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The role of learned helplessness in the onset of depression in obsessive compulsive personality disorder

Faucette, Robert Chester, Ph.D.

The University of North Carolina at Greensboro, 1994

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THE ROLE OF LEARNED HELPLESSNESS IN THE ONSET OF DEPRESSION IN OBSESSIVE COMPULSIVE PERSONALITY DISORDER

by

Robert C. Faucette

A Dissertation Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
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The characterological predisposition hypothesis posits that certain personality features may predispose certain individuals to depression. The general hypothesis of the present study was that obsessive compulsive personality disordered analogues (given the excessive importance of autonomy, achievement, and especially, personal control in their lives) would be more vulnernable, that is, characterologically predisposed, to the debilitating effects of perceived uncontrollability (i.e., learned helplessness) than a normal control group and an Anxious-Fearful Cluster personality disorder control group comprised of avoidant, dependent, and passive-aggressive personality disordered analogues.

A total of 136 subjects were selected for inclusion in the study based on their SCID-II personality profiles and were administered a two-phase learned helplessness procedure patterned after Hiroto's (1974) and Hiroto and Seligman's (1975) studies. Furthermore, subjects were administered the Attributional Style Questionnaire (ASQ) and the Tennessee Self-Concept Scale (TSCS) to assess attributional predispositions to depression and level of self-esteem in the three diagnostic groups.

Findings supported the basic learned helplessness effect that exposure to perceived uncontrollability results in performance/motivational deficits,

cognitive deficits, and affective disruptions. As predicted, the TSCS and the ASQ were found to be positively correlated with one another, and Anxious-Fearful analogues were found to have the lowest self-esteem of the diagnostic groups. Most importantly, findings revealed a vulnerability to learned helplessness in Obsessive Compulsive analogues and Anxious-Fearful personality counterparts based on ASQ scores; however, contrary to predictions, this diagnostic vulnerability was not demonstrated in the two-phase experimental procedure. Finally, modest support for Seligman's mood progression hypothesis was provided (i.e., perceived uncontrollability leads to anxiety which, in turn, leads to depression).

Implications and contributions of the study are highlighted, suggestions for future research are offered, and learned helplessness is critiqued and re-examined in light of the obtained findings.

APPROVAL PAGE

This dissertation has been approved by the following committee of the Faculty of the Graduate School at The University of North Carolina at Greensboro.

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CHAPTER I

INTRODUCTION

Depression

The label "depression" commonly is used when referring to a dysphoric affective state. Depression can be represented on a continuum and can manifest itself as a harmless and even adaptive symptom characterized as sadness or as a serious clinical syndrome characterized by a class of co-varying maladaptive symptoms. The consequences of depression to the individual experiencing this affective state also can range from a decline in optimal performance to complete debilitation and even suicide.

With regard to behavior in general, the line demarcating normal and abnormal behavior is not made by an exact science. Yet the importance of making such a distinction has generated many attempts to draw such a line. The Diagnostic and Statistical Manual for Mental Disorders: 3rd Ed. - Revised (DSM-III-R: American Psychiatric Association, 1987), the most commonly used taxonomy of psychopathology, defines abnormal behavior (mental disorders) as a "... a clinically significant behavioral or psychological syndrome or pattern that occurs in a person and that is associated with present distress (a painful symptom) or disability (impairment in one or more

important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or loss of freedom (p. xxii)." Furthermore, the disorder is not an expectable response to a situation (i.e., uncomplicated bereavement). Nor is it simply deviant behavior or behavior reflecting a conflict between individual and society.

With regard to depression, DSM-III-R assumes that normal individuals become depressed, but emphasizes that the degree, scope, and relative circumstances surrounding this "depression" determine whether or not it is categorized as a mental disorder. According to the DSM-III-R, which classifies behavior based on topographical differences, abnormal or clinical depression can take on several forms. Under the diagnostic category of Affective Disorders, major depression, dysthymia, bipolar disorder, and cyclothymia are found, each variations of abnormal affective states. The focus of the present dissertation is on major depression, the most common of the affective disorders, which afflicts 9 to 26% of adult females and 5 to 12% of adult males (APA, 1987) at some point in their lives and is reported to cost between $1.3 and $4 billion per year in terms of treatment and loss of productivity in the United States alone (Williams, Friedman, & Secunda, 1970). The DSM-III-R defines major depression as an Affective Disorder characterized by a response topography comprised by at least five of nine symptoms. One of these symptoms must be dysphoric mood and/or loss of interest (most everyday, for a majority of each day). The other symptoms comprising the diagnostic

criteria are a) weight loss/gain or appetite loss/gain, b) insomnia/hypersomnia, c) psychomotor agitation/retardation, d) loss of energy, e) feelings of worthlessness, guilt, lowered self-esteem, f) difficulty with thinking processes (e.g., concentration, memory), and g) thoughts of death and/or suicidal ideation, plans, or attempts. In order to qualify for the diagnosis, it must be determined that the symptoms have persisted for two weeks, that these symptoms represent a change for the worse in functioning, and that the response topography does not have an organic etiology.

Theories of Depression: A Brief Overview

DSM-III-R is an atheoretical classification nosology which affords it certain advantages (e.g., acceptance across theoretical orientations; focus on objective behavior). Being atheoretical and structural, however, it does not address the functional nature of psychopathology. Determining what "causes" or "maintains" certain disorders, including major depression, has been the special task of theory.

Historically, depression has been subtyped in various ways, sometimes consistently with the distinctions made in DSM-III-R and sometimes inconsistently. One distinction that has been proposed is the endogenous, or organically-based depression as opposed to the exogenous, or environmentally-based depression. Unlike the topographical distinctions made in DSM-III-R, such a distinction is based on hypothesized etiological origins. The conclusion drawn, however, from the unresolved nature-

nurture debate (Johnston, 1987) is that a distinct independence of organic and environmental influences on depression is unlikely. Thus, major depression, often referred to as a reactive-type depression is probably the product of, at least some combination of nature and nurture influences.

Today, there are many theories of depression which attempt to explain the response topography that DSM-III-R describes. For example, genetic factor theories utilize family pedigree analyses, twin studies, adoption studies, and linkage/recombinant DNA studies in pointing to a genetic contribution to depression (Carson & Carson, 1984). Biochemical explanations such as the biogenic amine hypothesis suggest that depression is associated with amine deficiencies in the brain (Carson & Carson, 1984). Traditional psychodynamic theory argues that depression is a reaction to the unconscious or imagined loss of an object (Freud, 1917) and the end product of anger turned inward. Ferster's (1973) functional analysis states that the depressed individual has a defective behavioral repertoire which decreases the likelihood of positive reinforcement and increases the likelihood of aversive consequences, resulting in behavioral decrements. Lewinsohn's (1986) behavioral theory of depression purports that having few interactions with positive outcomes causes one to feel depressed; the more depressed one feels, the less motivated he or she is to engage in activities which might have positive outcomes. This, of course, leads one to feel even more depressed, which in turn, causes one to become even less active, and so on. Beck's (1976, 1981) cognitive theory of

depression suggests that a pessimistic view of self, world, and future bias an individual's perceptions. According to this theory, these irrational cognitions, beliefs, or schemas directly cause the depressive emotional state. And Coyne's (1976) interpersonal theory of depression describes a "vicious circle" where depressed persons, who desire and seek social reinforcement from others, actually push others away as a result of their annoying "depressive" behavior. They then use this "rejection" as evidence to support their hypothesis that they are unloved or worthless. Upon making such an attribution, they then become more depressed and engage in more "depressive" behavior, which again pushes others even further away.

Each of the above theories, and many not mentioned here, provide a fresh perspective on depression. And, given their differing levels of analysis, such theories are not necessarily mutually exclusive. Just as there are many theories of depression, there are many treatments for depression, many of which logically follow from a given etiological theory (e.g., cognitive theory of depression-> cognitive therapy for depression).

The Learned Helplessness Model of Depression

A cognitive-behavioral etiological theory, Seligman's (1975) learned helplessness theory of depression, is the model being used to investigate major depression in the present study. The learned helplessness model is based on the premise that, when situational outcomes are not perceived as being contingent on an organism's actions, "helplessness" may result. In

other words, when the perceived probability of a particular outcome is the same regardless of whether or not a response is generated by an organism, this outcome is perceived as being independent of responding and, thus, is seen as "uncontrollable." It is this "uncontrollability" that produces anxiety, which, in turn, leads to what is being defined as "helplessness." This relationship between anxiety and helplessness (e.g., depression) is described by Miller, Rosellini, and Seligman (1986) in the following manner:

> [W]hen a man or animal is confronted with a threat or a loss, he initially responds with fear or anxiety. If he learns that the threat is wholly controllable, anxiety, having served its function, disappears. If he remains uncertain about his ability to control the threat, his anxiety remains. If he learns or is convinced that the threat is utterly uncontrollable, depression emerges (p. 195).

According to the original theoretical conceptualization of learned helplessness, when an organism is made helpless, certain performance/motivational deficits (e.g., slowed response initiation) and cognitive deficits (e.g., insensitivity to contingencies) accompany these affective disruptions. These deficits/disruptions are viewed by Seligman (1975) as symptoms of a reactive depression.

Note, according to Seligman, mere exposure to "uncontrollability" is not enough to produce "helplessness." An organism must come to perceive this noncontingency. Seligman asserts that both humans and non-humans acquire information about contingencies, and act based on these

representations. In this sense, the learned helplessness theory of depression is a mediational theory.

Animal Model

Early studies of this model focused on the debilitating effects that response/outcome independence had on voluntary response initiation, ability to learn, and emotionality in animals. In now classic studies, Seligman and Maier (1967) and Overmier and Seligman (1967) exposed dogs first to a series of inescapable shocks, and then to an escape/avoidance task where dogs could jump from one side of a shuttlebox, over the barrier, to the other side in order to escape or avoid a shock stimulus. Note that in the first phase of this experiment, shock was "uncontrollable," whereas in the second phase, the shock could be avoided if the proper response was emitted. In the second phase, when signalled that the aversive stimulus was forthcoming, a vast majority of the dogs, instead of leaping over the barrier to "safety," merely laid down, whimpered, and passively accepted the shock. These "helpless" dogs, not only demonstrated a lack of motivation, but also showed, what Seligman labeled, a cognitive deficit with regard to their ability to learn. Contrary to Phase 1, outcomes in Phase 2 were dependent on responding, yet the dogs failed to learn the new contingency that they could escape or avoid the shock. In addition to these motivational/performance and cognitive deficits, most dogs experienced heightened emotionality or affective disruptions. These disruptions were inferred by the early researchers from the

dogs' overt fear responses (e.g., whimpering). Other animal studies have assessed more rigorously this heightened emotionality by measuring blood pressure (Hokanson, DeGood, Forrest, & Brittain, 1971; cited in Seligman, 1975) and ulcer formation (Weiss, 1968; also cited in Seligman, 1975). This learned helplessness effect on emotion is often referred to as an affective "deficit" in the learned helplessness literature. However, in the following pages, it will be referred to as a "disruption," given that the term "deficit," which implies "absence of affect," may be misleading.

According to Seligman (1975), learned helplessness experiments have produced deficits/disruptions in many species other than dogs: fish (e.g., Padilla, Padilla, Ketterer, & Giacalone, 1970), rats (e.g., Maier, Seligman, & Solomon, 1969; Maier and Testa, 1975; Seligman & Beagley, 1975), cats (e.g., Thomas & Balter, 1974), and humans (e.g., Glass & Singer, 1972; Miller & Seligman, 1975), to name a few.

The Triadic Design

The original learned helplessness effect has been demonstrated using a variety of experimental procedures. Most have used variants of the triadic design. This rather elegant procedure was originally used in Seligman's dog studies to demonstrate that the helplessness assessed in these animals was, in fact, psychological in nature (i.e., the result of "perceived uncontrollability") and not merely a physical side effect of the shock administered to the dogs. This procedure, still popular in learned helplessness research, utilizes three

groups. Typically, Group A subjects are first exposed to a pretreatment situation where they can control the experimental outcome by responding in some specified manner. Group B subjects are also exposed to the pretreatment situation; however, they are "yoked" to Group A subjects, meaning that they receive the identical outcome as Group A subjects but have no means of controlling the outcome. Group C subjects do not receive the pretreatment. Finally, in a test phase, all three groups are tested on a particular task (different than the pretreatment task) and specific performance/motivational deficits, cognitive deficits, and affective disruptions are assessed. Typically, subjects in Group A--the "controllable" or response dependent group--and Group C--the "no pretreatment" group--show fewer deficits/disruptions than subjects in Group B--the "uncontrollable" or response independent group. In sum, Group B subjects are made, in essence, experimentally helpless as a result of the pretreatment, and the effects of this helplessness induction generalize to test phase performance where they are assessed. Past and current researchers have utilized this design, and helplessness induction has been reliably demonstrated. In fact, a recent meta-analysis by Villanova and Peterson (1991; cited in Peterson, Maier, & Seligman, 1993) which investigated 132 human helplessness studies concluded that "the learned helplessness effect in people appears to be as robust as most findings in the social sciences (p. 107)." Many of these studies have utilized some variant of the shuttlebox paradigm mentioned above; however, others have remained true to the

triadic design while incorporating different pretreatment and test phase tasks and/or aversive stimuli (See Appendix A for schematic representation of the triadic design).

Depressive Equivalent

As was mentioned above, proponents of the theory assert that learned helplessness is applicable to, and procedures effective on, a variety of species (e.g., fish, rats, cats, dogs, humans). Of course, the theory's applicability to humans makes the theory particularly interesting to individuals wanting better to understand human depression.

In an attempt to present evidence for a learned helplessness/human depression relationship, Seligman (1975) pointed to six symptoms of learned helplessness that have similar counterparts in depression: 1) lowered initiation of voluntary responses (i.e., response rate, latency), 2) negative cognitive set or difficulty in learning contingencies, 3) time course--symptoms subside if the aversive stimulus is not presented for multiple sessions, 4) lowered aggression, 5) loss of appetite, and 6) physiological changes (e.g., norepinephrine depletion).

He also drew a convincing etiological similarity between learned helplessness and depression, claiming that learned helplessness is caused by a perception or belief that any attempt to control the environment is futile, while similarly, certain reactive depressions are believed to be caused when an individual perceives work, family life, and friendships as uncontrollable.

Thus, according to Seligman (1975), both the dog in the shuttlebox and the depressed human may come to learn that they are "helpless," and manifest this learning through performance/motivational deficits, cognitive deficits, and affective disruptions.

Further support for viewing human depression from this learned helplessness perspective comes from therapeutic strategies that seek to cure and prevent resulting or future depressive symptoms. Studies have shown that the "helpless" dog who is dragged to the safe compartment of the shuttlebox is forced to learn that it has control over the shock in the test phase. Eventually, the effects of helplessness weaken and the dog is "cured." Likewise, the young man who is depressed and believes that he is unable to control his world successfully, may come to realize, through therapeutic intervention (e.g., cognitive therapy's hypothesis testing), that his earlier beliefs were irrational, and that outcomes in his life are, in fact, contingent upon his responses. Thus, similar strategies with the aim of getting the organism to experience response-dependent contingencies may be employed to alleviate symptoms of both "helplessness" as defined by Seligman and depression as defined by others (e.g., Beck).

Also, it has been suggested, both in the depression and learned helplessness literature, that early exposure to response independence predisposes an individual to later depressive symptoms, whereas early exposure to response dependence or "mastery" serves to immunize against

future depression.

According to Seligman (1975), taken together, this evidence justifies and lends support for a learned helplessness model of depression, at least depression of a reactive nature.

Human Studies

As the Villanova and Peterson (1991) meta-analysis attests, learned helplessness in humans has been reliably demonstrated in the laboratory. Using the triadic design in one of the first studies to focus on learned helplessness in humans, Hiroto (1974) exposed college students in the "controllable" group (Group A) to a loud aversive noise that could be terminated by simply pressing a button. The "yoked" group (Group B) received the same noise; however, as in the dog study, they had no way of controlling it. A third group (Group C) received no noise and were given no task. In the test phase, all subjects were instructed that they could escape the noise somehow. The actual response requirement was to move their hands from one compartment of a hand shuttlebox to another. This procedure was viewed as analogous to the original dog shuttlebox procedure. As predicted, students in the "uncontrollable" or "yoked" group performed the poorest on the task, at times passively accepting the noise, as the dogs had passively accepted the shock. Specifically, Group B subjects demonstrated a deficit in response initiation (i.e., their mean reaction times were slower), a deficit in ability to learn (i.e., it took them more trials to reach a learning criterion), and

a deficit on a measure that assessed both the performance/motivational and cognitive deficit (i.e., they failed a greater number of trials on the shuttlebox task). In addition to demonstrating the basic helplessness effect in humans, Hiroto explored the effect of locus of control and skill versus chance instructions on helplessness induction. He found that individuals with an external locus of control (i.e., those who perceive events as being controlled by outside forces as opposed to internal forces (see Rotter, 1966)) and individuals given chance instructions (i.e., those who were led to believe that pretreatment success or failure was determined by chance) were more vulnerable to helplessness induction.

In a different learned helplessness demonstration using humans, Miller and Seligman (1975) investigated the effect of helplessness-inducing procedures on both nondepressed and depressed subjects. The study utilized the same pretreatment task used in Hiroto (1974) but employed a different test phase task--a series of 20 solvable anagrams borrowed from Tresselt and Mayzner (1966). In addition to assessing deficits on anagram performance, the authors measured affect change using a self-report instrument, the Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin, 1965). The findings of the study supported the basic learned helplessness effect in nondepressed subjects (i.e., Group B subjects demonstrated deficits in their ability to solve the anagrams, were slower to initiate responses, became more depressed based on their self-report). Another finding was that Group C subjects within the

depressed group demonstrated greater deficits/disruptions than Group C subjects in the nondepressed group and at least as significant deficits as Group B subjects in the nondepressed group. The authors argued that this demonstrated that the effects of experimentally induced helplessness mimic the effects of naturally occurring depression. They concluded that the findings of the study provided further support of the learned helplessness model of depression. An interesting finding in their study that is referred to later in the present dissertation is that Group B subjects in the depressed group did not demonstrate greater deficits relative to Group A and C counterparts on the anagram measures as the triadic design predicts. In fact, depressed Group B subjects performed better on the anagram task than Group C counterparts and self-reported a slight mood elevation (i.e., became less depressed) on the depression scale of the MAACL. As in Hiroto (1974) where external locus of control (i.e., subject variable) and chance instructions (i.e., a procedural variation) appeared to influence helplessness induction, another subject variable was identified in Miller and Seligman (1975) which appeared to influence the effects of perceived uncontrollability; namely, the level of pre-experimental depression. This finding also will be referred to later in the text.

Hiroto and Seligman (1975) also tested the basic learned helplessness effect on nondepressed humans with a special focus on demonstrating the generality of the effect utilizing different experimental tasks. The pretreatment and test phase tasks in Hiroto's (1974) study were both seen as

"instrumental" procedures whereas the pretreatment task in Miller and Seligman (1975) was "instrumental" and the anagram test phase task was identified as "cognitive." In Hiroto and Seligman's study (1975), both "instrumental" and "cognitive" pretreatments and test phases were employed. The "cognitive" pretreatment was a discrimination learning task borrowed from Levine (1966, 1971) that was solvable for Group A subjects, unsolvable for Group B subjects, and not administered to Group C subjects. The "cognitive" test phase task was the same administered by Miller and Seligman (1975). The "instrumental" pretreatment and test phase tasks were the same as those employed by Hiroto (1974). Crossing type of task with experimental phase yielded four combinations, each of which were tested in a separate experiment. Hiroto's (1974) learned helplessness effect was replicated in the instrumental-pretreatment/instrumental-test phase experiment and Miller and Seligman's (1975) effect was replicated in the instrumental-pretreatment/cognitive-test phase. Furthermore, the effect was demonstrated in the other two variants: cognitive-pretreatment/cognitive-test phase experiment; and cognitive-pretreatment/instrumental test phase experiment. The authors argued, based on the findings, for a generality of learned helplessness in humans. In other words, the effect was not specific to particular pretreatment and test phase tasks. In Hiroto and Seligman (1975), an important modification was made in the instrumental-pretreatment/instrumental-test phase experiment that was

neglected in Hiroto (1974) and not utilized in Miller and Seligman (1975). Recall, in both studies utilizing the instrumental pretreatment task, Group C subjects were not administered a pretreatment task, nor were they administered aversive tones. Hiroto and Seligman (1975), however, determined that it was important that Group B and Group C differ only in perceived uncontrollability; therefore, their Group C subjects received the same aversive tones as their Group A and Group B counterparts. Recall, the triadic design prediction is that Group B will demonstrate deficits relative to both Group A and Group C. Interestingly, with this Group C modification in the instrumental-pretreatment/ instrumental-test phase experiment, Group B did not perform significantly more "helpless" than Group C on two of the three helplessness measures assessed. Furthermore, while not statistically significant, an inspection of the means revealed that Group C subjects took longer to learn the contingency, failed more trials, and demonstrated greater delay in response initiation than their Group A counterparts. This pattern of findings was not found in Hiroto (1974) or Miller and Seligman (1975). This Group C pretreatment modification and subsequent finding is being noted here because it is important in the pages that follow.

The Reformulated Model of Learned Helplessness

While there was empirical support for the original learned helplessness theory, certain problems began to emerge in the research, among them (a) learned helplessness could not always be induced, (b) helplessness

generalization from the pretreatment phase to the test phase was not always achieved, (c) extraneous "information" often influenced the instillation of helplessness, (d) different personality characteristics (e.g., locus of control) seemed to influence the likelihood of helplessness, (e) suicide could not be explained from the theory given that this behavior is not passive yet is a symptom of depression, and (f) the theory seemed to, by definition, exclude another "central" symptom of depression--self-blame. Hence, it was soon determined that learned helplessness was more complicated than first believed.

In an attempt to address this complexity, Abramson, Seligman, and Teasdale (1978) revised the original learned helplessness model of depression by adding the self-esteem deficit to the three original deficits/disruptions previously mentioned and by adding a second independent variable along side perceived response/outcome independence in the causal "equation." The inclusion of this second independent variable, "attributional style" or "explanatory style" addressed the original theory's neglect of the influence of attributions on learned helplessness. When an individual is exposed to a positive or negative event, he or she obviously attributes the occurrence of the event to certain causes. It follows, then, that how an individual explains positive and negative events will to a large degree determine whether or not helplessness will develop. Therefore, simply perceiving noncontingency may not be enough to produce helplessness. The perception of response

independence, however, accompanied by certain causal explanations for positive and negative events seems to be the prerequisite condition for the onset of helplessness. In other words, helplessness, as defined in this revised model, develops as the result of a diathesis-stress relationship. Possessing "at-risk" styles in isolation (without a "triggering" uncontrollable environment) is unlikely to produce helplessness. Similarly, an uncontrollable environment without the attributional predisposition is unlikely to produce helplessness. According to the theory, the two conditions must be present if helplessness is to develop (Seligman & Peterson, 1986). Peterson and Seligman (1984) suggest that depressive attributional styles are shaped by learning experiences. Specifically, imitation of one's primary caretaker, the effects of teachers' reactions to one's failures and successes, and one's initial attributions made to early traumatic losses, all influence the acquistion of attributional style.

This theoretical modification provides a more sophisticated explanation of the mechanism responsible for learned helplessness and appears to help account for the many inconsistencies that the old model failed to address. Abramson et al. (1978) have outlined three relevant explanatory dimensions (internal-external, stable-unstable, and global-specific). The cause of an event, as determined by the individual, may stem from within the self (internal) or from the outside world (external), may persist over time (stable) or be transient (unstable), and may affect a variety of circumstances (global) or

only a particular circumstance (specific). The reformulated theory states that individuals who consistently attribute internal, stable, and global reasons for negative events and external, unstable, and specific reasons for positive events may be defined as "at-risk" for developing a learned helplessness depression. Thus, the revised model offers an advantage of predicting helplessness by first identifying maladaptive attributional patterns or styles.

For example, the job applicant who attributes his failure to get a job to internal--"I was horrible in the interview," stable--"I am never good at interviews," and global reasons--"I am incompetent at everything" (Hollon & Garber, 1980) may therefore be defined as "at-risk" for depression. Similarly, the college student who attributes her making an "A" to external--"I was lucky, the teacher gave it to me," unstable--"I was lucky on this test, next time may be different," and specific reasons--"I am competent in this class, but in nothing else" may be defined as "at-risk" for depression. An obvious advantage of this theoretical revision is that potential depressives can be identified based on their self-reported attributional styles, retrained to utilize alternative attributions through therapeutic intervention, and ultimately protected from an unhealthy depression.

