ETIOLOGY OF EATING DISORDERS WITHIN A LEARNED HELPLESSNESS MODEL OF DEPRESSION

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ETIOLOGY OF EATING DISORDERS WITHIN A LEARNED HELPLESSNESS MODEL OF DEPRESSION

by

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A thesis submitted to the School of Graduate Studies in partial fulfilment of the Requirements for the degree of

Master of Educational Psychology

Department of Education
Memorial University of Newfoundland

January 2000

In Memory of Dr. James Y. Parsons

Abstract

This thesis is an attempt to parallel characteristics of anorexia (AN) and bulimia nervosa (BN: with those of depressive disorder in order to re-frame the etiology and progression of these eating disorders within a learned helplessness model of depression. In their 1993 publication, Seligman and his colleagues (Peterson, Maier, & Seligman) address the significance of the learned helpless theory in a contemporary society:

> "Learned helplessness is an important theory for the present because those of us in the Western world seem to be living in an era in which personal control is an overarching issue. We are also wary of the future. Because this incredible emphasis on personal control has its costs. We argue that the epidemic of depression among young adults represents a disorder of personal control. Generally speaking, the incredible selfishness of the American people can be phrased in terms of personal control" (p. 307).

It is proposed that the anorexic and the bulimic patients' symptomatology is rooted in a loss of a sense of control and of mastery, which can be traced to antecedent events. Consequently, these events or situations result in the eating-disordered patients' feeling helpless, powerless,

and not in control of circumstances affecting their lives. These feelings ultimately lead to a persistent negative affective state frequently reported in eating-disordered patients. It is suggested that the depressive state is attributable to learned helplessness depression as initially defined by Seligman (1975). The eating-disordered patient thwarts depressive feelings by focussing on her body, more specifically her weight. The patient seeks control over the only perceivably controllable aspect of her life, her weight. The thesis is presented in two parts: Part 1 is a literature review. It provides background information which examines existing research in order to elicit core themes and commonalities that parallel learned helplessness depression with AN and BN. Included is the proposed learned helplessness model of AN and BN. Part 2 is a proposal for empirical research to explore the relationship among three constructs: depression, hopelessness, and locus of control, in a clinically diagnosed eating-disordered population compared to a non-clinical, gender and age-matched student population.

Acknowledgements

I wish to acknowledge with sincere thanks my supervisor, Dr. Tim Seifert, for his support, direction, patience, and for the way he so generously made time available for my consultations during the many months this thesis was in progress.

I extend my appreciation to the Memorial University Education graduate administrative staff and computer staff for their kindness and support.

In addition, I extend a heartfelt thanks to my sister, Pat Jackson, and my cousin, Joseph Brennick and his wife, Eleanor, for their support and encouragement.

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Chapter 1

Introduction. .

Princess Diana - Case in Point

Increased growing public concern for certain maladies appears to have catapulted those particular disorders into heightened public awareness. Researchers, health care workers, and the media work frenetically to provide explanations of who, why, and how certain disorders come about. Eating disorders have come under particular scrutiny during the last decade. The most renowned struggle with this disorder was brought to light when the late Princess Diana in the early nineties openly revealed her long suffering experience with bulimia.

Generally, eating disorders are characterized by severe disturbances in eating behaviour. Anorexia nervosa (AN) is described as a refusal to maintain a minimally normal body weight. Bulimia nervosa (BN) is characterized by repeated episodes of binge eating followed by self-induced vomiting or misuse of laxative, diuretics, or other medications, fasting, or excessive exercise (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV, American Psychiatric Association, [APA], 1994). They are described

as psychosomatic disorders caused by biological, familial, psychological, and socio-cultural factors. The significance of the individual factors is not known (Kinzl, Mangwaeth, Traweger, & Biebl, 1997).

In writing this thesis I thought of using Princess Diana as an example of how I perceive learned helplessness to be the core clinical characteristic in the etiology and progression of eating pathology. However I will not elaborate as this information is meant merely to provide the reader with a frame of reference and not a case study. In addition, I refer only to brief excerpts from Andrew Morton's 1992 book, "Diana, Her True Story". Princess Diana had admitted to collaborating on the writing of the book which provided some credence to the validity of its contents.

As is the case with the majority, if not all, psychological dysfunction, infancy and early childhood are crucial phases in the development of pathology (Warren, 1996). Morton depicted the familial turmoil during Diana's early years and how parental conflicts impacted on the children's psychological development. Marital stressors were evident even before Diana's arrival. Her mother had given birth only to females. A disappointing situation inasmuch as a son was most desired to continue the long

royal heritage of the Viscount Althorp. At the insistence of her husband, the Viscountess made continued visits to the doctor to seek a cure for this peculiar malady. The Viscontess, by all accounts considered to be a "combative and tough-minded" woman, found these experiences to be "humiliating and unjust" (Morton, p. 10). Eventually Diana's parents divorced. Although her mother was cited as having had an extramarital affair which led to the marriage breakup, it was later discovered that her father, too, had been involved with other women while married. Diana's mother sued for custody of the children and lost. "The children became pawns in a bitter and acrimonious battle which turned mother against daughter and husband against wife" (Morton, p. 17).

Morton provided his readers with clues of a seemingly depressed young Diana when he wrote of her childhood experiences after the divorce of her parents "It was not just the adults who were scarred by this vicious legal battle....The reality was more traumatic than many had realized" (p. 18). According to Diana, it was difficult for her and her younger brother living with her father and stepmother. Her life seemed to centre around the loss of her mother and efforts to comfort her younger brother. One such instance is described in detail. Although Diana often

wanted to comfort her brother during the night, she was too frightened to negotiate the long, dark corridors to go to his side, and instead remained in her own bed listening to him sobbing for his mother.

It is significant that at one time in their lives both Sarah (Diana's older sister) and Diana both suffered from eating disorders, AN and BN, respectively. Morton wrote: "These illnesses are rooted in anxiety, and to use the jargon, 'malfunctioning' family life". Diana, in her own words, recalls the time, "My parents were busy sorting themselves out. I remember my mother crying. Daddy never spoke to us about it. We could never ask questions...too many nannies. The whole thing was very unstable" (p. 18). It is also interesting that of all the photographs depicted of the young Diana in the book, rarely is she smiling. Her face appears solemn and introspect. Diana's teacher remembered Diana "bursting into tears for no apparent reason during a painting class one afternoon. All her childhood "artwork was dedicated to Mummy and Daddy" (pp. 19 and 20).

A sense of powerlessness often is accompanied with depression, the onset of which emanates from the feeling that one lacks the ability to control or change significant aspects of one's life (Peterson, Maier, & Seligman, 1993). Certainly it is plausible to conceive of Diana as feeling

helpless to control her parents' situation which ultimately uprooted her own home stability, and of having had suffered depression as a result of it.

Fifteen years later, Diana was faced with another threatening and destructive situation which in some respects resembled that of her mother's ordeal. Even before her marriage to Prince Charles, Diana knew of her fiance's much talked about affair with Camilia Parker-Bowles. She named the woman as being a third party in the matrimonial bed. After her marriage to Prince Charles, the situation continued even after the birth of their children. Her marital difficulties worsened, and her sense of control over her fate (of which her dignity and self-esteem were inherently interwoven) was now threatened. Noted author on eating disorders, Russell Marx (1991) wrote: "The anorexic copes with her pain through disturbed eating because she fears that if she doesn't do something, anything, her life will get worse" (p. 5). Confronted with the thought of losing her husband and her celebrated royal role as Princess of Wales, in addition to having to contend with unrelenting pressures from her royal in-laws to conform to their expectations, Princess Diana was forced to live within an atmosphere of deceit and humiliation involving her husband's scandalous adultery, and yet maintain her public image as

Princess of Wales while providing for the emotional needs of her children. More commonly put, and in all respects, Princess Diana was "caught between a rock and a hard place".

It would not be implausible to assume that Princess Diana had once before felt helplessness with her inability to control vital aspects of her life when her parents divorced. Over a decade later she was then placed in a similar situation, and the feelings of helplessness and depression she had experienced early in her life once again emerged. It is interesting that people can learn to be helpless through the observation of another person encountering uncontrollable events (Peterson, Maier, & Seligman, 1993, p. 12). Observation of her mother's stressful situations might have also contributed to Diana's feelings of helplessness. Rollo May (1953) psychotherapist and philosopher observed:

> The chief problem of people of the 20th century is emptiness...the human being cannot live in a condition of emptiness for very long....The pent-up potentialities turn into morbidity and despair, and eventually into destructive activities....The feeling of emptiness...generally comes from people's feeling that they are powerless to do anything effective about their lives or the world they live in....When a person

> continually faces danger he is powerless to overcome, his final line of defence is at last to avoid even feeling the dangers (In Emmett, 1985, p. 316).

Relationship Between Eating Disorders and Depression..

The relationship between depression and eating pathology has been studied most extensively. Many behavioural accompaniments of starvation closely resemble symptoms of a primary depressive disorder (Keys, Brozak, Henschel, MicKelsen, & Taylor, 1950; Strober & Katz, 1988). It is well known that malnutrition intensifies the severity of depression (Leung & Steiger, 1991). As many as 91% of malnourished anorexics suffer from depressive disorders (Eckert, 1982; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983). Researchers have found 15% to 58% of patients continue to exhibit some degree of depressive disturbance after weight restoration, ranging from several months to 5 years (Cantwell, Sturzenberger, Burroughs, Salkin, & Green, 1977; Dally, 1969; Hudson, et al., 1983; Hsu, Crisp, & Callender, 1992; Leung & Steiger, 1990; Morgan & Russell, 1975; Theander, 1970; Rolvland, 1970).

The debate continues as to the nature of the relationship between mood and eating disorders. It is not clear whether depressive symptoms are secondary to weight loss and malnutrition or whether these weight loss and

malnutrition exist pre-morbidly, and thus may contribute to the pathogenesis of this illness (Deep, Nagy, Weltzin, Rao, & Kaye, 1994).

The literature includes three basic causal theories to explain the relationship between eating disorders and depression (Leung & Steiger, 1996).

The first theory attributes depressive symptoms to the biological or psychological effects of abnormal eating and weight loss (Crisp, 1980; Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). For example, it has been argued that depressive symptoms in anorexics are consequences of the gross physiological and psychological distortions that characterize this disorder (Crisp, 1980; Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). Similarly, Fairburn (1983) has contended that depression in BN is secondary to loss of control over eating, and that depressive symptoms remit as control over aberrant eating behaviors is established (Fairburn, 1983; Garner, et al., 1990; Williamson, et al., 1987).

The second viewpoint speculates that some unspecified "third variables" lead, in some individuals, to an increased vulnerability to both eating and affective disorders (Gershon, et al., 1984; Hudson, et al., 1987). These findings have led many researchers to believe that a common

genetic vulnerability is likely to be the third variable that simultaneously increases the likelihood of developing mood and eating disturbances (Devlin & Walsh, 1989). Findings from a study of first-degree relatives with AN (Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990) found an excess of affective disorders (mainly unipolar depression) among relatives of anorexic probands. The increased risk was accounted for largely by the subset of anorexics with coexisting depressive disorder.

Finally, there is the viewpoint that, in many cases, eating disorders are consequences of affective disturbances given the fact that numerous studies indicated that mood disorder preceded the eating disorder (Cantwell, Sturzenburger, Burroughs, Salkin, & Green, 1977; Gomez & Dally, 1980; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Piran, Kennedy, Garfinkel, & Owens, 1985). However Leung and Steiger (1990) have pointed out that most of these studies examined depression in eating disorder patents with a retrospective design. Therefore, they contend that it is unclear to what extent these findings might have been confounded by limits in subjects' accuracy of memories. These researchers, when investigating this phenomenon, found no relationship between depressive symptoms and eating abnormalities in their non-clinical population. The fact

that their population was non-clinical limited interpretation of their results.

This thesis re-examines the association of eating disorders and depression, and introduces another view. It is proposed that individuals who develop eating disorders have experienced learned helplessness depression through some means of development. The result is chronic anxiety and depression which leads to eating pathology. Human developmental researchers have studied extensively the effect of early development and later pathology. Warren (1992) suggested that:

> "All neurotic behaviour, including seriously disturbed eating behaviour arises from a basic anxiety formed from the fears developing in early childhood. That anxiety arises from a sense of being small and insignificant, powerless and endangered in a world that abuses, cheats, hurts, and betrays us".

The learned helplessness model of depression hypothesized that experience with uncontrollable events leads to difficulties in motivation, cognition, and emotion. Seligman (1975) suggested that it is not the extinction of negative reinforcers but lack of control over them that causes depression. For Seligman, "maternal deprivation results in a particularly crucial lack of control" (p. 145).

According to my thesis the depressive symptoms observed in eating disorders arise from the individual's early experiences of learned helplessness. That is, the individual had not the ability nor the power to control a vital aspect of her life, which resulted in the eating-disordered patient's feeling helpless and depressed. The ego-threatening experiences created an emotional scarring and served to frame all similar future experiences. The individual is left with a vulnerability. Relapses among eating disordered patients are accounted for by the depression subsiding, but not totally disappearing. The severity of the patient's depressive symptoms vacillate between those of unipolar depression and dysthymia. These two diagnoses are difficult to differentiate (DSM-IV, APA, 1994), which may contribute, perhaps, to some confusion on the part of the physician as to a proper diagnosis. If these two diagnoses are mistaken for each other, then it is conceivable that a more appropriate diagnosis, at least in cases of eating pathology, should have been learned helplessness depression. Symptoms associated with Dysthymia are similar to those of learned helplessness depression (Seligman, 1975, 1993). Once the "recovered" eating-disordered patient experiences an ego-threatening situation similar to the original experience or "template", depression

and eating pathology re-emerge. Researchers have found that both ego-threats and physical-threats are related to overeating among women with unhealthy eating attitudes (Schotte, 1992), and ego threats have been shown to be particularly salient (Heatherton, et al., 1991, 1992; Waller & Majatovich, 1996). This conceptualization coincides with that of the reformulated model of learned helplessness depression which states that "people develop a depressive explanatory style as a pre-existing disposition that can bring about symptoms of depression through its influence on expectations of helplessness; and people with depressive explanatory styles are at risk for future depression , (Peterson, Maier, & Seligman, 1993).

Clinical Characteristics of Anorexia and Bulimia

Eating disorders can occur at any age during the period of adolescent growth and maturation and carry the profound hazard of interrupting both somatic and psychological development (Strober, Freeman, & Morrell, 1997). These are insidious illnesses of unusual tenacity. More than 90% of reported cases of AN occur in females (DSM-IV, APA, 1994), and these are most commonly seen in adolescent females (DSM-IV, APA, 1994; Pollice, Kaye, Greeno, & Weltzin, 1996). Frombonne (1995) found incidence rates for AN in specific countries consistently peaked for the 15-19 year-olds and

decreased somewhat for the 20-24 year-olds. Recovery is lengthy and relapses are frequent, with nearly 30% of patients suffering relapses following hospital discharge (Strober, Freeman, & Morrell, 1997). Mortality rates exceed incidence of death from all causes in women 15-24 years of age by 12-fold (Sullivan, 1995; Strober, Freeman, & Morrell, 1997; Steinhausen, Rause-Mason, & Seidel, 1991). Of individuals admitted to university hospitals, the long-term mortality from AN is over 10%. Death most commonly results from starvation, suicide, or electrolyte imbalance (DSM-IV). Latest reports indicate that in Canada, 1%-2% of women between the ages of 14 to 25 years are anorexic and 3%-%% are bulimic (The National Association of Anorexia Nervosa and Associated Disorders (ANAD) and Statistics Canada, 1993).

Gender Differences

Although the incident reports of males diagnosed with eatng disorders is significantly lower than females, it is estimated that men probably represent only about 10% of the cases of AN and BN (DSM-IV). Several researchers have argued that men are susceptible to the debilitative affects of these disorders (Killen, et al., 1986; Lachenmeyer & Muni-Brander, 1988; Keel, et al., 1997; Crisp, Burns, & Bhat, 1986; Fichter & Daser, 1987; Steiger, 1989). Earlier

studies reported higher male-female ratios than more recent ones. Jones, et al., (1980) reported a ratio of 1:14, Szmukler, et al., (1986) of 1:12, and Lucas, et al., (1991) of 1:18 (Cited in Van't Hof, 1994). Rates are particularly high and growing among high school and college-aged males (Lachenmeyer & Muni-Brander, 1988).

Many studies indicate that eating disorders in males and females bear more similarities than differences (Crisp, Burns, & Bhat, 1986; Fichter & Daser, 1987; Steiger, 1989; Sterling & Segal, 1986; Scott, 1986; Van't Hof, 1984). Selme & Fioles (1990) showed that men and women differ very little in the amount that they think about food, eat for emotional comfort and the quantity of diet products that they consume. Drewnowski and Lee (1987) found that college freshmen reported that an equal number of males and females were dissatisfied with their bodies. All of the women studied reported that they wanted to be smaller, while half the men wanted to be thinner and the other half wanted to be more muscular. Some researchers contend that the "lack of familiarity" with AN in males leads to a delay in evaluation, diagnosis, and referral" (Siegel, Hardoff, Golden, & Shenker, 1995), and that the diagnose tends to be overlooked in males (Sterling and Segal, 1985; Scott, 1986; Van't Hof, 1994).

However, other researchers have suggested that a greater level of distress in men with eating disorders (BED) may be necessary before a decision to seek treatment is made (Tanofsky, Welfley, Spurrell, Welch, & Brownell). The inference is that current incidence figures may represent only the tip of the iceberg.

Prevalence

It is a widely held belief that the incidence of eating disorders has increased for both genders over the past few decades (Mitchell & Eckert, 1987; Duddle, 1973; Halmi, 1974; Jones, et al., 1980; Kendell, et al., 1973). Theander (1970) over thirty years ago in south Sweden, was the first to publish an epidemiological study to establish an increase in patients suffering from AN. Researchers in non-Western countries have reported increases in eating disorders (Abou-Saleh, Younis, & Karim, 1996; Ghubash, Hadi, & Bebbington, 1994). Ishikawa (1965) asserted that the number of patients in Japan had increased markedly since World War I. A more recent epidemiology study (Pawluch & Gorety, 1996) found countries having the highest incidence of AN were the United States, followed by Holland and Great Britain, respectively. Their findings also strongly suggested that, overall, the incidence of AN, particularly among those very young women at greatest risk of

experiencing it, had not increased significantly. However, the risk for women in their 20s and 30s appeared to increase significantly (Pawluck & Gorety, 1996).

Historical Overview

The term "anorexia nervosa" literally denotes a lack or absence of appetite of nervous origin. However, many have pointed out that anorexics do not suffer from a lack of appetite. AN was, according to some historians, first described in 1973 by Leseque in France and Gull in England (Bemporad, 1995). Contrary to this belief others believe that the existence of AN symptomatology has existed long before it was formally termed a disorder. Morton (1689) cited the first two cases of AN which, surprisingly, were male (Philpott, 1995;. Vandereycken & van Deth, 1994; Ziesat & Ferguson, 1984).

A wide variety of prominent figures have been identified as having AN, including the late medieval monarchs, Richard II and Mary Queen of Scots, and the nineteenth century poets Emily Bronte, and Emily Dickinson.

