimation of the significance of a traumatic event or of an individual's performance. Inexact labeling refers to the distorted cognitive labeling of an event that, in turn, negatively impacts depressives' affective reactions (Beck, 1963). Thus, the label assigned is not proportional to the intensity of an event. In response to these cognitive errors, the meanings of events tend to be extreme, judgmental, and negative that, in turn, tend to result in very negative emotional responses (Beck *et al.*, 1979). For a more extensive review of cognitive errors, the reader is referred to Beck (1963).

Support for the cognitive errors is found in some of Beck's early work which, incidentally, contributed to the formulation of these concepts (Beck, 1963, 1967). His research involved conducting face-to-face interviews with 50 depressed and 31 nondepressed subjects as regards their thoughts and feelings both prior to and during therapy sessions. Based on the data collected, Beck (1963, 1967) concluded that depression was characterized by certain themes such as low self-esteem, self-blame, and desires to escape. Further, depressed, as opposed to nondepressed, groups displayed all of the systematic errors hypothesized to contribute to their depressive themes. Caution is advised in interpretation of these results, however, as data were based on self-report, most of which was actually retrospective. Such self-report data have been found to be susceptible to a number of response biases—for example, attempting to please the experimenter (see Anastasi, 1982)—and are also known to be highly subjective and unverifiable in nature (Evans, 1986; Kulper and MacDonald, 1983).

Additional support for the occurrence of cognitive errors has been demonstrated in a study conducted by Blackburn and Eunson (1989). On examination of depressed subjects' self-reported thoughts, two raters were able to reliably label the five cognitive errors which Beck described. As noted above, however, such self-report data should be interpreted with cau-

tion due to the lack of objective verifiability of the data and the potential for response biases in such data (Anastasi, 1982; Evans, 1986; Kulper and MacDonald, 1983).

Beck has also theorized that negative/depressive affects are evoked by cognitive errors and unpleasant thoughts (Beck, 1963, 1967). This hypothesis was based on Beck's (1963) clinical observations and interviews that patients' negative thoughts, which generally contained cognitive errors, often preceded the negative affect. Beck hypothesized that when a thought resulted in a strong affective reaction, patients would have more difficulty in objectively appraising the cognition. These observations appear to be a foundation on which Beck developed his theory of the importance of cognitions in mediating affective states. However, as Beck typically had study subjects who felt negatively "think back" and recall the content of their previous cognitions, this technique may have resulted in misattributions regarding the relationships between their prior thoughts and current feelings.

Additionally, similar to what has been suggested for schemata, negative affect evoked via cognitive errors may maintain and/or facilitate further negative cognitions and cognitive errors (Beck, 1963, 1967). In turn, this may result in the downward spiral (deeper depression) frequently observed in depression (Beck, 1963, 1967).

Cognitive Therapy

Fundamental Concepts

To correspond to his cognitive theory of depression, Beck also formulated cognitive therapy for depression disorders that focuses primarily on patients' distorted, negative cognitions (Beck *et al.*, 1979). In general, cognitive therapy was typically designed for the treatment of unipolar, nonpsychotic depression. This short-term treatment usually involves 12–20 individual therapy sessions, initially held twice weekly, which then taper to once weekly after several weeks (Beck *et al.*, 1979; Wright and Beck, 1983). Cognitive therapy involves a multitude of techniques to modify depressive states. Initial techniques generally involve recognizing and testing maladaptive automatic thoughts, whereas later procedures include detection and modification of underlying schemata. As discussion of specific therapy methods is beyond the scope of this paper, the reader is referred to Beck's own writings (Beck, 1976; Beck *et al.*, 1979) for detailed coverage of cognitive therapy techniques for depression. Last, the goals of cognitive therapy typically involve modification and resolution of depressives' cognitions and symptoms as well as the teaching of new ways of thinking that may

promote positive cognitions and help prevent the return of depressive symptomatology (Beck *et al.*, 1979; Wright and Beck, 1983).

Mechanisms of Change

A diversity of studies have examined the specific processes/mechanisms that may contribute to cognitive therapy efficacy. Rush *et al.* (1981) examined symptom change in unipolar depressives treated with cognitive therapy or pharmacotherapy via cross-lagged point analysis to evaluate temporal relationships among symptoms. During cognitive therapy, results indicated that client improvement in hopelessness, views of the self, and mood tended to precede changes in vegetative and motivational symptoms, while no reliable change patterns were associated with the pharmacotherapy group. These findings were suggested to provide support for cognitive therapy in that alterations in negative cognitions (views of self and future) and mood generally preceded reductions in other depressive symptoms (i.e., vegetative and motivational symptoms). The relationship between automatic thoughts and mood changes during cognitive therapy sessions has also been examined (Persons and Burns, 1985). Findings indicated a strong positive association between decreases in patients' automatic thoughts and reductions in the intensity of their negative mood states. However, it should be noted that a good therapeutic relationship was also associated with intrasession mood improvements and may have contributed to these findings.

