MEASUREMENT OF EATING PATHOLOGY

MEASUREMENT OF EATING PATHOLOGY: DISTINCT ROLES OF THOUGHTS AND BEHAVIOURS IN THE ASSESSMENT OF RISK AND DETECTION OF EATING DISORDERS

By

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Preface

This thesis consists of studies that have been previously published or are currently under review for publication in peer-reviewed scientific journals. Chapter 2 contains the published article from *Eating Behaviors*, Vol. 7, pages 69-78 (2006), by J. L. Miller, L. A. Schmidt, T. Vaillancourt, P. McDougall, & M. Laliberté; *Neuroticism and introversion: A risky combination for disordered eating among a non-clinical sample of undergraduate women*, Copyright (2005) Elsevier Ltd. Chapter 3 contains the published article from *Eating Behaviors*, Vol. 9, pages 352-359 (2008), by J. L. Miller, L. A. Schmidt, & T. Vaillancourt; *Shyness, sociability, and eating problems in a non-clinical sample of female undergraduates*, Copyright (2008) Elsevier Ltd. The authors of both these published papers retain the rights without further permission from the publisher to use the published version of this article for personal and internal institutional use, not limited to but including use within a thesis or dissertation. The author of this thesis is the primary author of both these published works, including data collection and analysis, manuscript preparation and revisions. For Chapter 2, the thesis committee advisor, Louis Schmidt, is the second author, and the thesis supervisor, Tracy Vaillancourt, is the third author. The fourth author, Patricia McDougall, was a consultant on the analyses and the fifth author is a member of the thesis committee, Michele Laliberté. For Chapter 3 the thesis committee advisor, Louis Schmidt, is the second author, and the thesis supervisor, Tracy Vaillancourt, is the third author.

The data presented in Chapter 2 were collected in the spring 2004, and the data presented in Chapter 3 were collected in the spring of 2005.

Chapter 4 and 5 contain articles under review for publication. For Chapter 4, the author of the present thesis is the primary author of this work including building the theoretical proposal, reviewing the literature and manuscript preparation. The second author of the paper is the thesis supervisor. For Chapter 5, the author of this thesis is the primary author of the paper, including study and questionnaire design, data collection, analysis, and manuscript preparation. The second author of this paper is the thesis supervisor and the third author, Steven Hanna, was a statistical consultant for the analyses and software used in the study.

The data presented in Chapter 5 were collected across three occasions between 2005 and 2007.

Chapter 6 contains an article in preparation for submission to a journal. The thesis author is also the primary author on this manuscript, including secondary data analyses and manuscript preparation. The second author is the thesis supervisor.

The data presented in Chapter 6 are based on secondary data analysis from the Canadian Community Health Survey, Mental Health Supplement, collected in May 2002.

Abstract

In Part 1 of this thesis the interaction of personality variables in predicting risk of disordered eating is examined. Shyness and an interaction between neuroticism and introversion were found to increase risk of disordered eating in independent university samples. These findings highlight a potential genetic susceptibility to eating pathology by demonstrating similar personality vulnerabilities in clinical eating disorders, as in non-clinical disordered eating. However, the implications and generalizations that can be drawn from these first two studies are limited by the equivocal relationship between disordered eating and eating disorders.

Part 2 of the thesis examines the continuum theory of eating disorders. A theoretical hypothesis is introduced and offers a framework for thinking of thoughts separate from behaviours. Through a review of existing literature, it is noted that eating disorder thoughts are far more prevalent than the behaviours, and while the thoughts can occur independent of the behaviours, behaviours are necessarily tied to pathological thoughts. Preliminary empirical support for this hypothesis is presented by modeling eating disorder thoughts and behaviours as distinct latent constructs in a confirmatory factor analysis using data from a large sample of university females. Frequency analyses of eating disorder thoughts and behaviours offered support for the argument that eating disorder symptoms are not normally distributed across non-clinical settings. As predicted, thoughts occurred independent of the behaviours, and behaviours occurred only in conjunction with thoughts. The interaction of eating disorder thoughts with eating disorder behaviours provided the most robust predictor of psychopathology, although the relative contribution of thoughts and behaviours to psychopathology was not equivalent. These results were replicated in a national epidemiological sample of females ages 15 to 34 years, and the findings were consistent with results from the university sample. Behaviours were more consistently associated with psychopathology in both the university sample and the national sample. The contribution of this thesis to the field of epidemiological research in eating disorders is through the recommendation that screening instruments use behaviours to identify cases and a high threshold of thoughts in risk assessment.