Very early in the history of neurology and the study of brain and behaviour, Franz Joseph Gall, in the first half of the nineteenth century, proposed a new doctrine of cerebral localization which was later called "phrenology". Cerebral localization assigns different bodily functions to various

parts of the brain. The premise that the mind can influence bodily functioning indirectly set the first cornerstone for psychosomatic medicine.

Years later, in 1834, French physician Fleury Imbert published a neurophysiological study on the sensations of hunger and thirst. Imbert's principal tenet was that the "brain contains all vegetal, intellectual, affective, moral, and psyche functions or capacities". In other words, the brain executes bodily functions. In his book, Traite Theorique et Pratique des Maladies des Femmes, under the heading "Neuroses of the Stomach", Imbert described three eating disorders: AN, BN, and pica. AN is further subdivided: "anorexie gastrique" (or "nervous stomach"), and "anorexie nerveuse" which was associated with a brain dysfunction. Typically, patients with the latter experienced a loss of appetite due to the brain's incapacity to excite appetite. In addition to the loss of appetite, these patients also displayed neurotic symptoms, particularly change of temperament, i.e.,they became melancholic, angry, or frightened, or a combination thereof.

Decades later, in 1860, Louis-Victor Marce published an article depicting a particular form of hypochondriacal delirium, which was characterized by food refusal and occurred especially in pubertal girls. Marce argued that

"It is not the stomach that demands attention, but the delirious idea which causes the malady". Thus there is a departure from the cause being biological or physiological but instead owing to the patient's lack of motivation or resistance to eat. Marce suggested that if the patient refuses to eat, the physician should employ intimidation and even force. Vandereycken and van Deth (1994) pointed out that Marce's accurate description went unrecognized by the medical community of that time because the description did not include the term "hysteria" (p. 149).

Just about the same time, in 1859, William Stout Chipley, chief medical officer of the Eastern Lunatic Asylum of Kentucky, gave a talk on the causes and treatment of "sitomania" or "stiophobia", an intense dread of eating or aversion to food. For Chipley, these "hysterical" females refused to eat as a means of gaining attention from those surrounding them.

In that same year, Pierre Briquet demonstrated more insight than any of his predecessors, with the publication of Traite Clinique et Therapeutique de l'Hysterie, a book considered to be highly remarkable for its time. Briquet denounced the long-presumed cause of hysteria as being attributed to "frustrated sexual instincts or insatiable erotic desires", a disorder more commonly seen among

Parisian prostitutes. Briquet argued that hysteria should not be located in the womb. He supported his view by demonstrating with figures that hysteria might occur in men as well, although the frequency would be far less (one in twenty). In is clinical work, Briquet described symptoms of hysterical hyperaesthesia of digestion, and took note of a peculiarity:

> "Despite frequent vomiting some of the women succeeded in staying 'fresh' and some stout. Some of them have an insatiable hunger so that their day passes, literally speaking, by eating in order to replace what they had just vomited, and by vomiting in order to deliver what they had just eaten" (Vandereycken & Van Deth, 1994, p. 152).

The latter symptoms, of course, appear very much like those associated with BN or BED.

Modern medical history credits two physicians, with the actual discovery of AN as a "new syndrome", an Englishman, Sir William Wishey Gull, and a Frenchman, Dr. Ernest Charles Laseque. Sir Gull was a prominent physician in England, who included among his many titles, that of Physician Ordinary to the Queen. In 1868 he described the "new syndrome, Apepsia hysteria", at the British Medical Association annual meeting, which was later published in its entirety in "The

Lancet". An excerpt reads:. .

> At present our diagnosis is mostly one of inference, from our knowledge of the liability of the several organs to particular lesions; thus we avoid the error of supposing the presence of mesenteric disease in young women emaciated to the last degree through hysteric apepsia by our knowledge of the latter affection. And by the absence of tubercular disease elsewhere" (Vandereycken & Van Deth, 1994, p. 156).

Months later Gull acknowledged Leseque's article and conceded publicly that the phenomena would be more appropriately termed "Anorexia". Descriptions of the new syndrome later appeared in German, Italian, America, and Dutch literature (Habermas, 1991; Van Deth & Vandereycken, 1990, 1993; Van't Hof, 1994).

Diagnostic criteria for eating pathology has evolved in the last thirty years. The diagnostic term, "anorexia nervosa", had its beginnings in the nineteenth century. Historians parallel the emergence of the disorder with the changing socio-economical changes brought on by the industrial revolution. The transition from home to the competitive mainstream of the workplace impacted especially upon women. Today, AN is a well-established disorder which is considered to be a morbid desire for thinness where the

individual uses self-starvation as a method of controlling weight.

BN is a fairly current disorder. It emerged as a separate syndrome from other eating pathologies during the 1970s (Palmer, 1987; Vandereycken, 1994). The literature indicates one of the earliest descriptions of binge eating was offered by Stunkard in 1959. Gerald Russell, in 1979, documented this disorder to be both a common and serious condition (Mitchell, Maki, Adson, Ruskin, & Crow, 1996). BN is characterized by cyclical episodes of binging and purging (the latter including self-induced vomiting), laxatives, diuretics, and associated impulsive behaviours (Kendler, et al., 1991; Crowther, Wolf, & Sherwood, 1993).

Another eating disorder is Binge Eating Disorder (BED), a very recent diagnostic category which emanated from research in the obese. The two core criteria of BED are episodic overeating, and loss of control. An episode may or may not be planned in advance and is usually (but not always) characterized by rapid consumption. The binge eating often continues until the individual is uncomfortably, or even painfully, full. Binge eating is typically triggered by dysphoric mood states, interpersonal stressors, intense hunger following dietary restraint, or feelings related to body weight, body shape, and food.

Binge eating may transiently reduce dysphoria, but disparaging self-criticism and depressed mood often follow (DSM-IV).

During the last five years, AN and BN have been re-categorized and divided into various subgroups reflecting differences in symptoms, severity, and duration. AN is further subdivided into Restricting Type and Binge-Eating/Purging Type. BN is subdivided into Purging Type and Nonpurging Type (DSM-IV). (See Appendix A for the diagnostic criteria for AN and BN which include subtypes.)

Investigators have made distinctions between restricting type and bulimic-type AN (Casper, Eckert, Halmi, Goldberg, & David, 1980; Garfinkel, Molodofsky, & Garner, 1980). For example, purging anorexics are described as having greater incidence of affective disorders and a poorer outcome than nonpurging anorexics (Favaro, 1995; Garner, Garner, & Rosen, 1993; Vandereycken & Pierloot, 1983). Data shows restricters to be perfectionistic and emotionally over-controlled, and bingers/purgers to be impulsive and emotionally labile (Jabalpurwala, Champagne, & Stotland, 1994; Johnson & Wonderlich, 1993; Sohlberg & Strober, 1994; Vitousek & Manke, 1994). Patients with BN tend to be heavier, more sexually active, and sometimes have severe depressive symptoms and more histories of suicide attempts

(Russell, 1979). Bulimic anorexics tend to be older and have a longer duration of illness (DaCosta & Halmi, 1992).

There is some controversy as to whether there are valid distinctions among the subgroups (Nagata, McConaha, Rao, Sokol, & Kaye, 1996; Tobin, Alexandra, Griffing, & Griffing, 1996). For example, Johnson and his colleagues argued that in restrictive and bulimic eating disorder variants, different adaptive styles may explain different expressions of what is often the same core disturbance (Johnson, 1991; Johnson & Connors, 1987; Steiger, Jabalpurwala, Champagne, & Stotland, 1996). People with AN may convert from one subtype to another over the course of their illness (Kasselt, Gwirtsman, Kaye, Brabdt, & Jimerson, 1988). Bulimic symptoms develop within about 1.5 years after patients have started restricting and losing weight (Garfinkel, et al., 1980; Nagata, McConaha, Rao, Sokol, & Kaye, 1996; Russell, 1985). Clinical research indicates that approximately 50% of patients with AN have symptoms of BN (Garfinkel, Moldofsky, & Garner, 1980), and across time, individuals with AN often become BN (Hsu, 1988).

BN is distinguishable from AN in that the anorexic patient through self-starvation continues to lose weight whereas the bulimic patient's weight fluctuates. The essential features of the bulimic patient are binge eating

and inappropriate compensatory methods to prevent weight gain. The bulimic has a sense of lack of control over eating during the episode (e.g., feeling that one cannot stop eating or control what or how much one is eating.) Self-evaluation measures indicate they are excessively influenced by body shape and weight.

Diagnostic criteria (Diagnostic and Statistical Manual, (American Psychiatric Association, 1994) .

AN and BN are clinically categorized in the most current DSM-IV under the rubric of "Eating Disorders".

AN is characterized by a preoccupation with body weight and food. The individual refuses to maintain a minimally normal body weight, is intensely afraid of gaining weight, and exhibits a significant disturbance in the perception of the shape or size of her body. In addition, post-menarcheal females with this disorder are amenorrheic. The patient exhibits peculiar patterns of handling food. Anorexics diagnosed binge eating/purging type are more likely to have other impulse-control problems, and substance abuse. Their mood is more labile and they are more sexually active.

Other features associated with AN (restrictive type) include concerns about eating in public, feelings of ineffectiveness, a strong need to control one's environment, inflexible thinking, limited social spontaneity, and overly

restrained initiative and emotional expression. (See Appendix A for DSM-IV criteria which include complete description of subtypes). Anorexics also consistently display ritualism, perfectionism, meticulousness (Pollice, Kaye, Greeno, & Weltzin, 1996), and harm avoidance (Steiger, Stotland, Ghdirian, & Whitehead, 1994).

BN is distinguishable from AN in that the anorexic patient, through self-starvation, continues to lose weight whereas the bulimic patient's weight fluctuates. The essential features of the bulimic patient are binge eating and inappropriate compensatory methods to prevent weight gain. The bulimic has a sense of lack of control over eating during the episode (e.g., feeling that one cannot stop eating or control what or how mc one is eating.) Self-evaluation measures indicate they are excessively influenced by body shape and weight.

Purpose.

This research is an attempt to parallel characteristics of AN and BN with those of certain depressive disorders in order to re-frame the etiology and progression of these eating disorders within the context of Seligman's model of learned helplessness depression. In this model the anorexic and the bulimic patients' symptoms are thought to be rooted in a need for control and a sense of power, which can be

traced to situations which had left these individuals feeling helpless, powerless, and not in control of situations and circumstances affecting their lives. These feelings ultimately develop into a depressive state similar to that of learned helplessness depression, a disorder first described by Seligman in 1975.

The aim of my thesis is to gain further insights into the development of AN and BN by re-examining the relationship between these disorders and depression. In this paper, I bring together the eating disorder clinical and experimental reports, and identify elements which support a learned helplessness depression model of AN and BN. Both theory and empirical research suggest that eating-disordered patients may have experienced a number of persistent antecedent stressful events (Rosen, Compas, & Tacy, 1993; Compas, Howell, Phares, Williams, & Giunta, 1989; Shatford & evans, 1986; Wolf & Crowther, 1983) which impacted greatly on their lives, leaving them feeling powerless, ineffective, dependent, and helpless. Onset of the eating disorder is dependent upon the time of life the events occurred. For example, these antecedent events could have occurred in infancy (e.g., crisis occurring during separation-differential phases (Friedlander & Siegel, 1990), during childhood (Rorty, Yager, & Rossotto, 1994), or

adolescence (Burns & Crisp, 1984; Carlat & Carmago, 1991; Crisp & Burns, 1983; Kinzl, Mangweth, Traweger, & Biebl, 1996). In addition, the antecedent events markedly affected and distorted future cognitions (Cattanach & Rodin, 1988; Shatford & Evans, 1986). The anorexic patient, in order to thwart depressive symptoms associated with helplessness and uncontrollability "exercises extraordinary attempts to control her appetite and to provide a long coveted sense of mastery within the context of lifelong feelings of incompetence" (Marx, 1991). Vandereycken and Van Deth (1994) wrote: "A paralysing sense of helplessness and low self-esteem or negative self-image seriously impede their psycho-social functioning despite the successful emaciation".

To view AN and BN within a learned-helplessness paradigm, anxiety needs to be considered as concomitant with depression. Depression and anxiety are related (Peterson, Maier, & Seligman, 1993), and often occur simultaneously with episodes of both disorders following each other in the course of time (Dobson, 1985; Tyrer, 1988; Steptoe & Appels, 1989). Garber and Seligman (1980) argued that many depressives display symptoms of anxiety because in real life many undesirable and often largely uncontrollable events involve both actual loss and the threat of loss particularly

when they are assessed over time.

The theory of learned helplessness (Seligman, 1975) has been referred to in one study of "dietary helplessness" in eating disorders (Carmody, Brunner & St. Joir, 1984) in which obese versus non-obese adults, and adults whose weight fluctuates versus adults who maintained body weight, were able to modify their unhealthy eating habits. "Dietary helplessness" was defined as the level of doubt in one's ability to modify unhealthy eating habits.

To date no other study has applied the theory of learned helplessness depression within the context of the etiology and progression of AN and BN.

Chapter 2

Literature Review

Current Theories

More than a century of scientific interest in AN has given rise to a host of hypotheses and speculations on the cause and meaning of AN. Despite a dramatic increase in research in eating disorders, gaps remain in our understanding of the factors responsible for the development and maintenance of dysfunctional attitudes and behaviours related to weight and eating (Garber, 1991; Emmett, 1985; van't Hof, 1994). There are distinct theories but none offer a "complete" explanation for the occurrence of AN and BN (Vandereycken & Van Deth, 1994; van't Hof, 1994). Most researchers have concentrated on multivariate models for the occurrence of eating disorders.

In addition, many clinicians and researchers have attempted to cast eating disorders within their own theoretical bent. Literature supports the view that AN and BN are disorders with distinctive physiological (Meltzer, 1987) cognitive, intrapsychic, and familial features (Becker, Bell, & Billington, 1987; BN: Humphrey, 1986a, 1986b, 1986c; Johnson & Flack, 1985; Minuchen, Rosman, & Baker, 1978). However, most theorists propose that eating

disorders are heterogenous and multi-determined in the sense that various predisposing factors can precipitate symptoms in vulnerable individuals (Anderssen, 1979; Casper, 1982; Emmett, 1985 Garner & Garfinkel, 1985;). Table 1 shows four levels of precipitating factors, biological, psychological, familial, and socio-cultural,which are presumed to contribute to AN. These four levels relate to the following six current theories.

Genetically Determined Disorders

In 1973 Hilde Bruch wrote: "Observations on monozygotic twins, with discordant occurrence of anorexia nervosa, do not support the assumption of a genetic or constitutional factor." Over twenty years later, new technology provides a contrasting view. The DSM-IV (APA, 1994) indicates that there is an increased risk of AN among first-degree biological relatives of individuals with the disorder. Studies of AN in twins have found concordance rates for monozygotic twins to be significantly higher than those for dizygotic twins. In addition an increased risk of mood disorders has also been found among first-degree biological relatives of individuals with AN, particularly relatives of individuals of the Binge-eating/Purging subtype. Studies have shown lifetime prevalence of eating disorders to be elevated among the female relatives of anorexics (Strober,

Lampert, Morrel, Burroughs, & Jacobs, 1990), and bulimic patients' (Kassett, et al., 1989) probands suggest eating disorders are a familial transmitted syndrome. In contrast, Steiger, Stotland, Ghradirian, & Whitehead (1994) examined

Table 1- A Multifactorial Model of Anorexia Nervosa
Source: Karren (1986); Dutch National Health Council (1989) Vandereycken and Hock (1993); Sonja van't Hof (1994)

	Biological level	Psychological level	Familial level	Socio-cultural level
Predisposing factors	Caucasian race; female sex	female gender; identity and autonomy problems	middle and upper social class; family interactions	industrialization; consumer society; pressures to be slim
Precipitating factors	development of secondary sex features; neurochemical and endocrinological changes	stressful events; sexual maturation	familial conflicts separation problems; pressures to achieve	pressures to be slim; pressures to achieve; risktul occupations (modeling, ballet)
Perpetuating factors	neurochemical and endocrinological changes	isolation fear of loss of control	as above	as above

dimensions along which familial transmission of eating disorders might occur. They compared self-reported eating concerns and psychopathological traits among selected proband samples and then among probands' first-degree relatives. They found no tendencies for mothers, fathers, or siblings of eating disorder probands to report elevated eating concerns or psychopathological traits.

AN illustrates one of the great paradoxes of behaviour genetics. One researcher concluded: "According to the data from a combined twin and family study using a small sample (Holland, et al., 1988), it appears to be "almost entirely genetically heritable (broad sense heritability = 98%), it seems to be entirely environmental because it only occurs in certain cultures" Barber, 1996).

Investigators continue to search for a biological substrate for AN (Gordan, Lask, Bryant-Waugh, Christie, & Timimi, 1996). What is known is that "virtually every endocrine system is altered by anorexia nervosa, as it is by starvation" (Gordon, et al. 1996; Spack, 1985), AN is the only psychiatric condition that, at present, requires an endocrine-linked abnormality, namely primary or secondary amenorrhea, for diagnosis (Eisenberg, 1981; The Study Group on anorexia Nervosa, 1994). Disturbances of the limbic

system, and the hypothalamic-pituitary-gonadal axis have been most frequently cited as the likely sites for an organic factor contribution to the etiology of eating pathology (Gordon, Lask, Bryant-Waugh, Christie, & Timimi, 1996; Wakeling, 1985), although the hypothalamic-pituitary-adrenal (HPA) axis, the hypothalamic-pituitary-thyroid (HPT) axis, the growth hormone (GH/somatomedin C (IGF-1) system, and the central peripheral arginine vasopressin (AVP) systems have also been implicated (Newman & Halmi, 1988; The Study Group on Anorexia Nervosa, 1994).

Serotonin Deregulation

Serotonin remains historically the neurotransmitter most intimately involved with neuropsychopathology. Serotonin deregulation is associated with both AN and BN. Serotonin is found all over the body; however, brain cells must synthesize their own supply. The first step is the uptake of the amino acid tryptophan which is the primary substrate for the synthesis. Plasma tryptophan arises primarily from the diet, and elimination of dietary tryptophan can profoundly lower the levels of brain serotonin. Ingestion of L-tryptophan has been shown in animals to increase brain serotonin levels (Fernstrom & Wurtman, 1971, 1972), and to decrease carbohydrate intake (Li & Anderson, 1984; White, et al., 1987). A decrease in

total food intake following L-tryptophan administration has been reported in some animal studies (Lacy, Van Krey, Skewes, & Denbow, 1986; Morris, Li, MacMillan, & Anderson, 1987), but not others (Weinberger, Knapp, & Mandell, 1978; Peters, Bellissimo, & Harper, 1984). In normal humans, ingestion of L-tryptophan has been found to diminish food intake (Hrboticky, Leiter, & Anderson, 1985; Silverstone & Goodall, 1986), and carbohydrate consumption (Silverstone & Goodall, 1986 Wurtmann & Wurtmann, 1981), although these findings are also inconsistent (Rogers, Binnas, Marthur, & Blundel, 1979). The system is not cause and effect inasmuch as brain levels of tryptophan are determined not only by the plasma concentration of tryptophan but also by the plasma concentrations of competing neural amino acids such as tyrosine and phenylalanine. The pineal gland located in the brain contains all the enzymes required for the synthesis of serotonin. It contains more than 50 times as much serotonin per gram as the entire brain. Serotonin is also involved in the production of melatonin. Melatonin content, and its influence in suppressing the female gonads is reduced by environmental lighting and enhanced by darkness through sympathetic innervation.