Each of these dimensions helps define whether or not helplessness is likely to ensue given a particular negative or positive event. In addition, they determine the boundaries or scope of applicability. The internal-external dimension determines the degree of self-esteem loss, the stable-unstable

dimension affects the chronicity of the helplessness, and the global-specific dimension addresses the pervasiveness of perceived uncontrollability (Abramson, Garber, & Seligman, 1980).

The Attributional Style Questionnaire (ASQ) is a self-report instrument that was devised specifically to assess these three explanatory dimensions and identify "at-risk" attributional patterns. In the administration of this measure, adults are presented with 12 hypothetical events, 6 good and 6 bad, and are asked to provide causes for the events and rate their responses on 7-point scales that measure degree of internality, stability, and globality (Peterson, Semmel, von Baeyer, Abramson, Metalsky, & Seligman, 1982). The ASQ has generated convincing evidence that the internal, stable, and global attributional style for negative events is correlated with depressive symptoms. For example, according to Peterson and Seligman (1984), in the first investigation of the revised theory, Seligman, Abramson, Semmel, and von Baeyer (1979) tested 143 college students using the ASQ and a short form of the Beck Depression Inventory (BDI; Beck, Ward, Mendleson, Mock, & Erbaugh, 1961) and found that, as predicted, depressive symptoms correlated with the ASQ composite score ($r = .48$, $p < .001$). More specifically, when bad events were being explained, the BDI correlated with internal attributions ($r = .41$, $p < .001$), stable attributions ($r = .34$, $p < .001$), and global attributions ($r = .35$, $p < .001$). Other studies lend further support to the correlation (e.g., Eaves & Rush, 1984; Navarra, 1981; both cited in Peterson et

al., 1984).

While the ASQ has been successful in actually predicting depression based on internal, stable, and global attributional patterns for negative events, it has been less successful in predicting depression based on external, unstable, and specific attributional patterns for positive events (Peterson et al., 1984). Nevertheless, the addition of attributional style is viewed as an improvement upon the original theory as is evidenced by converging support for the reformulated theory using five research strategies: a) cross-sectional correlational studies, b) longitudinal studies, c) experiments of nature, d) laboratory experiments, and e) case studies, all of which utilize the ASQ and/or similar measures (Peterson et al., 1984).

Critique

While the reformulated theory is considered an improvement and addresses some of the problems inherent in the original model, it still has its critics. Even with the improvements made, some argue that the procedures used to induce and assess helplessness are too artificial. Others are concerned with the mixed findings that are difficult to account for. As was noted above, certain subject variables and procedural modifications seem to complicate matters. Still others are concerned that the theory is overly simplistic, that it is narrowly focused, and that does not fully capture the complexity of depression.

Clearly, all depressions do not emerge as a product of a learned helplessness mechanism, and learned helplessness theory does not purport to be the universal explanation for depression. Its proponents claim that it is applicable to a subgroup of reactive depressions only and that a learned helplessness mechanism is a sufficient condition for the diagnosis of depression, but certainly not a necessary one. Miller, Rosellini, and Seligman (1986) predict that this depression subgroup may one day become formalized diagnostically with the creation of a diagnostic category labeled "helplessness depression." Such a subgroup classification potentially could be beneficial, in that individuals manifesting this type of depression could better be matched with therapeutic interventions designed to overcome the debilitating effects of perceived response/outcome independence and maladaptive attributional styles.

It seems that helplessness theorists and researchers make no apologies for their theory's circumscribed focus. With regard to other criticisms, they continually assert that their theory is firmly grounded in and supported by substantial empirical research (See Villanova & Peterson, 1991). They argue that the research derived from the theory is reliable and valid and that helplessness theory does at least as good a job accounting for depression as other theories of depression.

In sum, the learned helplessness theory of depression has progressed from an initial focus on performance/motivational deficits, cognitive deficits,

and affective disruptions in animals to a reformulated "explanatory" model of depression that seems to apply across the human lifespan. As Peterson and Seligman (1984) imply, future research topics in the area of learned helplessness should focus on attempts to identify and "immunize" particular "at-risk" subgroups before the actual onset of depression. It is primarily with this "subgroup identification" that the present study is concerned. Given the fact that helplessness induction is influenced by certain subject variables (e.g., locus of control, attributional style, pre-experimental affective state), investigating the influence of "personality" on learned helplessness appears to be a logical area to explore in the pursuit of identifying vulnerable subgroups.

Personality Disorders

Nature and Classification of Personality and Personality Disorders

Like the construct of depression, "personality" can be defined in numerous ways. Traditionalists have viewed "personality" as a construct representing relatively enduring traits, while behaviorists prefer to view it as a summary term useful in describing behavior patterns. In either case, "personality" is often subtyped into topographically, and sometimes, functionally different variants. And just as "depression" can be conceived of as both normal and abnormal, personality, too, exists on a continuum, ranging from an adaptive behavior pattern/trait to a dysfunctional behavior disorder. Thus, a given personality feature is said to be Janus-faced in nature.

In moderation, it may benefit. In excess, it may harm. Furthermore, a given personality feature (e.g., histrionicity) may be deemed maladaptive in one environment (e.g., conservative business conference) yet quite adaptive in another environment (e.g., a Hollywood party or casting call).

According to the DSM-III-R (1987), personality disorders are defined as "enduring patterns of perceiving, relating to, and thinking about the world and oneself...[that are] inflexibile and maladaptive and cause either significant functional impairment or subjective distress (p. 335)." Again, DSM-III-R is atheoretical and thus categorizes personality disorders based on relatively objective topographical features. In DSM-III-R, the various personality disorders are grouped into one of three clusters. Cluster A, referred to as the Odd-Eccentric Cluster, is comprised of paranoid, schizoid, and schizotypal personality disorders; Cluster B, referred to as the Erratic-Dramatic Cluster, is comprised of antisocial, borderline, histrionic, and narcissistic personality disorders; and Cluster C, referred to as the Anxious-Fearful Cluster, is comprised of avoidant, dependent, passive aggressive, and obsessive compulsive personality disorders. The labels given to these clusters informally describe the personality features shared by members of the cluster (i.e., Cluster C disorders share the central feature of being "anxious" and "fearful"). Historically, personality disorders have been categorized in various ways. The current DSM-III-R clustering actually is quite consistent with the psychotic (i.e., similar to Cluster A), characterological (i.e., similar to Cluster

B), and neurotic clusters (i.e., similar to Cluster C) referred to in the literature (e.g., Kernberg, 1984) and with the psychoticism (i.e., similar to Cluster A), the extroversion (i.e., similar to Cluster B), and introversion clusters (i.e., similar to Cluster C) also mentioned in the literature (e.g., Vaillant, 1987). However, DSM's decision to group the disorders as it has was not purely intuitive or solely based in tradition. Factor analyses provide empirical support for such groupings (e.g., Hyler & Lyons, 1988).

There are numerous etiological theories that attempt, functionally, to account for the personality disorder topographies outlined in DSM-III-R. Among them: biological theories (e.g., Klar, Sievar, & Coccaro, 1988), psychodynamic theories (e.g., Stricker & Gold, 1988; Cashdan, 1988; Gunderson, 1984), behavioral theories (e.g., Turner & Turkat, 1988; Swenson, 1989), cognitive theories (e.g., Beck, Freeman, & Associates, 1990; Murray, 1988), social or interactional theories (e.g., Endler & Edwards, 1988; McClemore & Brokaw, 1987), and biosocial learning theory (i.e., Millon, 1981, Millon & Everly, 1985). Like the theories of depression, they approach the subject matter from different levels of analysis and likely each contribute to our understanding of personality disorders.

Relationship between Depression and Personality Disorders

The relatively high comorbidity of personality disorders and depression is well-documented in the literature (e.g., Shea, Glass, Pilkonis, Watkins, & Docherty, 1987). However, while there is considerable agreement

with regard to this high comorbidity, less consensus has been found with regard to the nature of this comorbid relationship.

Farmer and Nelson-Gray (1990) address some of the relationships proposed in the literature and these will be summarized briefly below. However, none of these proposed hypotheses attempting to explain this relationship is all inclusive. Each potentially can serve as a useful model to represent the relationship and characterize certain cases of comorbidity. Furthermore, the proposed hypotheses are not necessarily mutually exclusive. In other words, more than one may be used to describe the relationship of a particular case at different points in time. Interestingly, while the following hypotheses regarding the depression/personality disorder relationship have been proposed, most of the research addressing these proposals has been correlational in nature. Few studies actually have attempted to isolate experimentally the particular mechanisms responsible for these relationships.

One popular hypothesis, the complication hypothesis (Akiskal et al., 1983) suggests that personality characteristics are shaped by depressive experiences. The attenuation hypothesis (Akiskal et al., 1983; Gunderson & Elliott, 1985) suggests that similar genetic factors are responsible for both personality characteristics and depression, but that personality features are less extreme manifestations of these constitutional factors. The modification hypothesis (Akiskal et al., 1983) suggests that the presence of a personality

disorder may influence the clinical picture of depression, but this hypothesis does not necessarily address directional causality. The orthogonal hypothesis (Akiskal et al., 1983; Gunderson & Elliott, 1985) suggests that personality disorders and depression are independent from one another but commonly coexist. The coeffect hypothesis (Docherty, Fiester, & Shea, 1986) suggests that personality disorders and depression are independent with regard to their psychobiological origins, but co-occur as a result of some third variable. The heterogeniety hypothesis (Gunderson & Elliott, 1985) suggests that features of both a personality disorder and a depression within a given individual emerge from different sources, including different genetic endowments. And finally, the model being used in the present study to broadly describe the depression/personality disorder relationship--the characterological predisposition hypothesis (Akiskal, Hirschfield, & Yerevanian, 1983; Gunderson & Elliott, 1985)--suggests that depression is secondary to and emerges from primary personality characteristics.

Recall, the learned helplessness literature points to certain pre-experimental subject variables (i.e., primary personality characteristics) that have been implicated as having an influence on helplessness induction (i.e., secondary depressive characteristics). It follows, then, that specific personality disorders, due to certain personality characteristics that define these disorders, might be particularly susceptible to the debilitating effects of a learned helplessness depression. Since it is beyond the scope of the present study to examine all of

the personality disorders described in DSM-III-R, this study examines the anxious-fearful cluster with special attention being given to obsessive compulsive personality disorder.

Obsessive Compulsive Personality Disorder

For reasons to be highlighted below, the focus of the present study is on obsessive compulsive personality disorder and its susceptibility to a learned helplessness depression. DSM-III-R characterizes obsessive compulsive personality disorder, a specific personality disorder within the Anxious-Fearful Cluster or Cluster C, as "a pervasive pattern of perfectionism and inflexibility, beginning by early adulthood and present in a variety of contexts (p. 354)." To qualify for a diagnosis of obsessive compulsive personality disorder, at least five of the following nine criteria must be met:

> (1) perfectionism that interferes with task completion, e.g., inability to complete a project because own overly strict standards are not met
> (2) preoccupation with details, rules, lists, order, organization, or schedules to the extent that the major point of the activity is lost
> (3) unreasonable insistence that others submit to exactly his or her way or doing things, or unreasonable reluctance to allow others to do things because of the conviction that they will not do them correctly
> (4) excessive devotion to work and productivity to the exclusion of leisure activities and friendships (not accounted for by obvious economic necessity)
> (5) indecisiveness: decision making is either avoided, postponed, or protracted, e.g., the person cannot get assignments done on time because of ruminating about priorities (do not include if indecisiveness is due to excessive need for advice or reassurance from others)
> (6) overconscientiousness, scrupulousness, and inflexibility about matters of morality, ethics, or values (not accounted for by cultural or religious identification)
> (7) restricted expression of affection
> (8) lack of generosity in giving time, money, or gifts when no personal gain is likely to result

> (9) inability to discard worn-out or worthless objects even when they have no sentimental value (p. 356; APA, 1987).

DSM-III-R differentiates between obsessive compulsive personality disorder, an Axis II disorder, and obsessive compulsive disorder (i.e., historically referred to as obsessive compulsive neurosis), an Axis I disorder. Obsessive compulsive disordered individuals are described as having as their essential feature, recurrent and uncontrollable obsessions and/or repetitive compulsions which are sufficiently severe to cause marked distress, to be time-consuming, or to significantly interfere with functioning. While the personality disorder and the clinical syndrome share certain features, obsessive compulsive disorder, an anxiety disorder, is considered a more serious clinical syndrome and is clearly ego-dystonic.

The obsessive compulsive personality disorder that the most recent version of the DSM describes has been conceptualized in different ways and has been assigned different labels (i.e., anal personality, anankastic personality, obsessive personality disorder, compulsive personality disorder) over the last century. An unfortunate consequence of this evolution in psychiatric classification is that making comparisons across conceptualizations and diagnostic labels is often imprecise. Nevertheless, there are many points of similarity between the obsessive compulsive personality version described today the versions addressed in the past.

Several psychoanalytic conceptualizations have been offered. Freud (1908) was the first to refer to the "anal retentive" or "anal character." He used

these labels to describe a grouping a symptoms--obstinancy, parsimony, and orderliness--that were thought to be associated with the anal stage of psychosexual development and thought to be caused by strict toilet training. Erikson (1963), another psychoanalyst, noted a similar cluster of behaviors, but downplayed the influence of toilet training; instead, he pointed to the second stage of his social/emotional development theory--autonomy vs. shame and self-doubt--in explaining the etiology of these behaviors. He argued that, a child failing to develop basic trust and fearing criticism from significant others, would as a defense, repress emotions viewed as unacceptable to those significant others and instead focus on irrelevant details in his surroundings.

Horney (1937) looked to unfair, self-righteous, and authoritarian parenting practices as being responsible for the obsessive compulsives' neurotic perfectionism which she viewed as central to their character. She argued that hostile feelings toward the parents are controlled through self-control and through maintaining an idealized self-image.

Several cognitive conceptualizations of obsessive compulsive personality disorder have also been offered. Shapiro (1965) focused on the compulsive personality's cognitive style, arguing that it is characterized by rigidity in thinking (i.e. inability to shift modes of attention), tense motor activity designed to maintain focused attention, and a distorted view of autonomy where activity is under external sources of control such as moral

principles or rules. There is, in fact, empirical support for Shapiro's conceptualization in the literature (e.g., Reed, 1969, 1977; Marago & Smith, 1981; Schneidmiller, 1987). One study, for example, Schneidmiller (1987), took a radical behavioral approach in providing support for Shapiro's description of "cognitive" rigidity in thought. Namely, she found that obsessive compulsive analogues were more likely than histrionic personality analogues and normal controls to demonstrate dysfunctional rule-following behavior. Specifically, she found that obsessive compulsives who were provided an accurate rule to help them succeed on an experimental task, took longer to abandon the rule in an extinction phase where the rule was no longer accurate. Beck, another cognitive theorist and researcher, best known for his cognitive theory of depression, has extended his argument--underlying schemas or beliefs are responsible for psychopathology--to personality disorders (Beck et al., 1990). Specifically, he outlines a characteristic profile of obsessive-compulsive personality disorder, describing these individuals as having a "responsible, accountable, fastidious, and competent" view of themselves; as having an "irresponsible, casual, incompetent, and self-indulgent" view of others; as possessing main beliefs that "[they] know what is best...details are crucial...people should do better;" and as utilizing main strategies of "applying rules, being perfectionistic, evaluating and controlling events." Beck (1983) argues that this personality is characterized by "expression of values, goals, and drives relevant to self-definition, mastery of

bodily functioning, and acquistion of power and control over the environment (p. 2)."

In his biosocial learning theory, Millon describes dysfunctional compulsives as passive ambivalent individuals, meaning that they passively seek reinforcement from within themselves and from others. Millon's theory seems to address points made in both the psychoanalytic and cognitive conceptualizations. For example, according to Millon and Kotik (1985):

> by clinging grimly to the rules of society and insisting upon regularity and uniformity in relationships and life events, these individuals help restrain and protect themselves against their own aggressive impulses and independent strivings. Although the behavioral and cognitive rigidity may effectively shield the individual from intrapsychic conflict as well as social criticism, it may also preclude growth and change, cause alienation from inner feelings, and interfere with the formation of intimate and warm relationships (pp. 732-733).

As is evident, the above conceptualizations of obsessive compulsive personality disorder describe the behavioral topography of these individuals in similar ways while pointing to different, but perhaps overlapping, mechanisms responsible for such a topography.

A Brief Comparison and Contrast of Obsessive Compulsive Personality Disorder and Its Anxious-Fearful Counterparts

As mentioned above, the disorders which occupy the Anxious-Fearful Cluster of the DSM-III-R share certain features which justify their being grouped together. In the present dissertation, differences between obsessive compulsive personality disorder and its Anxious-Fearful counterparts--avoidant, dependent, and passive aggressive personality disorder--are

addressed. While detailed descriptions of these disorders and theories explaining their origins are not covered here, the DSM-III-R diagnostic criteria are presented below to highlight topographical differences.

According to DSM-III-R, avoidant personality is characterized by "[a] pervasive pattern of social discomfort, fear of negative evaluation, and timidity, beginning by early adulthood and present in a variety of contexts, as indicated by at least four of the following:

> (1) is easily hurt by criticism or disapproval
> (2) has no close friends or confidants (or only one) other than first-degree relatives
> (3) is unwilling to get involved with people unless certain of being liked
> (4) avoids social or occupational activities that involve significant interpersonal contact (e.g., refuses a promotion that will increase social demands)
> (5) is reticent in social situations because of a fear of saying something inappropriate or foolish, or of being unable to answer a question
> (6) fears being embarrassed by blushing, crying, or showing signs of anxiety in front of other people
> (7) exaggerates the potential difficulties, physical dangers, or risks involved in doing something ordinary but outside his or her usual routine, e.g., may cancel social plans because she anticipates being exhausted by the effort of getting there (pp. 352-353; APA,1987)."

Dependent personality disorder is characterized by "[a] pervasive pattern of dependent and submissive behavior, beginning by early adulthood and present in a variety of contexts, as indicated by at least five of the following:

(1) is unable to make everyday decisions without an excessive amount of advice or reassurance from others
(2) allows others to make most of his or her important decisions, e.g., whereto live, what job to take
(3) agrees with people even when he or she believes they are wrong, because of fear of being rejected
(4) has difficulty initiating projects or doing things on his or her own
(5) volunteers to do things that are unpleasant or demeaning in order to get other people to like him or her
(6) feels uncomfortable or helpless when alone, or goes to great lengths to avoid being alone
(7) feels devastated or helpless when close relationships end
(8) is frequently preoccupied with fears of being abandoned
(9) is easily hurt by criticism or disapproval (p. 354; APA, 1987)."

Passive aggressive personality disorder is characterized by "[a] pervasive pattern of passive resistance to demands for adequate social and occupational performance, beginning in early adulthood and present in a variety of contexts, as indicated by at least five of the following:

(1) procrastinates, i.e., puts off things that need to be done so that deadlines are not met
(2) becomes sulky, irritible, or argumentative when asked to do something he or she does not want to do
(3) seems to work deliberately slow or to do a bad job on tasks that he or she does not want to do
(4) protests, without justification, that others make unreasonable demands on him or her
(5) avoids obligations by claiming to have "forgotten"
(6) believes that he or she is doing a much better job than others think he or she is doing
(7) resents useful suggestions from others concerning how he or she could be more productive
(8) obstructs the efforts of others by failing to do his or her share of the work
(9) unreasonably criticizes or scorns people in positions of authority (pp. 357-358; APA, 1987)."

While there is symptom overlap and other obvious similarities among the disorders comprising Cluster C (i.e., that they are rigid, "neurotic," dysfunctional behavioral patterns characterized loosely as "anxious-fearful"), obsessive compulsive personality disorder can be contrasted with the others on several points. First, obsessive compulsive personality disordered individuals are more likely than their Anxious-Fearful counterparts to view themselves as competent (Millon, 1990, Beck et al., 1990). Second, according to Millon's (1987) circumplical model of DSM-III-R personality disorders, obsessive compulsive personality disordered individuals are classified as "autonomous" on the affiliation dimension, whereas their Anxious-Fearful counterparts are classified as more "enmeshed." Third, obsessive compulsive individuals are less likely to find their personality characteristics bothersome (i.e., symptoms are ego-syntonic) in contrast to dependent, avoidant, and passive aggressive personality disordered individuals. Of particular importance to the present dissertation, however, are central personality features of obsessive compulsive personality disorder--excessive concern with personal control, autonomy, and achievement--which play less of a role in avoidant, dependent, and passive aggressive personality disorder. Other important features that distinguish them from their Anxious-Fearful Cluster counterparts are the obsessive compulsive's rigidity in thought and tendency to be rule-bound.

Obsessive Compulsive Personality Disorder and Depression

As mentioned above, the comorbidity between personality disorders and depression is common. Furthermore, the comorbidity between depression and the Anxious-Fearful Cluster, in general, and obsessive compulsive personality disorder, in particular, is quite high. In fact, within outpatient populations, obsessive compulsive personality disorder's comorbidity with depression is the highest among personality disorders according to two studies (Shea et al., 1987; Tryer, Casey, & Gall, 1983; cited in Farmer and Nelson-Gray, 1990). Despite this high comorbidity, an argument can be made that certain personality features common to obsessive compulsive personality disordered individuals potentially could serve as a "defense" against the onset of such a dysfunctional affective state. Their internal locus of control, for example, and their relatively positive self-image are personality features not commonly associated with depression. Furthermore, Wittenborn and Maurer (1977) argue that the obsessive compulsive may react to overwhelming environmental stressors and a sense of impending loss of control, by intensifying certain behaviors (i.e., hostility; denial) as a defensive manuever. Additionally, Millon and Kotik (1985) argue that they may attempt to block or neutralize reactions to stressful events through brief periods of accelerated activity which are short-lived and which create considerable anxiety. Perhaps, these "defenses" succeed much of the time and depression is avoided. In behavioral terms, it could be argued that,

due to their learning history, controlling their environment is highly reinforcing for obsessive compulsives. And, because they have been reinforced for persisting and even, intensifying their obsessive compulsive behaviors in situations where difficult tasks are encountered and eventually solved, it follows that, when faced with an apparent uncontrollable situation, persistence, not depressive symptoms, would be observed.

Nevertheless, as the high comorbidity attests, these individuals do become depressed, and attempts have been made to clarify this relationship using the characterological predisposition hypothesis as a model.

According to Zettle and Hayes (1982), excessive rule and self-rule following behavior can be be likened to "irrational cognitions" in that they represent rigid thinking styles that may predispose individuals to depression. Given Schneidmiller's (1987) finding above and further evidence of rigidity in thought among obsessive compulsive personality disordered individuals, a logical deduction can be made that such personality features in this population make them susceptible to depression.

Another attempt at clarifying this relationship comes from Beck (1981) who asserts that obsessive compulsives, because of their emphasis on autonomy, control, and action, become depressed when they fail to reach important goals in their lives. He suggests that, upon failing to reach critical goals, the "self-sufficient, inner-directed, and active" obsessive compulsive eventually becomes "powerless, [and] devoid of initiative and self-control."

Similarly, Areiti and Bemporad (1980) argue that the obsessive compulsive is routinely rewarded with support and acceptance for achieving certain goals, and that when a "dominant goal" becomes unattainable, this threatens the obsessive compulsive's self-esteem and meaning in life. Also, Hammen, Ellicott, Gitlin, and Jamison (1989) argue that "a person who derives sense of worth, significance, and efficacy from autonomous achievement would be vulnerable to the impact of failure or goal frustration in the achievement domain (p. 155)." Such conceptualizations are consistent with the specificity hypothesis which argues that achievement loss may serve as a "trigger event" for depression in certain personality types while social loss may be more detrimental to other personality types (e.g., Neitzel & Harris, 1990). While research on the specificity hypothesis has generated mixed support, the loss of achievement trigger event has been implicated in the onset of depression in autonomous, achievement-oriented individuals (Neitzel & Harris, 1990). Taken together, the conceptualizations above argue that the strong achievement orientation of obsessive compulsives make them vulnerable to feelings of inadequacy and failure, and ultimately depression.

Related to, and perhaps underlying this strong achievement orientation is the excessive emphasis placed on perfection and personal control in the lives of obsessive compulsive personality disordered individuals. Beck et al. (1990) write:

> Due to their rigidity, perfectionism, and strong need to be in control of themselves, their environment, and their emotions, obsessives are very vulnerable to becoming overwhelmed, hopeless, and depressed when they experience their lives as having gotten out of control and their usual coping mechanisms as being ineffective (p. 320).

The above quote suggests that obsessive compulsive personality disordered individuals might be particularly vulnerable to the debilitating effects of perceived uncontrollability (i.e., learned helplessness).