Other studies that have examined cognitive therapy mechanisms have divided therapies into self-monitoring, logical analyses, and hypothesis-testing components (Jarrett and Nelson, 1987). As compared to self-monitoring, major depressives exposed to logical analysis and hypothesis-testing components were found to be associated with fewer self-reports of depression symptomatology and dysfunctional thoughts. Better interpersonal relationships and more pleasant experiences were also associated with these active components of cognitive therapy. Similarly, Teasdale and Fennell (1982) studied the immediate (within sessions) effects on depression of thought-exploring vs. a thought-change condition that encouraged depressives to reevaluate their depressive thinking by considering alternative explanations for events. Findings indicated that the thought-change condition resulted in more belief change and greater reductions in depressive mood as compared to the thought-exploration condition. These results suggest that certain cognitive therapy techniques may have immediate, positive impacts on depressive symptomatology. DeRubeis *et al.* (1990) has examined the association between changes in depression-relevant cognitions (i.e., automatic thoughts, hopelessness, underlying assumptions, and attributions)

and change in depressive symptomatology in patients with major depression disorder who were randomly assigned to cognitive therapy or pharmacotherapy. Although significant improvement in all four cognitive variables was noted from pretreatment to midtreatment in both treatment groups, this finding was predictive of change in depressive symptomatology only in the cognitive therapy group from midtreatment to posttreatment and not in the pharmacotherapy group. The results appear to provide additional support for cognitive variables playing a mediational role in cognitive therapy for depression.

While these studies appear to provide some support for various cognitive mechanisms that may be associated with cognitive therapy efficacy, there is presently a lack of data on the precise mechanisms responsible for the therapeutic success (Fennell, 1983). For example, it is likely that many nonspecific variables, such as the therapist/client relationship, may also play important mediational roles in cognitive therapy outcomes. Furthermore, other studies involving diverse treatments have found that depressives demonstrate similar changes in cognitive and mood variables after receiving different therapies (Simons *et al.*, 1984; Zeiss *et al.*, 1979). Simons *et al.* (1984) examined change process in moderately depressed outpatients who had been randomly assigned to either cognitive therapy or pharmacotherapy. Results indicated that patients in both treatment groups displayed very similar positive changes in mood, cognitive processes, and content. Such findings suggest that diverse treatments may have nonspecific effects and that cognitive changes may be due more to depression improvement rather than the specific result of cognitive therapy (Simons *et al.*, 1984; Zeiss *et al.*, 1979). However, Hollon *et al.* (1987) have stated that even if diverse treatments lead to similar outcomes (for example, similar mood and cognitive changes), the mediating factors (for example, cognitive processes or biochemical process) leading to such outcomes may be quite different across diverse depression therapies. Thus, cognitive variables may play mediating roles in modifying depression during cognitive therapy whereas other variables (for example, biochemical) may play roles in modifying depression in the course of other therapies (for example, pharmacotherapy) although the final outcomes will appear similar.

Mode-Specific Changes

In addition to a large diversity of outcome (see Dobson, 1989, for a meta-analysis review) and mechanism studies (presented above) that have demonstrated the effectiveness of cognitive therapy in reducing depressive symptomatology, other studies have found mode-specific changes associ-

ated with such treatment. Gauthier *et al.* (1983) compared the effectiveness of cognitive restructuring and cognitive rehearsal with self-observation and a waiting list control in enhancing self-esteem. Results indicated that only the cognitive therapy strategies were found to be significantly better than the waiting list for self-esteem enhancement and reduction of depressed mood. McNamara and Horan (1986) have examined cognitive vs. behavioral treatment effects in a sample of moderately depressed subjects. The authors found that cognitive therapy produced a significant and durable impact on a battery of measures reflecting cognitive aspects of depression, whereas behavior therapy resulted in no improvement on any cognitive or behavioral assessment measures. These findings of convergent and divergent results were suggested to demonstrate the construct-valid strength of cognitive therapy. The effects of cognitive-behavioral therapy plus pharmacotherapy or only pharmacotherapy were investigated in groups of major depressives with varying levels of cognitive dysfunction (Miller *et al.*, 1990). Findings indicated that the combined treatment resulted in significant decreases in the dysfunctional cognitions of highly cognitive dysfunctional patients whereas pharmacotherapy alone did not. These data suggest that cognitive-behavioral therapy has mode-specific effects of modifying dysfunctional cognitions, which may be important for treatment response. Similarly, Whisman *et al.* (1991) investigated the mode-specific changes on dysfunctional cognitions in patients with major depressive disorder who received either standard treatment (pharmacotherapy and milieu management) or this treatment plus cognitive therapy. Results indicated patients who also received cognitive therapy acknowledged less hopelessness and fewer cognitive biases posttreatment, and at six and 12-month follow-ups. In contrast to the above mode-specific findings, it should be noted that the NIMH Treatment of Depression Collaborative Research Program (Sotsky *et al.*, 1990) found relatively little support for mode-specific differences across a wide diversity of treatments, which include cognitive-behavioral therapy. These findings may reflect the possibility of either common factors operating across therapies or a lack of a more comprehensive and sensitive assessment battery for each therapy as only one measure specifically designed for each treatment was used. Mode-specific effects may have occurred during different therapies (rather than being present at treatment end) that resulted in similar improvement across assessment measures by treatment end (Hollon *et al.*, 1987). Thus, while the data on mode-specific changes is mixed, overall the data seems to support the notion that certain mode-specific changes are associated with cognitive therapy.