The physiology of eating, particularly postprandial satiety, is modulated by serotonin (Kaye & Weltzin, 1991).

Drugs that are agonists at serotonergic receptors or that increase intrasynaptic serotonin reduce food consumption. In low-weight anorexic patients, decreases in a variety of serotonin parameters have been consistently reported; those findings suggested reduced serotonin synthesis, uptake, and turnover, as well as reduced post-synaptic serotonin, receptor sensitivity (Kaye, Weltzin, 1991). Altered regulation of serotonin in patients with bulimia is evidenced by increased platelet serotonin uptake (Goldbloom, Hicks, & Garfinkel, 1990). Okamoto, et al. (1995) studied the intracellular calcium response to serotonin in BN and AN and reported enhanced calcium mobilization to serotonin in the bulimic group only. In contrast, Berk, Kessa, Szabo, and Butkow (1996) reported enhanced calcium mobilization in only the anorexic group.

Researchers point out difficulties and methodological issues in interpreting results from neurotransmitter research. The issue is to distinguish between state versus trait-related factors. State-related factors are those that are temporarily associated with weight loss and malnutrition whereas trait-related factors may contribute to the onset and maintenance of the disorders (The Study Group on anorexia Nervosa, 1994). Decreased central serotonin is suggested to contribute to associated symptom patterns

observed in bulimic patients, including episodes of major depression and impulsive behaviour patterns. However it is unclear whether central serotonergic changes in AN and BN suggested by the neurobiological studies are pre-existing factors, predisposing to the onset of the disorders (Jimerson, Lesem, Hegg, & Brewerton, 1990; Van't of, 1994). The neurochemical changes may also perpetuate the behaviour. It is claimed that the longer the disorder lasts, the more difficult it is to recover from it (Hsu, 1988; Van't Hof, 1994).

Socio-cultural and Feminist Theories

Van't Hof (1994) wrote: "Whenever a psychiatric disorder achieves social prominence, we are inclined to assume it is on the rise. And if it increasingly occurs, we assume it is related to particular cultural circumstances". The West German psychoanalysts Massing and Beckers (1974) felt that:

> "The traditional family was experiencing a clash with the values of a changing society. Additionally, emerging subcultures were causing changes in sexual attitudes and behaviour of the youth, notably girls, and ambivalent attitudes among teenagers about accepting a clearly defined sex role. Through magazines, television, and movies, the western culture

sends a constant and dangerous message".

In other words, the media message is "Thin wins", as more tersely described by Marx (1991).

Feminist/transcultural theory views the eating disorders as emanating from the stress women must endure to straddle two worlds, be it generational, work-family, cultural, or traditional and modern (Katzman & Lee, 1996). Carol Gilligan and her colleagues (1982) identified cultural pressures on women such as to be "nice" and to suppress their anger. According to these theorists, women are taught to value dependency and to define themselves through their relationships with others. The by-products of these pressures include depression, self-doubt, and decreased self-confidence (Marx, 1991). Thus, women employ food denial as an instrumental means of negotiating the transition, disconnection, and oppression, that they uniformly endure (Katzman & Lee, 1996). Another interesting concept along the same vein was developed by Barber (1996). Barber interprets AN and BN as "one nonfunctional manifestation in modern environments, of evolved psychological mechanisms which modulate the standard feminine bodily attractiveness as a response to economic conditions." He argued that women's changing roles from a primarily domestic and childbearing status, to a modern-day

competitive career status has been propelled by economics. He pointed out that the 19th century woman was heavier and more curvaceous, making her more appealing for childbearing and domesticity whereas the standard of modern day attractiveness tends to be "non-curvaceous" and serves to help women to fit in with the male-dominated realm of business by appearing both less feminine and more competent. He cites Silverstein, Perdue, and Peterson (1986) who suggested that there is prejudice against women in the work-place, in the sense that highly curvaceous women are perceived as lacking competence.

The American psychiatrist Sours (1969) theorized "that apparently a food rich culture is essential for anorexia nervosa as well as for politically coercive non-eating, if these behaviours are to have meaning to other people". In the last decade many studies have surfaced which examined eating disorders and our Western culture's role in emphasizing through the media the message that slenderness is a sign of sexuality, beauty, success and social competence. Schwartz, Thompson, and Johnson (1985) argued that AN is a "final common pathway" of a number of etiological factors, and "that the influence of social phenomena are not only an important part of the etiology, but may be the part that can explain the possible rise in

the incidence". Typically, eating disorders are thought to be a western "culture-bound syndrome" (Prince, 1983, 1985; Van't Hof, 1994; Russell, 1993). Prince (1985) defined a culture-bound syndrome as a "group of signs and symptoms of a disease that is restricted to certain cultures primarily by reason of distinctive psycho-social features of those cultures" (Van't Hof, 1994).

Researchers suggested that the westernization of certain cultures has given rise to increased incidence of eating disorders (Abou-Saleh, Younis, & Karim, 1996; Ghubash, Hamdi, & Bebbington, 1994). Other researchers after examining varying cultures found contrasting symptomatology among these populations (Abou-Saleh, Younis, & Karim, 1996; Kam & Lee, 1996; Lee, Lee, Leung, & Yu, 1997; Weiss, 1995). They suggested that the absence of certain characteristic features among AN and BN patients such as body distortion, fear of fatness, and the pursuit of thinness gives rise to alternative etiological theories. Although epidemiological studies differ in various international studies (Steinhausen, Winkler, & Meier, 1996), leading researchers in the field of eating disorders have argued against the use of restrictive diagnostic criteria, especially in comparative and epidemiological work (King & Bhugra, 1989; Lee, Lee, & Leung, 1996; Palmer, 1993; Patton

& Szmukler, 1995; Steiger, 1995) have recommended "a change of emphasis" in epidemiological research and propounded that the study of non-western groups is "likely to offer the greatest insight into the cultural determinants of eating disorders". The Study Group on Anorexia Nervosa (1994), in proposing directions for further research, likewise recommended that, "It is important to take a wide view and not to focus specifically on identifying cases defined on narrowly named western criteria". In addition, they emphasized the need to develop appropriate local instruments in the first instance.

Other researchers see through the seemingly obvious. For example, Vandereycken and Van Deth (1994) wrote: "A paralysing sense of helplessness and low self-esteem or negative self-image seriously impede their (anorexic patients') psycho-social functioning, despite the 'successful' emaciation". Bruch (1985) defined the paradox when she pointed out that social reinforcement does not adequately account for the development of AN since the emaciated state often achieved by most patients is well beyond the societal standards for shape. She further offers, "for the anorexic, hunger is associated with higher order accomplishment. The anorexic's attempt to control her appetite provides her a sense of mastery within the context

of lifelong feelings of incompetence".

More recently, researchers at The Hague Psychiatric Institute in the Netherlands (1998, [April 22nd] The Evening Telegram, p. 22) found the prevalence rates of AN in Curacao were similar to that reported in western countries. These researchers concluded that their findings challenged the idea that socio-cultural pressure to diet is a crucial factor in the causation of AN, and that it occurs only in western societies. (See Appendix B for newspaper article.)

Learning Theories

A learning theory perspective views AN as a kind of weight phobia which is rewarded in two ways: by losing weight fear of overweight is avoided and at the same time the anorexic is paid a great deal of attention particularly by her parents. It is important to note that AN is unlike a simple phobic disorder, since symptoms are maintained by positive as well as negative reinforcement. Weight loss provides not only a solution for avoiding the feared situation of fatness, but also a sense of gratification, control, and mastery in its own right. A higher weight is not simply avoided; a thinner weight is actively pursued through dieting. This view is linked to Marx' (1991) claim that anorexics are literally "starving for attention", and to systems theory, which regards AN as a sign of disturbed

structures and interactions within the family. Similarly, Slade (1982) viewed anorexic and bulimic behaviour as a response of the organism which, once precipitated, is powerfully reinforced by its consequences. Unlike Barber's (1996) "nonfunctioning" model of AN, Slade proposed a "functional" model of AN and BN. However, one might argue that both are similar inasmuch as both models describe maladaptive responses, or functioning, to precipitating and uncontrollable factors.

Slade (1982) suggested initial dieting behaviour is triggered by innocuous psycho-social stimuli in the context of major setting conditions of the individual's current-life situation. His functional analysis views current behaviour as a function of two sets of variables; namely antecedent events and consequences. Dieting behaviour, once commenced, leads to feelings of success and "being in control". It is reinforced both positively (by the resultant feelings of success/satisfaction, feelings of being in control) and negatively (through fear of weight gain and, alteration of body image, and avoidance of other problems, for example, physical threats (Schotte, 1992), and emotional threats (Heatherton & Baumeister, 1991; Herman & Polivy, 1980). Given the nature of the setting conditions, the positive and negative reinforcements accruing from dieting (weight

control) are sufficiently powerful to ensure that the behaviour is intensified with consequently increased contingent reinforcement of both a positive and negative type. Antecedent events are either general setting conditions such as dissatisfaction with life and low self-esteem, and perfectionistic tendencies. He suggested that general dissatisfaction with life and self, and perfectionist tendencies lead to the individual's need to control absolutely some aspect of his or her life situation, or attain some total control in at least one area. He argued that persons who have these psychological features will be predisposed toward body and weight control. Slade believed that adolescent conflicts/problems set the stage for self-esteem issues inasmuch as it is a critical transitional stage of development centering around issues of autonomy, and social anxiety which also involved peer pressure. The other factors which are hypothesized as contributing to the setting condition of general dissatisfaction with life and with self is that of stress and failure experiences. Studies have shown that stress and eating disorder symptoms are positively correlated (Shatford & Evans, 1986; Wolf & Crowther, 1983), Other researchers support the view that stress may be a significant antecedent to eating disorders (Cattanach & Rodin, 1988; Rosen, Compas,

& Tacy, 1993). The consequent downward spiralling of weight, together with the endocrine disorder eventually leads to AN. (See Table 2 for Slade's diagram of the etiology of AN.). Slade suggested BN as possibly developing from AN. The progression of the disorder is similar to that of AN as are the resultant feelings of being in control, and avoidance of other problems. (See Table 3 for Slade's diagram of the etiology of BN.).

Slade and Dewey (1984) later developed "SCANS" a 40-item, self-report questionnaire assessment instrument derived from the functional-analytic model of AN. They found that eating disordered individuals scored significantly higher on five components of the questionnaire: Dissatisfaction and loss of control, social and personal anxiety, perfectionism, adolescent problems and weight control. The two constructs considered to be "major" were general dissatisfaction and perfectionism. Further analysis of these constructs revealed that the general dissatisfaction scale provided reasonable discrimination between groups of eating disordered individuals and controls. Eighty-eight percent of eating disordered patients scored above the cutoff, and 78% of controls scored below the cutoff. In addition, on the perfectionism scale 95% of eating-disordered patients scored above the cutoff

Table 2. Slade's (1982) Diagram of the Etiology of Anorexia Nervosa

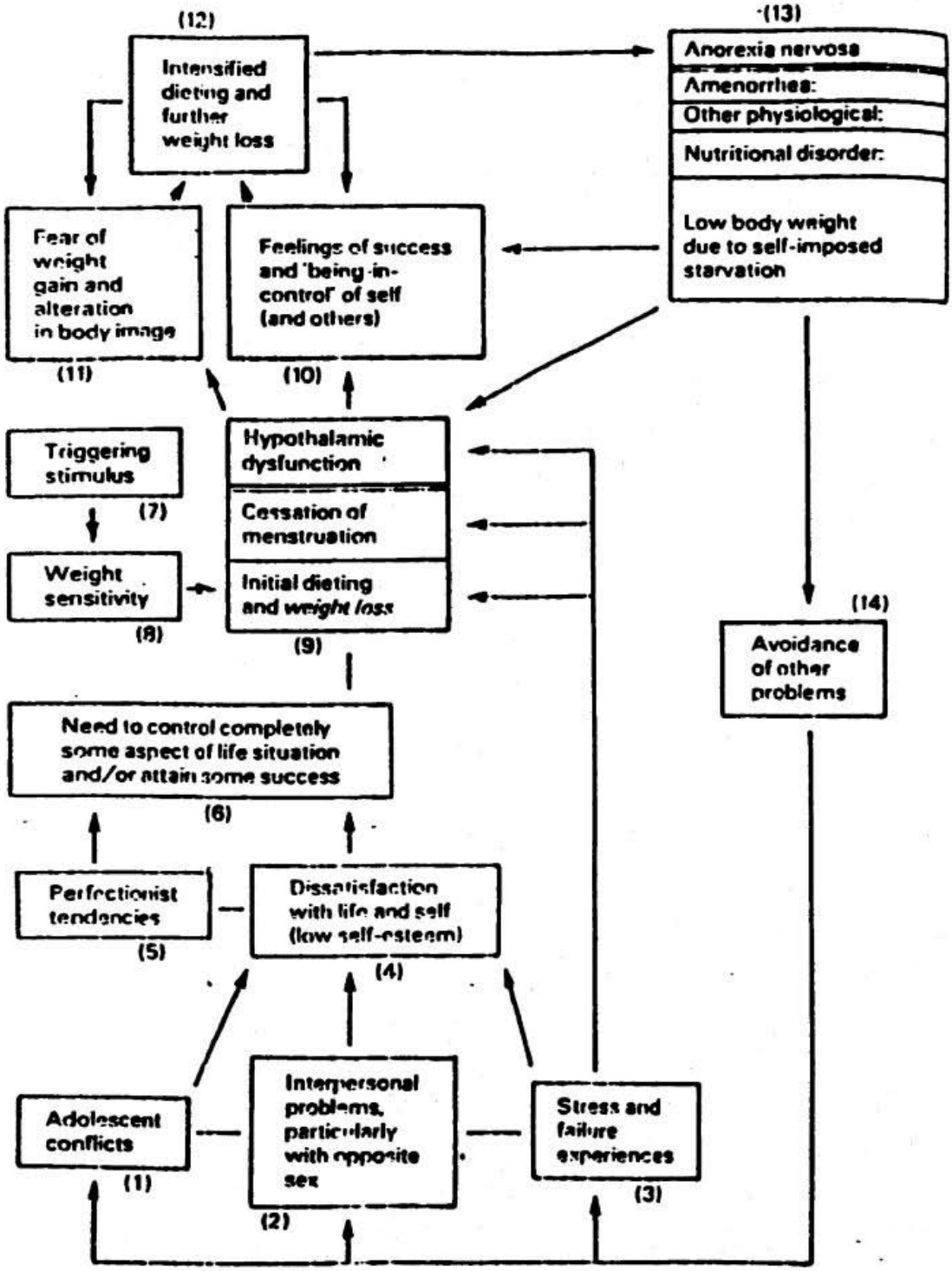
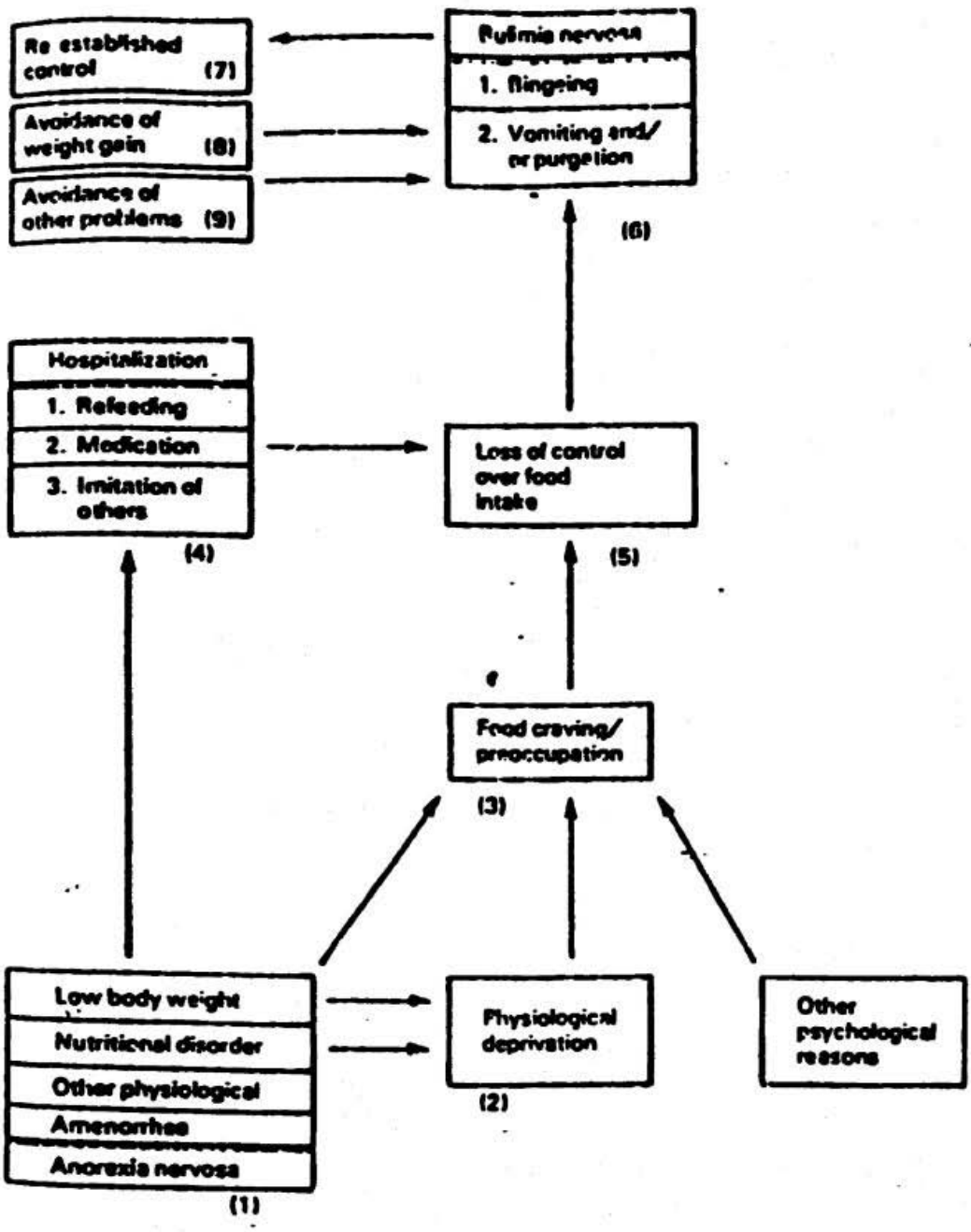


Table 3. Slade's (1982) Diagram of the Etiology of Bulimia Nervosa.



and 68% of controls scored below the cutoff point. They also found that the "need for control" construct turned out to be narrower and was restricted to two items concerning need for "weight and bodily control".

Family Theories

It has been speculated that the appearance and maintenance of eating-disordered behaviour may be attributed to certain familial characteristics which predispose vulnerable individuals to the development of these behaviours (Larson, 1991; Lindholm & Waters, 1991; Kinzl, Mangweth, Traweger, & Biebl, 1996). This idea dates back to the last century. Both Leseque (1873) and Gull (1968) regarded disordered family emotions an important factor in the course and outcome of AN.