Personality Disorders and Learned Helplessness

Recall that one of the early criticisms leveled against the original learned helplessness model of depression was that "extraneous information" such as personality characteristics seemed to influence the likelihood of helplessness induction. The reformulation attempted to address this and other concerns by highlighting the influence of attributional style on helplessness. Recall that this reformulation improved the theory's ability to predict which individuals would be more likely to respond in a helpless manner when faced with "perceived uncontrollability." This modification represented an acknowledgement that helplessness was more complex than first conceived and that personality variables (i.e., attributional style) needed to be addressed and included in the model.

Prior to the reformulation, personality influences had been demonstrated in learned helplessness research. Recall that Hiroto (1974) divided his subjects into personality groups based on locus of control and found that individuals with an external locus of control were more

vulnerable to helplessness induction than individuals with an internal locus of control. In addition, Thornton (1982) hypothesized that certain personality types were more prone to helplessness induction while others were more resistent. Thornton divided subjects into a prone-type group and resistant-type group based on a questionnaire that he devised which borrowed questions from the Minnesota Multiphasic Personality Inventory, the California Personality Inventory, and Rotter's Internal-External Locus of Control Scale. In line with his predictions, the prone group was more vulnerable to helplessness induction while the resistant group was less vulnerable.

Consistent with these studies, Seligman (1975) argues that certain environmental contingencies can shape a child who believes that he is helpless and that other environmental contingencies can shape a child who possesses a sense of "mastery." Specifically, Seligman writes, "those people who are particularly susceptible to depression may have had lives relatively devoid of mastery; their lives may have been full of situations in which they were helpless to influence their sources of suffering and relief (p. 104) ." In contrast, Seligman asserts that "the most resistant individuals to helplessness are those whose lives are filled with mastery and extensive experience controlling and manipulating sources of reinforcement (p. 104)." Thus, according to Seligman, a learning history shapes an organism in ways that will influence "perceived uncontrollability" and, hence, the impact of learned

helplessness.

While an argument can be made that obsessive-compulsive personality disordered individuals might be resistant to learned helplessness given the importance they place on "controlling and manipulating sources of reinforcement," a counterargument can be made that their excessive and pathological concern with "mastery" and personal control places them "at-risk" for depression when forced to perceive uncontrollable contingencies. In other words, when the obsessive compulsive's previously reinforced responses are no longer reinforced (i.e., his or her behavior does not control outcomes), something akin to extinction is likely to occur. It is this counterargument in this Janus-faced debate that is being taken in the present study. Just as in the specificity hypothesis where autonomous individuals become depressed when they experience a loss of reinforcement (i.e., when their autonomy is threatened) and where sociotropic individuals become depressed when they experience a loss of reinforcement (i.e., when their social status/acceptance is threatened), it is being proposed here, that obsessive compulsive personality disordered individuals will become depressed when they experience a loss of reinforcement (i.e., when their personal control is threatened). While other obsessive compulsive features--emphasis on autonomy and achievement; rigid, rule-bound thinking--probably contribute to a learned helplessness vulnerability, the obsessive compulsive's pathological emphasis on personal control is being viewed as

central to this vulnerability hypothesis, given that "perceived uncontrollability" is the central mechanism responsible for a learned helplessness depression.

Statement of Purpose

To date, most of the research addressing the relationship between personality disorders and depression has been correlational in nature. The present study attempted experimentally to investigate a particular relationship between obsessive compulsive personality disorder and depression--a relationship consistent with the characterological predisposition hypothesis. The learned helplessness model of depression was the theoretical perspective from which this comorbid relationship was analyzed.

The general hypothesis of the dissertation was that obsessive compulsive personality disordered analogues (given the excessive importance of autonomy, achievement, and especially, personal control in their lives) would be more vulnerable, that is, characterologically predisposed, to the debilitating effects of perceived uncontrollability (i.e., learned helplessness) than a normal control group and an Anxious-Fearful personality disordered control group comprised of avoidant, dependent, and/or passive-aggressive analogues. Converging support for such a hypothesis comes from research demonstrating that the obsessive compulsive personality style may be susceptible to perceived uncontrollability as a trigger event and from research demonstrating rigidity of thought and over-reliance on rules in this population. This vulnerability hypothesis was forwarded despite the fact that a plausible argument can be made that certain

obsessive compulsive personality features (i.e, internal locus of control, motivation to excel in achievement endeavors) could serve as protective features in the short-term. Reconciling these two arguments (vulnerability vs. resistance) in light of the obtained findings was attempted.

The present study attempted to demonstrate a learned helplessness vulnerability in obsessive compulsive personality disordered analogues using a partial replication of Hiroto (1974) and Hiroto and Seligman's (1975) escape-avoidance hand shuttlebox procedure. Specifically, a two-phase learned helplessness protocol was administered to all participants.

1. It was predicted that Group B subjects who are given a response-independent task in the pretreatment phase of the triadic design administration would show more pronounced affective disruptions and more pronounced performance/motivational and cognitive deficits on a response-dependent task administered in the test phase as compared to subjects given either a response-dependent task (Group A) or no task (Group C) in the pretreatment phase. In other words, it was predicted that Group B subjects made experimentally helpless in the pretreatment phase would perceive this uncontrollability, would predict that future outcomes are independent of their actions, would become more affectively distressed, would take longer to initiate responses and reach a learning criterion, and would perform more poorly on a solvable task compared to triadic

counterparts. Hence, the basic learned helplessness effect was predicted.

2. Furthermore, as mentioned above, it was predicted that obsessive compulsive Group B subjects would demonstrate greater learned helplessness deficits relative to Group B subjects in the two control groups.

3. In addition, it was predicted that both of the personality disorder analogue groups, especially the obsessive compulsive personality analogue group, would provide relatively more internal, stable, and global reasons for negative events and more external, unstable, and specific reasons for positive events on the Attributional Style Questionnaire. In other words, it was predicted that both analogue groups, particularly obsessive compulsives, would be identified as being more "at-risk" of developing a learned helplessness depression than normal controls. Furthermore, it was hypothesized that Group B subjects identified as more "at-risk" would actually behave in more helpless manner in reaction to perceived uncontrollability in the experiment.

4. The Tennessee Self-Concept Scale was also administered to clarify the relationship between self-esteem and attributional style. Specifically, a significant correlation between "trait" self-esteem and "trait" attributional style was predicted. As for specific diagnostic predictions, it was hypothesized

that Anxious-Fearful controls would self-report lower self-esteem than normal controls. Given past research on obsessive compulsive personality disordered individuals' self-perceptions, obsessive compulsive analogues in this study were not predicted to differ significantly from normal controls with regard to their self-esteem.

5. A secondary research hypothesis addressed by the present study concerned the hypothesized mood progression in the development of a learned helplessness depression described by Seligman (1975) and Miller, Rosellini, and Seligman (1986). It was hypothesized that, upon "perceiving uncontrollability," Group B subjects' scores on the Multiple Affect Adjective Checklist would indicate a progression in mood from anxiety to depression over the course of helplessness induction. Recall, Seligman argues that, when organisms are first exposed to uncontrollability, their behavior is characterized more by anxiety (i.e., they get agitated and become energized--responses which often help organisms overcome obstacles and regain control). However, after some time of experiencing continued failure to control outcomes, this anxious affective state is replaced by a depressive affective state and other accompanying learned helplessness deficits. Prior to this study, little research had attempted to capture this mood progression experimentally.

Given the relatively high comorbidity rate between obsessive compulsive personality disorder and major depression in outpatient samples, the present investigation has significant clinical implications. Namely, demonstrating this learned helplessness vulnerability would address directly Peterson and Seligman's (1984) charge for future learned helplessness research to identify "at-risk" subgroups and would identify a possible "trigger event" for depression in obsessive compulsive disordered individuals--"perceived uncontrollability." Furthermore, identifying uncontrollability as a "trigger event" in this group potentially could influence psychotherapeutic decisions designed to treat both the depression and the personality disorder, and might have implications for the prevention of both as well. These clinical considerations are addressed in the discussion.

CHAPTER II

METHOD

Subjects

A total of 136 female undergraduates enrolled in psychology courses at the University of North Carolina--Greensboro participated in the study. They were selected based on their scores on the self-report version of the Structured Clinical Interview for DSM-III-R (SCID-II; Spitzer, Williams, Gibbons, & First, 1990) which was administered during mass screening sessions at the beginning of several semesters (See Table 1 for participants' SCID-II scores; Table 1 and all other tables are located in Appendix B).

A total of 45 subjects comprised the Obsessive-Compulsive Personality Disorder analogue group (OCPD). These subjects were found to meet the diagnostic criteria on the Obsessive-Compulsive scale of the SCID-II (i.e., they self-reported at least 5 of 9 criteria). A subset of this group (n=23) were identified as "pure" OCPDs in that they did not meet the diagnostic criteria on any of the remaining personality scales. The remainder of this group (n=22) were identified as "non-pure" OCPDs in that they met the diagnostic criterion on one additional personality scale outside the Anxious-Fearful Cluster (i.e., Avoidant, Dependent, or Passive-Aggressive). Analyses conducted using only

the pure group generated a pattern of findings similar to analyses using the combined group, hence, justifying the use of the combined group. Utilizing this combined group increased the statistical power of the analyses. Therefore, the acronym "OCPD," used throughout the text, refers to the two subgroups combined.

A total of 45 subjects comprised the Anxious-Fearful Control group (AFC). These subjects were found to meet the diagnostic criteria on one or more of the Anxious-Fearful Cluster personality scales except for the Obsessive-Compulsive scale of the SCID-II (i.e., Avoidant--at least 4 of 7 criteria, Dependent--at least 5 of 9 criteria, and/or Passive-Aggressive scales--at least 5 of 9 criteria). A subset of this group (n= 24) were identified as "pure" AFCs in that they did not meet the diagnostic criteria on any of the remaining personality scales. The remainder of this group (n=21) were identified as "non-pure" AFCs in that they met the diagnostic criterion on one additional personality scale outside the Anxious-Fearful Cluster. Again, analyses conducted using only the pure group generated a pattern of findings similar to analyses using the combined group, hence, justifying the use of the combined group. Utilizing this combined group increased the statistical power of the analyses. Therefore, the acronym "AFC," used throughout the text, refers to the two subgroups combined.

A total of 46 subjects comprised the Normal Control group (NC). These subjects did not meet the diagnostic criteria on any of the SCID-II personality

scales.

In addition to the above selection criteria, all subjects were screened for depression using the Beck Depression Inventory immediately prior to participating in the experiment. Only non-severely depressed subjects with scores of 20 or below were actually included in the study. Two subjects (not included in the totals cited above) met the SCID-II criteria for inclusion in the study but scored above the BDI depression cut-off point used in this study. They were not administered the procedure but, instead, were given a special debriefing. Futhermore, only subjects who reported no significant hearing or ear problems were included in the study given that an aversive tone was presented periodically throughout the experimental procedure.

Within each diagnostic category (i.e., OCPD, AFC, NC), subjects were assigned to one of three triadic groups (i.e., A, B, or C). The number of subjects per cell when diagnostic group and triadic group are crossed follow: OCPD-Group A = 15; OCPD-Group B = 15; OCPD-Group C = 15; AFC-Group A = 16; AFC-Group B = 14; AFC-Group C = 15; NC-Group A = 16; NC-Group B = 15; NC-Group C = 15. Triadic groups were assumed comparable with regard to age and race by using simple random assignment within diagnostic group.

Subjects were compensated for their participation with either research credits or money. The treatment of all participants was in accordance with the ethical standards of the American Psychological Association (See Ethical Principles of Psychologists and Code of Conduct, APA, 1992).

Materials

Self-Report Instruments

The Personality Diagnostic Questionnaire--Revised (Hyler & Reider, 1984), also referred to as the self-report version of the Structured Clinical Interview for the DSM-III-R: Personality Disorders (SCID-II) (Spitzer et al., 1990), is a 130-item self-report screening instrument that assesses personality disorder symptoms which directly correspond to the personality disorder classification system of the DSM-III-R. Subjects simply respond affirmatively or negatively to the items. In contrast to the more thorough SCID-II Interview, the self-report version results in a high false positive rate (Reich, 1989). Thus, it is not appropriate to use this instrument in isolation to diagnose personality disorders. Instead, it should be used as a screening tool by clinicians in diagnosing individuals with suspected personality disorders. Due to its high false positive rate, results obtained from studies utilizing this self-report measure only are often diluted, however, and must be interpreted with this shortcoming in mind. Nevertheless, the advantages of the self-report version of the SCID-II are its correspondence to the DSM-III-R categories and the ease with which it can be administered. The self-report version of the SCID-II has been used as a screening measure in previous research (e.g., Levin & Hyler, 1986; Yager, Landsverk, Edelstein, & Hyler, 1989) and both adequate reliability and validity coefficients have been demonstrated (e.g., O'Boyle & Self, 1990; Reich, 1989; Spitzer et al., 1990).

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report measure designed to assess current severity of depression. For each item, respondents are required to choose the most self-descriptive statement from among four alternatives that characterizes their psychological state over the last two weeks. These statements are graded in severity and are assigned values (i.e., 0-3, 3=most severe) which are summed over all 21 items. Beck and Beamesderfer (1984) suggest that cut-off scores should be based on the purpose of the assessment and the decisions to be made as a result of the assessment. Different cut-off scores for different levels of depression have been suggested in the literature; however, there is general agreement that scores above 20 are commonly associated with severe depression (e.g., Steer, Beck, & Garrison, 1985). Of the three "triple response" modalities, the BDI emphasizes the cognitive-verbal channel, although affective, motivational, and physiological symptoms are assessed. The BDI has been found to have adequate test-retest reliability (e.g., Tennen, Herzberger, & Nelson, 1987), concurrent and predictive validity (e.g., Beck, 1967), and internal consistency (e.g., Tennen et al., 1987).

The Attributional Style Questionnaire (ASQ; Peterson, Semmel, von Baeyer, Abramson, Metalsky, & Seligman, 1982) is a self-report instrument that was devised specifically to assess the three "explanatory" dimensions proposed in the reformulated learned helplessness theory and identify "at-risk" attributional patterns. Adults are presented with 12 hypothetical events

(i.e., 6 with positive outcomes and 6 with negative outcomes) and then are asked to provide causes for the events and rate their responses on 7-point scales that measure the attributions' degree of internality, stability, globality, and importance. The ASQ's psychometric properties are sound and are reviewed in Tennen and Herzberger (1986).

The Tennessee Self-Concept Scale-Clinical and Research Form (TSCS; Fitts, 1965) is a 100-item self-report measure that assesses what an individual "is, does, likes, and feels" (p. 663) (Walsh, 1984). Respondents rate self-statements on a 5-point scale ranging from "completely false" to "completely true." This instrument provides 29 scores, including, among others, a global self-esteem measure and informative subscales that tap various internal aspects of self-concept (i.e., identity, behavior, and self-satisfaction) and external aspects of self-concept (i.e., moral-ethical, social, personal, physical, and family). It has been utilized extensively in both clinical work and research and is considered to have respectable reliability and validity (See Fitts, 1965, and Walsh, 1984, for extensive reviews of psychometric properties of the TSCS).

The Multiple Affect Adjective Checklist-Today Form (MAACL; Zuckerman, Lubin, Vogel, & Valerius, 1964) is a 132-item self-report instrument that yields three empirically derived affective state scales: Depression, Anxiety, Hostility. Respondents simply endorse adjectives that describe how they feel. This measure is sensitive to daily or "here-and-now"

fluctuations in mood. This advantage, along with the fact that it is easy and quick to administer, make it ideal for repeated measures. According to the literature, the MAACL has adequate reliability and validity (e.g., Zuckerman & Lubin, 1965; Zuckerman & Lubin, 1985). Its popularity among researchers is evidenced in Lubin, Zuckerman, and Woodward's (1985) bibliography of 716 published articles and dissertations that have used this measure.

The Depression Adjective Checklist (DACL; Lubin, 1966) is a 32-item self-report instrument that assesses state changes in depressive mood. While it does not provide multiple affective state assessment like the MAACL, its advantage is that it is even quicker to administer than the MAACL. Like the MAACL, it has adequate reliability and validity (e.g., Lubin & Himelstein, 1976). Seven parallel forms of this measure are available. The inter-form correlations are quite high (i.e., parallel form reliability) which justifies the use of alternate versions (Petzel, 1985). In the present study, only Form A was utilized given that the DACL was administered only once.

In the present study, two measures were devised by the experimenter. The first measure, an Expectancy Rating Form was used to assess the degree to which subjects had confidence in their future performance on a given task. They were asked to rate on an 11-point scale how well they thought they would do on an upcoming task to which they had not yet been exposed (i.e., 0=No confidence in success, 10=Complete confidence in success). The second measure devised by the investigator was a Post-Experiment Questionnaire

designed to assess various process measures that were included to clarify any predicted or unpredicted findings. Copies of both of these measures can be found in Appendix C and Appendix D, respectively.

Shuttlebox Apparatus

A CompuAdd 286 personal computer (including hard drive, monitor, and keyboard) was used to administer the experimental protocol. A black dot was affixed to both the space bar and the "H" key on the keyboard, a red dot was affixed to the "8" key on the number pad, and a blue dot was affixed to both the "4" and "6" keys on the number pad (an explanation for these dots is provided in the next section). A 15 1/2" by 8" cardboard keyboard cover was utilized that could be easily moved by the experimenter to expose or conceal the marked keys. Written on the keyboard cover was the following statement: "Do not remove this cover."

Several graphics were displayed on the computer monitor during the experimental protocol. These graphics are described in the next section.

The computer generated a tone which was directed through an attenuator (Hewlett Packard, 350 D Attenuator Set, 5W-55V, 600, DC-IMC), through an adaptor (Grayson-Stadler, Co., 400/600 Ohms) and finally through a set of headphones (Grayson-Stadler, Co., TDH39-10Z; MX-41/AR pads). The frequency of the tone was set at 3000Hz, but the intensity of the tone could be manipulated by the experimenter using the attenuator dial. Tone intensity could range from 60 to 100 dB in 5 dB increments.

A BASIC program was designed to administer the protocol and collect data. Data output was printed on an Epson LX-800 printer.

Procedure

A procedural schemata of the present study is presented in Appendix E and may be referred to as needed. Recall that the present study is a partial replication of the 2-phase learned helplessness protocol used by Hiroto (1974) and Hiroto and Seligman (1975). As the present study is detailed, attention is given to the primary differences between the present and above studies with regard to procedure. Furthermore, the rationale behind these modifications is given.

Testing time blocks of one and one-half hours were reserved for each participant, and details concerning date, time, and place of testing were discussed in advance. The protocol was administered by one of four trained experimenters. Experimenters followed a standardized administration procedure in order to lessen the possible effect of different administration "styles." Before testing began, an informed consent was signed by the participant (See Appendix F for copy of Informed Consent). Next, each subject was administered the BDI in order to rule out the presence of depression. If a subject scored above 20 on the BDI, the testing was discontinued and a special debriefing statement was given (See Appendix G for Special Debriefing A and B). If a subject scored 20 or below, testing was continued, and the ASQ and TSCS were administered in a randomly determined order for each subject.

Each subject read the instructions to these questionnaires herself and completed them at her own pace, alone in a laboratory room.

Next, the subject was escorted to the testing room where she was read the following instructions:

> Before we get started, there are a few more things that we need to do. First, we need to select a volume level that you find to be slightly unpleasant. This will be the volume level that will be presented throughout the experiment. I'm going to present a series of tones, starting at a low level and then, increasing in volume. I'd like you to raise your hand high when you find a particular noise slightly unpleasant. Again, I won't present any noises that are considered dangerous. We'll do this several times and I'll start each series from a different volume level. Remember, raise your hand high when you find the noise slightly unpleasant.

Recall that in Hiroto and Seligman's (1975) human version of Seligman's dog shuttlebox study, an aversive tone was used as the aversive stimulus instead of shock. A tone of 3000 Hz and 90 dB was administered to all subjects in their study. In the present study, all subjects were administered a tone of 3000 Hz, but were instructed to select a dB level that they personally found "slightly unpleasant." This modification was made for ethical reasons and to insure that subjects were equated on their subjective perception of aversiveness. Tones, ranging from 60 to (potentially) 100 dB, were presented in ascending order of 5 dB increments over four trials. Each trial began at a different dB level and was comprised of a series of tones. An average of the decibels levels selected over the four trials was calculated on each subject and was used as the "slightly unpleasant" noise level presented during the

procedure for that particular subject. No significant differences among triadic groups or diagnostic groups were noted with regard to decibel level selection.

After the decibel level was selected, the subject was given the first administration of the MAACL (MAACL1) and was read the following instructions:

> Now I'm going to give you another brief questionnaire. On this sheet you will find words which describe different kinds of moods and feelings. Mark an "X" in the boxes beside the words which describe how you feel now--today. Some of the words may sound alike, but we want you to mark all of the words that describe your feelings. Please read the words carefully, work rapidly, and tell me when you are through.

After the subject completed the checklist, she was given the first administration of the Expectancy Rating form (ER1) which asked the subject to rate on a scale from 0 to 10 how well she thought she was going to do on an upcoming task (0 = Poorly; 10 = Very Well).

Learned Helplessness Procedure

At this point in the protocol, the subject was administered the 2-phase learned helplessness procedure. The procedure differed depending on triadic group assignment. Recall, subjects in all three diagnostic groups were randomly assigned to one of three triadic groups, Group A--the response dependent group, Group B--the response independent group, and Group C--the "no-pretreatment" group. As mentioned above, this procedure was a partial replication of Hiroto (1974) and Hiroto and Seligman's (1975) study. As in both studies, Group A subjects were presented with a solvable

pretreatment task, Group B subjects were presented with an unsolvable pretreatment task, and Group C subjects were not administered a formal pretreatment. The present study utilized the aversive tone modification made in Hiroto and Seligman (1975) which corrected for the design flaw in Hiroto (1974). Namely, Group C subjects did not receive a pretreatment task, but were administered the same aversive tones as Group A and Group B. Recall, this modification was seen as important because it equated all groups on exposure to the physical stimulus and helped isolate perceived uncontrollability as the mechanism responsible for the noted deficits/disruptions. Finally, in the test phase, all subjects were administered a solvable hand shuttlebox task where learned helplessness deficits were assessed.

In the present study, several modifications were made in the procedure. Some attention now is paid to the differences between the present study and Hiroto's (1974) and Hiroto and Seligman's (1975) studies. It was decided to computerize the 2-phase procedure in the present study in order to make the administration of the task easier and data collection more precise. Instead of using an actual box with a knob protruding from the top in the test phase, the modified shuttlebox was graphically displayed on the computer monitor. Instead of sliding the knob from one end of the box to the other in order to terminate the noise, subjects were required to figure out a sequence of key presses that would move a cursor across the screen from one box to

another. The response requirement, determined during pilot work, was such that an adequate range of performance was produced (i.e., some subjects never learned the sequence, others caught on immediately, and most fell somewhere in between).

Several more modifications were made to the pretreatment phase of the procedure that were not part of Hiroto's (1974) or Hiroto and Seligman's (1975) studies. In piloting the procedure for the present study, it was found that many Group B subjects were making an external attribution regarding their failure during the pretreatment phase. For example, many subjects reported that they suspected that the computer was programmed to make terminating the noise impossible (i.e., given that they had exhausted all response options). It was reasoned that making such an external attribution might make helplessness induction and generalization less likely to occur. Subjects in the initial pilot work, like subjects in the Hiroto (1975) and Hiroto and Seligman (1975) studies, were presented with a single button and asked to "do something" to stop the noise. In further pilot work, the pretreatment phase was complicated by adding a second "dummy" key that actually had no control over the noise. Also, each time the space bar was pressed, a graphically displayed square appeared on the monitor. Group B pilot subjects exposed to this new procedure were more likely to make an internal attribution (i.e., to attribute their failure to their inability to "break the code" and not to the task being impossible).

Another modification was to divide the pretreatment phase into two distinct blocks. The procedure for the two blocks was the same, but they were separated by a short interval of time. This modification was made for two reasons. The first reason was to facilitate further an internal attribution for failure in Group B subjects. For example, if experimentally sophisticated subjects were initially likely to attribute their failure to the task being impossible and also suspected that, in the next phase, they would have control over outcomes, it was reasoned that their initial attributions would more likely change to internal attributions upon receiving a second identical block. Secondly, dividing the pretreatment into two blocks made possible administering the MAACL at the midpoint of the pretreatment phase in addition to the beginning and end of the pretreatment phase as had been done in previous research. Thus, affect in the first half of the pretreatment phase could now be compared with affect in the second half of the pretreatment phase to investigate experimentally the mood progression from anxiety to depression posited by Seligman (1975) and Miller, Rosellini, & Seligman (1986). To avoid confusion, the first half of the pretreatment phase will be referred to as "Block 1," the second half of the pretreatment phase will be referred to as "Block 2," and the test phase will be referred to as "Block 3."