For Emma. Be healthy and strong; love yourself from the inside out.

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CHAPTER 1

GENERAL INTRODUCTION

Eating disorders are severe psychiatric illnesses that affect more than 10 million people worldwide (Crowther, Wolf, & Sherwood, 1992; Fairburn, Cooper, & Shafran, 1993; Gordon, 1990; Hoek, 1995; Shisslak, Crago, & Estes, 1995). While especially prevalent among young North American females (Hoek & van Hoeken, 2003), eating disorders cut across age, gender, ethnicity, and even culture. Eating disorders are now the third most common chronic illness after obesity and asthma in adolescents and adults (Fisher et al., 1995; Lucas, Beard, O'Fallon, & Kurland, 1991). The mortality rates over the first 10 years from clinical presentation is 10%, with most deaths a direct result of medical complications (e.g., cardiac arrest) or due to suicide (Fairburn & Harrison, 2003).

Early identification of eating disorders is critical, as mortality rates increase linearly with duration of illness (Hoek, 2006) and recovery rates are linked to symptom severity at the onset of treatment (Bulik, Sullivan, Carter, McIntosh, & Joyce, 1999). In general, recovery rates vary depending on the definition of recovery, but a substantial number of individuals, especially those with anorexia, will remain chronic, will relapse following treatment, or will continue to struggle with psychological symptoms (e.g., fear of weight gain, body dissatisfaction) despite achieving physical recovery (Strober, Freeman, & Morrell, 1997). For these reasons many researchers have turned their focus to the prevention of eating disorders.

Detection of eating disorders is extremely difficult as they are vastly under-reported, partly due to the secretiveness and shame felt by those struggling with the illness (Hoek & van Hoeken, 2003; Fairburn & Cooper, 1982). Approximately one third of people with anorexia and only 6% of people with bulimia receive mental health care (Hoek & van Hoeken, 2003). Epidemiology research is useful for the detection of undiagnosed cases and for the prevention of new ones through identifying risk factors associated with the onset of an eating disorder (Ackard, Fulkerson, & Neumark-Sztainer, 2007). In order to identify the risk factors associated with eating syndromes and detect undiagnosed cases it is necessary to have valid, reliable measurement tools and clearly defined definitions of the variables being studied.

How do we define eating disorders? This is a difficult question to answer because the definition of an eating disorder has evolved with each revision of the Diagnostic and Statistical manual for Mental Disorders (DSM; Wilfley, Bishop, Wilson, & Agras, 2007). As well, the way in which eating disorders are defined continues to be re-evaluated at the clinical and research level because of lack of agreement, or lack of discussion, of a definition that clarifies the distinction between normal and abnormal eating behaviour. The field of abnormal and clinical psychology as a whole is predicated on the very assumption that we have clear and precise definitions of what is abnormal. Yet in truth, the greatest challenge within the field is in establishing consistent measurable criteria for what is and is not abnormal (Fairburn & Cooper, 2007).

There are two aspects of eating disorders that receive the most attention in defining the features of the disorder: pathological behaviours and dysfunctional thinking.

For example, the DSM-IV defines eating disorders as "severe disturbances in eating behaviour" (APA, 2000, pg 583) and Fairburn and Walsh (2002) define eating disorders as "a persistent disturbance of eating behaviour or behaviour intended to control weight, which significantly impairs physical health and psychosocial functioning" (pg. 171).

The DSM also notes the disturbance in perception of body shape and weight as an essential feature of both anorexia and bulimia nervosa. Fairburn and colleagues have argued that the disturbance in body image and weight preoccupation is the 'core psychopathology' of eating disorders (Fairburn et al., 2003; Fairburn & Harrison, 2003). Both the behavioural and cognitive dysfunctions of an eating disorder are necessary for a clinical diagnosis (APA, 2000). Unfortunately these definitions do not offer clearly operationalized constructs that we can apply to measurement instruments for use in detecting eating disorders in a general population study. Nor do the clinical criteria specified in the DSM definition of an eating disorder necessarily map onto the behavioural and psychological features that occur in a non-clinical or at-risk population.

Thus presents the challenge of epidemiologic research in the eating disorders. In order to detect eating disorders and identify factors related to their onset we must discover what features and at what levels, cause significant distress and impairment sufficient to warrant the title of 'disorder'.