According to Dare, LeGrange, Eisler, and Rutherford (1993), Minuchin and his colleagues (1975) provided "the most elegantly worked out description of the observed clinical features of anorexic and bulimic patients' families". Minuchin, et al, suggested that the eating-disordered child is physiologically vulnerable. The "sick" child plays an important role in the family's pattern of conflict avoidance, and this role is an important source of reinforcement. The child's family has four transactional

characteristics: enmeshment, overprotectiveness, rigidity, and lack of conflict resolution. Enmeshment is an extreme form of over-involvement (Minuchin et al., 1978) which gives rise to a low level of personal differentiation, of parental control over children, and of the boundaries between the generations. Families which are overprotective show a high degree of concern for each other's welfare (Minuchin, et al., 1978). Family members are preoccupied with nurturing each other. Thus family members are "hypersensitive" to signs of each other's distress. Critical remarks and demands are often accompanied by pacifying behaviours (Minuchen, 1975). Overprotectiveness also serves to reduce autonomy and differentiation of individual family members. Rigidity is the tendency to maintain the status quo. This characteristic serves to stagnate normal developmental growth processes. Psychological adaptation in response to physical growth and maturation become difficult. The rigid family is incapable of dealing with changing events (such as an adolescent negotiating individual autonomy) nor does it allow these events to surface to the point where they can be explored and where effective adjustments can be made. The last characteristic, lack of conflict resolution is derived from the families' rigidity, overprotectiveness, and enmeshment. Families adapt certain methods to avoid

conflict and therefore avoid methods of conflict resolution.

A wealth of clinical literature links eating disorders to dysfunctional familial development and structures. A few descriptive studies of anorexic clients' perceived and actual family relationships have indicated that family dysfunction is prevalent. These families are described as enmeshed, intrusive, hostile, and negating of the child's emotional needs (Strober & Humphrey, 1987). Dare, LeGrange, Eisler, and Rutherford (1993) in their study of 26 families of eating-disordered patients found low levels of emotional expressiveness. Families of anorexic patients were reportedly "characterized by conflict, avoidance, rigidity, and overprotectiveness. Families of bulimic patients were described as enmeshed but disengaged, with high levels of conflict and low emphasis on self-expression (Johnson, 1985); Hodges, Cochrane, and Brewerton's (1996) study of family characteristics of BED patients found a considerable amount of pathology, even higher than that reported in anorexic or bulimic patients, especially in the areas of family conflict, cohesiveness, and encouragement to express honest feelings. BED patients described their families of origin as limited in the emphasis of independence, and were more isolated and sedentary than anorexic or bulimic

patients. In addition, BED patients rated their families lower on organizational and control subscales with less structure, rules, and consistency or predictability than other eating-disordered groups.

Many of these studies involved clinical observation and the use of questionnaires. The most frequently used is the Family Environment Scale (FES: Moos & Moos, 1986), a 90-item self-report questionnaire. Thienemann and Steiner (1993) attempted to replicate the above findings using several measures, including the FES, and the Beck Depression Inventory (BDI: Beck, 1978) with an adolescent patient population. The BDI provides information about symptoms and the cognitive set of the individual. According to Beck, Rush, Shaw, and Emery (1979), depressed individuals selectively attend to internal and environmental stimuli in a consistently negative pattern. These researchers expected to find different family pathologies in accordance with different eating pathologies. Their results indicated that there were no significant relationships between the FES subscales and specific eating disorders. However, and quite interestingly, their results indicated a strong statistical relationship between the level of depression as measured by the BDI, and the negative report of family environment, independent of diagnosis. That is, the study found a

statistically significant association between the report of family environment and the degree of self-reported depression or depressive cognition. Thienemann and Steiner (1993) attributed the more negative cognitive set to the emotional immaturity of their population. They suggested that adolescents more readily regress to more primitive, polarized, negative thinking and reporting.

After ten years, a treatment team at the Children's Hospital Medical Centre in Boston, utilizing Minuchin's concepts of pathological family structures in addition to individual psychotherapy, concluded in 1980 that:

> "Many patients realistically did not fit with a redefinition of the anorexic symptomatology as exclusively a problem of family structure and interactional patterns....Predisposing vulnerability, intrapsychic conflict, and a developmental perspective must be added to family system considerations in formulating the pathology and appropriate therapeutic interventions in anorexia" (Piazza, Piazza, & Rollins (1980).

Benson and Futternan (1985) wrote that they do not believe family therapy by itself will insure permanent changes in the child's pathology, and that exploration of transactional patterns observed in early family sessions is achieved

largely in the individual work with the patient or the parents. Marx (1991) minimized the importance of using a label to characterize a family structure. He argued that:

> "There is no such thing as a 'typical' eating-disordered family. The same dynamic that triggers an eating disorder in one person may allow another to thrive. A family may unknowingly perpetuate an eating disorder. However, uncovering the hidden family meanings of a patient's anorexia or bulimia is often essential to her recovery" (p. 195).

Bruch (1982) was also not convinced of the sole contribution to the pathology that the family offers, when she wrote:

> "The stagnating patterns of family interaction must be clarified and unlocked, but this alone is not enough Regardless of what the family contribution to the illness has been in the past, the patient has integrated the abnormal patterns and misconceptions, and only individual intensive psychotherapy can correct the underlying, erroneous assumptions that are the precondition for the self-deceptive pseudosolution" (p. 305).

Psychodynamic Theories

The psychodynamic point of view states that anorexics are afraid of their sexual maturation and female growth. By

means of emaciation their growth and development are hampered in order to lead physically and mentally, a childish, asexual life (Vandereycken & Van Deth, 1994). With respect to AN both traditional (e.g., Bruch, 1985) and feminist (e.g., Orbach, 1981) psychoanalysts have emphasized the role of early disturbances in mother-infant relationships.

During the past ten years, a number of studies have been surfacing applying attachment theory (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969, 1973, 1980), and its antecedent, object relations theory or separation-individuation theory (Edward Ruskin, & Turrini, 1981; Mahler, Pine & Bergman, 1975), to eating pathology (Friedlander & Siegel, 1990; O'Kearney, 1995; Sharpe, et al., 1996). Attachment theory suggests that when a care-giver is consistently available and sensitive in response to an infant's attachment behaviours, the infant develops a sense of the care-giver as a secure base from which to explore the environment. Should danger arise, the attached infant is secure in the knowledge that there is a "safe haven" to return to (Armstrong & Roth, 1989; Bowlby, 1969; Hazan & Shaver, 1987). Such experiences make it more likely that the growing child will develop a sense of emotional comfort and self-competence. In contrast, those lacking a

secure foundation may develop a negative view of the self and may be at risk for the development of adverse outcomes (O'Kearney, 1995). Separation-individuation is the normal developmental sequence of achieving a sense of separate individual entity (Edward, et al., 1981).

Research encompassing these two psychodynamic theories emanated from family systems' clinical research. Palazzoli (1978) described a pathological relationship between the child and an intrusive, emotionally unresponsive and demanding mother, which results in the child's internalizing of mothering as a controlling function. Consequently the eating-disordered patient develops an insatiable need for control. Bruch (1985) provides her object-relations theoretical framework for the etiology of eating disorders which includes the clinical characteristics of "helplessness" and a need to "over-control". She wrote:

> "The absence of a responsive mother who consistently and appropriately meets her infant's needs, especially the need to eat, contributes to depriving the infant of the necessary foundation for a sense of body identity. As a result the growing child's perceptions of her own physiological functions become distorted. She fails to learn to differentiate biological disturbances from interpersonal and emotional ones. Such a child is

> typically not encouraged by her parents to be autonomous. Excessive obedience and conformity characterize the anorexic's personality. Later with the onset of puberty, she experiences a sense of helplessness in the face of increased biological urges (including hunger) and new emotional and social demands. Reactively, she may try to over-control (her) needs. Her obsessions and compulsions related to weight, eating and body-image may lead to self-starvation".

Masterson (1977) gives an account of the early mothering of the anorexic as rewarding of dependency, and at the same time threatening emotional abandonment when the child shows signs of separation and independence. The outcome for the child is an overwhelming fear of abandonment and confusion in attempts to separate and individuate at adolescence. This fear and confusion are resolved for the anorexic by an avoidance of physical maturity as an equivalent for delaying or avoiding independent psychological functioning. This concept coincides with that of Marx (1991) who contends that AN represents a way to avoid all aspects of maturity, i.e., physical, sexual, and social. By achieving thinness, the anorexic turns back the clock to a childlike physical appearance. Friedlander and

Siegel (199) tested the theoretical link between difficulties with separation-individuation and a set of cognitive-behavioural indicators characteristic of AN and BN. Their findings strongly supported a link, and indicated that predictive of anorexic behaviours were the pursuit of thinness, an inability to discriminate feelings and sensations, distrust of others, immaturity, and a belief about personal inadequacy. Their study also found that predictive of bulimic behaviours were dependency and a generally diminished sense of individuality, a general sense of inadequacy and worthlessness, and a poor sense of personal control.

Clinical Correlates

Issues regarding distinguishing subtypes of eating disorders has generated a number of studies on clinical correlates.

Body-Image Distortion

The diagnostic criteria (DSM-IV, APA, 1994) requires body-image distortion in the form of a specific psychopathology whereby a dread of fatness persists as an intrusive, over-valued idea. Body-image distortion refers to the patient's overestimation of their bodies. Bruch (1962) is credited as being the first to recognize dysfunctional body-image experiences as a core feature of

eating disorders, particularly AN (Cash, & Deagle, 1996). Rosen (1988) argued that body-image is multidimensional and that eating disorder pathology is secondary to body-image, a disorder unto itself labelled "Body Dysmorphic Disorder" (DSM-IV), which excludes AN as a criteria. Typically, researchers contend that eating-disordered patients do present with body dysmorphic complaints (Crisp, 1988; Sturmey & Slade, 1989). Others have suggested that general feelings of fatness are more typical among eating disorder patients (Thomas, 1987).

In recent years, researchers have documented the presence of body-image dysfunction and eating disturbance in adolescent females (Fabian & Thompson, 1989; Thompson, 1990, 1991). Despite extensive research over the past 30 years, body-image distortion and its role in eating disorders remain ill-defined and equivocal. Research generated from size estimation techniques have yielded inconsistent results (Hsu & Sobkiewicz, 1991; Szymanski & Seime, 1996). Williamson (in press) suggested that the body-image disturbances in eating disorders may be understood as information-processing biases. That is the overestimation of body size reflects a cognitive judgment bias rather than a purely perceptual bias (Cash & Deagle, 1996).

More current studies in other regions are finding that

the instruments which measure eating disorder symptoms may not be valid. For example, Chinese anorexic patients may score atypically low on the Chinese version of the Eating Attitudes Test-26 (Garner & Garfinkel, 1979). Although fear of fatness is common among modern young Chinese females (Lee, 1993), AN is not uniformly grounded in a desire to pursue slimness among Chinese patients (Lee, Ho, & Hsu, 1993). Western subcultures that anorexic patients experience are not homogeneously grounded in the fear of fatness (Banks, 1992; Lee, et al., 1993). Yager and Davis (1993) in the United States wrote that, "We are...also aware of how clinically different it can sometimes be to elicit a fat phobia in patients who have evolved a different type of attribution regarding why they are not eating". Steiger (1993) in Canada noted that "anyone who works with large numbers of anorexic sufferers knows that this disorder is not uniformly about a desire to be thin".

Some researchers (Cooper and Taylor, 1988; Cohen-Tovee, 1992) have suggested that depressed mood and low self-esteem could accentuate concerns with body shape, as a specific manifestation of a more general state of self-depreciation (Cohen-Tovee, 1992).

Risk Factors

Relative to other eating disorder research, few have

examined risk behaviour associated with AN and BN (Leung, Geller, & Katzman, 1995). Risk factors commonly associated with eating disorders are environmental pressures toward thinness (the western cultural influence), and parental psychopathology (Ledingham, 1990) where studies indicate that most major psychiatric disorders are more common in first-degree relatives of the affected person than would be expected by chance. Holzman (1982) proposed a risk-factor model consisting of two major types: biological and behavioural markers. Biological markers refer to biochemical, anatomical or neurophysiological traits, and neurochemical and endocrine abnormalities (Fova, Gopeland, Schweiger, & Herzog, 1989). Behavioural markers refer to observable behaviour patterns, signs, or symptoms (Leung, Geller, & Katzman, 1995). Some researchers (e.g., Nylander, 1971; Streigel-Moore, Silberstein, & Rodin, 1986) view eating disorders as part of a continuum, ranging from little concern with weight and normal eating to normative discontent with weight and moderately deregulated eating, to full-blown symptoms of AN or BN. Patton, et al, (1990) studied "attenuated eating symptoms". He found that at 12-month follow-up assessment girls who showed both eating and psychiataric symptoms at initial screening were more likely to have received a diagnosis of eating disorders than girls

who showed eating or psychiatric symptoms only. Schleimer (1983) observed that besides attenuated eating symptoms, all future AN cases also displayed multiple psychiatric symptoms associated with dysphoria and depression, and personality features such as low self-esteem during the premorbid phase of the disorder. As previously mentioned, Slade (1982) argued that eating pathology is a secondary adaptation to certain psychological "setting conditions". These conditions involve a combination of "general dissatisfaction with oneself and loss of control over one's life" and "perfectionist tendencies". One group of researchers (Kiemle, Slade, & Dewey, 1987; Slade & Dewey, 1986) attempted to identify women at risk for eating disorders on the basis of non-symptom psychological characteristics. These researchers examined two sets of hypothesized risk factors using the Eating Attitudes Test (EAT: Garner & Garfinkel, 1979). Their study found that individuals scoring above the critical cutoff points on the control/perfectionism measure scored significantly higher on all of the measures on the EAT questionnaire; while individuals who showed significant body dissatisfaction did not differ on as many factors concerning abnormal eating attitudes and behaviours compared with individuals who were more satisfied with their body image. Leung, Geller &

Katzman (1995) in their study of risk-factor models concluded that risk models are still in their formative years. They proposed that the non-symptom behaviour risk model represents one of the more promising avenues for future AN research.

Comorbidity

In the past ten years, investigators have been examining comorbidity of eating disorders with other psychiatric disorders (DSM-IV, APA, 1994), for example psychiatric disorders on Axis I and II (Lilenfeld, et al., 1997); Axis I, Schizophrenia (Deckelman, Dixon, & Conley, 1996; Kruger, Shugar, & Cooke, 1994); bipolar disorder (Kruger, Shugar & Cooke, 1994); the entire range of Axis II personality disorders within eating-disordered patients (Skodol, et al., 1993); and specifically within a BN population (Carroll, Touyz, Beumont, 1994). Wonderlich, Fullterton, Swift, and Klein (1993) examined the existence of personality disorders within an eating-disorder population at a five-year follow-up. Other researchers have narrowed their investigation to specific personality disorders, for example, obsessive-compulsive disorder (Pollice, Kaye, Greeno, & Weltzin, 1996; Steiger & Stotland, 1995); borderline personality disorder (Waller, 1992); Multiple personality disorder (Levin, Kahan, Lamm &

Spauster, 1992), and substance abuse (Wiederman, Pryor, 1995). One group of researchers examined eating pathology in a Down's syndrome patient (Raitasuo, Virtanen, & Raitasuo, 1996). Others have looked at generational indications of personality pathology in family members (Steiger, Liquornik, Chapman, & Hussain, 1990); and first-degree relatives of a bulimic population (Lilenfeld, et al., 1997). General psychopathology of Chinese anorexic patients had been demonstrated with the Minnesota Multiphasic Personality Inventory (MMPI: Hathaway & McKindley, 1967) which revealed abnormal elevation on the depression, schizophrenia, paranoia, and psychasthenia sub-scales (Lee, Lee, & Leung, 1996).

Swift and Stern (1992) suggested that eating-disordered patients can be broadly characterized as falling along a continuum of intrapsychic structures, ranging from borderline personality organization to identity conflicts. These researchers suggested the group of patients with "false self" organizations (Winnicott, 1965) are loosely grouped together by some of these common intrapsychic structures. In contrast, according to Vitousek & Manke (1994), "no consistent general personality variables or underlying core beliefs have been identified that uniquely characterize BN (Friedman & Whisman, 1996).

The most commonly observed psychopathologies are anxiety (Deep, Nagy, Weltzin, Rao, & Kay, 1994), and depressive symptoms (Cantwell, et al., 1977; Halmi, et al., 1973; Hsu, et al., 1979; Levy & Dixon, 1985). Deep, et al. (1994) suggested that childhood anxiety disorders may herald the first behavioural expression of a biological vulnerability in some individuals who later develop AN. Numerous studies have suggested that depression is prominent in the clinical presentation of both AN and BN (Hood, Moore, & Garner, 1982; Nolen-Hoeksema, 1987-1990; Skoog, Anderssen & Laufer, 1984; Strober, 1983-1985; Sykes, Gross, & Subishin, 1986; Wulff, 1932; Zutt, 1948).

Controversy surrounds the precise relationship between eating disorders and affective illness. Marx (1991) hypothesized that "the strong association between eating disorders and depression suggests a possible link somewhere in the brain." Some researchers have argued that eating disorders are atypical somatized expressions of depression (Cantwell, Sturzenberger, Burroughs, Salkin, & Green, 1993). Christensen and Somers (1995) studied the nutrient intake among depressed and non-depressed individuals and found increased carbohydrate consumption consistent with cravings characteristic of the depressed. These researchers suggested that this behaviour may relate to the development

or maintenance of depression and served to link behaviour to eating pathology commonly observed in BN and BED. Cooper and Taylor (1988) suggested depressed mood and low self-esteem could accentuate concerns with body shape. Tovees' (1992) study of a population of "normal young women" supports that view.

Various studies have reported that roughly 30-50% of patients with AN and BN suffer from concurrent major depression ; the fraction rises to one-half to three-quarters when past depression is included (Devlin & Walsh, 1989; Hudson, Pope, Yurgelun-Todd, Jonas, & Frankenburg, 1987; Swift, Andrews, & Barklage, 1986). Bulimics have been shown to be significantly more depressed than their non-eating disorder counterparts (Dykens & Gerrard, 1986; Katzman & Wolchick, 1984; Schmidt, Hodes, & Treasure, 1992; Yellowlees, 1985). Positive correlations between severity of depression and bulimic symptomatology have been reported (Casper, et al., 1980; Lee, et al., 1985; Strober, 1984). Fitzgibbon et al.'s (1997) study of binge eating in Hispanic, Black and White women found that binge eating severity was predicted by weight and depression in Hispanics and by depression in Whites. Nagata, et al. (1996) compared subgroups of inpatients with AN and found similar scores for depression and anxiety on restricting anorexics, bulimic

anorexics and anorexics with purging behaviours.