Block 1

After the first administration of the Expectancy Rating form, Group A and Group B subjects were read the following directions:

> Please listen to these instructions carefully. I am not allowed to give you additional information other than what I am about to tell you. I will answer any questions you may have at the end of the experiment. From time to time a slightly unpleasant noise will come on for awhile. When that noise comes on there is something you can do to stop it. I'm not going to tell you how to stop it, but I will give you a hint. It involves pressing one or both of the marked keys in some way. A feedback screen will appear on the computer monitor after each noise. The feedback screen will tell you how the noise on each trial was stopped. After any given noise, if you find the way to stop the noise, then the screen will say "Congratulations. You stopped the noise." After any given noise, if you don't stop the noise yourself, then the screen will say, "Sorry, the noise stopped automatically." Please be sure to look up at the screen after each noise to see how you did. It is possible to stop each noise yourself. Remember, the goal is to stop the noise yourself by doing something. Do you understand these instructions? Taking the headphones off, dismantling the apparatus, and removing the keyboard cover will not help you stop the noise so please don't do any of those things. I'll be next door and return when this part of the experiment is over.

The following reminder was printed on a sheet of paper and affixed below the computer monitor: "Your job is to stop the noise by doing something. Hint: Use one or both of the marked keys in some way." The experimenter then left the room and returned after 25 trials had been administered.

Group C subjects were read the following instructions instead:

> Please listen to these instructions carefully. I am not allowed to give you additional information other than what I am about to tell you. I will answer any questions that you may have at the end of the experiment. From time to time a slightly unpleasant noise will come on for awhile. Please sit and listen to it. I'll be next door and I'll return when this part of the experiment is over.

The following reminder was printed on a sheet of paper and affixed below the computer monitor: "Just sit and listen to the noise." Again, the experimenter then left the room and returned after 25 trials had been administered.

During Block 1, Group A and B subjects were administered a series of 25 self-selected tones. The intertrial interval duration between the offset of each tone and the onset of the next tone was predetermined and the same for all subjects. For Group A subjects, the duration of each tone was determined by the subject. Each Group A subject could terminate the noise by pressing the space bar (marked with a black dot) four consecutive times. If a correct response was not made, the tone terminated automatically after 5 seconds. At the offset of a given tone, Group A subjects were given accurate feedback on their performance. Each Group B subject was yoked to a Group A subject. In other words, each Group B subject received the same intertrial interval durations and tone durations as a Group A counterpart. However, Group B subjects always received feedback that they had failed to terminate the noise. No response on their part could terminate the noise. Group C subjects were also yoked to a Group A subject. They received the same intertrial interval durations and tone durations but were not given feedback on their performance given that they were not required to terminate the noise. In essence, they were not administered the pretreatment task but were equated with Group A and B subjects with regard to total time elapsed during Block 1

and with regard to exposure to the aversive stimulus. Again, the pretreatment phase in this experiment is where helplessness is induced in Group B subjects and where Group A and C subjects serve as controls, helping to isolate the effects of uncontrollability.

After the completion of the first half of the pretreatment phase, all subjects were given the second administration of the MAACL (MAACL2) and were read the following instructions:

> This is that same questionnaire you filled out earlier. Your answers may or may not be the same as they were before--that is not important. The important thing is to check off all those adjectives that describe how you are feeling right now--at this very moment. Please read the words carefully, work rapidly, and tell me when you are through.

Block 2

Next, the second half of the pretreatment was administered and the following instructions were read to all subjects:

> I'm going to start it up again. The instructions are the same as before. I will answer any questions you may have at the end of the experiment.

Block 2 was identical to Block 1. Another series of 25 tones was presented with the same instructions as in Block 1. Again, Group B subjects were being made experimentally helpless, and Group A and C subjects were serving as controls. After completion of the second half of the pretreatment phase, all subjects were given the third, and final, administration of the MAACL (MAACL3) and were read the following instructions:

> This is that same questionnaire you filled out earlier. Again, your answers may or may not be the same as they were before--that is not important. The important thing is to check off all those adjectives that describe how you are feeling right now--at this very moment. Please read the words carefully, work rapidly, and tell me when you are through.

Block 3

The pretreatment phase (Blocks 1 and 2) was designed to induce helplessness in Group B subjects and the test phase (Block 3) was designed to measure the generalization effects of this helplessness induction on a different task. In Block 3, the following instructions were read to all subjects:

> Please listen to these instructions carefully. These are different than the earlier set of instructions. I am not allowed to give you additional information other than what I am about to tell you. I will answer any questions that you may have at the end of the experiment. You will be given some trials in which a slightly unpleasant noise will be presented to you. Whenever you hear the noise come on there is something you can do to stop it. If you are unable to figure out the strategy, the noise will stop on its own after 5 seconds. The goal is to do something in order to keep the noise off as much as possible. You will not receive feedback on whether you are keeping the noise off or whether the noise is turning off automatically, but this should become obvious to you as you do the task. I'll give you two hints that might help you. First, use only the colored keys exposed on the keyboard. I'll show you those in just a second--they're under the cover right now. Second, it might help to occasionally look up at the screen while making a response to see the effect your response is having. Do you understand these instructions? [Again], taking the headphones off, dismantling the apparatus, and removing the keyboard cover will not help you stop the noise so please don't do those things. I'll answer any questions at the very end of the experiment. I'll be next door and I'll return when this part of the experiment is over.

A different reminder printed on a sheet of paper was posted below the computer monitor and read: "You are to figure out a strategy that will keep the noise off as much as possible: Hint: Use the marked keys in some way and, it may help to occasionally look up at the screen to see the effect your response is having."

A second Expectancy Rating Form (ER2) was administered at this point. Again, the experimenter asked the subject to rate on a scale from 0 to 10 how well she thought she was going to do on the upcoming task (0 = Poorly; 10 = Very Well).

As in Blocks 1 and 2, during Block 3 all subjects were administered a series of 25 self-selected tones. Again, the intertrial interval duration was predetermined and the same for all subjects. During Block 3, it was possible for all subjects, regardless of triadic group assignment, to figure out the strategy on the test phase task. At the beginning of each of the 25 trials, a graphically-presented shuttlebox appeared on the computer monitor. A cursor was preset in the left box. Once the tone came on, subjects could terminate the tone by moving the cursor from the left box, through an "alley", to the right box by pressing the top red key and right blue key (located on the number pad) in an alternating fashion. In order to move from the right box to the left box, subjects were required to press the top red key and left blue key in an alternating fashion. The initial cursor placement, and hence, correct response sequence alternated each trial. After the tone sounded, if a

subject was unable to move the cursor across the screen in five seconds, the tone stopped automatically. Five seconds prior to the onset of each tone, a "warning signal" appeared on the screen and was present until the tone sounded. Subjects were able to move the cursor as soon as the warning signal appeared and, if they responded quickly enough, could get to the opposite box prior to the tone ever sounding. Thus, they could avoid the noise altogether. To escape the tone was defined as terminating the noise within 5 seconds after it had sounded, and to avoid the noise was defined as making a correct response prior to tone onset.

Upon completion of the test phase, all subjects were administered a Post-Experiment questionnaire and underwent a formal debriefing (Appendix H). The DACL was administered for ethical purposes to identify subjects who might have been distressed as a result of the procedure, but were not willing to admit this to the experimenter when specifically asked. Two subjects were administered Special Debriefing B (Appendix G) as a precautionary measure to address suspected distress. However, these two subjects reported that they were upset due to reasons unrelated to their participation in the experiment.

After the debriefing, all subjects a) were asked for permission to use the data collected, b) were asked not to tell anyone about the details of the experiment so as to insure that future subjects would be naive, c) were either given research credit or financial compensation for their participation, and d) were thanked for their time and contribution.

Independent Variables

The primary independent variables used in the present study were diagnostic group and triadic design group. Diagnostic group was comprised of three levels (i.e., OCPD, AFC, and NC), as was triadic design group (i.e., Group A, Group B, and Group C). Crossing the two primary factors yielded a 3 by 3 factorial design. A secondary independent variable was created by converting the ASQ Composite (defined below) into a two-category variable so that it could be used as a factor. The two categories were ASQ Composite scores above the mean, and ASQ Composite scores below the mean.

Primary Dependent Variables

The following section operationally defines the primary dependent variables utilized in the study and indicates what high and/or low scores on various measures represent. It is meant to be used as convenient reference.

Expectancy Ratings

ER1-ER2--This variable represents the change in performance expectancy from the pretreatment task to the test phase task. Higher numbers suggest the perception of uncontrollability and reduced confidence.

Learned Helplessness Deficits/Disruptions

Affective Disruptions (MAACL)

MAACL Composite--This variable represents the change in overall affect from baseline to the end of the pretreatment phase. Higher scores represent an increase in overall affect.

D3-D1--This variable represents the change in depressive affect from baseline to the end of the pretreatment phase. Higher scores represent an increase in depressive affect.

A3-A1--This variable represents the change in anxious affect from baseline to the end of the pretreatment phase. Higher scores represent an increase in anxious affect.

H3-H1--This variable represents the change in hostile affect from baseline to the end of the pretreatment phase. Higher scores represent an increase in hostile affect.

Cognitive/Motivational Deficits (Shuttlebox Measures)

Number of Trials to Criterion-Escape Response (NTC-Escape)--This variable is operationally defined as the number of trials (out of 25) needed to meet the escape response learning criterion (3 consecutive escape responses). Lower scores represent quicker learning and presumably less depression.

Number of Failures (NF)--This variable is operationally defined as the number of trials (out of 25) on which the subject fails to make an escape response. Lower scores represent quicker learning, greater motivation, and presumably less depression.

Latency

a. Reaction Time on Trial 1 (RT1)--This variable is operationally defined as the latency (in seconds) for the subject to initiate a key press on Trial 1. Lower scores represent greater motivation and presumably less depression.

b. Mean Latency to Initiate a Response (MLI)--This variable is operationally defined as the mean latency to initiate a response (in seconds) over 25 trials for the subject to initiate a key press. Lower scores represent greater motivation and presumably less depression.

Number of Trials to Criterion-Avoid Response (NTC-Avoid)--This variable is operationally defined as the number of trials (out of 25) needed to meet the avoidance response criterion (3 consecutive avoidance responses). Lower scores represent quicker learning and presumably less depression.

Number of Avoidance Responses (NA)--This variable is operationally defined as the number of trials (out of 25) on which a subject successfully makes an avoidance response. Higher scores represent quicker learning, greater motivation, and presumably less depression.

Trait Measures

Attributional Style Questionnaire

ASQ Negative Events--According to the literature this is the most predictive index of the Attributional Style Questionnaire scales. Higher scores represent a more depressive attributional style when faced with negative events.

ASQ Positive Events--Lower scores represent a more depressive attributional style when faced with positive events.

ASQ Composite--ASQ-Negative Events minus ASQ-Positive Events. Higher scores represent a more depressive attributional style when faced with both positive and negative events.

ASQ Subscales--selected subscales will be addressed and defined later in the text.

Tennessee Self-Concept Scale

TSCS Total Positive--This score represents overall self-esteem. Higher scores indicate higher self-esteem.

Mood Progression

Percent Change in Depression in Block 1 from MAACL1 to MAACL2

Percent Change in Anxiety in Block 1 from MAACL1 to MAACL2

Percent Change in Depression in Block 2 from MAACL2 to MAACL3

Percent Change in Anxiety in Block 2 from MAACL2 to MAACL3

CHAPTER III

RESULTS

Overview

The findings of the present study are presented under seven subheadings. In the first section, expectancy ratings are analysed to satisfy the prerequisite for demonstrating the learned helplessness effect; namely, it must be established that Triadic Group B subjects "perceive uncontrollability" in the pretreatment phase. In the second section, the overall helplessness construct is assessed using multivariate analyses with special attention being paid to the role of personality and attributional style influences on the basic learned helplessness effect. In the third section, the component deficits and disruptions comprising this overall construct, as well as the impact of personality characteristics on helplessness induction, are analysed using univariate statistics. In the fourth section, diagnostic groups are compared on attributional style and self-esteem trait measures, using univariate analyses, and these two measures are correlated using Pearson's product moment correlation coefficient. In the fifth section, descriptive and inferential statistics are utilized in an attempt to assess the hypothesized learned helplessness mood progression. In the sixth section, post-experiment questionnaire ratings, designed to clarify predicted and unpredicted results, are analyzed

using univariate methods. And finally, in the seventh section, ancillary analyses which are used to illustrate the arguments made in the discussion section are presented.

Expectancy Ratings

It was predicted that Group B subjects would perceive uncontrollability and report being less confident about their future performance on a different task to which they had not yet been exposed. A two-way ANOVA on change in expectancy ratings (ER1-ER2) was performed with triadic group and diagnostic group serving as factors. This analysis yielded a statistically significant triadic group effect, F (2,125) = 24.69, p = .0001. Triadic group means were as follows: Group A = -.17, Group C = .49, Group B = 1.64 (Table 2). According to Scheffe's test of multiple comparisons, all three groups were significantly different from one another (Table 3). Thus, Group B demonstrated less confidence and greater perceived uncontrollability than both Group A and Group C as predicted. Interestingly, an unpredicted finding was that Group C demonstrated less confidence and greater perceived uncontrollability than Group A. No significant diagnostic effect or interaction was obtained.

Multivariate Analyses

Several multivariate analyses were performed to assess the effects of the independent variables on combined dependent variables. These global tests address a larger "helplessness" construct (i.e., the overall learned

helplessness effect), whereas the more molecular analyses, to be covered below, address the component deficits/disruptions in isolation. Interestingly, past learned helplessness research has often inspected only the individual variables, some which have yielded statistical significance and some which have not, in determining whether a basic effect has been obtained. Such a determination, at times, has been quite subjective in that researchers have argued for obtaining the effect based on an arbitrary number of the analyses yielding significant differences. The advantage of the MANOVA is that it addresses such a determination statistically, taking into account the correlation among the various dependent variables included in the model.

In the present study, it was predicted that Group B subjects would demonstrate greater learned helplessness deficits than Group A and C counterparts and that OCPD would be more vulnerable to helplessness induction than AFC and NC. Furthermore, it was predicted that adding ASQ to the model, modified as a categorical factor, would demonstrate that attributional style does, in fact, influence helplessness induction. Specifically, it was predicted that subjects identified as having attributional styles "at-risk" for depression would demonstrate more pronounced learned helplessness deficits/disruptions in the experimental demonstration than subjects with nondepressive attributional styles.

a. MANOVA #1: A two-way MANOVA with diagnostic group and triadic group as factors yielded a significant triadic group main effect, Wilks'

Lambda = .832, equivalent to F (6, 244) = 3.915, p = .0009 (Table 4) when the combined contribution of three dependent variables representing each of the traditional learned helplessness deficits was analyzed (i.e., MAACL Composite--Affective Disruption; NTC-Escape--Cognitive/Learning Deficit; RT1--Motivational/Performance Deficit). An inspection of the means indicated that Group C demonstrated greater learned helplessness deficits/disruptions than Group A. However, the statistical significance of the difference between Group A and B could not be determined using this model, but is addressed using a modified model that is described below. No significant diagnostic main effect or interaction was found in MANOVA#1. The three dependent variables named above were selected for this MANOVA for the following reasons. MAACL-Composite was selected to represent the affective domain in this analysis because it is a global measure of affect. While learned helplessness is a theory of "depression," research argues that perceived uncontrollability produces anxiety, hostility, and depression, all of which are assessed by MAACL-Composite (Peterson, Maier, & Seligman, 1993). The dependent variables, NTC-Escape and RT1, were identified as the best representatives of the cognitive and performance/motivational deficits, respectively, in that conceptually, they are the purest measures of their respective domains. NTC-Escape has been used as a measure of "ability to learn" in many previous studies including Hiroto's (1974) and Hiroto's and Seligman's (1975) studies. RT1, or reaction time to initiate a response on Trial

1, was selected in this study because it appeared to be the purest measure of motivation. The other reaction time measure assessed in this study, MLI, is an average of latencies over 25 trials. Measures similar to MLI have been used in previous research (Hiroto, 1974; Hiroto & Seligman, 1975); however, this measure of "motivation" appears to be influenced greatly by "ability to learn." For example, an individual may be highly motivated, but because she does not learn the response requirement as quickly as other subjects, her long MLI reaction time may be misleading. Conversely, an individual who learns the response requirement quickly and learns that responding prior to the tone is possible, may actually be quite unmotivated, and yet, may generate a MLI that suggests that she is highly motivated. Therefore, RT1 was selected as the representative measure of motivation because it is independent of learning the response criterion.

b. MANOVA #2: Another MANOVA was employed to determine if there was a significant difference between the two critical triadic groups, Group A and Group B. Thus, Group C was eliminated from the analysis. Demonstrating such a difference between Group A and B is necessary to argue for a basic learned helplessness effect. In addition to the modified triadic group factor, a categorized ASQ factor was added to the model to determine if triadic group differences are affected by attributional style. Because no significant diagnostic effect or triadic/diagnostic interaction was obtained in MANOVA #1, the diagnostic group factor was removed from the model in

MANOVA #2. Therefore, a two-way MANOVA with triadic group and categorized ASQ as factors was conducted. This analysis yielded a significant triadic group main effect, Wilks' Lambda = .909, equivalent to F (3, 83) = 2.769, p = .0468 (Table 5) when the combined contribution of the three representative dependent variables was analyzed. Group B subjects were, indeed, found to be more helpless than Group A subjects as predicted, but no significant categorized ASQ main effect or interaction was obtained.

c. MANOVA #3: A third MANOVA was employed as an alternative to MANOVA #2. Whereas in MANOVA #2, the cognitive deficit was represented by the shuttlebox measure, NTC-Escape; in MANOVA #3, this deficit was represented by Expectancy Rating Change over the course of the pretreatment (ER1-ER2). Whereas NTC-Escape assessed the actual learning curve, ER1-ER2 assessed "belief that actions would not lead to success." The independent variables in MANOVA #3 were the same as in MANOVA #2. Thus, a two-way MANOVA, with triadic group and categorized ASQ as factors, was conducted. This analysis yielded a significant triadic group main effect, Wilks' Lambda = .577, equivalent to F (3,83) = 20.281, p = .0001 (Table 6) when the combined contribution of the three representative dependent variables was analyzed. Again, Group B subjects were found to be more helpless than Group A subjects, and no significant categorized ASQ main effect or interaction was obtained. Here, in MANOVA #3, the cognitive deficit was defined more as an "irrational expectancy" as opposed to a "slowed ability

to learn." In the learned helplessness literature, it has been defined both ways. In the present study, ER1-ER2 was designed primarily as a measure to assess perceived uncontrollability in the pretreatment. But, given that it explicitly assesses expectancy of test phase performance, using it as an "irrational cognition" measure in this model appears justified. If Group B subjects make a pessimistic judgement on their upcoming performance without being exposed to the actual contingencies, such a judgement might be considered a cognitive deficit. Modifying the model in this way actually produced a more robust basic effect, again confirming the hypothesis. But, again, attributional style did not appear to effect performance.

The conclusions drawn from the MANOVA findings are that (a) the basic learned helplessness effect was demonstrated, thus confirming the hypothesis, but that (b) personality features and attributional style did not influence this induction differentially as was predicted. An interesting finding was the presentation of "helplessness" symptoms in triadic Group C subjects--a finding that is addressed in greater detail later in the text.

Learned Helplessness Deficits/Disruptions

While several MANOVAs were employed to assess the overall learned helplessness effect, separate analyses were performed to pinpoint which of the representative dependent variables were predominately responsible for the MANOVA findings. In addition, other variables from the three deficit/disruption domains were analysed, and these analyses are presented

below.

Several two-way ANOVAs with triadic group and diagnostic group as factors were performed to assess the learned helplessness effects of perceived uncontrollability (i.e., affective disruptions, cognitive deficits, performance/motivational deficits). It was predicted that Group B subjects would demonstrate greater learned helplessness than Group A and C counterparts, and that OCPD would be more vulnerable to helplessness induction than AFC and NC.

Affective Disruptions

With regard to the overall affect change score on the MAACL (MAACL-Composite), a significant triadic group main effect was obtained, F (2,126) = 4.30, p = .0156 (Table 7). Triadic group means were as follows: Group A = 5.83, Group C = 11.4, Group B = 13.21. Group A and B were significantly different from one another based on Scheffe's test of multiple comparisons (Table 8), and Group A and C were significantly different from one another based on a more liberal Fisher's PLSD test of multiple comparisons (Table 8). Group B and C were more distressed overall than Group A but were not statistically different from one another. No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: OCPD = 9.57, NC = 14.6, AFC = 15.36.

With regard to the depression change score on the MAACL (D3-D1), a significant triadic group main effect was obtained, F (2,127) = 3.60, p = .03

(Table 7). Triadic group means were as follows: Group A = 2.13, Group C = 4.31, Group B = 5.18. Only Groups A and B were significantly different from one another based on Scheffe's test of multiple comparisons (Table 8). Group B was more depressed than Group A. No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: OCPD = 3.53, NC = 6.0, AFC = 6.07.

With regard to the anxiety change score on the MAACL (A3-A1), the F value approached statistical significance, F (2,127) = 2.97, p = .055 (Table 7). Triadic group means were as follows: Group A = 1.74, Group C = 3.11, Group B = 3.45. A significant triadic group effect was obtained, however, when the model used only triadic group as a factor, F (2,133) = 3.08, p = .0495. Only Groups A and B were significantly different from one another based on Fisher's PLSD test of multiple comparisons (Table 8). Group B was more anxious than Group A. No significant diagnostic main effect or interaction was obtained using the two-way ANOVA. The diagnostic group means of Group B subjects follow: OCPD = 2.93, AFC = 3.64, NC = 3.80.

With regard to the hostility change score on the MAACL (H3-H1), a significant triadic group main effect was obtained, F (2,127) = 4.16, p = .0178 (Table 7). Triadic group means were as follows: Group A = 1.96, Group C = 3.98, Group B = 4.43. Group A and B were significantly different from one another based on Scheffe's test of multiple comparisons (Table 8), and Group A and C were significantly different from one another based on Fisher's PLSD

test of multiple comparisons (Table 8). Group B and C were more hostile than Group A. No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: OCPD = 2.93, NC = 4.8, AFC = 5.64.

The above findings confirm the hypothesis that perceived uncontrollability produces affective disruptions, and thus, they support the basic learned helplessness effect. Interestingly, Group C subjects became quite distressed simply listening to the tones, however, less so than Group B. The prediction that OCPD Group B subjects would become relatively more distressed was not confirmed. However, an interesting diagnostic finding was that, within AFC, the percent change in anxiety for Group A subjects (M = 42% increase) was statistically greater than the percent change in anxiety for Group B subjects (M = 2% decrease) during the second half of the pretreatment using Fisher's PLSD test of multiple comparison, F (2, 42) = 2.211, p = .1222 (Table 9 for ANOVA and Table 10 for test of multiple comparison). Such a finding was not noted when the other two diagnostic groups were analyzed separately. This interesting finding is addressed in greater detail later in the text.

Cognitive and Performance/Motivational Deficits

SHUTTLEBOX MEASURES

With regard to NTC-Escape, a significant triadic group effect was obtained, F (2,127) = 5.35, p = .0059 (Table 11). Triadic group means were as

follows: Group A = 11.45, Group B = 12.07, Group C = 15.98. According to Scheffe's test of multiple comparisons, Group C was significantly different (i.e., slower to learn) than both Group A and B, but Group A and B, despite being in the predicted direction, were not significantly different from one another (Table 12). No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: AFC = 10.79, OCPD = 12.0, NC = 13.33. Interestingly, when the model was modified by excluding OCPD subjects, a significant interaction was obtained, F (2,85) = 3.319, p = .034 (Table 11). An inspection of the means reveals that, NC Group B subjects (M = 13.333), as predicted, and NC Group C subjects (M = 18.133) take longer to learn the criterion than their NC Group A counterparts (M = 9.188). However, the opposite is the case with regard to AFC. AFC Group A subjects actually take longer (M = 14.188) than their Group B (M = 10.786) and Group C (M = 13.867) counterparts. Thus, personality factors appear to interact with helplessness induction on this measure, albeit not in a manner predicted.