Predictors of Response

A number of patient variables have been associated with response to cognitive therapy. Beckham (1989), in his examination of mildly to severely depressed patients treated via cognitive therapy, found the greatest amount of improvement in mild to moderately depressed patients, whereas persons who were severely depressed at treatment start exhibited no significant improvement as therapy progressed. Alternatively, another study (Thase, Simon, Cahalane, McGeary, and Harden, 1991) found that severity of depression did not predict differential levels of depressive symptomatology at end of cognitive behavior therapy. However, the severely depressed group did display more depressive symptoms across therapy sessions and also tended to remit less completely. Further, Jarrett *et al.* (1991) found that higher symptom severity of unipolar depressives was predictive of poorer responses. The authors also found that married depressives and subjects with low levels of cognitive distortions responded better to cognitive therapy than did single patients or ones with high levels of cognitive distortions. Other studies (Sotsky *et al.*, 1991) have likewise found better treatment responses in depressives with low cognitive dysfunction whereas Keller (1983) found high levels of dysfunctional attitudes predicted poor response to cognitive therapy. High learned resourcefulness has also been found to predict better cognitive therapy response (Simons *et al.*, 1985). This suggests that cognitive therapy may capitalize on a preexisting repertoire of cognitive skills possessed by depressives. Finally, cognitive therapy has been demonstrated to be effective with depressed patients who demonstrate endogenous features (Thase, Simons, Cahalane, and McGeary, 1991; Thase, Simons, Cahalane, McGeary, and Harden, 1991). Although the studies cited above may aid in predicting who will benefit the most from cognitive therapy, these studies do not specifically explain why such characteristics may predict better treatment success.

Summary

In conclusion, a host of investigations have provided a reasonable degree of support for Beck's cognitive theory and therapy of depression. While these studies suggest that cognitions may play a mediational role in depression, as Beck has noted, it is farfetched to state that cognitions cause depression since other factors, such as environmental stressors and biochemical alterations, are likely involved in the development of the disorder (Beck, 1987; Beck, 1991; Wright and Beck, 1983).

Furthermore, a number of methodological problems that tend to occur throughout the majority of cognitive studies precludes the determination of cause-and-effect relationships between cognitions and depression. For example, many investigations have relied upon patient self-report. Self-report, as noted previously, is subject to a number of possible response biases and is also limited by an individual's awareness and perceptions of his/her problems (Kulper and MacDonald, 1983). Such data is also highly subjective and unverifiable, and thus many questions may arise as to the validity of this type of information. Additionally, the majority of cognitive studies have been correlational in nature; for example, changes in cognitions are often found to be only "associated" with changes in depressed mood. It is widely known that such studies do not lend themselves to the determination of temporal cause-and-effect relationships. Cognitive studies have also not typically controlled for possible confounding variables, such as the client–therapist relationship, that may be at least partly responsible for the studies' outcomes. Thus, there appears to be a relative absence of sound methodological data regarding the precise cause-and-effect relationship between cognitions and depression.

NEUROPSYCHOLOGICAL IMPLICATIONS FOR COGNITIVE THEORY AND THERAPY OF DEPRESSION

Theory Support

A diversity of neuropsychological evidence appears to provide credibility to Beck's cognitive theory for depression. As discussed herewithin, the RH, especially the frontal region, appears to be differentially specialized for processing negative affect. This right frontal region has also been hypothesized to be associated with avoidance/withdrawal behaviors. In depression, the RH has been associated with heightened activation relative to that at the LH. Induced emotion studies have demonstrated that presentation of negatively intoned stimuli is also associated with greater RH activity. As Otto *et al.* (1987) has briefly alluded, based on these neuropsychological studies, it may be that in depression-susceptible individuals, negative cognitions differentially activate the RH. Alternatively, the RH might first exhibit increased activation, which in turn promotes negative affect and fuels negative cognitions. Once activated, however, the RH may facilitate a negative affective/depressive state that predisposes such individuals to experience additional negative cognitions. It appears possible that a continued cycle of such negative cognitions and RH activation might produce the downward spiral (deeper depression) hypothesized by Beck (1963,

1967). This scenario appears similar to Beck's hypothesis that evoked affect may maintain and/or facilitate further negative cognitions.