It is also evident that depression antedates the onset of eating disorders in many patients (Hudson, et al., 1983; Lee, et al., 1985; Piran, et al., 1985; Walsh, Roose, Glassman, Gladdis, & Sadik, 1985; DSM-IV, 1994). Binging appears to follow negative affective states (anger, depression, anxiety), serving the function of reducing awareness of those emotions (Lacey, 1986; Waller & Daman, 1996). Although malnutrition brought on by self-starvation and binging intensifies the severity of depression, anxiety, and obsessionality in anorexic patients (Pollice, Kaye, Greeno, & Weltzin, 1996), other studies indicate that depressive symptoms continue in anorexic patients despite weight recovery (Cantwell, et al., 1977; Halmi, et al., 1973; Hsu, et al., 1979; Levy & Dixon, 1985). Hudson, Pope, Jonas, and Yurgelun-Todd (1983) reported that among their eating-disordered patients with a lifetime major depression, the mood disorder preceded the onset of eating disorder in 40% of the casaes. Piran, Kennedy, Garfinkel, and Owens (1985) found that 36% of their 33 bulimic patients had an episode of major depression during their lives and the onset of depression preceded the eating disorder in 44% of these individuals. Walsh, Roose, Glassman, Gladdis, and Sadik (1985) also found that in at least 25 of their bulimic

patients with a history of depression, the first depressive episode clearly preceded the onset of the eating disorder. These statistics could even possibly be higher. Leung and Steiger (1990) suggested that "The problem with most studies is that depression in eating disorder-patients is usually examined with a retrospective design. It is therefore unclear to what extent these findings might have been confounded by limits in subjects' accuracy as historians".

The type of depression most often associated with eating disorders is unipolar depression. Research does not support the bipolar depression as a concomitant of eating disorders (Rubenstein, Steiner, Pratt, & Koran, 1989) inasmuch as most studies report zero or near zero lifetime occurrence of mania in eating disorders. On the other hand lifetime rates of major depression in BN are 47-73% (Hudson, Pope, & Jonas, 1983; Hudson, Pope, Yurgelun-Todd, Jonas, & Frankenburg, 1987; Walsh, Roose, & Glassman, 1985), and lifetime rates in BED are between 24% and 51% (Marcus, et al., 1990; Yanovski, Nelson, Dubbert, & Spitzer, 1993). In addition, patients with eating disorder pathology have been found to differ from those with major depressive disorder. For instance, Crow, Zander, Crosby, and Mitchell (1995) compared BN, BED, and major depressive disorder patients and found that BN, and BED patients showed greater concern with

weight and shape, and heightened sensitivity to the views of others in this regard. These characteristics are more closely associated with a low self-esteem and the use of external standards to judge self-worth (Boskind-Lodahl, 1976; Steiner-Adait, 1986).

Another interesting area of investigation is melatonin disturbances in patients with eating disorders and Seasonal Affective Disorder (SAD: Rosenthal, et al., 1984). The pineal gland releases melatonin into the blood stream in response to sympathetic noradrenergic stimulation of pinealocytes. This process is inhibited by light via the retino-hypothalamic-pineal pathway, which means that melatonin is predominantly released in darkness. Under normal conditions the pineal gland secretes melatonin in a circadian pattern with peak blood levels occurring during the night whereas daytime levels are low or below detectable values Lewy, 1984). SAD is a form of depression characterized by recurrent fall and winter episodes marked by hypersomnia, overeating and carbohydrate craving (Ash, Piazza, & Anderson, 1997; Jacobson, Sack, Wehr, Rogers, & Rosenthal, 1987). There is considerable interest in melatonin output in patients with mood disorders (Wbeck-Friis, Von Rosen, Kjellman, Ljumgren, & Wetterberg, 1984; Brown, et al., 1985; Wetterberg, 1978), and eating disorders

(Kent & Lacey, 1990; Lam, Goldner, & Grewal, 1994). Bright light therapy has been shown to be an effective treatment of SAD (Blehar & Lewy, 1990), and of AN (Ash, Piazza, & Anderson, 1997; Kennedy, 1993) and BN (Levitan, Kaplan, Levitt, & Joffe, 1993).

Menorrhea is an essential diagnostic characteristic of AN (DSM-IV, 1994). Both the amount and circadian pattern of melatonin output have been investigated in a number of physical and psychiatric conditions including major depression and reproductive dysfunction. Changes in many hormonal circadian rhythms have been described in both AN and BN (Kennedy & Garfinkel, 1987; Goldbloom & Kennedy, 1993). Neuroendocrine abnormalities including hypercortisolism and a blunted corticotropic response to corticotropin releasing factor (CRF) occur in patient groups with both AN and major depressive disorder (Gold, Loriaux, Roy, Kellner, et al., 1984; Gold, Loriaux, Roy, Lling, et al., 1984). Gotestam, et al. (1996) investigated the prevalence of eating disorders in Norwegian somatic hospitals for the years 1990-1994. A total of 3.3 million admissions were examined. They found a significant number of increased admissions beginning in the winter months of March and a minimum number of admissions in June when daylight hours are longer.

Depression

For the purposes of this thesis concentration will focus on three depressive disorders, two of which are indicated in the DSM-IV (APA, 1994); unipolar depression (see Table 4) and dysthymia (see Table 5). Both have been associated with eating pathology, and successfully treated with antidepressants. The third is Seligman's, et al. (1975-1993) learned helplessness depression. Seligman and his colleagues argued that:

> "Depression is wholly continuous with normal depression, whereas the medical community insists unipolar depression is an illness and normal depression is a transient demoralization that has no clinical significance" (Peterson, Maier & Seligman, 1993, p. 184).

Thus learned helplessness depression is not recognized to be a disorder per se, and therefore it is not indicated in the current DSM-IV as such. However, the symptoms of learned helplessness depression are similar to the aforementioned depressive disorders and more specifically to dysthymia. Symptoms associated with learned helplessness depression have also been successfully treated with antidepressants.

Table 4 Diagnostic Criteria for Unipolar Depression, DSM-IV (APA, 1994).

Unipolar (Major) Depression

Depression is categorized as a mood disorder. Essential features of depressive episode is either depressed mood or the loss of interest or pleasure in nearly all activities. In children and adolescents, the mood may be irritable rather than sad.

Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning: at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

(1) depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). Note: in children and adolescents, can be irritable mood.

(2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).

(3) significant weight loss when not dieting or weight gain (e.g., change of more tan 5% of body weight in a month), or decrease or increase in appetite nearly every day.

(4) insomnia or hypersomnia nearly every day.

(5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

(6) fatigue or loss of energy nearly every day.

(7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).

(8) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan,

or a suicide attempt or a specific plan.

The symptoms do not meet criteria for a Mixed Episode.

The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

The symptoms are not due to the direct physiological effects of a substance (e.g., a drug, alcohol, or medication abuse) or a general medical condition (e.g., hypothyroidism).

The symptoms are not better accounted for by bereavement, i.e., after the loss of a loved one; the symptoms persist for longer than 2 months, or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Table 5 Diagnostic Criteria for Dysthymia, DSM-IV (APA, 1994).

Dysthymic disorder is characterized by chronic, less severe depressive symptoms that have been present for many years. When dysthymic disorder is of many years duration, the mood disturbance may not be easily distinguished from the person's usual functioning. In children and adolescents, the mood can be irritable rather than sad.

An individual is diagnosed with dysthymia if "while depressed", two (or more) of the following criteria is met:

(1) poor appetite or overeating

(2) insomnia or hypersomnia

(3) low energy or fatigue

(4) low self-esteem

(5) poor concentration or difficulty making decisions.

(6) feelings of hopelessness.

The DSM-IV, includes: "The differential diagnosis between

Dysthymic Disorder and Major Depressive Disorder is made particularly difficult by the facts that the two disorders share similar symptoms and that the difference between them in onset, duration, persistence, and severity are not easy to evaluate retrospectively" (p. 348).

Learned Helplessness Depression

Seligman and his colleagues believed "unipolar depression and normal depression to be wholly continuous, the same phenomenon, differing only in the number and severity of symptoms" (Peterson, Maier, & Seligman, 1993, p. 184). However, my thesis parallels more closely learned helplessness depression behavioural symptoms with that of dysthymia (See Table 6). Symptoms include:

(1) Depressed mood: The individual feels sad, unhappy, discouraged.

(2) Anxiety. Mildly and moderately depressed people are almost always anxious. Anxiety may be an energizing response to danger, whereas depression is a de-energizing, conserving response. When danger threatens a person, his anxiety will be aroused and will fuel planning and action, or as long as he believes that some action might be able to help him. Once he expects with some certainty that he will be helpless, anxiety may drop out and be replaced with depression. Anxiety and depression can coexist when an

individual oscillates between the expectation that he will

Table 6. Learned Helplessness and Depression Parallels between Symptoms of Learned Helplessness and Depression. Source: Peterson, Maier, & Seligman (1993), Rosenhan, & Seligman (1989). Abnormal Psychology (2nd ed.), New York: Norton, 1989 By W. W. Norton & Company, Inc.

	Learned Helplessness	Depression
Symptoms	Passivity	Passivity
	Cognitive deficits	Negative cognitive triad
	Self-esteem deficits	Low self-esteem
	Sadness, hostility, and anxiety	Sadness, hostility, and anxiety
	Loss of appetite	Loss of appetite
	Reduced aggression	Reduced aggression
	Sleep loss	Sleep loss
	Norepinephrine and serotonin depletion	Norepinephrine and serotonin depletion
Causes	Learned belief that responding is independent of outcomes	Generalized belief that responding will be ineffective
Treatments	Change belief in response futility	Cognitive and behavioral therapy
	ECT, antidepressants	ECT, antidepressants
	REM deprivation	REM deprivation
	Time	Time
Prevention	Immunization	Invulnerability factors

be helpless and the hope that he might be able to do something about his situation (Garber, Miller, & Abramson, 1980).

(3) Lack of Hostility: Unlike Freud's (1917) view that *depression results from hostility turned inwards*, the learned helplessness model conceptualizes hostility as being suspended. According to this model, the absence of hostility among depressives is part of the global deficits in initiating voluntary action that characterize the disorder.

(4) Thought Dysfunction: Seligman acknowledged Beck's description of thought dysfunction as being the most accurate. For Beck (1967), depression is mediated by cognitions of helplessness, hopelessness, and pessimism. Beck (1967) described depressive thinking in terms of the negative cognitive triad: that is, negative thoughts about the self, ongoing experience, and the future. Beck argued that depressed people make errors of logic. For example, depressed people habitually "jump to conclusions from a single fact".

Learned Helplessness Theory

Although the learned helplessness model of depression was first formulated in the early 60s, psychodynamic

theorists have long recognized the role of helplessness and hopelessness in depression. For example, E. Bibring (1953) wrote:

> "What has been described as the basic mechanism of depression, the ego's checking awareness of its helplessness in regard to its aspirations, is assumed to represent the core of normal, neurotic, and probably, also psychotic depression" (Seligman, 1975, p. 95).

P. Lichtenberg (1957) viewed hopelessness as the defining characteristic of depression:

> "Depression is defined as a manifestation of felt hopelessness regarding the attainment of goals when responsibility for the hopelessness is attributed to one's personal defects. In this context hope is conceived to be a function of the perceived probability of success with respect to goal attainment".

In 1969, two attachment theorists, F.T. Melges and J. Bowlby, viewed pessimism and hopelessness as having a significant role in the development of depression when they wrote:

> "Our thesis is that while a depressed patient's goals remain relatively unchanged, his estimate of the likelihood of achieving them and his confidence in the

> efficacy of his own skilled actions are both diminished....the depressed person believes that his plans of action are no longer effective in reaching his continuing and long range goals...From this state of mind is derived, we believe, much depressive symptomatology, including...inability to act, and...feelings of worthlessness." (Seligman, 1975, p. 96).

Seligman formulated the theory of learned helplessness which began with the study of animal behaviour in the early 1960s. Experiments involved temporal variations of administering shock to animals. When shock is inescapable, the animal learns that it is unable to exert control over the shock by means of any of its voluntary behaviours. It expects this to be the case in the future, and this expectation of uncontrollability causes it to fail to learn the future. Seligman and his colleagues further suggested that the expectancy reduces the dog's incentive to attempt to escape, thereby producing a deficit in its response initiation. When the animal learns that it has no control and expects this to be true in the future, it undergoes motivational and cognitive changes that are responsible for its failure to learn to escape (Maier, Seligman, & Solomon, 1969; Seligman & Maier, 1967; Seligman, Maier, & Solomon,

1971). Cognitively there is an alteration in the manner in which the animal processes information concerning the learning task rather than just a diminution in its response initiation or movement. (Seligman, 1975). Animals previously exposed to inescapable shock may not register the contiguity between their response and the cessation of shock. Exposing an animal to a random relationship between a cue and a reinforcer interferes with it developing an association between them if they are subsequently made contingent. Animals previously exposed to uncontrollable shock may accurately register the co-occurrence of their behaviour with termination of shock but then not expect the relationship to hold in the future. It interferes with the actual learning of response-shock termination relationships, thereby producing a cognitive deficit (Peterson, Maier, & Seligman, 1993). Uncontrollability distorts the perception of control (Seligman, 1975). Aversive helplessness retards the solution of non-aversive cognitive problems (Hiroto & Seligman, 1974). In addition, Maier and Seligman (1976) proposed that learning that one has no control over aversive events leads to anxiety that is then followed by depression if the experience continues. This differs from the original behaviourist or Pavlovian stimulus-response paradigm which acknowledged sensitivity only to the temporal conjunction of

the response and the reinforcer. In contrast, learned helplessness theory proposed that the animal is able to detect cause-effect relationships, separating momentary noncausal relationships from more enduring true ones (Peterson, Maier, & Seligman, 1993).

The theory of learned helplessness involves three essential components: the first is contingency. This refers to the objective relationship between the person's actions and the outcomes that he then experiences. The most important contingency is uncontrollability, defined as a random relationship between an individual's actions and outcomes. The second component is cognition which involves the individual's perception of the contingency. A person's perception may, or may not be accurate. The last component is the person's behavioural response.

Typically, helplessness studies measure someone's passivity versus activity in a situation different from the one in which uncontrollability was first encountered, meaning the individual has "given up" and failed to initiate action. Helplessness theory claims that other consequences may also follow as well from the individual's expectation of future helplessness, i.e., low self-esteem, lack of aggression, sadness, an depression (Peterson, Maier, & Seligman, 1993). The existence of a strong relationship

between self-esteem and negative self-appraisal, and depression is well known and documented. Self-esteem is substantially lowered among depressives. The view of self-esteem as a symptom is supported by a large body of evidence showing that recovered depressives generally regain normal levels of self-esteem (Ingham, et al., 1986; Steptoe & Appels, 1989).

The behaviourist believes that depression is caused by a loss of reinforcers or the extinction of responding. Extinction refers to the contingency in which reinforcement is withdrawn altogether, so that the individual's response (as well as lack of response) no longer produces reinforcement. Loss of reinforcers, as in the case of the death of a loved one, can be viewed as extinction. In Seligman's (1975) view, it is not the extinction of reinforcers but lack of control over them that causes depression. Reinforcement may also occur with a probability greater than zero, and still be independent of responding. This is the typical helplessness paradigm; such contingencies cause already established responding to decrease in probability. The helplessness model, which refers to independence between responding and reinforcement subsumes the extinction view ad suggests that even conditions under which reinforcers occur, but independently

of responding, are not under the individual's control can also cause depression. In addition, Seligman (1975) suggested that what produces self-esteem and a sense of competence, and protects against depression, is not only the absolute quality of experience, but the perception that one's own actions controlled the experience. The degree to which uncontrollable events occur, either traumatic or positive, depression will be predisposed and ego strength undermined. To the degree that controllable events occur, a sense of mastery and resistance to depression will result (Seligman, 1975).

Reformulated Model of Learned Helplessness Depression

Since Seligman first formulated the learned helplessness theory in the 1960s using animal experimentation, several theorists have applied the theory to various human phenomena. The transition from animal experiments to human behaviour came under much scrutiny that created considerable controversy (Abramson, Seligman, & Teasdale, 1978), especially when applying the theory to depressive disorders (Depue & Montroe, 1978; Rizley, 1978; Wortman & Dintzer, 1978). Critics argued that the original learned helplessness model does not explain the self-esteem loss frequently observed among depressed people. Another question raised by critics was "What determines the

chronicity ad generality of helplessness and depression?" Abramson, Seligman, & Teasdale (1978) addressed these shortcomings by reformulating the helplessness theory to include the individual's causal explanations of the original aversive events (Peterson, Maier, & Seligman, 1993). According to the reformulated model, (Abramson, Seligman, & Teasdale, 1978), the attribution chosen influences whether expectation of future helplessness will be chronic or acute, broad or narrow, and whether helplessness will lower self-esteem or not.

The reformulated model of learned helplessness combines two prominent theoretical veins: that of attribution theory (Heider, 1958; Kelley, 1967) and locus of control theory (Rotter, 1966). Kelley (1967) suggested people perceive the factor that consistently covaries with an outcome as the cause. Rotter (1966) argued that people's beliefs about causality can be arrayed along the dimension of locus of control, with "internals" tending to believe outcomes are caused by their own responding , and "externals" tending to believe outcomes are not caused by their own responding but by luck, chance, or fate. The reformulated model (Abramson, et al., 1978) assigns three dimensions or parameters of causal explanation: internal-external, stable-unstable, and global-specific. Heider (1958) was one of the early

theorists who introduced the distinction between internal and external causes of events. Weiner (1972, 1974) is credited with introducing stable versus unstable causes, and the dimension of global versus specific was first introduced by Kelley (1967, 1972).

The first parameter makes the distinction between internal versus external causes. An internal explanation for uncontrollable events is associated with personal helplessness, because the uncontrollability is attributed to something about the particular person. An external explanation is associated with universal helplessness, because the uncontrollability is attributed to something about the situation or circumstance that would affect anybody placed in it. The second parameter is a causal distinction between stable-over time-versus those situations that are unstable. In other words, will the aversive situation last permanently or will it be transitory, or short-lived? The third parameter distinguishes between causes that are global (affecting a variety of outcomes and situations) and those that are specific (affecting few outcomes and situations). When perceived helplessness deficits occur in a broad range of situations, they are referred to as being global; and when the perceived deficits occur in a narrow range of situations they are called

specific.

The attributional reformulation of helplessness theory resolves two problems: Universal versus personal helplessness (and the role of self-esteem) is explained by introducing internal versus external causal explanations for uncontrollability. Generality of helplessness deficits are explained by introducing stable versus unstable causal explanations (influencing generality across time) and global versus specific explanations (influencing generality across situations).

Fundamentally, the reformulated model of learned helplessness, claims that depressed affect is a consequence of learning that outcomes are uncontrollable (Abramson, Seligman, & Teasdale, 1978). If an experience is less global it will be less likely to recur in a new experience or environment. Abramson, Seligman, and Teasdale (1978) posited that people, in the wake of uncontrollable events, ask "Why did this happen to me?" The reformulated model proposes that people susceptible to depression interpret these bad events in internal, stable, and global terms. And if reality is ambiguous enough, a person may project his or her habitual explanations onto it. Research indicates that it doesn't matter if the individuals are explaining hypothetical events or real events that have just happened

to them in the laboratory. Sweeney, Anderson, and Bailey (1986) performed meta analysis of 104 studies involving over 15,000 individuals that tested the relationship of explanatory style to depression. Depressed individuals were found to make more internal, stable, and global explanations for bad events (By between .34 and .44 standard deviations, than do non-depressed individuals. And depressed individuals make more external, unstable, and specific explanations for good events (by between .12 and .36 standard deviations) than do non-depressed individuals.