With regard to NF, a significant triadic group effect was obtained, F (2,126) = 5.39, p = .0057 (Table 11). Triadic group means were as follows: Group A = 8.6, Group B = 9.47, Group C = 13.53. According to Scheffe's test of multiple comparisons, Group C was significantly different (i.e., more failures) than both Group A and B, but, despite being in the predicted direction, Group A and B means were not significantly different from one another (Table 12).

No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: AFC = 8.54, OCPD = 9.73, NC = 10.0. Interestingly, when the model was modified by excluding OCPD subjects, the interaction approached statistical significance, $\underline{F}$ (2,84) = 2,513, $\underline{p}$ = .0871 (Table 11). An inspection of the means reveals that, NC Group B subjects ($\underline{M}$ = 10.00), as predicted, and NC Group C subjects ($\underline{M}$ = 15.667) fail more trials than their NC Group A counterparts ($\underline{M}$ = 6.5). However, with regard to the AFC Group A and Group B means, the opposite is true. AFC Group A subjects actually fail more trials ($\underline{M}$ = 11.00) than their Group B ($\underline{M}$ = 8.538) counterparts.

With regard to RT1, a significant triadic group effect was obtained, $\underline{F}$ (2,125) = 4.95, $\underline{p}$ = .0085 (Table 11). Triadic group means were as follows: Group A = 4.93, Group B = 5.2, Group C = 6.16. Group C was significantly different (i.e., slower reaction time) than Group A using Scheffe's test of multiple comparisons (Table 12) and significantly different than Group B using Fisher's PLSD test of multiple comparisons (Table 12). Group A and B were not statistically different from one another, but means were in the prediction direction. No significant diagnostic main effect or interaction was obtained. The diagnostic group means of Group B subjects follow: AFC = 4.43, OCPD = 5.25, NC = 5.81. Again, when the model was modified by excluding OCPD subjects, the interaction approached statistical significance, $\underline{F}$ (2,83) = 2.188, $\underline{p}$ = .1186 (Table 11). An inspection of the means reveals that, NC Group B

subjects' (M = 5.814 sec.), as predicted, and NC Group C subjects' (M = 5.901 sec.) reaction times are slower than their NC Group A counterparts (M = 4.946 sec.). However, with regard to the AFC Group A and Group B means, the opposite is true. AFC Group A subjects' reaction times are actually slower (M = 5.324 sec.) than their Group B (M = 4.431 sec.) counterparts.

With regard to MLI, no significant triadic group main effect, diagnostic main effect, or interaction was obtained (Table 11). Triadic group means were as follows: Group C = 3.69 sec., Group A = 4.3 sec., Group B = 4.32 sec.. The diagnostic group means of Group B subjects follow: NC = 4.1 sec., AFC = 4.26 sec., OCPD = 4.59 sec..

With regard to NTC-Avoid, no significant triadic group main effect, diagnostic main effect, or interaction was obtained (Table 11). Triadic group means were as follows: Group C = 20.56, Group B = 20.95, Group C = 21.77. The diagnostic group means of Group B subjects follow: AFC = 19.64, NC = 21.0, OCPD = 22.13.

With regard to NA, no significant triadic group main effect, diagnostic main effect, or interaction was obtained (Table 11). Triadic group means were as follows: Group A = 4.36, Group B = 4.81, Group C = 5.71. The diagnostic group means of Group B subjects follow: OCPD = 3.47, NC = 5.53, AFC = 5.54.

While the MANOVA results confirmed the learned helplessness basic effect by assessing the deficit/disruption domains collectively, the above shuttlebox measures, taken separately, yielded mixed results. Nevertheless,

upon inspection of the representative measures, NTC-Escape and RT1, a trend in the predicted direction was noted with Group B subjects demonstrating greater cognitive and performance/motivational deficits than Group A subjects. Such a trend is worthy of note given that it does, in fact, contribute to the significant MANOVA findings. Interestingly, however, Group C subjects performed in a manner significantly more "helpless" than either Group A or B on NTC-Escape, NF, and RT1. This finding was not predicted, but is intriguing and is addressed later in the text. The hypothesis of an OCPD vulnerability was not confirmed. However, an interesting diagnostic finding was that, while Group A and Group B NC subjects performed in the predicted direction on NTC-Escape, Group A and Group B AFC subjects performed in the opposite direction as was evidenced by a significant interaction (Table 11). A similar pattern of results was obtained on NF and RT1; however, on these measures the interactions only approached statistical significance. This interesting finding is discussed later in the text as well.

Recall from above, ER1-ER2 was also used as a cognitive deficit measure in MANOVA #3. Thus, the statistically significant expectancy rating finding noted above (Table 2) also supports the basic learned helplessness effect. An argument can be made that ER1-ER2 also might be used to represent a "self-esteem deficit." Recall that in the reformulated learned helplessness theory, in addition to the three traditional deficits/disruptions, a

fourth deficit--self-esteem--was added. While this is not always directly assessed in learned helplessness research, "lowered expectancy" in Group B subjects might be interpreted as a "lowered confidence in ability," thus adding further support to the basic effect. Note that self-esteem as measured by ER1-ER2 is a "state" measure subject to situational changes, whereas self-esteem assessed by the TSCS is a "trait" measure.

Trait Measures

Several two-way ANOVAs with triadic group and diagnostic group as factors were performed to detect any significant diagnostic, triadic, or interactive effects on the ASQ and TSCS. It was predicted that the personality disorder analogue groups, particularly OCPD, would self-report more depressive attributional styles. It was also predicted that AFC would demonstrate poor self-esteem relative to NC, but no specific hypothesis was made with regard to OCPD's self-esteem. It was predicted that no triadic effects or interactions would be obtained given that these are trait measures administered prior to the experimental treatment.

Attributional Style Questionnaire

With regard to ASQ--Negative Events, a significant diagnostic effect was obtained, F (2,127) = 9.18, p = .0002 (Table 13). Group means were as follows: NC = 3.75, OCPD = 4.1, AFC = 4.2. Both OCPD and AFC were statistically different, and thus, more "at-risk" for depression than NC, but were not statistically different from one another using Scheffe's test of

multiple comparisons (Table 14). No significant triadic group effect or interaction was obtained.

With regard to ASQ--Positive Events, a significant diagnostic effect was obtained, F (2,127) = 6.09, p = .003 (Table 13). Group means were as follows: AFC = 5.06, OCPD = 5.35, NC = 5.47. AFC was statistically different, and more "at-risk" for depression, than NC using Scheffe's test of multiple comparison (Table 14), and AFC was statistically different, and more "at-risk" for depression than OCPD, using Fisher's PLSD test of multiple comparisons (Table 14). OCPD and NC were not statistically different from one another based on either test of multiple comparisons. No significant triadic group effect or interaction was obtained.

With regard to ASQ--Composite, a significant diagnostic effect was obtained, F (2,127) = 12.47, p = .0001 (Table 13). Group means were as follows: NC = -1.72, OCPD = -1.25, AFC = -.85. These means are derived by subtracting the ASQ-Positive score from the ASQ-Negative score. These means are less than zero because, for all three diagnostic groups, the ASQ-Positive Events mean was greater than the ASQ-Negative Events mean. With regard to ASQ-Composite, OCPD and AFC were statistically different, and more "at-risk" for depression, than NC, but not statistically different from one another using Scheffe's test of multiple comparisons (Table 14). They were statistically different from one another, however, using the more liberal Fisher's PLSD test of multiple comparisons (Table 14). Using this comparison, AFC was

more "at-risk" for depression than OCPD. No significant triadic group effect or interaction was obtained.

Inspection of the ASQ subscales revealed the following significant comparisons: (a) AFC was determined to attribute more internal reasons to negative events, F (2,127) = 5.26, p = .0064 (Table 15 for ANOVA and Table 16 for test of multiple comparisons) and to be more unstable with regard to their attributions for positive events, F (2,127) = 4.57, p = .0121 (Table 15 for ANOVA and Table 16 for test of multiple comparisons) than both NC and OCPD, (b) OCPD's attributions were determined to be more global than NC with respect to achievement events regardless of whether the event was positive or negative, F (2,127) = 2.77, p = .0662 (Table 15 for ANOVA and Table 16 for test of multiple comparisons), and (c) personality analogues (i.e., OCPD, AFC) were determined to make more external attributions for positive events, F (2,127) =7.16, p = .0011 (Table 15 for ANOVA and Table 16 for test of multiple comparisons) and more global attributions for negative events than NC, F (2,127) = 6.72, p = .0017 (Table 15 for ANOVA and Table 16 for test of multiple comparisons). No significant triadic group effects or interactions were obtained on any of the subtest analyses highlighted above.

Tennessee Self-Concept Scale

With regard to TSCS--Total Positive, a significant diagnostic effect was obtained, F (2,126) = 10.161, p = .0001 (Table 17). Diagnostic group means were as follows: NC = 367.22, OCPD = 364.80, AFC = 343.89. AFC was determined to

be statistically different (i.e., possessing lower self-esteem) than NC and OCPD using Scheffe's test of multiple comparisons (Table 18). No significant triadic group effect or interaction was obtained.

Consistent with past research, a significant Pearson's product moment correlation coefficient was obtained when ASQ--Composite was correlated with TSCS--Total Positive, r (136) = -.461, $p < .001$). This indicates that at-risk attributional styles are associated with low self-esteem.

Overall, the trait results suggest that the two analogue groups, as predicted, are more susceptible to a learned helplessness depression based on ASQ, but that OCPD are no more "at-risk" for depression than AFC as was hypothesized. In fact, with regard to ASQ--Composite, AFC are significantly more vulnerable than OCPD using the liberal Fisher's PLSD test of multiple comparisons. As was mentioned above, however, neither diagnosis nor attributional style predicted actual performance during the two-phase helplessness procedure. Also, AFC subjects were found to have lower self-esteem than NC, as predicted, and lower self-esteem than OCPD.

Mood Progression

Recall, it was predicted that, within Group B subjects, a mood progression from perceived uncontrollability to anxiety to depression would be noted. This progression would serve as an experimental microcosm of the mood progression that is purported to occur in the development of a learned helplessness depression.

During the first half of the pretreatment phase (Block 1), Group B subjects' anxiety affect increased 75% as compared to a 48% increase in depression affect (i.e., Change in Anxiety: M A2 - M A1/ A1 M; Change in Depression: M D2 - M D1/
M D1). During the second half of the pretreatment phase (Block 2), Group B subjects' anxiety affect increased 4% as compared to a 7% increase in depression affect.

Note that most affect change took place in Block 1. Nevertheless, anxiety increased more than depression in Block 1, and depression increased more than anxiety in Block 2. Despite these descriptive statistics, the percent increase in anxiety relative to depression in Block 1 and the percent increase in depression relative to anxiety in Block 2 was not statistically significant when paired t-tests were used; Block 1: t (43) = .376, p = .3543 (one-tailed), Depression M = 159%, Anxiety M = 177%; Block 2: t (43) = .518, p = .3035 (one-tailed), Depression M = 7%, Anxiety M = 11%. Note, in fact, that the means in Block 2 are opposite of the descriptive statistics and predictions (i.e., in Block 2, Group B subjects became relatively more anxious than depressed although this difference was not statistically significant).

Given that Group C reported significant subjective distress during the pretreatment relative to Group A subjects, their mood progression was also investigated. An inspection of the means revealed that, like Group B subjects, Group C subjects' anxiety increased more during Block 1 relative to their

depression, and, consistent with predictions, their depression increased more during Block 2 relative to their anxiety. Again, neither t-test used to make the comparision yielded a statistically significant difference; however, the anxiety/depression difference during Block 1 approached statistical significance, Block 1: t (44) = 1.555, p = .0636 (one-tailed), Depression M= 73%, Anxiety M= 113%; Block 2: t (44) = -.473, p = .3193 (one-tailed), Depression M = 28%, Anxiety M = 23%.

Hence, while an inspection of the means suggested that anxiety increased more than depression in Block 1 for both Group B and Group C, Group C data were more consistent with the overall directional predictions of the mood progression hypothesis. Overall, the above findings provide modest support for the mood progression hypothesis.

Post-Experiment Questionnaire Analyses

A debriefing questionnaire was administered to tap certain exploratory "process" variables that were hoped would clarify predicted and unpredicted results. One question asked was, "in the first part of the experiment, what strategy did you use to turn off the noise?" It was clear based on informal inspection of the verbal reports of Group A subjects that they had, in fact, discovered the required pattern of key presses responsible for terminating the noise. Group B subjects reported that they had attempted many strategies, but had failed to discover the correct one.

Another question asked was "how confident are you that your strategy stopped the noise in the first part of the experiment?" Subjects were asked to rate their response on a scale from 0 (not confident at all) to 10 (very confident). A two-way ANOVA with triadic group and diagnostic group as factors yielded a significant triadic group effect, $F(1,85) = 340.89$, $p = .0001$ (Table 19a). No significant diagnostic effect or interaction was obtained. Group A subjects ($M = 8.72$) produced significantly higher confidence ratings than Group B subjects ($M = 1.13$), lending further support to the hypothesis that Group B subjects would perceive uncontrollability. Group C subjects were not administered the pretreatment task, and thus, were not asked to rate the above question.

Subjects were also asked to rate the unpleasantness of the tone during the first part of the experiment (i.e., Block 1 and 2) on a scale from 0 (not unpleasant at all) to 10 (extremely unpleasant). A two-way ANOVA with triadic group and diagnostic group as factors yielded no significant triadic group effect, diagnostic group effect, or interaction (Table 19b). Triadic group means were the following: Group A=5.29, Group B=5.09, C=5.16. Diagnostic group means were the following: OCPD=4.80, AFC=5.47, NC=5.28). Therefore, subjects' subjective experience of the tone during the pretreatment phase was not significantly influenced by their triadic group or diagnostic group assignment. It also should be mentioned that no triadic group effect, diagnostic group effect, or interaction was found using a two-way ANOVA to

analyze actual decibel level selection of subjects (Table 19c).

Also, with regard to the pretreatment phase, two-way ANOVAs with triadic group and diagnostic group as factors were conducted on ratings (i.e., 0 to 10) of the following post-experiment questions: 1) "how important was it for you to figure out the strategy?," 2) "how hard did you try to stop the noise?" and 3) "how well would you expect to do if you were asked to do the same task again?" Group A subjects rated their pretreatment performance as more important than Group B subjects, $\underline{F}$ (1,84) = 23.28, $\underline{p}$ = .0001; Group A $\underline{M}$ = 7.59; Group B $\underline{M}$ = 5.33 (Table 19d). And Group A reported that they thought they would do better on the task compared to Group B subjects were it to start up again, $\underline{F}$ (1,84) = 254.00, $\underline{p}$ = .0001; Group A $\underline{M}$ = 8.96; Group B $\underline{M}$ = 2.54 (Table 19e). No triadic group effect was found on the question regarding effort (Table 19f). And no diagnostic group effect or interaction was noted on any of the above ratings.

Identical questions were asked in regard to subjects' performance on the test phase task. Given that Group C subjects were administered the same test phase task, they were asked to rate the following questions as well. No diagnostic or triadic group differences were noted with regard to confidence in strategy ratings (Table 20a), effort ratings (Table 20b), or expectancy ratings (Table 20c). However, Group B subjects judged the tone to be less aversive than Group A subjects using Fisher's PLSD test of multiple comparisons, $\underline{F}$ (2,133) = 2.37, $\underline{p}$ = .09; Group A $\underline{M}$ = 5.77; Group B $\underline{M}$ = 4.636, Group C $\underline{M}$ = 5.07

(Table 20d for ANOVA and Table 21 for test of multiple comparisons). Also, Group A subjects rated their performance as more important relative to Group B subjects, using Fisher's PLSD test of multiple comparisons, F (2,132) = 2.55, p = .08; Group A M = 7.76; Group B M = 6.77 (See Table 20e for ANOVA and Table 21 for test of multiple comparisons).

In addition, two-way ANOVAs with triadic group and diagnostic group as factors were conducted on ratings of subjects' attributions for Block 3 failures: (a) lack of ability (Table 20f), lack of effort (Table 20g), or the impossibility of the task (Table 20h). Furthermore, two-way ANOVAs were conducted on the ratings of the following questions: "how well did you think you would be able to stop the noise when you first saw the shuttlebox appear on the screen?" (Table 20i), "how important was it for you to make a good impression on the experimenter?" (Table 20j), and "how interesting was the entire experiment to you?" (Table 20k). Of these comparisons, only the following were statistically significant: Group C demonstrated greater confidence than Group B, using Fisher's test of multiple comparisons, with regard to being able to stop the noise when first exposed to the shuttlebox, F (2,133) = 2.26, p = .11; Group A M = 6.00; Group B M = 5.43, Group C M = 6.29 (Table 20i for ANOVA and Table 21 for test of multiple comparisons). Group A was more likely than Group B, using Fisher's test of multiple comparisons, to attribute failures to their inability, F (2,124) = 2.689, p = .0719; Group A M = 7.35; Group B M = 6.00, Group C M = 6.93 (Table 20f for ANOVA and Table 21

for test of multiple comparisons). AFC subjects were more likely than NC subjects, using Fisher's test of multiple comparisons, to attribute their failures to their lack of effort, F (2,126) = 1.966, p = .1443; AFC M = 3.82; NC M = 2.69, OCPD M = 3.16 (Table 20g for ANOVA and Table 21 for test of multiple comparisons). Group A was more likely than Group B, using Fisher's test of multiple comparisons, to report that they found the experiment interesting, F (2,127) = 2.323, p = .1021; Group A M = 6.79; Group B M = 5.86, Group C M = 6.36 (Table 20k for ANOVA and Table 21 for test of multiple comparisons). Finally, subjects were asked if they knew the specific purpose and hypotheses of the study, and if they did, to elaborate. While some subjects offered hypotheses that approximated that of the study's, none was specific enough, according to an informal inspection of subjects' written responses, to suggest that the results were compromised.

Ancillary Analyses

The following analyses were conducted post hoc to provide support for arguments made in the discussion section. They are presented here without elaboration. Their purpose and contribution is clarified later in the text.

A one-way analysis of variance revealed that OCPD Group B subjects' percent increase in depression during Block 2 (M = -1 %) was less than NC Group B subjects' percent increase in depression (M = 16 %) using Fisher's PLSD test of multiple comparison, F (2,41) = 2.374, p = .1058 (Table 22 for ANOVA and Table 23 for test of multiple comparisons).

A one-way analysis of variance on BDI scores with diagnostic group as the factor was statistically significant, F (2,133) = 8.44, p = .0004 (Table 24). AFC subjects were determined to be significantly more depressed (M = 4.93) than NC (M = 2.07) and OCPD (M = 2.69) using Scheffe's test of multiple comparisons (Table 25).

A one-way analysis of variance on ER1 with diagnostic group as the factor (OCPD excluded from the model) approached statistical significance, F (1,89) = 3.743, p = .0562 (Table 26). Diagnostic group means follow: AFC M = 6.222; NC M = 6.848.

A one-way analysis of variance on MAACL-Composite with triadic group as the factor (only AFC subjects included) was statistically significant, F (2,42) = 3.387, p = .0433 (Table 27). AFC Group A subjects were determined to be significantly less distressed (M = 3.938) than AFC Group B subjects (M = 15.357) using Scheffe's test of multiple comparisons (Table 28).

A one-way analysis of variance on MAACL-Composite during Block 2 only with triadic group as the factor approached statistical significance, F (2,133) = 2.99, p = .0537 (Table 29). Group C subjects were determined to be significantly more distressed (M = 4.93) than Group B counterparts (M = 2.02) using Fisher PLSD test of multiple comparison (Table 30).

CHAPTER IV

DISCUSSION

Overall Interpretation of the Analyses

Consistent with past research, the basic learned helplessness effect was demonstrated. Also, OCPD analogues were found to possess a more at-risk attributional style based on the ASQ than normal controls, which is suggestive of a vulnerability to perceived uncontrollability. This finding supports the hypothesis that OCPD might be identified as an at-risk group. However, their attributional style was less at-risk for depression than their AFC counterparts'. Thus, it can be concluded that the Anxious-Fearful Cluster of personality disorders taken collectively (i.e, avoidant, dependent, passive-aggressive and obsessive compulsive) are more at-risk for depression than normal controls based on their attributional styles.

Attributional style, however, did not predict behavior in the actual experiment. Neither OCPD nor AFC demonstrated greater learned helplessness deficits/disruptions than normal controls. OCPD responded similarly to NC (i.e., Group B subjects demonstrated deficits/disruptions relative to Group A). However, an interesting and perplexing diagnostic effect was that AFC responded in a manner opposite of what is predicted based on learned helplessness theory and past research. Another interesting finding

was that, Group C, overall, differed significantly from Groups A and B on the shuttlebox measures, demonstrating a more pronounced helplessness response topography than either of the other triadic groups. This was not predicted, but is not altogether unexpected for reasons that will be addressed below. While not a central component of the study, modest support for the mood progression hypothesis that perceived uncontrollability leads to anxiety, which, in turn, results in depression was provided.

Explanations for Obtained Findings

Diagnostic Findings: OCPD

While OCPD and NC Group B subjects became helpless overall, the effect was actually less pronounced among OCPD subjects based on an inspection of the means. Interestingly, while NC Group B subjects reported an increase in depression from MAACL2 to MAACL3, OCPD Group B subjects reported a decrease in depression (Table 22 and 23). This finding, coupled with the fact that the differences between OCPD Group A and Group B means on NTC-Escape, NF, and RT1 were less than the differences between NC Group A and Group B means on the same measures (Table 31), suggests that personality features of OCPD analogues (e.g., goal-orientation, perseverance, emotional constriction) actually facilitated shuttlebox performance of OCPD Group B subjects relative to NC Group B subjects and served as a "coping mechanism" against affective disruption (i.e., depression) in the present study. The obtained findings are more consistent with the Thornton (1982)

and Wittenborn and Maurer (1977) arguments made earlier which suggested that individuals similar to OCPD might be resistent to helplessness induction.

Perhaps the two hypotheses--OCPD as vulnerable (i.e., as suggested by the ASQ findings) versus OCPD as resistant (i.e., as suggested by the experimental findings) can be reconciled. Perhaps there exists a linear relationship between degree or duration of perceived uncontrollability and degree of depression. And perhaps, within OCPD, the manifestation of obsessive compulsive behavior varies as a function of the degree and/or duration of perceived uncontrollability. It has been argued that OCPDs engage in their characteristic manner to avoid aversiveness. In certain environmental contexts (e.g., where the degree of perceived uncontrollability is at a minimum or the duration of perceived uncontrollability is short), perhaps OC-like behavior is at a minimum. In such an environment, depression is not induced and employing such a defense is not adaptive. However, in a slightly more "helpless" environment (i.e., greater duration, greater degree of perceived uncontrollability), OC-like behavior is utilized, and is successful in warding off the possible onset of depression. Such an environment may be representative of the present study's environment. In yet a more "helpless" environment, it is conceivable that OC-like behavior is at a maximum but is unsuccessful in warding off a depression. Here, the defense is utilized but is not effective. Finally, in a highly "helpless" environment, the OC-like behavior is perhaps extinguished and depression is

established. The last scenario appears to be more consistent with the learned helplessness conceptualization and the predictions made in the present study. Thus, in loose terminology, the environment in force (i.e., which influences the degree and/or duration of perceived uncontrollability) will influence the topography of the personality/depression relationship. The present study, of course, does not test experimentally this relationship. And, as proposed here, such a hypothesis is only speculative. Yet, there is precedence for such speculation.

Pittman and Pittman (1979), in their study which investigated the effects of differing degrees of helplessness training and locus of control on mood and performance, found that individuals with internal loci of control exhibited greater learned helplessness symptoms in high helplessness situations (i.e., noncontingent feedback was given for each pretreatment trial) than individuals with external loci of control. Furthermore, in low helplessness situations (i.e., no feedback was given on some pretreatment trials and noncontingent feedback was given on others), internals performed in a less helpless manner than controls. Thus, in Pittman and Pittman's study, a personality variable--locus of control--and degree of helplessness induction interacted with one another. Given that an internal locus of control is a defining feature of obsessive compulsive personality disorder, Pittman and Pittman's findings are quite applicable to the present study and consistent with the relationship proposed above.

If such a relationship between OCPD and learned helplessness exists, a behavioral account might implicate extinction as the mechanism responsible for both "resistance" and "vulnerability." It is perhaps easy to implicate extinction as being responsible for the latter response topography, but more difficult to see its role in the former topography. According to the definition of extinction, when positive reinforcement (i.e., controllability) is withheld over time, previously reinforced behavior decreases (i.e., extinguishes). Such an effect on the behavioral repertoire may be analogous to "becoming depressed." Yet, the "resistance" topography predicted in this curvilinear relationship also might be explained by extinction. Specifically, perhaps the "overcompensation" described by Thornton (1982) and the "intensification of symptoms" described by Wittenborn and Maurer (1977) are analogous in some respects to the extinction burst phenonemon where the initial effect of reinforcement loss is an increase in the previously reinforced behavior. This OCPD/helplessness relationship is interesting and plausible and should be addressed experimentally in future research (See Appendix I for a schematic representation of this relationship). While the present results suggest either no vulnerability or a resistance to learned helplessness in OCPD, until further research examines the possibility of the relationship proposed above, no definitive statement can be made with regard to the relationship between OCPD and learned helplessness.