It seems probable that the underlying negative schemata hypothesized by Beck (1964) may be due, at least in part, to heightened RH activation that colors an individual's perceptions, interpretations, and cognitions in a negative depressotypic manner. Beck has suggested that schemata tend to become more and more active in depression, which results in events becoming increasingly negative and distorted. Thus, similar to the cycle noted above, it seems possible that heightened RH activation relative to that at the LH may result in schemata becoming increasingly depressogenic that, in turn, promotes increased RH activation and continuation of the downward spiral.

Similar to depressotypic schemata, the negative cognitive triad might also be maintained by heightened RH activation relative to that at the LH. Beck (1967) has stated that negative views of the self, world, and future may also feed back into a depressed individual's thinking and actually strengthen negative schemata. Thus, it seems probable that such negative views might fuel additional RH activation that, in turn, strengthens both negative views and depressotypic schemata. Negative automatic thoughts might also be fueled by a similar cycle.

Cognitive errors may arise, at least in part, from heightened activation of the RH, which appears specialized for processing negative affect. These errors may help maintain the heightened RH activation as they are typically associated with negative affect (Wright and Beck, 1983). Thus, it seems likely that a cycle, similar to the ones described above, may develop, which results in the maintenance and facilitation of depression, negative affect, and negative cognitive errors. The withdrawal and avoidance behaviors that have been hypothesized to be related to right frontal activation may also reaffirm these cognitive errors. For example, depressives may conclude that no one desires to be around them when, in actuality, they are actually the ones who have avoided and withdrawn from others. Thus, this might be an example of arbitrary reference (incorrect conclusions) that occurred in response to avoidance behaviors secondary to RH activation.

Alternatively, the left frontal region hypoactivation, relative to that at the RH, which has been associated with depression via EEG (for example, Henriques and Davidson, 1991) and PET scan (for example, Baxter *et al.*, 1989) studies may likely contribute to several types of frontal lobe dysfunctions that, in turn, may promote distorted, negative cognitions as proposed by cognitive theory. Although it is unknown at present whether such relative decreases in left frontal activation may precede cognitive distortions, it seems likely that once this hypoactivation does occur, it may promote additional maladaptive cognitions. Furthermore, a number of studies (i.e.,

Fisher *et al.*, 1986; Savard *et al.*, 1980; Shipley *et al.*, 1981) have found neuropsychological test deficits in depressives that have been suggestive of frontal lobe dysfunction.

It is possible that frontal lobe dysfunction might result in deficits of the logical self-talk mechanisms that have been suggested to be associated with the LH (Luria, 1973). Depressed individuals may be unable to adequately apply self-talk mechanisms in a logical manner, which results in distorted cognitions and cognitive errors. Errors in reasoning and judgment are frequent correlates of left frontal dysfunction; more specifically, left frontal dysfunction has been suggested to promote concrete attitudes and thinking (Luria, 1973; Stuss and Benson, 1984). Neuropsychological tests have also indicated impairments in depressives' abstraction abilities (for example, Braff and Beck, 1974; Newman and Sweet, 1986), which may be suggestive of frontal lobe impairment. Thus, a depressed individual with left frontal hypoactivation might make very literal interpretations of their experiences, where events are taken at their most obvious face value and little abstract thinking occurs. This may result in cognitive distortions and errors regarding depressives' experiences as they may not fully appreciate all the underlying dynamics and components that are associated with events nor be able to think of events in abstract terms. Depressed individuals with left frontal dysfunction may also be unable to appreciate their impact on others or be able to appropriately analyze social situations (see Lezak, 1976; Stuss and Benson, 1984). This deficit may lead to cognitive errors and erroneous views of themselves and their relationships with others. Perseveration, which has been found in studies of depressives' neuropsychological test performances (for example, Martin *et al.*, 1991) may be another sequelae to prefrontal dysfunction (Kolb and Whishaw, 1990; Luria, 1973; Stuss and Benson, 1984). Depressed individuals with prefrontal dysfunction may be prone to make the same responses or think the same distorted negative thoughts repeatedly and be unable to shift cognitive sets. Finally, as the RH may be somewhat activated relative to that at the LH, this could promote the negative/depressotypic tone of distorted cognitions/errors, which then may be facilitated further by the LH dysfunction.

It should be noted that, just as it is unknown at present whether the relative increased RH activation/decreased LH activation results in depression or vice versa, it is unknown if negative cognitions might produce this pattern of heightened RH activation relative to the LH (and depression) or if such activation patterns (and depression) may alternatively result in negative cognitions. Further, as the precise roles of cognitions in the neuropsychology of depression have remained virtually uninvestigated, the preceding conclusions should be regarded as speculative.