The reformulated model of learned helplessness was again revised by Peterson and Seligman in 1984. They fine-tuned the original terminology: "Attributional style" became "explanatory style" which allows for individual variation in response to uncontrollability. A key concept of the reformulated theory is explanatory style, defined as "the habitual lending to offer the same sorts of explanations for diverse bad events". In 1973 Kelley described a causal schema; an already abstract belief about the causes of particular events. When people encounter these events they draw on their causal schema to identify the cause. A causal schema is presumed to be imparted to an individual during socialization through a variety of means. Repeated experience with events that lead one to make the same

explanations should eventually produce a consistent explanatory style. Peterson, Maier, and Seligman (1993) believe that explanatory style can be learned as a whole and research supports this notion (Plous & Zimbardo, 1986; Seligman, et a., 1984). According to these authors explanatory style may even be "heritable" which involves environmental influences on genetic composition. In addition, these authors elucidated on the association between learned helplessness depression and the construct of controllability dimension. They posited that "controllability is not completely reducible to the locus and stability dimensions, and it could play an independent role in the etiology and description of learned helplessness and depression." Garber, Miller, and Abramson (1980) supported this view and argued that individuals who develop a generalized expectation of inability to control future outcomes are particularly vulnerable to the development of learned helplessness. They also suggested that individuals who believe that desired outcomes are not contingent on acts in their repertoires but are contingent on acts in the repertoires of others, will show lower self-esteem than individuals who believe the desired outcomes are totally unavailable.

Further, Peterson, Maier, and Seligman (1993) concluded

that:

> "Explanatory style is far upstream in the chain that causes depressive symptoms. A person need not actually experience repeated events in order for them to produce helplessness. All that is needed is for the person to expect that events will be controllable. Once someone expects responses and outcomes to be independent, he seeks a causal explanation from this non-contingency, which in turn determines the nature of helplessness deficits. Causal explanations influence expectations, and expectations are sufficient for helplessness deficits. The person's expectation that bad events are uncontrollable is the immediate cause of his depression (p. 212).

Chapter 3

Proposed Learned Helplessness Depression Model of Eating Disorders

As indicated in the previous section on comorbidity, controversy surrounds the precise relationship between eating disorders and affective illness. Some researchers have argued that eating disorders are atypical somatized expressions of depression (Cantwell, Sturzenberger, Burroughs, Salkin, & Green, 1993). Hudson, Pope, and Jonas (1983) hypothesized that BN could be conceptualized as an affective disorder variant. Under investigation is the question as to whether or not eating disorders are variants of depressive disorders or whether depressive symptoms occur secondary to eating disorders. Strober and Katz (1988) postulated that:

> "Depressive disorder, when it occurs in certain personality types, increases risk for the development of weight preoccupation culminating in eating disturbance. The connecting link here may be the enhanced sense of personal effectiveness associated

with dietary restriction that defends the individual against the painful and ego-disruptive feelings of emergent depression. Alternatively, changes in appetitive behaviour and weight loss brought on by incipient depression may expedite the transition into more pathological extremes of dieting behaviour by meeting certain preexisting intrapsychic needs and eliciting social reinforcement" (p. 101).

However, Strober and Katz (1988), along with many others (Altschuler & Weiner, 1985; Halmi, 1985; Swift, et al., 1986) were of the opinion that attempts to link eating disorders to affective disorders were premature.

Research generated since the 1980s have revealed numerous similarities between eating disorders and depression which overwhelmingly support the notion that eating disorders are variants of depressive disorders.

Gender differences exist in both eating disorder pathology and depression with women being far more likely to report these disorders. Studies on sex and depression found approximately a two-fold risk for females (DSM-IV, American Psychiatric Association, 1994). Differences also exist in the reporting of symptoms of depression in the general population (Byrne, 1981). Consistently, clinical and epidemiological studies have indicated that women report

this disorder to a greater extent than men (Broverman, et al., 1970; Byrne, 1981; Cooperstock, 1971; Gove & Tudo, 1973; Philips & Segal, 1979; Weissman & Klerman, 1977).

As stated in chapter 2, eating disorders have been associated with decreased levels of serotonin (5-HT), and Seasonal Affective Disorder (SAD). It has been suggested that the overall function of the serotonergic system is to enable the organism to ward off feelings of fear, helplessness, and depression. This concept of the role of 5-HT is consistent with the evidence that 5-HT has a significant role in the core behaviours that are disturbed in the affective disorders such as mood, sexual activity, appetite, and cognitive function (Meltzer & Lowy, 1987). Decreases in brain 5-HT concentrations can precipitate depression in recovered depressed patients (Meltzer, 1987).

Similar to eating disorders, research has shown numerous disturbances in circadian rhythm in depressed patients, for example the HPA axis (Sachar, Hellman, Roffwarg, Halpern, Fukushima, & Gallagher, 1973), the sleep-wake cycle (Wehr, & Goodwin, 1981), and body temperature (Avery, Wildschiodtz & Rafaelsen, 1982),

Recently psychiatrists at the University of Pittsburgh Medical Centre's Western Psychiatric Institute and Clinic, reported using Prozac (fluoxetine), an antidepressant drug,

for recovered anorexic and bulimic patients to prevent them from relapsing. Other physicians are prescribing Prozac as initial treatment as well as to prevent relapses (1997, May 27, The New York Times; see Appendix B). Clinically, fluoxetine is indicated in the Compendium of Pharmaceuticals and Specialties (CPS: 1998, Thirty-Third Edition, Canadian Pharmacists Association) as an anti-depressant, anti-obsessional, and anti-bulimic agent inasmuch as the action of fluoxetine is presumed to be linked to its ability to selectively inhibit the neuronal uptake of serotonin into human platelets. These physicians have cited success with the use of this drug. However, fluoxetine is not the treatment of choice for eating disorder patients. The CPS lists several precautions for the administering of the drug which include the pre-existence of anxiety and insomnia, significant weight loss, especially in underweight depressed patients. It reads, "Fluoxetine should be given with caution to patients suffering from anorexia nervosa and only if the expected benefits (e.g., co-morbid depression) markedly outweigh the potential weight reducing effect of the drug".

Pope and Hudson (1985) suggested future use of tricyclic antidepressants (TCA) for the use of AN. TCA's have been associated with excessive weight gain (Garland,

Remick & Zis, 1988; Russ & Ackerman, 1988). The mechanism of weight gain associated with TCAs is unknown and the relevant data are sparse (Russ, Ackerman, Schwartz, Shindledecker, & Smith, 1990). Studies of peripherally administered TCAs in animals have failed to demonstrate an increase in food intake (Blavet & DeFeudis, 1982; Nobrega & Coscina, 1987; Storlien, Higson, Bleeson, Smythe, & Atrens, 1985). However, infusion of TCAs into the paraventricular nucleus of the medial hypothalamus has been shown to increase food intake (especially carbohydrate intake) in rats (Leibowitz, Aromao, & Hamer, 1978). TCAs have side effects which can be quite annoying to an emaciated patient which include drymouth, blurred vision, constipation, and memory lapses (Pope & Hudson, 1985). Pope and Hudson (1985) pointed out that patients weighing only 88 pounds might require 120-160 milligrams per day, a dosage considerably higher than would be prescribed normally to achieve adequate antidepressant blood levels.

The existence of a strong relationship between self-esteem and negative self-appraisal, and depression is well known and documented. Self-esteem is substantially lowered among depressives. This view of self-esteem as a symptom is supported by a large body of evidence showing that recovered depressives generally regain normal levels of self-esteem

(Ingham, et al., 1986; Steptoe & Appels, 1989). Depressed mood and low self-esteem have also been found to accentuate concerns with body shape and weight (Cooper & Taylor, 1988; Cohen-Tovee, 1992) Low self-esteem is a result of the depressed individual's feelings of being inferior or ineffective. It is these core symptoms that create a bridge between eating pathology and learned helplessness depression. Within the context of learned helplessness depression, these symptoms arise from the individual's experiencing and attempting to cope with significant uncontrollable stressful events.

Seligman and his colleagues (1993) contend the central concept of learned helplessness depression is controllability, and many bad events are a subset of uncontrollable ones (Peterson & Seligman, 1984). The learned helplessness model posits that depressed individuals perceive themselves as unable to exert any volitional control to increase the probability of desired outcomes. The individual perceives herself as ineffective because she is ineffective at attaining goals which appear to be beyond her control. The lack of control is presumed to be internalized prior to the onset of depression in clinical patients (Litman-Adizes, 1978; Schwartz, 1964).

Similar to Bruch's (1985) object-relations theoretical

framework for the etiology of eating pathology, Seligman views early development to be crucial in the etiology of learned helplessness. In Seligman's view (1979), the "mother is the primary partner in the dance of development, the fountain of synchronies with the infant's responses and the main object of her contingency analysis". Seligman suggested that maternal deprivation results in a particularly crucial lack of control. The infant's sense of mastery or of helplessness develops from the information provided by mother's responsive reactions. If mother is absent, a profound sense of helplessness should arise.

The reformulated model of learned helplessness depression proposes that people susceptible to depression interpret bad events in stable and global terms. In line with this concept, current research indicates that eating disordered patients process information related to self-worth, especially in times of threat, according to stable-appearance related beliefs (Vitousek & Hollon, 1990). The eating-disordered individual uses external standards to judge self-worth. Bosking-Lodahl (1976) found that women suffering from eating disorders were high in passivity, dependence, and need for approval from others (Steiner-Adait, 1986). Weight and shape become grouped with other characteristics which would normally be associated with

self-worth. Eating-disordered patients have generalized this one aspect of their life to represent most others which are normally associated with an individual's self-worth. This reliance of external frames of reference in making self evaluations is reinforced by current cultural values; however in patients with eating disorders, the cognitions become crystalized and generalized (Garner & Bemis, 1982, 1985). In addition, anorexics have been described as having a perfectionistic personality style (Bauer & Anderson, 1989; Bruch, 1973; Cole & Edelmann, 1987; DSM-IV, 1994; Garner, 1986; Garner, Olmstead, & Polivy, 1983; Halmi, Goldberg, Eckert, Casper, & Davis, 1977; Slade, 1982). Burns (1980) described perfectionism in terms of a cognitive style characterized by dichotomous thinking and over-generalization, characteristics often observed in depressed individuals. A component of perfectionism is the proclivity for individuals to measure personal worth in terms of performance. Bruch (1978) made the clinical observation that anorexics usually excel in their school performance, and that the excellent academic achievements are not uncommonly the result of great effort. However, perfectionism, in most cases, is an unrealistic goal. Thus, highly perfectionistic individuals are prone to experience failure inasmuch as they have stringent criteria (Hewitt,

Flett, & Ediger, 1995). For the eating-disordered individual, perfectionism in scholastic pursuits becomes a means of trying to compensate for feelings of inadequacy and lowered self-esteem.

There have been a number of studies which have indicated eating pathology is associated with both an internal locus of control and an external locus of control. Individuals diagnosed as anorexic or bulimic, in comparison with non-eating-disordered individuals, have evidenced significant elevations on global measures of perceived internal control (Hood, Moore, & Garner, 1982; Strober, Salkin, Burroughs, & Morel, 1982), and perceived external control (Dunn & Onerci, 1981; Grace, Jacobson, & Fullager, 1985; Harding & Lachenmeyer, 1986; Rost, Newhaus, & Florin, 1982). Other studies have reported no group differences for either internal or external locus of control (Garner, Garfinkel, Stander, & Moldofsky, 1976; Hood, Moore, & Garner, 1982). Roth and Armstrong (1990), using a college sample found that issues of control differ in males and females, with women feeling less control over eating behaviours. Thus, they concluded that perceived control over specific eating behaviours is of core importance and is not associated with a more general sense of behavioural helplessness. However, they added that generalizing to a

clinical population raises some issues. One researcher, King (1989) concluded that the locus of control construct does not have a useful role in explaining eating problems. This would appear to contradict my thesis as the literature related to the reformulated model of learned helplessness depression indicates an internal locus of control to be a factor. However, the locus of control explanatory style does not entirely delineate the helplessness model of depression. Since the original locus of control theory was first formulated, other investigators have illuminated on its complexity. The construct was once thought to be unidimensional. More recently researchers have argued that the construct of personal control (Roth & Armstrong, 1990), and Rotter's (1966) Internal-External Locus of Control Scale are multidimensional and include several sub-factors (Abramowitz, 1973; Abramson, Schuludermann, & Schuldermann, 1973; Dixon, McKee, & McRae,1976; Mirels, 1970; Reid & Ware, 1973; Viney, 1974; Paulhus & Christie, 1982). Rotter (1975), himself, later altered his view about the complexity of the construct. Lazarus and Folkman (1984) argued that there is no single construct of control, rather it has many meanings and is used differently by different writers and even by the same writer at different times (Steptoe & appels, 1989).

To explain the internal-external locus of control attribution difficulty, we need only revisit the original animal experimental model of learned helplessness. Uncontrollable shocks produce learned helplessness in animals. The uncontrollability of the situation was the major contributing factor to produce the end result. Presumably the animal's realization was that the shock was in the external environment beyond it's control, which resulted in symptomatology looking very much like that seen in depression.

Fritz Heider (1958) believed that people look for meaning in their experiences. That is, a concern for causality is a basic aspect of human nature (Peterson, Maier, & Seligman, 1993). Waller (1996) suggested that consideration should be given to how perceived control varies as a product of the individual's experience. This concept is relevant to eating pathology inasmuch as perceived control does influence feelings of helplessness. Within the context of eating disorders, external locus of control is construed as the patient's perception of her lack of control over significant aspects of her life. The literature is replete with references to problems of control and dyscontrol in eating-disordered patients. A number of researchers (Garner, Garfinkel, & Shaugnessy, 1985; King,

1989) have demonstrated that women with established eating psycho-pathology have a relatively external locus of control (i.e., they see themselves as having low levels of control over events and their own lives. Feminist theories have argued that the inferior status of women and their early socialization lead them to be dependent and passive and to expect that they will be "helpless to control important events in their lives" (Nolen-Hoeksma, 1990). As indicated, Peterson, Maier, and Seligman (1993) posited that all that is needed for helplessness deficits to occur is the expectation that bad events are uncontrollable.

The causal chain to hopelessness and depression is hypothesized to begin with the occurrence of a negative life event (Abramson, Seligman, & Teasdale, 1978). According to the reformulated model individuals develop personality predispositions characterized by the expectancy or lack of control over life stress, and hence, experience feelings associated with learned helplessness (Abramson, et al., 1989). Stress is experienced when the situation is appraised as relevant to well-being and as outstripping coping resources. Lazarus (1984) defined stress as the relationship between an individual and the environment that is appraised as taxing or exceeding that individual's resources and as endangering his or her well-being. In

situations of uncontrollable stress, enhanced responding aimed at asserting control over an uncontrollable stressor must prove ineffective in the long run, for extended exposure eventually leads to the perception that no relationship exists between responses and outcomes. It is this "giving up" pattern which has been termed "learned helplessness" (Steptoe & Appels, 1989). A parallel can be drawn between stress related to learned helplessness depression and eating disorders. Some theorists have referred to the stress associated with cultural transition. The Dutch psychiatrist, Lafeber (1968) postulated that the status and role of women have quickly become very complicated causing uncertainty in traditionally reared girls as to what was expected of them. Steiner-Adait (1986) argued women develop eating problems as a way of meeting the social demands for thinness, controlling their own bodies, and coping with stress of self-doubts. In early writings many authors referred to reactive depression which was believed to be caused by environmental stress impacting upon the individual as opposed to endogenous depression which is caused by a biological chemical imbalance. Seligman (1975) paralleled reactive depression with learned helplessness. He suggested that reactive depression, as well as learned helplessness have their roots in the belief that valued

outcomes are uncontrollable. A similarity can also be drawn between DiNocola's (1990) term "cultural-reactive syndrome" when he referred to AN. He and others have observed that AN occurs mainly during conditions of rapid culture change, which could explain the increased incidence of eating disorders in immigrant groups. Ishikawa (1965) argued that anorexic patients tend to come from traditional ethnic families who try to adapt to a modern life style, but do not succeed.

Literature on eating pathology indicates that eating disorders occur modally at two crucial and stress-related developmental transitions: during the passage into adolescence and during the movement out of adolescence into young adulthood. The transition involves the integration of a changing body image into one's self-representation; the loosening of childhood bonds to parents and greater psychological and physical autonomy; the development of sexual relationships, the internalization of achievement values; and the organization of relatively stable and cohesive self-structure for the regulation of mood, impulse, and self-esteem (Attie, 1990).

Several studies have shown stress and eating disorder symptoms to be positively correlated (Shatford & Evans, 1986; Wolf & Crowther, 1983). Stressful life events or

difficulties have been shown to precede the onset of AN and BN in most cases (Schmidt, Tiller, Andres, Blanchard, & Treasure, 1997). Women with eating disorders also reported experiencing more stress currently (Soukup, Beile, & Terrell, 1990). In addition, stressful daily and major events are related prospectively to psychological and behavioural problems in adolescents (Compas, Howell, Phares, Williams, & Giunta, 1989; Wagner, Compas, & Howell, 1988) the population most at risk for eating disorders. Stress is believed to be an important antecedent to eating disorders, in particular, because it may disrupt normal appetite regulation, intensify body image preoccupation, or provoke maladaptive coping responses such as binge eating (Cattanach & Rodin, 1988); Compas, & Tacy, 1995; Shatford & Evans, 1986). Studies have indicated that adverse childhood experiences may lead to ego weakness, deficient self-esteem, maladaptive coping mechanisms, such as binging or chronic overeating, and labilization when the individual is faced with some stressors (Kinzl, Mangweth, Traweger, & Biebl, 1996). Slade (1982) emphasized the role of various stressors (e.g., failure experience, interpersonal problems, adolescent conflicts) which are posited to be one type of "setting condition" for eating disorders. He suggested that eating behaviour can have functional value in coping with

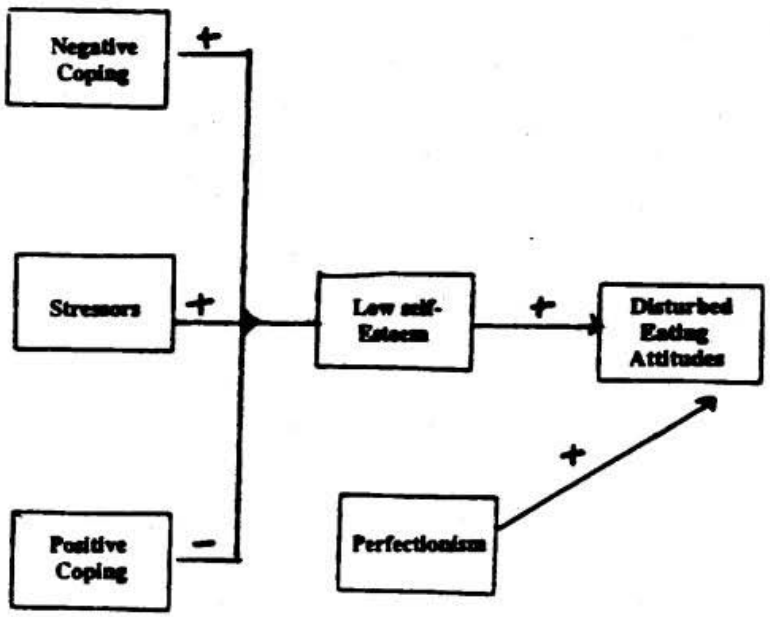
stress. He predicted that the experience of stress results in low self-esteem, which leads to disturbed eating attitudes. Fryer, Waller, and Kroese (1996) explored Slade's hypothesis. Results of their study indicated that increased stress coupled with maladaptive coping resulted in low self-esteem which in turn led to more disturbed eating attitudes, as illustrated in Table 7.