Diagnostic Findings: AFC

One potential explanation for the unusual findings that AFC performed opposite of the basic learned helplessness effect is that their performance on the shuttlebox task had something to do with their pre-experimental emotional state. While subjects with serious depression were screened out of the study, AFC subjects were significantly more depressed than OCPD and NC subjects based on the BDI prior to testing (Table 24 and Table 25). This is interesting in light of the Miller and Seligman (1975) study where clinically depressed subjects performed somewhat differently than normal controls in a helplessness induction scenario. If such a pre-experiment "state" was influential in the present study, it influenced shuttlebox performance only (i.e., AFC MAACL change scores and expectancy ratings were in line with predictions).

Perhaps AFC subjects exhibit unique personality characteristics or "traits" that were responsible for this unusual finding. AFC subjects were, in fact, determined to have significantly lower "trait" self-esteem than NC and OCPD subjects prior to the experiment. And the literature describes AFC subjects as feeling incompetent, helpless, and weak as opposed to OCPD and NC who have a more positive self-image. Perhaps such personality characteristics influenced the way AFC subjects perceived uncontrollability, responded to failure, and explained their failures. If this is true, then it is clear that attributional style alone is inadequate to predict vulnerability.

Perhaps personal control is deemphasized in AFC subjects as compared to NC and OCPD subjects, thus, leading AFC subjects to respond in such an unexpected manner. The high co-morbidity between AFC and depression is well-documented in the literature; however, loss of personal control may not contribute significantly to this demonstrated high comorbidity. In fact, the specificity hypothesis briefly addressed in the introduction argues that subjects valuing autonomy and control (i.e., OCPD) are more likely to become depressed when faced with achievement failure and uncontrollability; whereas subjects who value social approval and attention (e.g., dependent personality disorder) are more likely to become depressed when faced with social loss. Thus, mechanisms other than perceived uncontrollability may be more responsible for inducing depressions in AFC. This might explain AFC Group B's resistance to helplessness induction, but it does not fully address the fact that AFC Group A subjects performed worse than Group B subjects on the shuttlebox measures.

Perhaps AFC Group A subjects entered the experimental situation with low expectations about their performance due to their low self-esteem. In fact, inspection of baseline expectancy ratings supports this contention; however, the difference between AFC and NC means only approached statistical significance (Table 26). Furthermore, assume that AFC Group A subjects' immediate success on the pretreatment task was at odds with their pessimistic expectations, and, thus they became more "anxious and fearful" of eventual

failure. Perhaps this anticipation of failure had a debilitating effect on performance during the test phase. Given that their baseline was "success," they may have believed that they had "everything to lose and nothing to gain." And so when faced for the first time with failure in the test phase, they became relatively more distressed than their Group B counterparts, and this distress translated into poorer shuttlebox performance. If this hypothesis is true, one would expect this "anxious-fearful" affective state to be reflected in the pretreatment mood measures. Upon first glance, the MAACL data do not appear to support such a hypothesis. Overall, AFC Group A is significantly less distressed according to MAACL-Composite than AFC Group B during the pretreatment (Table 27 and Table 28). However, Group A is less anxious than Group B in Block 1 of the pretreatment, but <u>becomes significantly more anxious</u> (i.e., percent increase) from MAACL2 to MAACL3 than Group B. Interestingly, Group B actually experiences a mild <u>reduction</u> in anxiety during the second half of the pretreatment (Table 9). This "acceleration" in AFC Group A affect is interesting and seems consistent with the above speculation.

Perhaps Group B also entered the experimental situation with low expectations about their performance. Their failure on the pretreatment task, however, was consistent with their expectations, and thus they easily accepted the failure and emotionally habituated to it. Given that their baseline was "failure," they may have believed that they had "nothing to lose and everything to gain." And so, when faced with failure for the first time in the

test phase, they were less distressed than their Group A counterparts, and this relatively calm affective state translated into better shuttlebox performance. Again, the fact that AFC Group B subjects became less anxious than AFC Group A subjects in Block 2 suggests that their affective state was "decelerating" which is consistent with the above hypothesis.

Thus, in contrast to OCPD and normal controls, it could be argued that controllable scenarios are actually more aversive and that uncontrollable scenarios are actually less aversive to AFC individuals. Thus, perceived uncontrollability might impact AFC differently than the other diagnostic groups. Again, AFC's low self-esteem, pre-experimental affective state, deemphasis on personal control, and/or other personality characteristics may account for this finding. Obviously, further research is needed to examine these hypotheses.

Triadic Group C Dilemma

While the basic effect was demonstrated between Group A and B in the present study (i.e, greater helplessness in Group B), the findings that Group C (a) demonstrated even significantly greater deficits than Groups A and B on NTC-Escape, NF, and RT1, (b) generated significantly lower expectancy ratings than Group A, and (c) became significantly more affectively distressed than Group A during the pretreatment phase, all beg for an explanation.

Group C as an Experimentally Helpless Group

The MAACL disruptions and lowered expectancy ratings in Group C subjects suggest that something clearly happened to these subjects during the pretreatment phase. They became affectively distressed (i.e., even significantly more than Group B subjects in Block 2 (Table 29 and Table 30) and became significantly less confident about their upcoming performance in Block 3 (Table 2 and Table 3). While it can be argued that Group C subjects were not asked to terminate the noise and, therefore, did not perceive uncontrollability, an alternative interpretation may be made. Perhaps Group C, like Group B, was, indeed, made experimentally helpless during the pretreatment phase. While their "perceived uncontrollability" was never assessed directly, an argument can be made that their pretreatment experience was response-independent and that their deficits/disruptions noted in Block 3 were functionally, as well as, topographically similar to learned helplessness deficits/disruptions.

By definition, an aversive stimulus is one whose termination is negatively reinforcing. The layman's definition of "aversion," according to Random House College Dictionary (Stein, 1975) is "a strong desire to avoid because of dislike; repugnance; a turning away or preventing (p. 93)." In the present study, Group C subjects selected and were administered an aversive tone and, like Group B subjects, and unlike Group A subjects, had no means to control the tone. If a stimulus is intense enough to warrant the label

"aversive" and if the underlying assumption is that the stimulus' termination is reinforcing, then a logical deduction is that subjects who are not given an opportunity to terminate the stimulus would, in fact, "perceive uncontrollability." Support for this perceived uncontrollability in Group C comes from their verbal reports assessed by the post-experiment questionnaire. Group C subjects were asked to generate thoughts that they had during the pretreatment while "simply sitting and listening to the tones." Not surprisingly, many subjects reported that the experience was unpleasant, that they had attempted to "figure the experiment out" despite not being asked to do so, that they they wanted to leave, and that they wondered how much longer they would have to endure the tones. While no formal analyses were conducted on these verbal reports, they imply a "perception" of aversiveness and a "desire" for avoidance.

If Group C was made experimentally helpless in the present study, one would expect that previous research would have obtained similar findings. However, recall that the present procedure, like Hiroto and Seligman's (1975) procedure, differed somewhat from many past triadic designs in that Group C was equated with Group A and B on habituation to the tone. It is being argued here that removing the aversiveness from the pretreatment situation by not exposing Group C to the tone, also may remove the perception of uncontrollability (i.e., there is nothing to control in the situation). As in the present study, Hiroto and Seligman (1975) found the basic helplessness effect

between Group A and B and also noted that their Group C's shuttlebox performance was more consistent with Group B than Group A. Group B subjects still demonstrated the greatest deficits of the three groups, but some unaddressed mechanism was responsible for Group C's deficits relative to Group A.

It could be argued that perceived uncontrollability is a function of the context in which it occurs. A context in which subjects are exposed to an aversive stimulus but are provided with a response-dependent opportunity to terminate the stimulus (i.e., Group A) may be less aversive and less likely to produce perceived uncontrollability than other contexts. A context in which subjects are exposed to an aversive stimulus but are provided with a response-independent opportunity to terminate the stimulus may be more aversive and more likely to produce perceived uncontrollability (i.e., Group B). However, a context in which subjects are exposed to an aversive stimulus but not even provided an opportunity to terminate the stimulus may be the most aversive and most likely to produce perceived uncontrollability (i.e., Group C). Intuitively, subjects exposed to the last scenario would be more likely to make an external attribution for the uncontrollability (i.e., Some outside force controls the tones) as opposed to the internal attributions made for failures by Group B subjects (i.e., I should be able to control the tones). While the reformulation predicts that internal attributional styles for negative events are associated with learned helplessness, past research (i.e.,

Hiroto, 1974) also demonstrates that external loci of control and failure under chance conditions are more likely to produce helplessness. Thus, the potential for such an external attribution producing helplessness deficits in a context similar to Group C's in the present study finds support in the literature. Interestingly, learned helplessness applications to actual social problems have often described helplessness induction scenarios similar to the Group C scenario being proposed here. For example, in their learned helplessness studies on noise pollution, Cohen, Evans, Krantz, and Stokols (1980) and Cohen, Evans, Krantz, Stokols, and Kelly (1981) reported that school children whose classrooms were exposed to uncontrollable noise produced by airplanes that regularly flew overhead performed more poorly in school than students who were not directly under the flight paths of airplanes. Like Group C subjects in the present study, these children were exposed to an aversive stimulus and were not provided with an opportunity to terminate the stimulus. Future studies should further investigate perceived uncontrollability in light of perceived opportunity to control outcomes.

One might argue that Group C's pronounced shuttlebox deficits were due to the fact that they were placed at a disadvantage by not being exposed to the pretreatment task. Several points, however, argue against such a differential exposure effect. First, one might expect that such an effect would influence diagnostic groups equally. Such was not the case. AFC Group C

subjects actually performed better on the shuttlebox task than Group A subjects who had mastered the key pressing strategy in the pretreatment phase (Table 11). Second, if a lack of exposure to keyboard manipulation was solely responsible for Group C's shuttlebox deficits, one would not expect to see the MAACL affective disruptions and lowered expectancy ratings during the pretreatment. Clearly, Group C subjects were adversely affected by the pretreatment in some manner. Exposing Group A and B to the pretreatment task presumably could improve Group A and B's performance on the test phase task, but denying Group C the same opportunity should not adversely affect Group C subjects' expectations or affective state. Third, Group A subjects were observed to press keys in Block 3 using a response set shaped up in Blocks 1 and 2 that actually hindered performance on the shuttlebox task. Specifically, in the pretreatment phase, subjects were required to press a single key multiple times whereas, in the test phase, subjects were required to press different keys in an alternating fashion. Thus, it could be argued that a "practice effect" might place Groups A and B, not at an advantage, but at a disadvantage due to the "set" established early on. Finally, most learned helplessness studies utilizing the triadic design, require that Group A and B subjects attempt to solve some type of task in the pretreatment while Group C is unengaged. One might argue that attempting to solve a task in the pretreatment phase influences subjects' performance in the test phase as a result of behavioral momentum (e.g., this gets them thinking about

strategies, keeps them alert, keeps them moving). However, if such an influence is a practice effect, then it is not specific to the present study. Taken together, the above counterarguments appear to rule out differential exposure to the pretreatment task as an explanation for the Group C finding.

Critique of the Study

A general criticism of the study might be that the computerized experimental task used in the present study may have induced "computer fear" in some subjects which may have accounted for the unusual diagnostic and triadic findings. However, given that subjects were randomly assigned to triadic groups within each diagnostic group and given that such a fear conceivably could be found in all three diagnostic groups, this criticism is not of great concern. Furthermore, there is precedence in the learned helplessness literature for utilizing computerized experimental tasks (e.g., Peterson et al., 1993). Other points that must be acknowledged when evaluating the present study are a) that, as in any experiment, the possibility exists that the predicted and unpredicted findings were the result of Type I and Type II errors, b) that the ecological validity of the study is debatable, c) that the methodological constraints placed on the study may have made it difficult to capture hypothesized results experimentally with regard to some predictions (e.g., OCPD vulnerability) and more likely to obtain hypothesized results with regard to other predictions (e.g., triadic group differences), and d) that the statistical power needed to demonstrate certain actual group differences may

have been inadequate.

Another possible limitation of the present study may have been the use of analogues. The high false positive selection rate of the SCID-II could have diluted the sample, making true distinctions undetectable. Furthermore, while psychopathology is on a continuum and the use of analogues is justified, the Janus-faced nature of personality may have made OCPD analogues more resistant than true obsessive compulsive personality disordered individuals to the effects of helplessness induction. However, utilizing this sample did not prevent detecting the vulnerablity marker assessed by the ASQ. Failing to detect differences in attributional style would have been predicted in addition to failing to detect differences on the other measures if using a heterogenous sample were determined to be problematic. Therefore, the use of analogues screened using the self-report version of the SCID-II is not viewed as major concern.

Overall, the present study is valuable in that it raises important questions and concerns about learned helplessness research and theory. It is argued, here, that continued healthy debate and input from fresh, alternative perspectives would at the very least correct flaws and improve the status of learned helplessness as a viable theory of depression (See Appendix J for an alternative perspective on learned helplessness).

Implications for Treatment and Prevention

A major focus of the present study was to answer Peterson and Seligman's (1984) call to identify certain groups "at-risk" for depression. While the study identified the anxious-fearful cluster as a whole as being vulnerable based on the ASQ, the experimental findings did not. Nevertheless, the future identification of such groups has important treatment and prevention implications. For example, if treating a depressed client with certain personality features known to make one at-risk to helplessness induction, certain therapeutic strategies might be indicated. One general strategy might be to help instill or reinstill a sense of control or response- dependence in an individual. Treatments such as Beck's (1976; 1979) cognitive therapy, Ellis' (1973) rational-emotive therapy, or reattributional or expectancy training therapies (e.g., Hollon & Garber, 1980) are therapeutic approaches whose functional mechanisms seem consistent with the "deficit reversal" strategy in the learned helplessness literature. For example, if the actual contingencies in the client's environment are response-dependent but the client "perceives uncontrollability," this irrational cognition could be put to the test and eventually rejected, just as Seligman's dogs were dragged to the opposite compartment of the shuttlebox and forced to experience the actual contingencies. However, in situations where "perceived uncontrollability" is an accurate reflection of at least some of the environmental contingencies, alternative strategies may be indicated. For

example, the helpless client exposed to truly uncontrollable events (i.e., terminal cancer) might be helped to discriminate between those situations that are uncontrollable and those that are controllable. In such a treatment plan, those uncontrollable events might be addressed by having the client learn new behaviors or coping mechanisms that could help him or her accept certain inevitabilities (i.e., death). Also, in such a treatment plan, highlighting and capitalizing upon certain response-dependent outcomes (i.e., engaging in behaviors that may influence quality of life) could reinstill a sense of control and lessen the debilitating effects of helplessness.

The Group C finding in the present study suggests that individuals who are exposed to aversive stimulation but are not given an opportunity to control this stimulation may become helpless in a manner similar to Group B subjects. Therefore, it follows that the above treatments would be equally applicable to clients whose depression is acquired in a context similar to the Group C context. For example, similar measures could be used to address the depression in a child who has experienced a series of deaths or the depression in an adult who lives in a violent, crime-ridden neighborhood.

With regard to treating the obsessive compulsive, whose problem is not with a deficit in ego-strength or a lack of a response independent world view, but instead, with such "excesses," measures could be taken to deemphasize the importance placed on control, autonomy, and achievement, so that when perceived uncontrollability occurs (which inevitably it will),

such individuals will be better prepared for such an event. Such measures might include attributional retraining, cognitive therapy, and systematic desensitization to aversive uncontrollable situations.

With regard to treating the obsessive compulsives' counterparts in the anxious-fearful cluster, the findings in the present study suggest that they may benefit from treatment designed to target their low self-esteem and, in contrast to obsessive compulsives, target their avoidance and perceived aversiveness of situations where personal control is required. As with obsessive compulsives, attributional retraining, cognitive therapy, and systematic desensitization may be indicated. However, unlike obsessive compulsives, their desensitization might target aversive controllable situations.

If a learned helplessness mechanism does, in fact, come into play in the personality disorder/depression relationship, theoretically, preventive measures could be employed to block the onset of a depression in a personality disordered individual. Or better yet, these measures could be extended to construct an environment that prevents the development of certain personality features which might render one susceptible to a learned helplessness depression. How one might employ such preventive measures in the lives of individuals has been well-documented in the literature (e.g., Seligman, 1975; Peterson & Seligman, 1984). Just as treatment interventions might become more effective by instilling "controllability" or "enhanced

perception of response-dependence," so too might prevention strategies. Constructing an environment, for example, for a child, so as to promote "mastery" over one's world, might in a sense, immunize the child against a potentially detrimental learned helplessness depression in the future. This "mastery," according to Seligman (1975), may facilitate ego-strength. Or, again, in the case of the obsessive compulsive personality disordered individual who demonstrates excessive ego-strength, deemphasing personal control while maintaining a healthy world view of response-dependence might prevent the onset of a depression resulting from "perceived uncontrollability."

Thus, depending on the unique personality of the individual, different strategies might be indicated in preventing the onset of depression or treating the depression once established. In conclusion, further research designed to clarify the relationship between personality disorders and depression is needed. Such research endeavors may continue to help alleviate and prevent the suffering of individuals.

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APPENDIX A

Schematic Representation of the Triadic Design

	GROUP A	GROUP B	GROUP C
	|	|	|
	|	|	|
Pretreatment Phase	Response-Dependent Task A	Response-Independent Task A **	No Task
	|	|	|
	|	|	|
Test Phase	Response-Dependent Task B	Response-Dependent Task B	Response-Dependent Task B

** Helplessness Induction

APPENDIX B

Tables

Table 1

SCID-II Profiles of Participants

Acronym	Cutoff*	Personality Disorder Scale
AVD	(4)	Avoidant
DEP	(5)	Dependent
O-C	(5)	Obsessive Compulsive
P-A	(5)	Passive Aggressive
SDT	(5)	Self-Defeating (Scale not used)
PAR	(4)	Paranoid
STY	(5)	Schizotypal
SZD	(4)	Schizoid
HIS	(4)	Histrionic
NAR	(5)	Narcissistic
BRD	(5)	Borderline
ASL	<18 (4) and >18 (4)	Antisocial

* Scores at or above cutoff meet scale criterion

S	AVD	DEP	O-C	P-A	SDT	PAR	STY	SZD	HIS	NAR	BRD	ASL
A01	3	5	4	2	3	1	3	1	2	4	1	0
B01	4	3	1	1	0	1	2	3	1	4	2	0
C01	3	1	2	5	1	3	3	2	2	4	4	0
A02	5	3	3	3	4	3	2	4	2	3	3	0
C02	6	3	3	2	1	1	3	2	3	6	3	2
A03	1	5	3	0	3	2	4	1	3	2	3	1
B03	4	4	3	3	5	1	4	1	2	4	3	1
C03	7	6	4	2	1	1	2	2	0	2	1	0
A04	5	5	4	5	5	2	3	2	0	3	4	1
B04	2	6	4	2	1	0	2	2	1	2	2	3
C04	2	3	4	5	3	2	3	1	3	3	4	0
A05	4	2	1	0	0	2	1	1	0	2	3	0
B05	2	5	2	5	2	3	4	3	0	1	1	0
C05	7	5	4	3	4	3	4	2	2	4	4	0
A06	4	1	1	3	1	1	2	1	2	3	0	0
B06	4	5	2	1	2	0	3	2	1	1	3	0
C06	3	5	2	3	2	1	2	1	3	3	3	0
A07	3	8	2	4	4	1	3	2	3	4	4	0
B07	1	5	4	3	2	2	2	1	3	2	2	0
C07	3	2	3	7	3	3	3	2	3	3	2	0
A08	2	5	4	2	2	3	2	1	2	2	4	0

S	AVD	DEP	O-C	P-A	SDT	PAR	STY	SZD	HIS	NAR	BRD	ASL
B08	0	2	4	5	3	3	3	2	3	3	0	1
C08	7	7	4	5	3	3	5	3	3	4	4	0
A09	5	1	3	2	1	2	2	2	2	3	2	1
B09	4	2	4	3	1	3	2	1	4	3	0	0
C09	4	1	4	2	2	2	2	2	1	2	4	0
A16	0	2	4	3	3	2	4	1	2	3	1	0
B16	0	2	4	0	3	0	1	1	2	3	1	0
C16	1	1	4	1	1	0	3	3	2	3	0	0
A17	0	2	3	0	1	0	0	1	0	2	2	0
B17	0	3	4	2	2	1	2	2	1	2	2	0
C17	2	2	1	1	1	0	1	2	2	2	1	0
A18	0	2	2	2	3	3	2	1	3	2	1	0
B18	1	3	4	2	2	1	2	1	2	2	3	0
C18	3	0	1	3	3	0	3	1	1	2	0	0
A19	0	1	2	2	1	2	2	1	3	4	1	0
B19	2	2	4	2	3	2	2	3	2	1	1	0
C19	2	2	3	2	1	2	0	1	2	2	1	0
A20	2	0	2	0	2	1	3	2	2	1	0	0
B20	0	2	2	2	1	1	0	0	2	.	2	2
C20	0	1	1	0	1	1	2	1	2	3	2	.
A21	0	1	0	3	3	1	1	1	1	2	4	1
B21	2	2	3	0	3	3	1	3	2	3	2	0
C21	0	3	4	2	2	2	2	0	2	2	3	0
A22	0	0	2	1	0	3	1	1	1	1	1	0
B22	0	2	1	0	1	0	1	1	1	3	0	0
C22	0	1	3	1	1	2	3	0	0	2	3	0
A23	0	0	1	0	1	0	0	1	2	1	0	1
B23	0	1	2	3	0	0	0	0	0	0	0	0
C23	0	0	4	2	1	2	3	0	2	3	1	0
A24	0	0	0	0	0	0	0	0	1	2	0	1
B24	1	1	4	1	0	0	1	2	1	1	0	0
C24	1	0	3	3	1	3	2	2	3	4	4	1
A25	2	3	2	1	2	0	1	2	2	2	1	0
B25	1	3	0	0	0	0	0	1	2	1	1	4
C25	0	1	3	4	1	0	0	0	1	3	2	0
A26	2	2	4	4	2	2	1	0	2	2	2	0
B26	3	3	4	0	3	0	3	1	1	4	2	0
C26	0	1	2	3	2	2	3	2	2	3	1	0
A27	0	0	3	1	0	1	2	1	1	0	0	0
B27	1	0	4	2	1	1	1	1	2	2	1	0
C27	0	3	2	0	2	0	0	1	2	2	3	1
A28	1	3	4	0	3	2	2	3	2	1	2	0

S	AVD	DEP	O-C	P-A	SDT	PAR	STY	SZD	HIS	NAR	BRD	ASL
B28	1	3	2	3	0	0	0	2	0	4	1	0
C28	0	0	3	1	1	1	0	1	1	0	2	4
A29	0	3	2	1	2	3	3	1	2	3	1	1
B29	1	1	4	4	3	3	2	2	3	3	3	2
C29	0	0	1	0	0	2	0	0	1	2	1	0
A30	0	1	3	0	0	0	1	2	2	3	1	1
B30	0	2	4	4	2	3	3	2	2	4	4	3
C30	0	2	3	0	2	0	1	2	3	2	3	0
A31	3	2	5	2	1	3	4	2	2	4	4	0
B31	1	1	6	2	4	1	4	3	1	4	3	0
C31	2	3	5	2	3	1	3	2	2	4	2	1
A32	2	4	6	1	4	2	2	1	1	3	4	0
B32	1	1	6	0	3	2	1	3	2	2	0	0
C32	3	2	6	4	2	3	3	2	3	4	0	1
A33	1	0	6	4	0	1	4	3	3	3	1	2
B33	0	4	5	2	4	1	2	1	3	4	0	0
C33	1	2	5	1	1	2	4	2	2	2	0	0
A34	1	2	7	3	2	1	2	3	2	2	2	2
B34	2	2	5	2	2	2	4	2	2	4	2	0
C34	1	2	5	4	4	2	3	3	3	5	0	0
A35	1	2	5	1	1	0	1	0	1	3	0	0
B35	0	2	5	3	2	1	3	1	1	4	1	3
C35	0	1	8	2	3	2	2	1	1	3	2	1
A36	0	1	5	1	2	0	1	1	2	4	0	2
B36	2	4	5	0	0	3	1	1	2	2	2	0
C36	1	1	5	1	4	1	2	3	1	1	1	0
A37	0	3	5	2	1	0	2	2	2	2	3	1
B37	0	2	6	0	1	1	2	1	1	2	0	0
C37	2	1	5	3	1	0	1	1	2	4	0	0
A38	1	0	6	0	3	0	1	2	2	1	2	0
B38	2	2	5	2	3	1	2	2	3	4	3	0
C38	1	2	5	1	0	1	2	2	1	2	0	0
A39	2	3	6	2	3	4	4	1	2	4	0	0
B39	0	4	5	3	3	3	0	2	2	2	1	3
C39	0	2	5	0	5	3	2	2	4	2	2	0
A46	2	3	3	5	4	5	1	2	1	3	3	3
B46	6	3	4	1	2	2	6	2	3	4	4	0
C46	4	2	3	3	3	3	5	3	3	4	3	1
A47	0	1	4	5	2	4	4	3	0	4	3	0
B47	0	4	2	5	2	2	3	0	6	4	4	0
C47	5	2	3	1	3	2	5	2	0	4	4	0
A48	2	2	3	5	4	7	1	3	3	3	2	0