Therapy Support

Cognitive therapy is also supported by neuropsychological evidence. Cognitive therapy may likely be effective in alleviating depression and negative affective states by modifying the decreased left frontal activation relative to the right frontal region that is frequently seen with depression. For example, based on studies that have demonstrated the LH priming (increased activation) effects of verbal tasks (Davidson *et al.*, 1987), it seems probable that as cognitive therapy is a verbal talk therapy, such strategies may likewise serve to prime and increase the activation of the LH. Verbal priming may help offset the relatively heightened RH activation that is hypothesized to promote negative affect and cognitions. Thus, the LH (via increased activation) might come to exert more control over the RH and help restore an activation balance between the hemispheres, which subsequently serves to decrease the predominance of negative affect/cognitions as well as the avoidance behaviors often seen with depression. By increasing the activity of the LH, cognitive talk therapy might also promote increases in approach behaviors and positive affect/cognitions that have been found to be associated with the LH.

Neuropsychological studies may shed light on why certain components of cognitive therapy appear more efficacious in modifying depressive symptomatology as compared to other cognitive techniques. For example, Jarrett and Nelson (1987) found that when cognitive therapy was divided into three components, both logical analysis and hypothesis-testing parts were associated with less depressive symptomatology and fewer dysfunctional thoughts as compared with a self-monitoring component. These two more efficacious components were associated with more pleasant experiences and better interpersonal relationships than the self-monitoring component. It is probable that as both the logical analysis and hypothesis-testing components focused more on logical thoughts and reasoning, functions that have been associated with the LH and especially the left frontal region (Luria, 1973), such techniques might serve to increase the relative activation of the LH and establish more of a balance between the left and right hemispheres' levels of activation. This increased left frontal activation also may be responsible for the increase in pleasant experiences and better interpersonal relationships found in this study as the left frontal region has been suggested to be differentially specialized for positive affect and approach behaviors. Alternatively, self-monitoring might possibly result in depressed individuals focusing more on their negative, depressotypic cognitions and feelings. This may promote a relative increase in RH activation as compared to that at the LH that, in turn, may fuel additional negative cognitions and affect.

Similarly, Teasdale and Fennell (1982) examined the effects of thought-exploring vs. a thought-change condition on belief change and mood. Results indicated that the thought-change condition resulted in more belief changes and greater reductions in depressive mood as compared to the thought-exploration condition. It may have been that as the thought-change condition likely focused more on reason and logical thoughts, this may have served to activate the LH to a greater degree than the thought-exploring condition and, in turn, offset the relatively heightened RH activation frequently seen in depression. While thought exploration, which involved use of verbal linguistic abilities, may have resulted in some LH priming, it seems that such a technique may have also activated the RH as the thoughts of depressives that were explored were likely negative. Such a cognitive technique and the hypothesized increased RH activation may have served to fuel additional negative affect and cognitions which subsequently resulted in significantly less mood change.

Hence, it is hypothesized that different cognitive therapy techniques may differentially activate the left or right hemispheres and result in varying levels of belief and depressive symptomatology change. It is suggested that logical cognitive techniques, which may not focus exclusively on individuals' negative cognitions and feelings, are associated with greater LH activation and corresponding mood change. Additionally, it seems plausible that modifying negative cognitions via logical strategies may likely decrease RH activation by breaking the cycle of negative cognitions to increased RH activation that, in turn, fuels more negative cognitions. Thus, it may be important, at least initially in cognitive therapy, to concentrate on rational, logical, and positive verbal cognitive therapy strategies that may differentially prime the LH and promote more positive cognitions and affect as opposed to negative cognitions, which may be promoted by techniques that focus on depressives' current (negative) thoughts and feelings.

The neuropsychology of depression may also help explain certain mode-specific changes associated with cognitive therapy. For example, Gauthier *et al.* (1983) found that cognitive restructuring and rehearsal, as compared to a waiting list, resulted in significant improvements in self-esteem and mood. As noted above, such cognitive therapy techniques may differentially prime the LH, resulting in more positive views of the self. Further, an increase in the activation level of the LH may result in more of an activation balance between the hemispheres that, in turn, may promote more positive logical cognitions vs. negative distorted cognitions and emotions that are likely fueled by increased RH activation relative to that at the LH. This type of scenario may also be responsible for the decrease in dysfunctional (negative) cognitions (Miller *et al.*, 1990; Whisman *et al.*, 1991) and hopelessness (Whisman *et al.*, 1991) seen in other mode-specific

studies that have compared cognitive therapy to other depression treatments.

Therapy Ineffectiveness

While it is widely known that cognitive therapy is not effective with all depressed clients, neuropsychological evidence may help explain why such treatment may prove unbeneficial in certain cases. Possible therapeutic remedies for such cases are also discussed.