The central theme of this thesis is in how we define eating pathology because without an understanding of what we are measuring, our best efforts at detection and prevention will fail. This thesis will address two of the barriers to advancing epidemiological research of eating disorders. First, a clear and comprehensive definition

of eating pathology is complicated by the fact that not all the features of an eating disorder meet abnormal criteria. Disordered eating thoughts are a) not statistically infrequent and b) do not violate social norms.

Prototypical eating disorders themselves are certainly infrequent; they occur among approximately 1% of the population (Fairburn & Harrison, 2003; Hoek, 2006), but the behavioural and psychological components characteristic of eating disorders, differ in frequency. Extreme dissatisfaction with shape and weight is extremely prevalent in non-clinical populations (Ackard et al., 2007; Cash, Morrow, Hrabosky & Perry, 2004; Chamay-Weber, Narring, & Michaud, 2005) but behaviours such as vomiting, laxative use, binge eating and fasting have a much lower prevalence than the psychological features (Ackard et al., 2007; Chamay-Weber et al., 2005; McVey, Tweed, & Blackmore, 2004). A 'fear of fat' was once considered a pathological feature of a rare psychiatric disorder in the 1980's, but is now a normal feature found among normal-weight non-eating disorder populations (Rodin, Silberstein, & Striegel-Moore, 1984).

The second feature of disordered eating thoughts that does not meet abnormal criteria is the violation of social norms. Extreme weight concern does not violate societal norms, rather, feeling fat and hating your body is part of the 'culture of thinness' (Polivy & Herman, 1987). According to Rodin et al. (1984), feeling fat is just part of being a woman in today's society. Most women feel dissatisfied with their bodies and this is not limited to overweight women (Smolak, 2006). Weight concern, feeling fat and body dissatisfaction do not violate social norms, they conform to them. The social

marginalization, stigmatization, and victimization of overweight and obese children and adults, attests to this culture of thinness (Puhl & Latner).

The second barrier to advancing epidemiological research of eating disorders is that identifying eating disorder symptoms in non-clinical samples in order to identify groups at risk assumes a continuous relationship between eating pathology and eating disorders; and the answer to this question has not been successfully resolved in the literature.

A continuum of eating disorders represents the theoretical view that eating disorders are best conceptualized as existing along a dimension of severity, from healthy (no weight preoccupation no unhealthy behaviours) to mildly weight-preoccupied individuals (with or without weight loss strategies), to sub-threshold eating disorders and at the most extreme end of the continuum – the full syndrome eating disorders; anorexia nervosa, bulimia nervosa and binge eating disorder. Yet if eating disorders are discrete from eating-related problems such as dieting and body dissatisfaction then studying non-clinical samples will not tell us anything about the etiology or risk factors of clinical eating disorders.

The purpose of this thesis is to address these barriers to epidemiological research in eating disorders by first examining personality vulnerability to eating pathology using variables known to be personality features found among clinical eating disorder populations; and second by generating a new discussion of the continuum theory of eating disorders using a framework of dimensional thoughts and discrete behaviours.

Outline of Empirical and Theoretical Chapters

Chapters 2 and 3 examine the interaction of personality variables in predicting risk of disordered eating; variables that have previously been shown to increase risk for other dysregulated behaviours/disorders. The published empirical paper in Chapter 2 represents the first study to link the interaction of neuroticism and introversion to eating pathology. In Chapter 3 we explore the role of shyness in eating pathology. Shyness is a robust predictor of eating disorders but no research had previously examined its role in non-clinical disordered eating.

Part 2 of this thesis examines a theoretical hypothesis that eating disorder thoughts are on a continuum with normalcy but eating disorder behaviours are not. In Chapter 4, the theoretical hypothesis is introduced and offered as framework for thinking of thoughts separate from behaviours for screening purposes in epidemiological research. We review existing literature in the field and demonstrate that a) eating disorder thoughts are more prevalent than the behaviours; b) thoughts can occur independently of the behaviours in non-clinical groups, but thoughts and behaviours are synonymous in clinical groups; c) empirical tests of discrete versus dimensional constructs indicate a categorical model of eating disorders when the indicators used in the models are behaviours, but more evidence for a dimensional model is shown when models use psychological indicators; and d) recovery from an eating disorder is different for the behavioural versus the psychological features, both in timing, and in the relation to relapse.