S	AVD	DEP	O-C	P-A	SDT	PAR	STY	SZD	HIS	NAR	BRD	ASL
B48	0	6	2	3	5	2	1	0	3	5	3	0
C48	4	5	4	1	4	3	4	1	2	3	3	1
A49	2	6	2	6	3	3	2	2	3	5	3	8
B49	0	5	3	4	5	2	4	1	2	3	5	2
C49	4	5	3	2	4	3	3	3	1	2	5	0
A50	3	6	4	4	4	2	4	2	3	5	4	1
B50	2	3	3	5	2	2	3	3	5	3	4	1
C50	4	8	4	4	6	4	4	2	3	4	4	0
A51	2	2	5	2	2	5	3	3	2	4	2	0
B51	0	3	6	2	1	1	0	1	4	4	0	0
C51	3	4	6	4	1	3	3	1	2	7	4	0
A52	2	3	7	4	4	0	3	2	3	5	3	0
B52	2	3	6	1	3	0	0	3	2	4	1	3
C52	0	4	5	2	2	2	1	1	2	5	1	1
A53	1	1	7	2	4	5	4	1	1	4	1	2
B53	0	4	5	2	1	3	2	4	1	2	0	0
C53	2	3	6	2	3	2	3	2	3	8	3	1
A54	2	2	5	2	2	0	3	4	1	4	0	1
B54	3	3	6	3	6	4	3	3	3	1	2	0
C54	2	3	5	1	3	1	5	3	2	3	2	6
A55	3	2	6	4	4	3	1	2	3	6	1	3
B55	0	3	6	1	3	2	2	3	4	4	2	0
C55	1	4	6	3	1	3	1	1	4	3	1	0
A56	1	4	5	3	3	1	2	2	4	3	0	0
B56	0	1	5	2	3	2	3	0	4	2	2	0
C56	0	0	5	4	2	1	2	2	3	6	2	1
A60	0	1	3	0	1	2	3	2	2	2	2	2
A61	6	6	4	5	4	3	3	2	2	4	5	0
B61	4	5	4	7	5	3	4	0	1	3	6	1
C61	3	7	3	4	1	3	4	5	3	4	1	0
A62	4	3	4	3	2	2	4	2	5	4	3	0

Table 2

Two-way Analysis of Variance on Change in Expectancy (ER1-ER2)

with Triadic Group and Diagnostic Group as Factors

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.86	2.43	1.59	.2079
Triadic Group	2	75.48	37.74	24.69	.0001*
Interaction	4	5.94	1.49	.97	.4255
Error	125	191.05	1.53		

* Statistically Significant at .05 level of significance

Table 3

Test of Multiple Comparisons of Triadic Group on Change in Expectancy Ratings

Comparison	Mean Diff.	Scheffe F-test
A vs. B	-1.81	24.10 *
A vs. C	-.66	3.17 *
B vs. C	1.15	9.31 *

* Statistically significant at .05 level of significance

Table 4

Multivariate Analysis of Variance on MAACL, NTC-Escape, RT1 with Triadic Group and Diagnostic Group as Factors (MANOVA #1)

Source	Wilks' Lambda	Num df	Den df	F	p
Diagnosis	.986	6	244	.279	.9465
Triadic Group	.832	6	244	3.915	.0009 *
Interaction	.879	12	323.073	1.349	.1896

* Statistically Significant at .05 level of significance

Table 5

Multivariate Analysis of Variance on MAACL, NTC-Escape, RT1 with Modified Triadic Group and Categorized ASQ-Composite as Factors (MANOVA #2)

Source	Wilks' Lambda	Num df	Den df	F	p
Categorized ASQ-Composite	.976	3	83	.674	.5701
Modified Triadic Group	.909	3	83	2.769	.0468 *
Interaction	.955	3	83	1.289	.2837

* Statistically Significant at .05 level of significance

Table 6

Multivariate Analysis of Variance on MAACL, ER1-ER2, RT1 with Modified Triadic Group and Categorized ASQ-Composite as Factors

(MANOVA #3)

Source	Wilks' Lambda	Num df	Den df	F	p
Categorized ASQ-Composite	.992	3	83	.218	.8837
Modified Triadic Group	.577	3	83	20.281	.0001 *
Interaction	.978	3	83	.623	.6024

* Statistically Significant at .05 level of significance

Table 7

Two-way Analyses of Variance on MAACL Scores
with Triadic Group and Diagnostic Group as Factors

ANOVA for Change in Overall Affect (MAACL3-MAACL1)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	176.10	88.05	.57	.5663
Triadic Group	2	1325.73	662.86	4.30	.0156*
Interaction	4	379.42	94.85	.62	.6523
Error	126	19421.82	154.14		

ANOVA for Change in Depression (D3-D1)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	32.39	16.20	.51	.5992
Triadic Group	2	227.12	113.56	3.60	.03*
Interaction	4	85.80	21.45	.68	.6064
Error	127	4000.80	31.50		

ANOVA for Change in Anxiety (A3-A1)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	7.47	3.73	.30	.7436
Triadic Group	2	74.64	37.32	2.97	.055
Interaction	4	22.04	5.51	.44	.7808
Error	127	1597.12	12.58		

ANOVA for Change in Hostility (H3-H1)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	30.81	15.40	.80	.4522
Triadic Group	2	160.37	80.18	4.16	.0178*
Interaction	4	73.38	18.35	.95	.4368
Error	127	2449.27	19.29		

* Statistically Significant at .05 level of significance

Table 8

Tests of Multiple Comparisons of MAACL Change Scores

Test of Multiple Comparisons of Triadic Group on Overall Change in Affect Scores

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
A vs. B	-7.38	5.14 *	4.04 *
A vs. C	-5.57	5.08 *	2.36
B vs. C	1.81	5.19	.24

Test of Multiple Comparisons of Triadic Group on Depression (D3-D1) Change Scores

Comparison	Mean Diff.	Scheffe F-test
A vs. B	-3.05	3.42 *
A vs. C	-2.18	1.77
B vs. C	.87	.27

(cont.)

Table 8 (cont.)

Test of Multiple Comparisons of Triadic Group on Anxiety (A3-A1) Change Scores

Comparison	Mean Diff.	Fisher PLSD
A vs. B	-1.71	1.45 *
A vs. C	-1.37	1.44
B vs. C	.34	1.47

Test of Multiple Comparisons of Triadic Group on Hostility (H3-H1) Change Scores

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
A vs. B	-2.47	1.82 *	3.62 *
A vs. C	-2.02	1.81 *	2.44
B vs. C	.45	1.84	.12

* Statistically Significant at .05 level of significance

Table 9

One-way Analysis of Variance on Percent Change in Anxiety

During Block 2 with Triadic Group as the Factor
(AFC ONLY)

Source	df	Sums of Squares	Mean Square	F	p
Between groups	2	1.453	.727	2.211	.1222
Within groups	42	13.803	.329		
Total	44	15.256			

Table 10

Test of Multiple Comparisons of Triadic Group on

Percent Change in Anxiety During Block 2 (AFC Only)

Comparison	Mean Diff.	Fisher F-test
A vs. B	.439	.423 *
A vs. C	.244	.416
B vs. C	-.195	.430

* Statistically Significant at .05 level of significance

Table 11

Two-way Analyses of Variance on Shuttlebox (Block 3) Measures with Triadic Group and Diagnostic Group as Factors

ANOVA for Number of Trials to Reach Escape Criterion (NTC-E)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	10.96	5.48	.11	.8993
Triadic Group	2	551.92	275.96	5.35	.0059 *
Interaction	4	381.79	95.45	1.85	.1232
Error	127	6549.90	51.57		

ANOVA for Number of Trials to Reach Escape Criterion (NTC-E): Modified Model (OCPD Excluded)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	1	8.303	8.303	.154	.6954
Triadic Group	2	346.008	173.004	3.215	.0451 *
Interaction	2	378.732	189.366	3.519	.0340 *
Error	85	4574.032	53.812		

ANOVA for Number of Failures (NF)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.19	2.09	.04	.9651
Triadic Group	2	635.11	317.56	5.39	.0057 *
Interaction	4	319.95	79.99	1.36	.2523
Error	126	7421.36	58.90		

(cont.)

Table 11 (cont.)

ANOVA for Number of Failures (NF)
Modified Model (OCPD Excluded)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	1	4.172	4.172	.066	.7973
Triadic Group	2	410.197	205.098	3.265	.0431 *
Interaction	2	315.724	157.862	2.513	.0871
Error	84	5275.897	62.808		

ANOVA for Reaction Time-Trial One (RT1)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	1.38	.69	.18	.8352
Triadic Group	2	38.00	19.00	4.95	.0085*
Interaction	4	18.85	4.71	1.23	.3026
Error	125	479.93	3.84		

ANOVA for Reaction Time-Trial One (RT1)
Modified Model (OCPD Excluded)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	1	.83	.83	.239	.6263
Triadic Group	2	18.898	9.449	2.719	.0718
Interaction	2	15.207	7.603	2.188	.1186
Error	83	288.434	3.475		

ANOVA for Mean Latency to Initiate Response (MLI)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.20	2.10	.64	.5314
Triadic Group	2	11.26	5.63	1.70	.1860
Interaction	4	2.32	.58	.18	.9507
Error	125	412.69	3.30		

(cont.)

Table 11 (cont.)

ANOVA for Number of Trials to Avoidance Criterion (NTC-A)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	8.00	4.00	.11	.8916
Triadic Group	2	35.72	17.86	.51	.6004
Interaction	4	63.64	15.91	.46	.7676
Error	127	4427.90	34.87		

ANOVA for Number of Correct Avoidance Responses (NA)

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	34.70	17.35	.36	.7001
Triadic Group	2	43.10	21.55	.44	.6424
Interaction	4	56.94	14.23	.29	.8818
Error	126	6112.91	48.52		

* Statistically Significant at the .05 level of significance

Table 12

Tests of Multiple Comparisons of Shuttlebox (Block 3) Measures

Test of Multiple Comparisons of Triadic Group on NTC-Escape Scores

Comparison	Mean Diff.	Scheffe F-test
A vs. B	-.62	.08
A vs. C	-4.53	4.52 *
B vs. C	-3.91	3.26 *

Test of Multiple Comparisons of Triadic Group NF Scores

Comparison	Mean Diff.	Scheffe F-test
A vs. B	-.87	.14
A vs. C	-4.94	4.78 *
B vs. C	-4.07	3.10 *

Test of Multiple Comparisons of Triadic Group RT1 Scores

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
A vs. B	-.27	.82	.21
A vs. C	-1.23	.81 *	4.48 *
B vs. C	-.96	.83 *	2.62

* Statistically Significant at .05 level of significance

Table 13

Two-way Analyses of Variance on Primary ASQ Scale Scores with Triadic Group and Diagnostic Group as Factors

ANOVA for ASQ--Negative Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	5.21	2.6	9.18	.0002*
Triadic Group	2	.36	.18	.63	.5325
Interaction	4	.23	.06	.20	.9355
Error	127	36.00	.28		

ANOVA for ASQ--Positive Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.18	2.09	6.09	.003*
Triadic Group	2	.59	.29	.85	.4289
Interaction	4	.70	.18	.51	.7288
Error	127	43.64	.34		

ANOVA for ASQ--Composite

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	17.38	8.69	12.47	.0001*
Triadic Group	2	.56	.28	.40	.6708
Interaction	4	.35	.09	.12	.9736
Error	127	88.50	.70		

* Statistically Significant at .05 level of significance

Table 14

Tests of Multiple Comparisons of Primary ASQ Scale Scores

Test of Multiple Comparisons of Diagnostic Group on ASQ-Negative Scores

Comparison	Mean Diff.	Scheffe F-test
AFC vs. NC	.45	8.52 *
AFC vs. OCPD	.10	.39
NC vs. OCPD	-.36	5.25 *

Test of Multiple Comparisons of Diagnostic Group on ASQ-Positive Scores

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
AFC vs. NC	-.41	.24 *	5.80 *
AFC vs. OCPD	-.29	.24 *	2.89
NC vs. OCPD	.12	.24	.49

Test of Multiple Comparisons of Diagnostic Group on ASQ-Composite Scores

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
AFC vs. NC	.87	.34 *	12.77 *
AFC vs. OCPD	.39	.34 *	2.57
NC vs. OCPD	-.48	.34 *	3.84 *

* Statistically significant at .05 level of significance

Table 15

Two-way Analyses of Variance on Selected ASQ Subscales with Triadic Group and Diagnostic Group as Factors

ANOVA of Internality for Negative Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	7.18	3.59	5.26	.0064 *
Triadic Group	2	.07	.03	.05	.9515
Interaction	4	3.81	.95	1.40	.2383
Error	127	86.59	.68		

ANOVA of Stability for Positive Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.46	2.23	4.57	.0121 *
Triadic Group	2	1.24	.62	1.27	.2833
Interaction	4	1.54	.39	.79	.5336
Error	127	61.89	.49		

ANOVA of Globality for Achievement Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	3.85	1.93	2.77	.0662
Triadic Group	2	2.36	1.18	1.70	.1874
Interaction	4	2.21	.55	.80	.5289
Error	127	88.17	.69		

ANOVA of Internality for Positive Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	7.84	3.92	7.16	.0011 *
Triadic Group	2	.21	.11	.19	.8238
Interaction	4	1.22	.31	.56	.6934
Error	127	69.54	.55		

(cont.)

Table 15 (cont.)

ANOVA of Internality for Positive Events

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	10.04	5.02	6.72	.0017 *
Triadic Group	2	3.10	1.55	2.08	.1297
Interaction	4	1.33	.33	.45	.7754
Error	127	94.91	.75		

* Statistically Significant at .05 level of significance

Table 16

Selected Tests of Multiple Comparisons of ASQ Subscales

Test of Multiple Comparisons of Diagnostic Group on Internality for Negative Events

Comparison	Mean Diff.	Scheffe F-test
AFC vs. NC	.56	5.32 *
AFC vs. OCPD	.25	1.03
NC vs. OCPD	-.31	1.65

Test of Multiple Comparisons of Diagnostic Group on Stability for Positive Events

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
AFC vs. NC	-.41	.29 *	3.87 *
AFC vs. OCPD	-.35	.29 *	2.90
NC vs. OCPD	.05	.29	.06

(cont.)

Table 16 (cont.)

Test of Multiple Comparisons of Diagnostic Group on Globality for Achievement Events

Comparison	Mean Diff.	Fisher PLSD
AFC vs. NC	.05	.35
AFC vs. OCPD	-.34	.35
NC vs. OCPD	-.39	.35 *

Test of Multiple Comparisons of Diagnostic Group on Internality for Positive Events

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
AFC vs. NC	-.57	.30 *	6.96 *
AFC vs. OCPD	-.20	.30	.87
NC vs. OCPD	.37	.30 *	2.89

Test of Multiple Comparisons of Diagnostic Group on Globality for Negative Events

Comparison	Mean Diff.	Fisher PLSD	Scheffe F-test
AFC vs. NC	.54	.36 *	4.41 *
AFC vs. OCPD	-.06	.36	.06
NC vs. OCPD	-.60	.36 *	5.53 *

* Statistically Significant at .05 level of significance

Table 17

Two-way Analysis of Variance on TSCS-Total Postive Scores

with Triadic Group and Diagnostic Group as Factors

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	14636.744	7318.372	10.617	.0001*
Error	132	90985.449	689.284		
Total	134	105622.193			

* Statistically Significant at .05 level of significance

Table 18

Test of Multiple Comparisons of Diagnostic Group on TSCS-Total Positive Scores

Comparison	Mean Diff.	Scheffe F-test
AFC vs. NC	-23.336	8.788 *
AFC vs. OCPD	-20.918	7.138 *
NC vs. OCPD	2.418	.096

*Statistically significant at .05 level of significance

Table 19

Two-way Analyses of Variance on Post-Experiment Questionnaire Ratings with Triadic Group and Diagnostic Group as Factors (Questions Regarding Pretreatment)

Table 19a
ANOVA of Pretreatment Confidence in Strategy Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	13.379	6.69	1.742	.1813
Triadic Group	1	1309.05	1309.05	340.896	.0001 *
Interaction	2	2.946	1.473	.384	.6826
Error	85	326.402	3.84		

Table 19b
ANOVA of Pretreatment Tone Aversiveness Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	10.62	5.31	1.085	.3411
Triadic Group	2	.927	.463	.095	.9097
Interaction	4	7.093	1.773	.362	.8351
Error	127	621.74	4.896		

Table 19c
ANOVA of Decibel Levels

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	67.307	33.653	.361	.6977
Triadic Group	2	223.033	111.516	1.196	.3057
Interaction	4	144.875	36.219	.389	.8165
Error	127	11839.464	93.224		

(cont.)

Table 19 (cont.)

Table 19d
ANOVA of Pretreatment Importance Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	4.204	2.102	.425	.6551
Triadic Group	1	15.116	115.116	23.285	.0001 *
Interaction	2	1.122	.561	.114	.8928
Error	84	415.285	4.944		

Table 19e
ANOVA of Pretreatment Performance Expectancy Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	13.467	6.734	1.836	.1658
Triadic Group	1	931.557	931.557	254.008	.0001 *
Interaction	2	10.173	5.086	1.387	.2555
Error	84	308.064	3.667		

Table 19f
ANOVA of Pretreatment Effort Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	3.502	1.751	.244	.7844
Triadic Group	1	6.909	6.909	.961	.3298
Interaction	2	16.862	8.431	1.173	.3146
Error	84	308.064	3.667		

* Statistically Significant at .05 level of significance

Table 20

Two-way Analyses of Variance on Post-Experiment Questionnaire Ratings with Triadic Group and Diagnostic Group as Factors (Questions Regarding Test Phase)

Table 20a
ANOVA of Test Phase Confidence in Strategy Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	17.086	8.543	1.04	.3563
Triadic Group	2	4.031	2.015	.245	.7827
Interaction	4	17.478	4.369	.532	.7123
Error	127	1042.766	8.211		

Table 20b
ANOVA of Test Phase Effort Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	1.419	.709	.169	.8446
Triadic Group	2	3.519	1.759	.419	.6583
Interaction	4	18.632	4.658	1.111	.3545
Error	126	528.433	4.194		

Table 20c
ANOVA of Test Phase Performance Expectancy Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	13.545	6.773	1.182	.3100
Triadic Group	2	3.078	1.539	.269	.7649
Interaction	4	19.064	4.766	.832	.5074
Error	126	721.952	5.73		

(cont.)

Table 20 (cont.)

Table 20d
ANOVA of Test Phase Tone Aversiveness Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	21.241	10.621	1.731	.1812
Triadic Group	2	29.916	14.958	2.439	.0914
Interaction	4	33.250	8.312	1.355	.2533
Error	127	779.033	6.134		

Table 20e
ANOVA of Test Phase Importance Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	1.473	.737	.165	.8481
Triadic Group	2	22.184	11.092	2.485	.0874
Interaction	4	5.878	1.469	.329	.8579
Error	126	562.429	4.464		

Table 20f
ANOVA of Test Phase Inability Attribution Ratings for Failures

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	33.289	16.644	1.984	.1419
Triadic Group	2	45.120	22.560	2.689	.0719
Interaction	4	5.246	1.312	.156	.9598
Error	124	1040.509	8.391		

Table 20g
ANOVA of Test Phase Lack of Effort Attribution Ratings for Failures

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	29.562	14.781	1.966	.1443
Triadic Group	2	4.214	2.107	.28	.7561
Interaction	4	4.547	1.137	.151	.9622
Error	126	947.414	7.519		

(cont.)

Table 20 (cont.)

Table 20h
ANOVA of Test Phase Impossibility Ratings for Failures

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	1.62	.81	.109	.8972
Triadic Group	2	9.765	4.883	.655	.5214
Interaction	4	56.931	14.233	1.908	.1131
Error	127	947.366	7.46		

Table 20i
ANOVA of Test Phase Initial Confidence Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	.917	.459	.12	.8867
Triadic Group	2	16.638	8.319	2.183	.1169
Interaction	4	13.115	3.279	.86	.4898
Error	127	483.952	3.811		

Table 20j
ANOVA of Impression Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	14.288	7.144	1.344	.2644
Triadic Group	2	23.352	11.676	2.197	.1153
Interaction	4	22.001	5.5	1.035	.3919
Error	127	674.952	5.315		

Table 20k
ANOVA of Interest in the Experiment Ratings

Source	df	Sums of Squares	Mean Square	F	p
Diagnosis	2	9.897	4.948	1.143	.3222
Triadic Group	2	20.118	10.059	2.323	.1021
Interaction	4	11.517	2.879	.665	.6175
Error	127	549.945	4.330		

* Statistically Significant at .05 level of significance

Table 21

Selected Tests of Multiple Comparisons of Post-Experiment Questionnaire Ratings (Pretreatment and Test Phase)

Test of Multiple Comparisons of Triadic Group on Test Phase Tone Aversiveness

Comparison	Mean Diff.	Fisher PLSD
A vs. B	1.13	1.039 *
A vs. C	.699	1.033
B vs. C	-.43	1.05

Test of Multiple Comparisons of Triadic Group on Test Phase Importance Ratings

Comparison	Mean Diff.	Fisher PLSD
A vs. B	.988	.867 *
A vs. C	.450	.860
B vs. C	-.538	.871

(cont.)

Table 21 (cont.)

Test of Multiple Comparisons of Triadic Group on
Test Phase Initial Confidence Ratings

Comparison	Mean Diff.	Fisher PLSD
A vs. B	.568	.803
A vs. C	-.289	.798
B vs. C	-.857	.812 *

Test of Multiple Comparisons of Triadic Group on
Test Phase Inability Attribution for Failure

Comparison	Mean Diff.	Fisher PLSD
A vs. B	1.348	1.202 *
A vs. C	.418	1.209
B vs. C	-.930	1.222

Test of Multiple Comparisons of Diagnostic Group on
Test Phase Lack of Effort Attribution for Failure

Comparison	Mean Diff.	Fisher PLSD
AFC vs. NC	1.133	1.122 *
AFC vs. OCPD	.667	1.122
NC vs. OCPD	-.467	1.122

(cont.)

Table 21 (cont.)