Stress involved with familial dysfunction and pathology have also been shown to contribute significantly to eating pathology. Mangweth, Traweger, & Biebl (1996) found an increased risk for eating disorders in men with adverse family backgrounds. Studies of anorexic and bulimic patients' perceived and actual family relationships have indicated that family dysfunction is prevalent. These families are enmeshed, intrusive, hostile, and negating of the child's emotional needs (Strober & Humphrey, 1987). Women with eating disorders reported having had significantly more physical punishment and perceived their discipline to have been more harsh and capricious than women in a control group (Rorty, Yager, & Rossotto, 1995).

Seligman (1975) hypothesized that helplessness may generalize readily from more traumatic and important events to less traumatic or unimportant ones, but not vice versa (Seligman, 1975). Women with unhealthy eating attitudes are

more likely to overeat following exposure to particular

Table 7. Proposed Model of the Etiology of Disturbed Eating Attitudes Adapted by Slade (1982), and Illustrated by Frye, Waller, & Kroese (1996).



types of threat (Waller & Meyer, 1996). Studies have examined perceived external and internal threats among eating-disordered people. Externally derived threats include separation or the loss of a relationship (Pyle, Mitchell, & Eckert, 1981), victimization (Root & Fallon, 1989), and fear of abandonment (Patton, 1992). Internal threats impact on self-esteem and centre on negative evaluation of self (Heatherton & Baumeister, 1991; Heatherton, Herman, & Polivy, 1991, 1992). Waller and Mijatovich (1996) studied 60 non-eating disordered women and found that women with healthy eating attitudes ate slightly more after exposure to ego threat, but not physical threat. In contrast, the group with "unhealthy" eating attitudes ate more after exposure to both forms of threat.

Dansky (1995) suggested that issues such as anger and powerlessness are associated with both eating disorders and the aftermath of victimization. Waller (1996) contends that poor perceived control may suggest a traumatic history in women with eating disorders. Vandereyckin, Van Dyck, & Vetommen (1992) concluded from their clinical work that the eating pathology started as a survival mechanism to escape from the psychological damage of overwhelming trauma.

An increasing number of studies refer to high incidence

rates of traumatic experiences (mostly physical or sexual abuse) in early childhood and adolescence of eating-disordered patients (Chandara & Malla, 1989; Finn, Hartman, Leon, & Lawson, 1986; Oppenheimer, Howels, Palmer, & Chaloner, 1983; Palmer, Oppenheimer, Dignon, Chaloner, & Howells, 1990; Root & Fallon, 1988; Schechter, Schwartz, & Greenfield, 1987; Sloan & Leighner, 1986; Waller, 1991; Wooley & Kearney-Cooke, 1986). Although there is no clear link to diagnostic status, reported sexual abuse has been shown to be associated with eating psychopathology (Everill & Waller, 1995; Waller, 1995; Wooley, 1994) particularly where the eating problem involved a bulimic component. Such abuse has also been linked to poor perceived personal control, especially if it involved experiences of childhood incest (Finkelhor & Browne, 1986). Waller (1996) found that eating-disordered women who reported any abuse had a core external locus of control than other women even though there was no difference in the extent of their eating pathology. According to this research the locus of control depends on the presence or severity of childhood abuse.

The primary thrust of the learned helplessness model of depression is that the depressed individual believes that active coping efforts are futile (Seligman, et al., 1975; Rizley, 1978). Coping refers to the constantly changing

thoughts and behaviours in which people engage in order to manage, tolerate, or reduce internal or external demands (Lazarus & Folkman, 1984). Peterson, Maier, and Seligman (1993) speculated that causal explanations and coping may be two sides of the same coin, and suggested that these constructs could be combined into one operational construct. Several schemas have been used to classify coping strategies; among these are an approach/avoidance model, which differentiates between coping responses that attempt to deal with stress through active behavioural or cognitive strategies and those that attempt to avoid dealing with the stress (Billings & Moos, 1981), and a problem/emotional focussed model, which differentiates between coping responses that focus on managing or modifying stress, and those that focus on regulating emotional responses to stress (Folkman & Lazarus, 1980). In general, the use of problem- or approach-oriented strategies is associated with better psychological functioning than the use of emotional- or avoidance-oriented strategies. Studies have shown that women are more apt to employ emotion-focussed and avoidance-oriented strategies than men (Endler & Parke, 1990; Ptacek, Smith, & Zanas, 1992; Koff & Sangani, 1996).

Eating disorder statistics which indicate a much higher prevalence of AN and BN among women than men. Koff and

Sangani (1996) found individuals with emotion-oriented coping should be considered high risk for eating disturbances. The question posed then is: Are women more vulnerable to learned helplessness depression and eating disorders because of the way in which they cope with stressful event? Also, it is widely recognized that avoidance behaviour is resistant to extinction because it insulates the individual from recognizing when adversive contingencies are no longer in operation. This could possibly explain the frequent relapses experienced by eating-disordered patients. Studies have shown anorexic women to have greater reward dependence, and anorexic and bulimic women to have high harm avoidance (Lacey, 1986; Waller & Osman, 1996). Some theorists have concluded that the anorexics avoidance behaviour appears to be unique to the disorder (Garner & Bemis, 1982) and that they make greater use of avoidance coping (Heatherton & Baumeister, 1991). According to Strober's (1991) model there are three personality components for the core of eating disorder vulnerability: low novelty-seeking, excessive harm avoidance, and reward dependence. Chesler (1996) reported on four cases of pathological coping. In each case the patient attempted to assuage panic with food, and instead exacerbated both the panic and eating disorder

symptomatology. Waller and Mijatovich (1996) suggested that preconscious activation of elaborate threat-related schemata among women with relatively unhealthy eating attitudes leads to subsequent "escape" behaviour (eating).

A theory of learned helplessness depression as the underlying premise associated with eating pathology requires a somewhat different perspective than the reformulated model of depression. In addition, in some respects, the theory must revert to earlier learned helplessness paradigms. According to Seligman (1975) depression is a convenient diagnostic label that embraces a family of symptoms, no one of which is necessary. Learned helplessness need not characterize the whole spectrum of depression, but only those primarily in which the individual is slow to initiate responses, believes herself to be powerless and hopeless, and sees her future as bleak, perceptions which began as a reaction to having lost her control over gratification and relief from suffering. Events that set off reactive depression are failure at work and school, death of a loved one, rejection or separation from friends and loved ones, physical disease, financial difficulty, being faced with unsolvable problems, and growing old (Paykel, Myers, Dienelt, Klerman, Lindenthal, & Pepper, 1968). Seligman (1975) believed that what links these experiences and lies

at the heart of depression is unitary: the depressed patient believes or has learned that she cannot control those elements of her life that relieve suffering, bring gratification, or provide nurturance. In short, she believes she is helpless.

Many writers prior to 1990 believed that linking eating disorders with affective disorders was premature (Altschuler & Weiner, 1985; Halmi, 1985; Strober & Katz, 1988; Swift, Andrews, & Barklage, 1986). Strober and Katz (1988) wrote: "The total body of empirical evidence accumulated to date yields little support for the overarching hypothesis that anorexia nervosa and bulimia nervosa are variants of unipolar or bipolar affective disorders". Seligman (1975) believed that cognitions are the core cause of depression. In recent years, researchers have turned their efforts to examining cognitive correlates of eating psychopathology (Fairburn & Cooper, 1989; Pike, Loeb, & Vitousek, 1996; Waller & Meyer, 1996). An increasing number of reports are becoming available which minimize the role of other variables (e.g., western culturalization, familial dysfunction) as contributing factors. My thesis contends that the last decade has generated considerable evidence to suggest that eating pathology is indeed a variant of depressive disorder, and more specifically, that which is

observed in symptomatology parallel those of learned helplessness depression as shown in Table 8.

Significance

AN and BN present a major therapeutic challenge because of the frequent chronicity and a mortality rate of up to 18% (Steinhausen, Rause-Mason, & Seidel, 1991). The significance of this research lies in the further understanding of the etiology of these two disorders, and in doing so, provides another focus for treatment.

Results from the treatment outcome literature for eating-disordered patients indicate that approximately two-thirds are significantly improved at one-year follow-up after receiving some type of traditional psychotherapy such as cognitive/behavioural, interpersonal, psycho-dynamic, psycho-educational, or psycho-pharmacological (Garfinkel & Garner, 1982, 1987; Johnson & Connors, 1987; Hsu, 1990). The remaining one-third appear to be more resistant to treatment (& Sansone, 1993). Two factors that have been found to be associated with predicting a poor treatment outcome are discord in the family environment (Blouin et al.), and the presence of Axis I and Axis II comorbidity (Mitchell & Groat, 1984; Johnson, Tobin, & Enright, 1990).

Researchers have stressed identification of differences between subgroups of eating disorders will lead to treatment

which could enable more appropriate decisions to be made

Table 8. Proposed Learned Helplessness Depression Model of Eating Disorders: Parallels between Symptoms of Learned Helplessness Depression and Anorexia Nervosa and Bulimia Nervosa.

Eating Disorders	Learned Helplessness Depression
Antecedent Events (Perceived or real stressors	Antecedent Events (Perceived or real stressors
Passivity	Passivity
Feelings of helplessness, hostility	Feelings of helplessness, hostility
Anxiety	Anxiety
Sleep loss	Sleep loss
Low self-esteem	Low self-esteem
Cognitive deficits Learned belief that responding is independent of outcomes	Cognitive deficits Learned belief that responding is independent of outcomes
Maladaptive coping strategies	Maladaptive coping strategies
Low energy, fatigue	Low energy, fatigue
Decreased serotonin	Decreased serotonin
Responds to antidepressants	Responds to antidepressants

(Carroll, Touyz, & Beumont, 1994). Marx (1991) argued that

> "a diagnosis means more than just giving a name to the disorder. It is also the beginning of the effort to find out why the disorder exists in this particular person at this particular time in her life. Once we have discovered why, the patient and I can then begin to find out how we might solve the problem".

Which one of the disorders, either AN or BN, or any of their subgroups, seems to depend upon the clinicians or researchers. In general, the treatment of AN has had limited efficacy, with many patients relapsing and having a chronic (Gordon, et al., 1996), and unpredictable course and outcome.(Katz, 1985; Rathner, 1992; Russell, 1992; Yates, 1990). Herpertz-Dahlmann, Wetzer, Schulz, & Remschmidt (1995) found patients with persistent eating disorders mostly suffered from restrictive symptoms. Purging anorexics are described as having greater incidence of affective disorders and a poorer outcome than non-purging anorexics (Favaro, 1995; Garner, Garner, & Rosen, 1993; Vandereycken & Pierloot, 1983). Bulimic patients have also been described as more difficult (Nagata, McConalia, Rao, Sokol, & Kaye, 1996; Russell, 1979). Although bulimic patients respond to psychotherapy, outcomes are quite variable. One-third of cases achieve abstinence from binge-

purge episodes during short-term treatment, but another one-third remain full symptomatic (Garner, 1987; Mitchell, 1991). Bulimic patients tend to be older with a longer duration of illness (DaCosta & Halmi, 1992). Clinicians and researchers argued that by the time these patients have been seen by specialists, many already have had long histories of the disorders (Ogg, Millar, Pusztai, & Thom, 1996). Studies have suggested that only a small percentage of women who suffer from BN or BED seek treatment for their condition (Shitaker, et al., 1990; Wells, Garvin, Dohm, & Striegel-Moore, 1996).

Current Therapeutic Interventions

Over the past decade much effort has been directed toward the development of effective therapeutic techniques to treat patients with eating disorders (Cooper, Coker, & Fleming, 1994; Schmidt, Tiller, Andrews, Blanchard, & Treasure, 1993; Treasure et al., 1994; Turnbull, Schmidt, Troop, Tiller, Todd, & Treasure, 1996). Pharmacological therapy has been used effectively for eating-disordered patients similar to those used for depression. A recent seminar sponsored by the National Institute of Mental Health (Bethesda, MD), and consisting of an international consortium of members meeting in Geneva on the topic of AN concluded that one of the promising pharmacological

treatments may be serotonin-specific drugs (The Study Group on Anorexia Nervosa, 1994). In the last few years, clinicians and researchers have been treating BN and AN with Prozac (fluoxetine), a very commonly prescribed drug used in the treatment of depression. The CPS (1998) indicates that Prozac is an effective treatment for BN but not AN, It is significant that Dr. Walter Kaye of the University of Pittsburgh Medical Centre's Western Psychiatric Institute and Clinic, found that Prozac can help keep people who have recovered from AN from falling back into self-starvation. (See Appendix B for news item on the use of Prozac). One might infer that it is quite probable that depression predates the onset of AN. It is only at the point where the individual has drastically reduced her weight that a drug such as Prozac would not be recommended.

Psycho-dynamic approaches to the treatment of An and BN have paralleled both the evolution of psychoanalytic theory and the development of psychotherapeutic technique. Treatment of choice evolved from classical psychoanalysis to the more contemporary therapeutic interventions such as family therapy which is often used in conjunction with Cognitive Behavioural Therapy (CBT) or Interpersonal Psychotherapy (IPT) (Benson & Futterman, 1985). CBT usually consisting of 12 group sessions is widely used in the

treatment of BN and BED: however reports indicated that these treatments offer only partial success (Agras, et al., 1995; Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990; Wilson & Fairburn, 1993). Similar results have been found for 12-session IPT (Agras, Telch, Arnow, Eldredge, Detzer, Henderson, & Marnell, 1995). Eldredge, et al., (1996) studied the feasibility of extending group CBT beyond the 12-session program. Their results indicated that a longer (24-session) program would likely maximize the number of potential responders to treatment.

Some clinicians and researchers have re-examined their treatment approaches. For example, Raitasuo, Virtanen, & Raitasuo (1996) described a complex clinical case involving a Down's syndrome patient with a diagnosis of major depression, AN, and obsessive-compulsive disorder. These authors suggested that perhaps the patient developed AN because "He was somehow trying to psychologically control his own body and inner life". They further hypothesized that "Anorexia developed because treatment of his depression was insufficient". The patient was eventually successfully treated for depression and along with it the eating disorder. Johnson and Sansone (1993) found that several of their patients after an unsuccessful long-term multi-modal treatment program, became involved with Alcoholics Anonymous

or Overeaters Anonymous and made remarkable recoveries. These researchers analyzed the 12-step program offered by these two groups and created one for eating disorders (See Table 9). The authors, themselves, admitted to being far removed from those pressures often experienced by their patients, and thus could not immediately appreciate the 12-steps of the AA program. The first item, which includes the patient's being "powerless" seemed to perplex them, and even evoke controversy from therapists with a feminist bent. They acknowledged that the concept of powerlessness became more palatable once they were able to understand the concept as the patient's effort to identify the things that can be controlled and the things that cannot be controlled. They maintained that the popular serenity prayer, which reads, "God grant me the serenity to accept the things I cannot change. Courage to change the things I can, and wisdom to know the difference (Neibuhr, 1932), captures the "spirit" or the "intent" of the concept of powerlessness.

The essence of the concept is to create a reality test for the individual. It creates an awareness which focusses on the patient's egocentricity. In other words, the patient soon learns to recognize her limitations, and in doing so,

Table 9. Twelve-Step Treatment Program for Eating Pathology Developed by Craig L. Johnson And Randy A. Sansone (1993) and Compared to Alcoholics Anonymous Original 12-Step Program.

1. We admitted we were powerless over Alcohol that our lives had become Unmanageable (A).	1. Confront denial.
2. Came to believe that a power greater than ourselves could restore us to sanity. (B)	2. Establish hope and faith.
3. Made a decision to turn our will and our lives over to the care of God as we understand him	3. Confront grandiosity/ omnipotence push to establish dependency away from substance and into rational sphere.
4. Made a searching and fearless moral inventory of ourselves.	4. Challenge to begin process of introspection.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.	5.
6. We're entirely ready to have God remove all those defects of character.	6. Confession and catharsis.
7. Humbly asked him to remove our shortcomings.	7.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.	8.
9. Made direct amends to such people wherever possible, except when to do so Would injure them or others.	9. Making amends (penance and undoing) in non-selfish manner.
10. Continued to take personal inventory and when we were wrong promptly admitted it.	10. A mechanism to continue catharsis and undoing.
11. Sought through prayer and mediation to improve our conscious contact with God as we understand Him, praying only for knowledge of His will for us and the power to carry that out.	11. Continuing confirmation of a new image of oneself.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics and to practice these principles in all our affairs.	12. Giving back through helping others and recruiting new members, redirection of energy through altruism and sublimation.

(A) Overeaters Anonymous substitute food for alcohol.
(B) Some groups substitute the word "Higher Power".

relieves herself of the pressures, and anxiety of having to engage in extraordinary and maladaptive methods to gain control of some situation which in reality is not within her control. The patient is not asked to give up something or to relinquish a right, but to realistically evaluate that which is within her control and that which is not.

Other researchers have argued for proper methods for earlier diagnoses and preventative measures to be implemented (Ogg, Millar, Pusztai, & Thom, 1995). A few have suggested education as a means of achieving primary prevention of eating disorders (Killen, et al., 1993; Moreno & Thelen, 1993; Moriarty, Shore, & Maxim, 1990; Paxton, 1993; Rosen, 1989; Shisslak, Crago, & Neal, 1990). Later analysis of these school-based interventions indicated that these programs may do more harm than good. In their evaluation of a school-based eating disorder prevention program designed to reduce dietary restraint among adolescents, Carter, et al. (1996) found that there was an increase in the level of knowledge about the nature and consequences of eating disorders which included methods to resist social pressures to diet. However, unexpectedly, at 6-month follow-up, while knowledge remained significantly higher than it had been at baseline, the level of eating disorder features returned to baseline levels. The level of

dietary restraint, the target variable, was higher than it was to begin with. This was the reverse of what they had hoped to achieve since dieting among adolescent girls appears to increase the risk of the development of an eating disorder (Carter, Stewart, Dunn, & Fairburn, 1996; Kindler, et al., 1991; Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990). Carter, et al. (1996) suggested that these prevention programs may be counterproductive in the longer term.

Another category of treatments is the self-help programs which have become more popular in the last decade. Increasing health-care costs in the United States, for instance, has prompted a move towards the less-expensive, briefer, and more symptom-focussed treatments. A few pilot studies offered initial information about the feasibility and effectiveness of self-help programs for the treatment of BN (Schmidt, Tiller, ad Treasure, 1993). Findings suggested that offering guidance along with these self-help programs may enhance the efficacy of pure unguided self-help interventions. Fairburn and Carter (1996) found that women who participated in 12-brief office sessions with a lay therapist reported greater binging and purging abstinence and lower levels of depression (Wells, Garvin, Dohn, & Striegel-Moore, 1996).

Schema-Focussed Cognitive Therapy was developed for the treatment of personality disorders (Beck & Young, 1989). The rationale for this method of therapy is that all cognitions and behaviour patterns can be changed, and the patient has access to feelings, thoughts, and images. It is a brief therapy lasting 16 to 20 sessions. The therapist forms a therapeutic alliance with the patients in a collaborative working relationship. In line with this treatment is the Cognitive-Behavioural Body-Image Therapy which involves appearance-related schemas, strength of internalized physical ideals, appearance-management behaviour, and effect (i.e., *situational body-image* dysphoria). This therapy has been successfully applied to eating-disordered populations. Researchers agree that controlled investigations of the independent and additive-efficacy of body-image, schema-focussed treatments for eating *disorders are lacking and greatly* needed (Cash, 1992; Cash & Deagle, 1996; Cash & Grant, 1996; Rosen, 1990).