In Chapters 5 and 6 I examine the theoretical hypothesis of eating disorder thoughts and behaviours both as distinct latent constructs, and as independent predictors of risk for psychopathology. Distinct latent variables for thoughts and behaviours of eating disorders using confirmatory factor analysis (CFA) are demonstrated in Chapter 5, and show that behaviours are more consistently predictive of psychopathology (depression) compared to thoughts. In Chapter 6, the CFA model from Chapter 5 is replicated but this time using a national epidemiological sample instead of a convenience university sample and fewer indicators of eating disorder thoughts and behaviours are used. The results converge with the findings reported in Chapter 5. Again behaviours were more consistently associated with psychopathology. These findings are robust given that we used half the indicators used in the previous CFA model in Chapter 5 and given that we used a nationally representative sample of females ranging in age from adolescence to early adulthood.

Overlap in Chapters

The material presented in Chapters 2 and 3 both test interactions between conceptually similar personality constructs, shyness and sociability; neuroticism and introversion, in relation to eating pathology. Both studies are based on the same theoretical hypothesis proposed by Schmidt (1999; 2003) and therefore, the introductions of both published papers overlap in the background to the theoretical model.

The material presented in Chapter 4 is original material by the author of this thesis and lays the framework for the empirical papers in Chapters 5 and 6. Chapter 4 is referred to extensively throughout Chapter 5 as it is the first empirical paper to test the model put

forth in Chapter 4. Chapter 6 is a replication of Chapter 5 and thus Chapter 5 results are extensively referenced in Chapter 6. The theoretical model from Chapter 4 is mentioned in Chapter 6 but to a much lesser extent than in Chapter 5.

Summary of Thesis Contributions

The first part of the thesis advances our understanding of the personality traits that increase vulnerability to psychopathology. Both shyness and neuroticism are associated with clinical eating disorders, and the studies from Chapters 2 and 3 extend these clinical findings to non-clinical populations. These studies demonstrate that the same personality variables that predict eating disorders also predict risk for disordered eating and these results offer support for a continuum model of eating pathology.

The second part of this thesis also addresses the issue of a continuum of eating pathology and has both theoretical and applied implications. First, understanding the nature of the relationship between disordered eating and eating disorders advances the theoretical underpinning of eating disorder research, as classification of mental illness is at the heart of all psychiatric diagnoses and subsequent treatment protocols. Establishing valid categories for eating disorders is clinically important as it enables accurate diagnostic instruments to be used and creates standards for diagnosis which is an essential part of interpretation and communication among clinicians and researchers. Addressing the question of continuity versus discontinuity in eating disorders will inform our measurement of eating disorder symptoms in at-risk groups. Improving our measurement in epidemiological research will help reduce the number of false positives

in two-stage screening studies, increasing efficiency in research protocols, reducing costs from unnecessary testing, lessen participant burden incurred by lengthy interviews and free up valuable professional resources that can be directed towards prevention and treatment.

The most significant contribution to the literature is the theoretical and empirical support presented throughout the thesis that it is the eating disorder *behaviours*, not thoughts, which are pathognomonic. The presence of clinically significant eating disorder behaviours is the best indicator that the disorder is present; the reason being that eating disorder thoughts are far too prevalent in non-clinical populations. This thesis presents the first measurement model of eating thoughts and behaviours and provides the field of epidemiology a new perspective on screening for eating disorders and the tools with which to test it.

Based on the findings contained within this thesis, the following recommendations are given: the detection of eating disorders in non-clinical populations will be greatest when we screen using eating disorder behaviours and a high threshold of thoughts, although behaviours alone will perform just as well as behaviours with thoughts. High levels of thoughts may be useful when screening for risk groups even without the presence of behaviours. Instruments that rely predominantly on eating disorder thoughts to screen for cases will yield high rates of false positives, and will suffer a trade-off of low specificity for high sensitivity.

CHAPTER 2

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EATING BEHAVIORS

Neuroticism and introversion: A risky combination for disordered eating among a non-clinical sample of undergraduate women

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Abstract

Recent evidence suggests that people who score low on measures of sociability may be at risk for certain types of psychopathology, including mood and anxiety disorders. In an attempt to extend these findings to other forms of psychopathology, we examined levels of neuroticism and extraversion in relation to eating problems in a non-clinical sample of undergraduate women. The Eysenck Personality Questionnaire (EPQ), Eating Disorders Inventory (EDI), and the Eating Attitudes Test (EAT-26) were completed by 196 first-year undergraduate females. We found that high neuroticism was related to high scores on both of the EDI subscales (Bulimia and Drive for Thinness) as well as high scores on the EAT-26 measure, replicating previous work. In addition, neuroticism served as a moderator such that lower extraversion (i.e., introversion) was related to greater disordered eating, but only for those women who scored high on neuroticism. Thus, a combination of neuroticism *and* introversion may be a risk-factor for symptoms of eating disorders in a non-clinical sample of university women.
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Keywords: Disordered eating; Personality; Bulimia; Anorexia; Interactions