Test of Multiple Comparisons of Triadic Group on Interest in Experiment Ratings

Comparison	Mean Diff.	Fisher PLSD
A vs. B	.924	.860 *
A vs. C	.432	.855
B vs. C	-.492	.869

* Statistically Significant at .05 level of significance

Table 22

One-way Analysis of Variance of Diagnostic Group on Percent Increase in Depressive Affect During Block 2 (Group B Only)

Source	df	Sums of Squares	Mean Square	F	p
Between Groups	2	.236	.118	2.374	.1058
Within Groups	41	2.041	.05		
Total	43	2.277			

Table 23

Test of Multiple Comparisons of Diagnostic Group on Percent Increase in Depressive Affect During Block 2 (Group B Only)

Comparison	Mean Diff.	Fisher PLSD
AFC vs. NC	-.109	.167
AFC vs. OCPD	.067	.167
NC vs. OCPD	.176	.165 *

* Statistically Significant at .05 level of significance

Table 24

One-way Analysis of Variance of Diagnostic Group on BDI Scores

Source	df	Sums of Squares	Mean Square	F	p
Between Groups	2	206.13	103.07	8.44	.0004 *
Within Groups	133	1623.25	12.20		
Total	135	1829.38			

* Statistically Significant at the .05 level of significance

Table 25

Test of Multiple Comparisons of Diagnostic Group on BDI Scores

Comparison	Mean Diff.	Scheffe F-test
AFC vs. NC	2.87	7.67 *
AFC vs. OCPD	2.24	4.64 *
NC vs. OCPD	-.62	.36

* Statistically Significant at .05 level of significance

Table 26

One-way Analysis of Variance of Diagnostic Group on ER1 Scores

(OCPD Excluded from the Model)

Source	df	Sums of Squares	Mean Square	F	p
Between Groups	1	8.903	8.903	3.743	.0562
Within Groups	89	211.713	2.379		
Total	90	220.615			

Table 27

One-way Analysis of Variance of Triadic Group on MAACL-Composite (AFC ONLY)

Source	df	Sums of Squares	Mean Square	F	p
Between Groups	2	977.315	488.657	3.387	.0433 *
Within Groups	42	6059.885	144.283		
Total	44	7037.2			

* Statistically Significant at .05 level of significance

Table 28

Test of Multiple Comparisons of Triadic Group on MAACL-Composite (AFC Only)

Comparison	Mean Diff.	Scheffe F-test
A vs. B	-11.42	3.374 *
A vs. C	-5.929	.943
B vs. C	5.49	.756

* Statistically Significant at .05 level of significance

Table 29

One-way Analysis of Variance of Triadic Group on Change in MAACL-Composite During Block 2

Source	df	Sums of Squares	Mean Square	F	p
Between Groups	2	188.79	94.39	2.99	.0537
Within Groups	133	4199.1	31.57		
Total	135	4387.88			

Table 30

Test of Multiple Comparisons of Triadic Group on MAACL-Composite During Block 2

Comparison	Mean Diff.	Fisher PLSD
A vs. B	1.57	2.33
A vs. C	-1.34	2.32
B vs. C	-2.91	2.36 *

* Statistically Significant at .05 level of significance

Table 31

Group B Means of OCPD and NC on NTC-Escape, NF, and RT1

	OCPD		NC	
	Group A	Group B	Group A	Group B
NTC-Escape	10.93	12.00	9.19	13.33
NF	8.27	9.73	6.50	10.00
RT1	4.49	5.25	4.95	5.81

APPENDIX C

Expectancy Rating Forms

ER1

Even though you haven't been exposed to the task yet, how well do you expect to do on the upcoming task?

PLEASE READ THE WORDS CAREFULLY AND CIRCLE YOUR RATING

0	1	2	3	4	5	6	7	8	9	10
I don't expect to do well at all										I expect to do very well

ER2

How well do you expect to do on the next part of the experiment?

PLEASE READ THE WORDS CAREFULLY AND CIRCLE YOUR RATING

0	1	2	3	4	5	6	7	8	9	10
I don't expect to do well at all										I expect to do very well

APPENDIX D

Post-Experiment Questionnaire

THINK BACK TO THE FIRST PHASE OF THE EXPERIMENT
WHILE ANSWERING THE FOLLOWING QUESTIONS:

REMEMBER: THE FIRST PHASE WAS DIVIDED INTO TWO SECTIONS. THE INSTRUCTIONS WERE THE SAME.

1. In the first part of the experiment, what strategy did you use to turn off the noise?

PLEASE EXPLAIN IN DETAIL:

---

2. How confident are you that your strategy stopped the noise in the first part of the experiment?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not confident at all Very Confident

---

3. How unpleasant was the noise to you when it was on in the first part of the experiment?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not unpleasant at all Extremely Unpleasant

---

4. How well did you do on the first part of the experiment?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Extremely bad Extremely good

(Continued on next page)

5. How important was it for you to actually figure out the strategy and stop the noise yourself in the first part of the experiment?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not important at all Very Important

---

6. How hard did you try to stop the noise yourself in the first part of the experiment?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not hard at all Very hard

---

7. How well would you expect to do if the task were to start up again?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

I wouldn't expect to do well at all I'd expect to do very well

---

8. On #7 above, why did you circle the number you circled?

PLEASE EXPLAIN IN DETAIL:

NOTE: GROUP C subjects were not administered the above questions. Instead, they were asked to write down their thoughts during the pretreatment phase. All subjects were administered the following questions:

Questions 9-16 were identical to 1-8 EXCEPT they refer to performance in Block 3

(Continued on next page)

17. In the last task, when you first saw the shuttlebox appear on the computer monitor, what were you thinking? (in other words, WHAT WERE YOU THINKING BEFORE YOU EVEN TRIED TO STOP THE NOISE?)

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

I definitely will not be able to stop the noise — I definitely will be able to stop the noise

---

18. On #18 above, why did you circle the number you circled?

PLEASE EXPLAIN IN DETAIL:

---

19. Were you thinking anything else when you first started the last task?

PLEASE EXPLAIN IN DETAIL:

---

20. If you ever failed to stop the noise during the shuttlebox task, this was primarily due to the fact that:

RATE HOW STRONGLY YOU BELIEVE EACH STATEMENT

A. You did not figure out or use the correct strategy

0 1 2 3 4 5 6 7 8 9 10

Strongly disagree — Strongly Agree

****

B. It was impossible to stop the noise

0 1 2 3 4 5 6 7 8 9 10

Strongly disagree — Strongly Agree

****

(Continued on next page)

C. You did not try hard enough

0 1 2 3 4 5 6 7 8 9 10

Strongly disagree Strongly Agree

****

D. _____

Fill in another reason if you desire

0 1 2 3 4 5 6 7 8 9 10

Strongly disagree Strongly Agree

****

---

21. How important was it for you to make a good impression on the experimenter during the entire experimenter?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not important at all Very important

---

22. How interesting was the entire experiment to you?

PLEASE READ THE CHOICES CAREFULLY AND CIRCLE YOUR RATING

0 1 2 3 4 5 6 7 8 9 10

Not interesting at all Very interesting

---

23. Other than what the experimenter has already told you, do you think you know the specific purpose or the specific predictions of the experiment?

CIRCLE ONE

YES NO

IF "YES," PLEASE ELABORATE:

APPENDIX E

Schematic Representation of the Method

	Obsessive Compulsives OCPD	Personality Controls AFC	Normal Controls NC
Triadic Group	A B C	A B C	A B C

ADMINISTRATION OF ASQ AND TSCS

TONE SELECTION PROCEDURE

ADMINISTRATION OF MAACL1 AND EXPECTANCY RATING 1

BLOCK 1 (PRETREATMENT PHASE)

ADMINISTRATION OF MAACL2

BLOCK 2 (PRETREATMENT PHASE)

ADMINISTRATION OF MAACL3 AND EXPECTANCY RATING 2

BLOCK 3 (TEST PHASE)

POST-EXPERIMENT QUESTIONNAIRE

DEBRIEFING

APPENDIX F

Informed Consent

One of the questionnaires that you filled out during Mass Testing gave us some information about your general personality style. The general purpose of the experiment is to find out how people with certain personality styles solve a variety of tasks. Your responses to that earlier questionnaire as well as your performance during this experiment are completely confidential.

During the upcoming experiment, you will be exposed to a slightly unpleasant noise which will be administered through a set of headphones. The noise is not considered dangerous, but individuals with ear or hearing problems should refrain from participating in the study. Prior to the experiment, you will be asked to select a tone that you judge to be "slightly unpleasant." This will be the tone intensity that will be administered periodically throughout the experiment. During the experiment, you will be asked to sit and listen to these tones, and you may, at times, be asked to figure out a way to turn off the tones. Also, you will be asked to complete several brief questionnaires as part of the experiment.

This project will benefit you in that you will be able to experience psychological research firsthand, as an experimental subject. You will also be allowed to ask questions about the project at the end of the experiment. This project will benefit mankind in that it may add to what we currently know about certain personality types and their behavior.

The entire experiment should take approximately one and a half hours. You will receive <u>two</u> research experience credits for your participation or financial compensation as discussed by you and the individual who contacted you about participating in this project. You are free to withdraw from this experiment at any time and you will still receive research credit and/or compensation should you choose to withdraw. All data obtained from you will be assigned a code number, and only the experimenter will have access to these data. Upon completion of this project, all identifying information will be destroyed and all data will be secured by the experimenter. Any questions that you may have will be answered by the experimenter at the end of the study.

(Continued on next page)

APPENDIX F (cont.)

Informed Consent

THE UNIVERSITY OF NORTH CAROLINA AT GREENSBORO

Consent to Act as a Human Subject

Subject's Name_____

Date of Consent_____

I hereby consent to participate in the research project entitled Personality Style and Task Performance. An explanation of the procedures and/or investigations to be followed and their purpose, including any experimental procedures was provided to me by _____. I was also informed about any risks, benefits, or discomforts that I might expect. I was given the opportunity to ask questions regarding the research and was assured that I am free to withdraw my consent to participate in the project at any time without penalty or prejudice. I understand that I will not be identified by name as a participant in this project.

I have been assured that that the explanation I have received regarding this project and this consent form have been approved by the University Institutional Review Board which ensures that research projects involving human subjects follow federal regulations. If you have any questions about this, I have been told to call the Office of Research Services at (919) 334-5878.

I understand that any new information that develops during the project will be provided to me if that information might affect my willingness to continue participation in the project. In addition, I have been informed of the compensation/treatment or absence of compensation/treatment should I be injured in this project.

_____ _____

Subject's Signature Witness to Oral Presentation

APPENDIX G

Special Debriefings

(A)

**READ THIS ONLY IF SUBJECT SCORES OVER 20 ON THE BDI.

Unfortunately, due to your responses on the questionnaire I just gave you, I won't be able to present the whole experiment to you. But I will (give you the research credit/money) I promised anyway.

Also, just in case you ever want to talk with someone, I'll give you a list of agencies and their phone numbers. Thank you for showing up today.

(B)

**READ THIS ONLY IF SUBJECT BECOMES VERY DISTRESSED AS A RESULT OF PARTICIPATING IN THE EXPERIMENT OR IF SUBJECT'S RESPONSES ON THE DACL INDICATE THE NEED FOR SPECIAL ATTENTION.

I apologize for this being upsetting to you. I want to do whatever it takes to make sure you feel alright about this or at least have an opportunity to talk with someone about this. Let me give you a few names and phone numbers. Please feel free to contact any of these individuals or agencies if you so desire.

APPENDIX H

Debriefing

Thank you for your participation in this study. The main purpose of this study is to determine if individuals with certain personality styles perform worse on the shuttlebox task (the last phase of the experiment) and become more depressed when they are first exposed to an uncontrollable tone. Some experimental subjects are able to terminate the noise in the first phase by pressing the space bar four times. Other subjects are told that they can control the tone somehow, when, in actuality, they can't. A third group of subjects is just told to simply sit and listen to the tones in the first part of the experiment. They are not told to try to terminate the noise.
In the first phase of the experiment, you were in the group that
_____.

In the last phase of the experiment, it was possible for all subjects, including you, to turn off the noise by moving the cursor on the computer monitor, from the left box to the right box by pressing the right key and the top key in an alternating fashion. To move from the right box to the left box, you had to alternate the left and top keys. Did you ever figure out that you could completely avoid the noise by moving the cursor across the screen when that big square warning signal came on?

YES NO

If YES, ask the following: When you first moved the cursor before the noise came on , what was the reason:

A. Because you accidently hit a certain key before the noise came on and noticed that the line of stars appeared?

B. Because you were just playing around with the keys and noticed that the line of stars appeared.

C. You actually thought that there might be a way to completely avoid the noise, so you purposely tried to respond before the tone came on.

The questionnaire you filed out during Mass Testing (SCID-II--Self-Report) helpled us determine what your personality type is. However, this questionnaire only gives us an indication of your personality and it is not entirely accurate. Therefore, it would be irresponsible for me to provide you with individual feedback on your personality.

GROUP B SUBJECTS ONLY

Again, you were in the first phase group that could not terminate the noise. Nobody in your group can terminate the noise. I'm sorry that I led you on by telling you that you could terminate the noise. I hope that it wasn't too frustrating for you. It was important for me to make you believe that you could terminate the noise, so I had to deceive you a little. People react in different ways when I do this and it would be helpless to hear about your thoughts and feelings on this. ____. How do you feel about the fact that I deceived you? _____. Does this bother you? _____.

IF SUBJECT IS VERY UPSET, GO TO SPECIAL DEBRIEFING (B)

ALL SUBJECTS

Was there anything that bothered you about the experiment?
Do you want to talk about it?
Any comments or questions about anything?

____ Administer DACL (If DACL indicates the need for special attention or if the subject is noticeably upset, go to Special Debriefing (B))

IMPORTANT TO SAY TO ALL SUBJECTS:

**Please do not tell your friends about this experiment. I test alot of students here at UNCG and I want to be sure that all of my research subjects know nothing about the details of the experiment before they participate. If a research subject knows what the experiment is all about, that could mess up my experiment. Do you understand?

**Before going, I just wanted to get your permission again to use the information we've collected on you today. Is it alright?

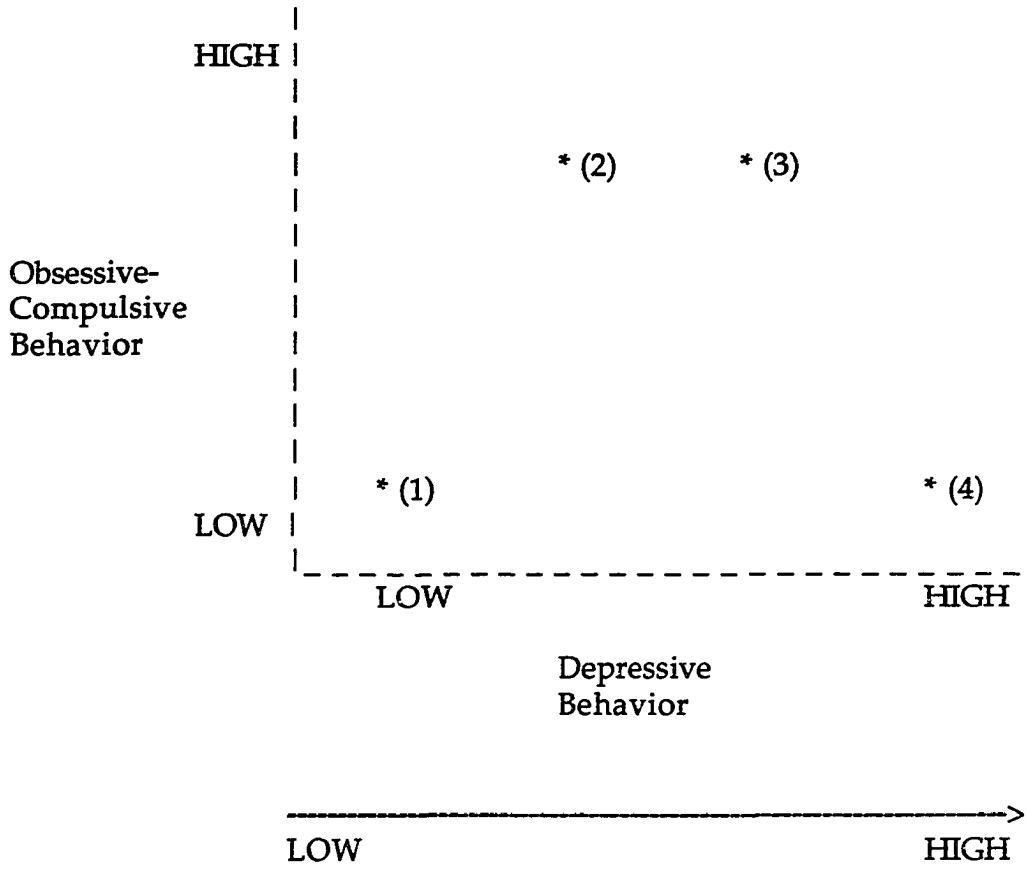
YES NO

**Thanks again for participating in our experiment.

Sign Research Participation Form or Compensate Subject

APPENDIX I

Schematic Representation of Possible Relationship Between OCPD and Depression



Environmental Context	Response Topography
1. "LOW"	LOW OC Behavior, LOW Depression
2. "MODERATELY LOW"	HIGH OC Behavior, LOW Depression +
3. "MODERATELY HIGH"	HIGH OC Behavior, HIGH Depression
4. "HIGH"	LOW OC Behavior, HIGH Depression ^

+ OC Behavior successfully "defends" against depression.
^ Learned Helplessness: OC Behavior is extinguished.

APPENDIX J

Radical Behaviorism and Learned Helplessness

Investigating the helplessness phenomenon from alternative perspectives could potentially strengthen the theory outlined by Seligman (1975). Alternative methods, language systems, and theoretical emphases might serve to address many of the criticisms leveled against the theory and improve the theory's ability to predict and control behavior without necessarily having to completely abandon it. Most theorists, while perhaps differing on details, probably agree that the purported mechanism--"perceived uncontrollability"--however defined, does, in fact, influence the onset of depression in certain individuals. One such alternative perspective on this mechanism, a radical behavioral perspective, will be briefly offered here for illustrative purposes. Other perspectives should be explored and capitalized upon as well.

If one were to stretch his or her imagination and translate the cognitive language of learned helplessness into radical behavioral terms, she or he might be able to conceptualize learned helplessness as one specific example of Ferster's more general functional analysis of depression--a theory briefly mentioned in the introduction and one which suggests that the depressed individual has a defective behavioral repertoire due to, for example, sudden environmental changes (e.g., uncontrollability). This defective repertoire

decreases the likelihood of positive reinforcement and increases the chances of aversive consequences, eventually resulting in extinction. Perhaps the learned helplessness deficits/disruptions discussed in the literature are in some way analogous to this extinction. If this is true, then Ferster's more general analysis could perhaps account for learned helplessness in its entirity. Some might argue then, that learned helplessness theory does not add anything to the more general analysis, and thus, is superfluous. However, in defense of the learned helplessness theory, it could be argued that its narrower scope still sheds light on particular cases of depression, which may have important practical treatment and prevention implications.

Radical behaviorism, a non-mediational approach, avoids such cognitive-behavioral notions as "perceived uncontrollability." Yet, it still may be able to address this mechanism from within its theoretical framework, capitalizing upon Skinner's rule governed behavior. Recall, rule-governed behavior was briefly addressed in the introduction when rigidity of thought was argued to be a behavior within the obsessive compulsive's repertoire. Skinner (1966, 1969) defines "rules" as verbal behaviors that serve as "contingency-specifying stimuli." They are provided by a "speaker" and either followed or not followed by a "listener." The listener is, in essence, governed by two sets of contingencies; one set, the actual contingencies that the rule specifies, and the other set, the socially-mediated consequences of rule-following. Rule-governed behavior is distinguised from contingency-shaped

behavior in that the listener, in rule governed behavior, may have never been exposed to the natural contingencies that the rule specifies before the rule was actually delivered. In contingency-shaped behavior, behavior is entirely controlled by the natural consequences of the situation.

Rules are followed by most people for several reasons. It may be quite efficient for an individual to follow rules due to the fact that that rule-governed behavior (unlike contingency-shaped behavior) does not necessarily rely on a detailed history of reinforcement. Also, an individual may follow rules because of the pliance contingencies added by the rules that may actually control responding and render the individual "insensitive" to the natural tracking contingencies. Zettle and Hayes (1982) define "pliance" as "rule-governed behavior primarily under the control of apparent speaker-mediated consequences for correspondence between a rule and relevant behavior (p. 9)" and they define "tracking" as "rule-governed behavior under control of the apparent correspondence between a rule and the way the world is arranged (p. 10)."

These two types of rule-following, pliance and tracking, may be either beneficial (e.g., promote adaptive functioning) or detrimental (e.g., lead to the development of irrational cognitions).

According to Skinner (1969), "self"-rules are similar to rules except for the fact that the "speaker" and "listener" are the same person. In other words, "self"-rules are unique in that they are created by (i.e., "extracted from" the

natural contingencies) and adhered to by the same individual. No doubt, however, the distinction between the role of the "speaker" and "listener" within the individual is often blurred.

As Zettle and Hayes (1982) assert, "self"-rules are often complicated in that they cannot be directly manipulated like ordinary rules that involve an interchange between at least two people. In addition, it may be extremely difficult to determine whether a "self"-rule controls a given response or is only a "collateral response" to another variable.

Like ordinary rules, "self"-rules may be followed primarily because of pliance contingencies or tracking contingencies. Also, like ordinary rules, forming and following "self"-rules may be advantageous in that this may increase the efficency of responding and guarantee that effective behaviors will be maintained under adverse contingency-shaped conditions. However, it is quite possible that "self"-rules, like ordinary rules, may have a negative effect on behavior as well. It is feasible to suggest that some forms of psychopathology may, in fact, result from the formulation of and/or following of "self"-rules.

To elaborate on the study cited earlier, Schneidmiller (1987), in her dissertation, investigated the role of rule-governed behavior in obsessive compulsive and histrionic personality disordered analogues. An experimental procedure originated by Hayes, Brownstein, Haas, and Greenway (1986) was utilized. Specifically, Schneidmiller exposed personality

disordered analogues and a control group to an Acquisition/Extinction computer task, where all subjects were given an accurate rule that helpled them successfully manipulate two telegraph keys under two different alternating conditions (FR/DRL MULT). Successfully manipulating the telegraph keys enabled subjects to move a Plus Sign presented in the upper left hand cell of a 5 by 5 grid displayed on a computer monitor to the lower right hand cell of the grid, resulting in the accumulation of points. Subjects first mastered this acquistion phase of the experiment. Next all subjects were placed on extinction, where the rule was no longer accurate and the task was no longer controllable. On the one hand, in this phase, as predicted, Schneidmiller found that the behavior of the obsessive compulsive analogues was more under the control of the rule rather than under the control of the natural contingencies, given that their responding was slow to extinguish. On the other hand, she found that the behavior of the histrionic analogues was more under the control of the natural contingencies rather than the rule, given that their responding was quick to extinguish. The extinction curve of the control subjects fell between the two analogues. Schneidmiller argued that that these two disorders might be related to dysfunctional rule-governed behavior, with obsessive compulsives demonstrating "excessive" rule-governed behavior and histrionics demonstrating "deficits" in this area.

Zettle and Hayes (1982) suggest that in addition to a relationship with

personality disorders, perhaps a relationship between dysfunctional ("self"-) rule formulation/following and depression can be drawn. They speculate that Beck's conception of irrational schemas/cognitions (e.g., "I most be loved by everyone") can be reinterpreted in radical behavioral language as dysfunctional rule-governed, verbal behavior. They argue that this reinterpretation could potentially offer certain advantages, among them: (a) Skinner's radical behaviorism might offer a fresh alternative framework from which to conceptualize psychopathology and the treatment of psychopathology; (b) it might serve to expand the scope of traditional behavioral therapies; and (c) it might encourage the investigation of environmental sources of phenomena traditionally viewed as innate or automatic (i.e., schemas, attributional style).

Many of the cognitive theories of depression (e.g., Beck, Seligman) argue that inflexible cognitive schemas, cognitive sets, or attributional styles are at least partly responsible for the onset of depression; that these "cognitions" color the perceptions of the organism, decreasing the probability of tacting "reality," and hence, increasing the probability of a depressive event.

Perhaps, as Zettle and Hayes suggest with regard to Beck's conception of irrational cognitions, the "perceived uncontrollability"--the critical mechanism for helplessness induction--is also an overextended rule derived from the verbal community and/or a maladaptive "self-rule" extracted from

the natural contingencies. This rule or self-rule, perhaps one time based in "truth," currently renders the organism "insensitive" to the natural contingencies, and prevents the organism from capitalizing on the potential reinforcements inherent in these contingencies. Interestingly, treatments derived from both Beck's and Seligman's models encourage the client to dispute irrationality and to tact the natural contingencies. "Dragging the helpless dog" to the safe compartment of the shuttlebox is in many respects similar to "hypothesis testing" in cognitive therapy--in both, the old, resistant, and faulty contingency "shackles" are removed, freeing the organism to tact new, "healthy," more reinforcing contingencies.

Critics of Seligman as well as the results of the present dissertation illustrate some inadequacies of the learned helplessness model. While the helplessness attributional reformulation determined it necessary to address the organismic variable of explanatory style in order better to predict helplessness, a radical behavioral perspective would probably go a step further, insisting on a more thorough functional analysis including an investigation of the individual's learning history. The present study, while not radical behavioral in its focus, attempted to investigate other organismic variables--personality factors shaped up through a learning history--that increase the probability of helplessness induction and generalization. This study points to the importance of personality factors (i.e., learning history) as a mediator in the onset of helplessness. But it is also clear based on the

findings that the phenomenon learned helplessness attempts to address is complex and difficult to produce, predict, and assess under the best of conditions. Considerable attention will need to be paid to addressing these complexities and difficulties and alternative perspectives may be instrumental in such an endeavor.