First, cognitive therapy may be ineffective due to the heightened RH activation/LH hypoactivation that may possibly impair intra- and interhemispheric processing and comprehension of cognitions and concepts discussed in therapy, as well as impede the integration of these cognitions with the corresponding affects. For example, the logical self-talk/reasoning functions (as well as the abilities to abstract and shift cognitive sets) of the left frontal region may be impaired and prohibit accurate conceptualizations and processing of logical cognitions and new ways of thinking that are discussed in therapy. These impairments may also inhibit integration of such logical cognitions and corresponding positive affects. Thus, it may be important for cognitive therapists to encourage clients' verbalizations of rational thoughts, which may differentially prime the LH and, in turn, may promote remission of the depression and resolution of the neuropsychological deficits (McAllister, 1981). Frequent verbal reinforcement and discussions of logical/rational and abstract ways of thinking may also promote increased activation of the LH and decrease depression.

Heightened RH activation, which has been associated with depression, negative affect, and avoidance behaviors, may also result in cognitive therapy ineffectiveness. Such activation may not only fuel negative feelings and cognitions toward the potential benefits of therapy but also promote resistance and avoidance of therapy techniques. For example, depressed individuals may resist the therapist's advice, avoid complying with homework assignments, neglect practicing cognitive techniques, or even avoid maintaining appointments due to this heightened RH activation. Hence, it may be important to specifically encourage depressed clients' participation in therapy as well as to enlist clients' significant others who may likewise encourage clients to take an active role in therapy and practice therapy techniques between sessions.

Cognitive therapy efficacy may be negatively influenced by neuropsychological deficits such as decreased attention, concentration, and vigilance, which have been associated with depression (for example, Abrams and Taylor, 1987; Cassens *et al.*, 1990; Frith *et al.*, 1983). These impairments may

decrease a client's abilities to comprehend and absorb concepts discussed within therapy sessions. Thus, repetition of therapy concepts to the client may prove helpful as well as requesting that the depressed person repeat concepts discussed to the therapist to assure that the client has indeed comprehended the ideas presented.

Memory impairments that have been associated with depression (for example, Breslow *et al.*, 1980; Cassens *et al.*, 1990; Fromm and Schopflocher, 1984) may also preclude cognitive therapy's effectiveness. Depressed clients may not only have difficulties with comprehension but once therapy concepts are absorbed, depressives may be unable to fully retain or remember all aspects of an idea or technique. Such memory deficits could easily prevent clients from accurately practicing cognitive techniques between sessions or performing homework assignments. Thus, it may be beneficial to instruct depressives with memory impairments to write down therapy concepts. The therapist might also prepare written guidelines or instructions that clients could carry with them.

Finally, once or twice weekly cognitive therapy session(s), which are typically recommended, may not provide the level of intensive therapy contact that may be required to effectively modify the heightened RH activation/LH hypoactivation nor any of the related functional impairments reflected in neuropsychological test studies. For example, while the levels of activation between the left and right hemispheres might possibly become more balanced during and shortly after therapy sessions (due to therapy effectiveness), based on the potential frontal lobe and memory dysfunctions often seen in depressives, as well as the RH-related, negative avoidance behaviors that may persist, in-session gains may gradually dissipate once sessions are concluded. It seems especially important for depressives, who display a number of neuropsychological deficits, that therapy sessions occur as frequently as possible, at least during the first few weeks of therapy. Written therapy instructions/guidelines, and involvement of significant others, may also prove beneficial in maintaining therapy gains between sessions.

Response to Therapy

At present, the neuropsychology of depression cannot reliably predict those depressed individuals (or subtypes) who will be good candidates for cognitive therapy. A primary reason for this is that there has been an absence of longitudinal, prospective studies that have assessed and followed the neuropsychological functioning (and therapy effectiveness) of depressives throughout a period of cognitive therapy. However, it is hypothesized

that depressed subjects who display neuropsychological deficits and/or heightened hemispheric activation asymmetries over the frontal regions may be at increased risk for not benefiting from cognitive therapy for the reasons noted above.

Intuitively, it seems likely that the more impaired neuropsychological test performances that are demonstrated by depressives, the greater their chance of nonresponse to therapy. Thus, as more severe depressives tend to be associated with more deficits (Miller, 1975; and possibly increased hemispheric activation asymmetries over the frontal lobe), severely depressed persons may be at greater risk for therapy failure. Several cognitive therapy studies have supported this hypothesis that severely depressed clients, as opposed to other depression levels, tend to have poorer treatment responses (Beckham, 1989; Jarrett *et al.*, 1991; Thase, Simons, Cahalane, McGeary, and Harden, 1991). However, different subtypes of depressives (for example, mild/severe) tend to display similar deficits that differ only as regards degree of impairment (Miller, 1975). Thus, those depressed subjects with relatively high degrees of impairment and multiple deficits may be most prone to therapy failure.