Due to the greater number of females with eating disorders, and the importance of the patient's concern with body-image, over-involvement with mother, ambivalence about *gender identity*, and the need for a role model, some authors concluded that women therapists have more to offer than males when treating eating-disordered women (Stockwell &

Dolan, 1994; Zunino, Agoos, & Davis, 1991). Kopp (1994) suggested that there are difficulties for both male and female therapists who work with the eating-disordered population. Waller and Katzman (1996) assessed the client and clinician characteristics associated with choosing a male or a female therapist for eating-disordered individuals. Twenty-seven clinicians experienced with working with eating-disordered patients were asked to judge whether they would be more likely to recommend a male or a female therapist for an adolescent client presenting with eating problems given different clinical features. Results of this study indicated that client characteristics associated with recommendation for a female therapist included: a history of paternal sexual abuse, body-image issues, and an overprotective mother. In addition, the recommendation of a female therapist was more likely if the participants were older, and less likely if the clinicians were medically qualified.

Treatment of Learned Helplessness Depression

Seligman (1975) suggested that reactive depression as well as learned helplessness have their roots in the belief that valued outcomes are uncontrollable. Forced exposure to the fact that responding produces reinforcement is the most effective way of breaking up learned helplessness. The

central goal, then, in successful therapy is to have the patient come to believe that her responses produce the gratification she desires; that she is an effective human being. Many have commented on the fact that Princess Diana's advocacy work had become foremost in her life, second only to the care of her children. When she became increasingly more involved with her charity work she began to recognize the value of her own contributions. Eventually the opportunity of doing her advocacy work afforded her the proper, public exposure and acceptance which provided her with a more comfortable vehicle in which to speak openly about her eating disorder. In the words of her couturier, Catherine Walker, "Diana, by developing her status as a charity worker, was regaining her self-esteem, mending her heart. She was at peace with herself" (1998, September 7, People Weekly).

Seligman (1981) hypothesized that explanatory style could be changed by the therapist in at least two ways: First, the therapist underscores the possibility of causal explanations other than those chronically favored by the individual; and, secondly, the therapist directly challenges the explanations the individuals make, as Beck described in his cognitively-based psychotherapy. Beck's (1970-1971) cognitive therapy is aimed at changing the negative

expectation of the depressed patient to a more optimistic one, in which the patient comes to believe that his responses will produce the outcomes he wants. Psychoanalytic techniques to treat depression also involve helping the client free herself from the conditions that led her to become hopeless, to recognize unused opportunities, to identify more realistic and reachable goals, and to adopt more effective alternative plans.

More recently, Beck (1989) developed upwards of 15 schemas which include various clusters of cognitions termed "domains". For instance, the "domain" of autonomy, involves schemas having to do with dependence, subjugation or lack of individuation, vulnerability to harm and illness, fear of losing self-control, etc. Wells and Mathews (1994) have noted that cognitive theory and therapy are often based on an inadequate understanding of the content of the presumed schemata. This thesis suggests a Helplessness/Hopelessness domain which would include combinations of many of the existing clusters, as well as some additional ones involving the need for control, and power.

Concluding Remarks

I am reminded of a quote: "We are card-carrying simplophiles in a field dominated by complophiles" (Peterson, Maier, & Seligman, 1993). I have presented this

thesis in much the same manner. Unlike previous theories which assume a multidetermined etiology for a AN and BN, my thesis views the disorders as emanating from a core feeling of helplessness which results in a persistent depressive or negative temperament. My thesis proposes a common denominator among all the variables presented in the literature. The core theory is that eating pathology is characterized by learned helplessness depression, the severity of which encompasses a spectrum of depression, but more commonly dysthymia. It suggests that learned helplessness depression is the "depression variant" referred to by several researchers. Also, in situations where the eating-disordered patient does not readily display depressive symptoms, the symptoms are masked by the eating disorder. Strober and Katz (1988) suggested a convincing explanation for the phenomena: "The connecting link (to depression) here may be the enhanced sense of personal effectiveness associated with dietary restriction that defends the individual against the painful ego-disruptive feelings of emergent depression."

Although my thesis may appear to be simplistic through generalizing a core explanation for the numerous variables involved in eating pathology, most prominently the paucity of information concerning eating pathology in males, it is

the belief of this writer, and others (Tanofsky, Wilfley, Spurrell, Welch, & Brownell, 1995) that the pathogenic pathway resulting in eating disorders in females is similar in males diagnosed with eating disorders. When eating disorders are present in males, they, too, suffer the effects of learned helplessness but their symptomatology is not as easily detectable. For example, males who present for treatment for BED are very similar to females, but it is suggested that males may need to experience a greater level of distress before seeking treatment (Tanofsky, Wilfley, Spurrell, Welch, & Brownell, 1995).

This thesis may, on the surface, appear contrary to Russell Marx's (1991) view regarding treatment.

> "The (medical)...approach takes each person's individuality into account and looks at all the forces that may be contributing to the disorder. It also lets the physician choose from many therapeutic strategies to determine the ones that have the best chance of working. Such an approach is vastly superior to one in which a doctor assumes from the beginning that all eating disorders result from one cause - for example, a defective sense of self-esteem. Such restrictive, simple-minded thinking can lead to one-dimensional therapy that fails to address the multifaceted,

perhaps...human nature of the disorder" (p. 11).

Yet, Marx (1991), in his book, It's Not Your Fault, illustrates the fundamental basis of the patient's cognitions. It is obvious that the common underlying thread is the patient's desire for control and mastery. The last quote illustrates the continuous sense of helplessness that pervades the anorexic's life, and her attempt to regain control of aspects of her life other than just weight. He illustrates the difference between the anorexic and the bulimic:

> "In the adolescent girl, 'The notion that..."There is something wrong with me" becomes...."There is something wrong with my body -it's fat'....Ironically, her "realization' that she is fat, though distorted and incorrect, produces a tremendous sense of relief....She has identified the problem, and now she can begin to manage it....She successfully loses weight, which provides a sense of accomplishment. Feelings of insecurity fade, replaced by a sense of mastery, competence, and self-control....No longer is she a passive, helpless victim of her inadequacy....She is regaining control over her life (pp. 34-35).
>
> "The bulimic thinks 'It's all my fault....If I were

> stronger, I'd be more in control. "I'd be thin." In bulimia, overcontrol produces a lack of control. Trying to govern something that is ungovernable-the need to eat-just leads to the feeling that one is out of control-the cycle begins. Bulimia lets her control at least one aspect of her life-eating-to make up for lack of control elsewhere. Achieving a sense of thinness can increase feelings of power and self-worth" (p. 39).

In another quote, Marx (1991) reflects the anorexics anxiety and perception of hopelessness: "The anorexic copes with her pain through disturbed eating because she fears that if she doesn't do something, anything her life will get worse."

My thesis does not dismiss the multitude of variables that contribute to the pathology, nor a multifaceted treatment approach, but contends that whatever the mode of therapy, be it educational, behavioral, cognitive-behavioral, or family, the therapeutic objective needs to address the individual's feelings of powerlessness, and helplessness. The therapist needs to help his or her patient regain a sense of personal control and of hope. This notion coincides with Waller (1996) when he suggested that "Clinical work with eating-disordered women who report a history of sexual abuse may derive particular benefit from

a focus on cognition regarding personal control", but Waller narrowed the focus to only those patients with a history of sexual abuse.

PART II

Proposed Empirical Research

This thesis is organized in two parts: The first is a literature review which provides the basis for the conceptualization for the etiology of eating disorders within a learned helplessness model of depression. The second part is a proposal for empirical research which will explore the relationship between AN and BN and hopelessness in order to determine the appropriateness of reframing AN or BN within such a model.

Methodology

It is hypothesized that antecedent events occurring at different stages of development, more commonly during infancy and adolescence, contribute to the etiology and progression of AN and BN. These antecedent events create a residue of distorted cognitions and maladaptive coping mechanisms in an attempt to overcome present feelings of helplessness and uncontrollability.

According to learned helplessness theory (Abramson, Seligman & Teasdale, 1978), the construct of hopelessness can be viewed as a global attribution of helplessness which occurs across situations and across time, reaching far into the future. That is, the learned helpless individual

inappropriately generalizes the expectation of noncontingency to a future, controllable situation. Hopelessness has been identified as one of the core characteristics of depression (Beck, 1963, 1967; Melges & Bowlby, 1969), and learned helplessness depression (Abramson, 1989).

Procedure

This research will compare a non-clinical population to a clinical eating-disordered population, to establish an inter- and intra-relationship among four variables: 1. the presence of eating-disorder behaviours and symptomatology (e.g., bulimia and anorexia, perfectionism, and feelings of ineffectivenss), as measured by the Eating Disorder Inventory (EDI: Garner, Olmstead, & Polivy, 1983); 2. the presence and severity of depression as measured by the Beck Depression Inventory (BDI: Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); 3. the relationship between aspects of the locus of control construct using the Adult Nowicki-Stuckland Internal-External Control Scale (ANS-IE), (Nowicki & Duke, 1974); and 4. the presence of the hopelessness construct, defined as a negative expectancy about the future (Minkoff, Bergman, Beck & Beck, 1973) as measured by the Beck Hopelessness Scale (BHS: Beck, 1978-88). The study will be implemented in two phases. The first will involve

dissemination of the four questionnaires to a volunteer population comprised of both male and female undergraduate and graduate university students. Data will be collected and analysed. Two groups will be selected: an anorexic-type group and a bulimic-type group, as distinguished by the EDI-1. Intra-relationships between these two groups and the scores on the three measures, the BDI, the ANS-IE, and the BHS will be analysed using ANOVA statistical formulae.

The second phase will involve the distribution of the four questionnaires to a clinical eating-disordered population. All participants will have met the diagnostic criteria for either AN or BN according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). This population will be drawn from the out-patient departments in local hospitals, and private clinics. The data will then be analysed similar to the first study.

Finally, data from the selected anorexic-type and bulimic-type population will be gender- and age-matched to the non-clinical population. Scores from both the student group and the clinical group will be compared.

Measurements

1. The Eating Disorders Inventory-1 (EDI-1: Garner, 1991) is the most widely used multidimensional instrument, with demonstrated utility for both clinical and nonclinical

purposes. It consists of 64 items that make up eight subscales: Drive for Thinness (DT); Bulimia (B); Body Dissatisfaction (BD); Inefficacy (I); Perfectionism (P); Interpersonal Distrust (ID); Interoceptive Awareness (A); and Maturity Fears (MF). The EDI-1 assesses cognitive-behavioural symptomatology commonly found in both AN and BN, as well as psychological correlates present in but not exclusive to these disorders (Garner, 1983, 1991).

2. The Beck Depression Inventory (BDI: Beck, Ward,Mendelson, Mock, & Erbaugh, 1961) is a 21-item, self-administered, self-report format. The BDI is a widely used test which, collectively, corresponds reasonably well to the symptoms of depression listed in the DSM-IV, and according to the authors has the ability to differentiate psychiatric patients from normal patients with dysthymic disorder, and major depressive disorder. The instrument may be used with adolescents and adults in clinical and non-clinical populations. For each item, subjects are presented with four statements and asked to indicate the one that best describes the way they fell. Scores on this inventory can range from zero to 63 with higher scores indicating greater depression

3. The Adult Nowicki-Strickland Internal-External Control Scale (ANS-IE) is a 40-item instrument to which the subjects respond yes or no to items they believe to be true

of themselves. Reported split-half reliability varies from .74 to .86, and a test re-test reliability of .83 for a six-week period. Construct validity correlations with The Rotter Internal-External Locus of Control Scale (Rotter, 1966) have been shown to be in the range of .59 - .68 (Norwicki & Duke, 1974).

4) The Beck Hopelessness Scale (BHS: Beck, 1978-88) is a self-administered 20-item forced-choice format, which is used for detecting hopelessness. It is appropriate for ages 17-years and over. The internal consistency reliability is "excellent" with coefficients between .82 and .93 reported for seven different normal groups. The test-retest reliability is .69 after 1 week, and .66 after 6 weeks.

Hypothesis .

Based on this thesis, it is hypothesized that:

1. The non-clinical participants who show high scores on the Bulimic and Anorexic sub-scales of the EDI-1 will also show high scores on both the BDI and BHS.

2. The clinical population will show high scores on both the Bulimic and Anorexic sub-scales of the EDI; and these high scores will correspond to high scores on the BHS and the BDI measures, and will

be higher than the non-clinical population.

3. Scores on the BHS and the BDI will positively correlate with the EDI sub-scales of either Anorexia or Bulimia, or both, perfectionism, Inefficacy, in both the clinical and non-clinical populations; and

4. Consistent with the literature, both the clinical anorexic or bulimic populations and the anorexic-type and bulimic-type non-clinical populations will show more external locus of control as measured by the ANS-IE instrument.

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APPENDIX A

DIAGNOSTIC CRITERIA FOR ANOREXIA NERVOSA
Diagnostic and Statistical Manual of
Mental Disorders (DSM-IV: APA, 1994)

A. Refusal to maintain body weight at or above minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).

B. Intense fear of gaining weight or becoming fat, even though under weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.

D. In post-menarcheal females, menorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have menorrhea if her periods occur only following hormone, e.g., estrogen administration).

Specify type:

Restricting Type: during the current episode of anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas).

Binge-Eating/Purging Type: during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

APPENDIX A

DIAGNOSTIC CRITERIA FOR BULIMIA NERVOSA
Diagnostic Statistical Manual of
Mental Disorders (DSM-IV: APA, 1994)

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), and amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications, fasting, or excessive exercise.

C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months.

APPENDIX B

The Evening Telegram, Saturday, April 28, 1998

Anorexia not exclusive to western women, Dutch study finds

BOSTON (Reuters) — A review of hospital records is challenging the conventional wisdom anorexia nervosa, a sometimes fatal eating disorder, is caused by the preoccupation with thinness in western culture.

The disease, which killed singer Karen Carpenter, prompts sufferers to starve themselves, or use vomiting or laxatives, in an effort to become increasingly thin. Victims develop a false perception of their bodies, where they cannot see they have become skin and bones.

Anorexia nervosa is "considered to be a western-culture-bound syndrome occurring mainly in young, white women," the research group led by Dr. Hans Wijbrand Hoek of The Hague Psychiatric Institute in the Netherlands wrote in a letter in Friday's New England Journal of Medicine.

"It is thought to be very rare outside the western world and in black women in industrialized countries."

The researchers examined the records of 44,192 people admitted to Curacao General Hospital between 1987 and 1989. They said they were expecting to find few, if any, cases of anorexia on the Caribbean island, "where overweight is socially acceptable."

They found six cases, a rate that "is within the range of rates reported in western countries."

Hoek's team said the six women were all born and living on Curacao. Five were Creole (mixed-race) and the other was of Portuguese origin, the researchers said.

"Our finding challenges the ideas that sociocultural pressure to diet is a crucial factor in the causation of anorexia nervosa and that it occurs only in western societies," the researchers said.

APPENDIX B

New York Times, May 5, 1997

Drug Helps Recovering Anorexics, Study Says

SAN DIEGO, May 28 (AP) — Prozac can help keep people who have recovered from anorexia nervosa from falling back into self-starvation, a study has found.

Many psychiatrists have been prescribing the drug for that purpose in the last few years. One expert called the work the best evidence yet that Prozac can help.

"This is very promising and it's very exciting" because relapses are common in anorexia, said Dr. Walter Kaye, the main author of the study.

But Dr. Kaye, a psychiatry professor at the University of Pittsburgh Medical Center's Western Psychiatric Institute and Clinic, cautioned that Prozac could not treat the disorder itself. And he said that other studies suggested that Prozac would not help if an anorexia nervosa patient was still underweight.

The work was paid for in part by Eli Lilly & Company, which makes Prozac. It was presented last week at a meeting of the American Psychiatric Association.

Prozac is best known as an antidepressant, but it is also approved for treating bulimia nervosa — in which people binge, then make themselves vomit — and obsessive-compulsive disorder.

Anorexia nervosa affects about one-half of 1 percent to 1 percent of adolescent girls and young women. Rates are much lower in men. People with the disorder can starve themselves to death, driven by an irrational belief that they are fat or a fear of getting fat.

Some eat too little or exercise too much, while others go on eating binges and then make themselves vomit. The new study focused on people in the first category, which accounts for about half of anorexia nervosa patients.

Many psychiatrists are prescribing Prozac as part of initial treatment as well as to prevent relapse, said Dr. Joel Yager, a psychiatry professor at the University of New Mexico School of Medicine.

Dr. Kaye's study is the strongest evidence yet of Prozac's usefulness in the disorder, Dr. Yager said.

Author takes new approach to eating

Saturday, October 4, 1997

disorders

TORONTO (CP) — Peggy Claude-Pierre asks you to forget everything you think you know about eating disorders.

Set aside such possible "triggers" as the prevalence of ultra-thin supermodels, physical or emotional abuse, or other trauma; the stereotypes — selfish, spoiled, perfection-seeking, attention-craving, vanity-driven girls struggling with low self-esteem.

Try instead to imagine that anorexics and bulimics are at war with themselves. They are of two minds, the Actual Mind and the Negative Mind, and the Negative Mind, the eating disorder mind, has taken over.

Once the patient is helped to distinguish herself from the illness — herself from the Negative Mind — she is able to fight it. But for the disorder to be beaten, every negative thought must be answered with a positive one.

If it sounds far-fetched, Claude-Pierre, in her 40s, asks that you take a closer look. She's revealed her theory and treatment in The Secret Language Of Eating Disorders (Random House, $32).

This mother of two became convinced that two minds were at work in the cases of the adolescent girls whom she freed more than a decade ago from the grips of eating disorders. No other explanation made sense, no professional intervention worked.

PEGGY CLAUDE-PIERRE

"I'm not so different, not so special but I had to look at the possibility of another way," she says.

Claude-Pierre considers her approach unconventional.

Dr. Blake Woodside, a psychiatrist and director of the eating disorder inpatient program at the Toronto Hospital, calls it unproven.

"There is no scientific information about whether it works or not," notes Woodside. "It is an unproven treatment that I would neither encourage nor discourage anyone from seeking out."

Beginning in 1988 Claude-Pierre used another way in her own treatment practice and, since 1993, at her Montreux Counselling Centre in Victoria. With 70 or so other careworkers, the centre often tends to the most extreme cases — patients near death — all, she says, referred to her by doctors.

The Montreux treatment facility is a private, user-pay centre. During the the first two stages of the five-stage program it costs about $1,000 a day. Sixty per cent of patients are from the United States, 30 per cent from Europe and 10 per cent from Canada.

Since appearances on TV's 20/20 and Oprah, Claude-Pierre says she's been flooded with requests for information by parents and medical professionals around the world.

"I couldn't possibly build enough clinics," she offers. The centre takes about 25 residential cases and about an equal number of out-patient cases per year.

Conventional treatments most based on behavior modification are largely ineffective because they put the responsibility for recovery on the sufferer, Claude-Pierre says.

In the acute stages of their disorders, patients are "emotional infants," in need of around-the-clock nurturing, unconditional support and hand-feeding.