While a number of response predictors have been found via cognitively based studies, these predictors can possibly be explained in terms of the neuropsychology of depression. For example, studies have found low vs. high cognitive dysfunction in depression is predictive of better responses to cognitive therapy (Jarrett *et al.*, 1991; Keller, 1983). Such a predictor might be indicative of depressives who have few/no neuropsychological dysfunctions, especially over the frontal regions. Low cognitive dysfunction may also be indicative of heightened left frontal activation relative to depressives with high cognitive dysfunction. Finally, as married depressives have been shown to respond better to cognitive therapy than single clients (Jarrett *et al.*, 1991), this may indirectly support the hypothesis that significant others may be beneficial in providing encouragement and reminders regarding active therapy participation to depressed persons who may exhibit avoidance behaviors and/or memory problems. As these relationships between cognitive therapy predictors and the neuropsychology of depression remain unproven at present, these conclusions should be regarded as speculative.

Other Therapies

While the specific implications of this model for therapies other than Beck's cognitive therapy are beyond the scope of this paper, to the extent that other talk therapies share mechanisms of change/techniques similar to cognitive therapy, then the neuropsychological hypotheses proposed to ex-

plain cognitive therapy's effectiveness/ineffectiveness may also be valid for these other therapies. Specifically, therapies that utilize techniques (similar to those described for cognitive therapy) that have been theorized to differentially activate the LH and offset the left–right activation asymmetry proposed to be present in depressed patients, may likewise be efficacious in decreasing depression and result in success rates similar to cognitive therapy. For example, depression therapies that employ verbal dialogue, which focuses on logical thoughts and reasoning, may promote LH priming, decreased anterior cerebrum asymmetry, and consequently, less depression. Alternatively, to the extent that other talk therapies are used with depressed patients who exhibit neuropsychological deficits (e.g., memory problems), or use techniques that have been hypothesized to differentially activate the RH (and reduce effectiveness), such as focusing on patients' negative thoughts/feelings, then these may result in therapy ineffectiveness similar to that previously described for cognitive therapy.

In sum, while other talk therapies may vary in specific methods or constructs, it is hypothesized that a critical criterion for therapy effectiveness (comparable with cognitive therapy) is that the techniques involved maximize utilization of functions associated with the LH, and especially the left anterior cerebrum, that, in turn, may differentially prime/activate the left cerebral hemisphere and result in decreased depression. In contrast, it is proposed that usage of techniques that may further activate the RH (and increase negative cognitions/affect) should be minimized.

Assessment Implications

Although, at present, neuropsychology cannot reliably predict good cognitive therapy candidates, neuropsychological assessment may allow identification of functional, neurocognitive deficits that may impede cognitive therapy's effectiveness. Such assessment may serve as a crucial adjunct to the specific measures of depression and dysfunctional attitudes/cognitions typically included in the evaluation of depressed clients by cognitive therapists. Via the inclusion of neuropsychological tests and measures, these tools will assist the therapist to individually tailor and modify cognitive therapy strategies to depressed clients based on their specific strengths and neurocognitive deficits. This idiosyncratic approach will likely promote increased effectiveness of cognitive therapy.

A diversity of neuropsychological tests may prove beneficial in assessing depressives' functional impairments, especially those of the frontal lobes and memory processes, which have been associated with depression. Examples of suggested neuropsychological tests follow. Each of these has been

included as they are relatively easily and quickly administered, interpreted, and scored, require limited materials/equipment and costs, and are generally standardized and supported by normative data. Psychologists may become proficient with these tests with minimal training and can easily administer a series of these tests within one therapy session.

A number of tests might be given that have been suggested to be sensitive to frontal lobe functioning. The Booklet Category Test (DeFilippis and Campbell, 1979) and the Wisconsin Card Sorting Test (Heaton, 1981) may be used to assess such functions as abstraction, perseveration, and ability to maintain sets. If deficits in abstraction abilities are apparent, then the therapist would likely want to concentrate on more concrete discussions of topics in therapy and avoid complex or abstract concepts or interpretations. Depressed patients who exhibit significant perseveration errors and impairments of shift of set abilities may have a difficult time changing the way they think and feel. This information may be important to the therapist in planning cognitive techniques such as thought-stopping strategies that might effectively interrupt depressed patients' persistent negative cognitions and affect. Likewise, the Stroop Color Word Test (Golden, 1978) could also be used to assess perseveration and increase reliability of the other tests of perseveration. The Stroop Test can likewise be used to assess deficits in response inhibition (Kolb and Whishaw, 1990). For example, if depressed subjects exhibit impaired response inhibition, then they may likely have difficulty inhibiting negative affect. This information may prove valuable in formulating cognitive techniques that are more logical as opposed to affective in nature, thus helping patients to inhibit negative affect via logical cognition. Verbal fluency (and perseveration) can easily be assessed via the Chicago Word Fluency Test, FAS Test, or Controlled Oral Word Association Test (see Kolb and Whishaw, 1990; Lezak, 1976). If decreased verbal fluency is observed, such findings can promote therapist awareness that the depressed patients may have difficulty verbally expressing their underlying cognitions and emotions. The Trail Making Test (Parts A and B) can be used to suggest impairments in cognitive processing speed, flexibility, and sequencing (Reitan, 1979; Reitan and Wolfson, 1993). If such deficits are found, the therapist may need to slow the process of cognitive therapy to compensate for the depressed patient's slowed processing capabilities. Similarly, impairments by depressed patients on these tasks may indicate that they are unable to successfully sequence a chain of therapeutic ideas. Hence, the therapist may need to slowly educate the depressed patient in cognition strategies by teaching them only a few simple steps at a time or by writing down technique steps to ensure that depressives perform the strategies in the proper sequence.

The Wechsler Adult Intelligence Scale—Revised Digit Span (Wechsler, 1981) subtest or a Serial Sevens test can be used as classic tests of attention (Lezak, 1976). If depressives demonstrate poor performances on these tasks, the therapist may need to shorten the duration of sessions and the amount of material covered to match the patient's attention span or else the sessions will be for naught.

Memory tests should also be considered crucial in neuropsychological screenings of depressives. Verbal learning and recall may be assessed via the Rey Auditory Verbal Learning Test (see Bigler, 1988) or the California Verbal Learning Test (Delis *et al.*, 1987). The widely used Wechsler Memory Scale (Wechsler, 1987), which has been used previously with depressives, can assess short-term contextual/meaningful verbal and figural memory (see also, Lezak, 1976). The Denman Neuropsychology Memory Scale (Denman, 1984) might also be administered to assess both verbal and nonverbal memory. As noted previously, memory impairments can easily prevent clients from retaining or remembering topics and techniques discussed in therapy and hence the practicing of these techniques between sessions. Knowledge of such impairments is crucial so that the therapist may assist his/her client's memory by providing him/her with written guidelines, instructions, or obtaining the assistance of family members to increase therapy compliance.

Additionally, to assess expression and perception of affect, a quick Affect Screen might be administered. This would involve instructing a depressed client to make both happy and sad faces and then to judge the affective facial expressions of the examiner. This information can provide insights into depressives' ability to accurately express, process, and perceive emotion. If such abilities are found to be deficit, the therapist can increase his/her focus on assisting the client to more accurately perceive and express affect that, in turn, may positively impact the patient's depression cognitions.

Ideally, as with all psychological evaluations, neuropsychological screening should consist of assessment of different functional brain areas via use of multiple measures. To increase confidence in assessment results, it is important to demonstrate reliable findings across measures that assess similar brain regions.

Future Research

While research in many diverse areas remains to be conducted to confirm the integrative hypotheses presented herewithin, there are several research avenues that appear particularly important. One line of research is

needed that directly links the neuropsychology of depression to Beck's cognitive theory and therapy of depression. Specifically, longitudinal prospective studies are needed that examine the neurological/neuropsychological functioning (via neuroimaging techniques and self-report/neuropsychological screening tests) of depressives prior to, during, and after the conclusion of cognitive therapy. We hope this research will provide evidence for the hypothesized differential effects of cognitive therapy on the activation levels of the left and right cerebral hemispheres of depressed individuals.

Studies then could be conducted that specifically examine the potential differential effects of various cognitive therapy techniques (e.g., logical analysis, hypothesis testing, self-monitoring) on the activation levels of the left and right cerebral hemispheres of depressives. Such studies would likely shed light on the neuropsychological mechanisms of change involved in various cognitive therapy techniques and aid in the determination of which techniques are most effective in modifying the left–right cerebral asymmetries that may be present.

To provide neuropsychological support for components of Beck's cognitive theory of depression, neuroimaging technology, such as EEG/brain mapping and/or PET scans, might also be utilized that monitor/map areas of hemispheric hypo/hyperactivation in depressed subjects during periods of active production of distorted cognitions, automatic thoughts, and negative self-talk. These studies could also observe patterns of differential hemispheric activation that might occur during production of more logical and positive cognitions. The goal of this research would be the identification of hemispheric activation patterns that have been hypothesized to be associated with such positive and negative cognitions.

Large-scale studies are needed that examine the specific neuropsychological deficits that may coincide with depression and the predictive value of such deficits on the outcome of cognitive therapy. Likewise, the efficacy of various cognitive techniques could be examined in individuals with different neuropsychological impairments in order to determine the therapeutic strategies that are most effective for depressives with particular types of neuropsychological deficits.

Finally, the left parietal region, which has been associated with relational thoughts (i.e., greater/less than, should/shouldn't; Luria, 1973) has received a relative lack of attention during periods in which depressed subjects are actively engaged in thinking distorted/negative relational thoughts. Thus, it seems important to investigate the role of this region (during such thoughts) via neuroimaging techniques to determine what impact, if any, this region may have on the hemisphere asymmetries typically associated with depression.

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