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1. Introduction

Over the last decade, there has been a dramatic increase in attention paid to eating disorders by researchers, clinicians, and the general public. Given the detrimental effects of eating disorders on an individual's physical and mental health, the study of eating disorders is of critical importance. Prevalence of bulimic behaviors, such as self-induced vomiting or episodes of binge–purge eating, are reportedly as high as 25–35% (Bulik, Sullivan, & Kendler, 2000; Sullivan, Bulik, & Kendler, 1998). The highest rates are found among females aged 15–19, who constitute over 40% of all diagnosed cases (Hoek & van Hoeken, 2003). Research also suggests that as many as 75% of women consider themselves too fat (Scarano & Kalodner-Martin, 1994), 80% have dieted before the age of 18 (Zerbe, 1995), and 35% have engaged in binge eating (Murray, 2003). Among a nationally representative sample of 15,349 grade 9 to 12 students, Forman-Hoffman (2004) found that 1-in-4 female and 1-in-10 male high school students in the U.S. engage in abnormal eating and weight control behaviors.

Prevalence rates of clinically-diagnosed eating disorders present more conservative estimates (0.3% anorexia nervosa, 1% bulimia nervosa and binge eating disorder; Hoek & van Hoeken, 2003) than studies examining those who are symptomatic or members of an at-risk population. However, even in "sub-clinical" cases, the accompanying pathology of individuals exhibiting symptoms of eating disorders has been found to resemble the pathology observed in individuals with full-blown eating disorders, bringing into question the utility of standard diagnostic criteria (Garfinkel et al., 1995; Zaider, Johnson, & Cockell, 2000). Furthermore, given the notion that eating disorders exist along a continuum, early detection of patterns of disordered eating may help in the prevention of more serious syndromes of clinically-diagnosed eating disorders (Forman-Hoffman, 2004). Accordingly, an important purpose of the present study was to examine symptoms, rather than disorders, of eating pathology in a non-clinical population.

In identifying at-risk populations, one approach is to distinguish among the various personality traits of individuals who exhibit disordered eating (e.g., Bulik, Sullivan, Joyce, & Carter, 1995; Kleifield, Sunday, Hurt, & Halmi, 1994). For example, Strober (1980) found individuals diagnosed with anorexia nervosa were obsessional, interpersonally insecure, highly dependent, excessively conformist and regimented. Casper (1990) found that individuals with anorexia nervosa (restricting types) scored high in emotional restraint, low on impulsivity, and were extremely conventional compared to a normal population. Kleifield, Sunday, Hurt, and Halmi (1993) noted that individuals with bulimia nervosa exhibited tendencies toward impulsivity and low sociability. Still others have examined interpersonal functioning among individuals with bulimia nervosa and found they were socially dependent and fearful of rejection (Hayaki, Friedman, Whisman, Delinsky, & Brownwell, 2003).

The personality dimensions neuroticism and extraversion in relation to eating disorders have received much attention. While neuroticism has long been implicated as a predictor of eating disorders and, in particular, of bulimia nervosa (Cervera et al., 2003; Davis, 1997; Heaven, Mulligan, Merrilees, Woods, & Fairooz, 2001; Kendler et al., 1991; Pearlstein, 2002; Wade, Tiggemann, Heath, Abraham, & Martin, 1995), the extant literature is less consistent in identifying the role that extraversion (or sociability) may play. Studies of clinical populations have found low levels of extraversion (i.e., introversion) to be related to disordered eating (De Silva & Eysenck, 1987; Feldman & Eysenck, 1986; Kleifield et al., 1993). Similarly, Bruce, Steiger, Koerner, Israel, and Young (2004) linked bulimia nervosa to social avoidance among a clinical sample of 59 women. Exceptions to these studies

are findings by Wade et al. (1995) who reported no relation between extraversion and bulimia nervosa among a clinical sample (N=58) and a study by Brookings and Wilson (1994) who found extraversion to be *positively* correlated with EDI-Drive for Thinness and the EAT-26, and to hold no relation to EDI-Bulimia, in a sample of non-clinical participants. Although the results by Brookings and Wilson appear to contradict previous research, it is important to consider that these authors used a different measure of extraversion and neuroticism (i.e., NEO Personality Inventory; Costa & McCrae, 1985) in a *non-clinical* population.

Given the inconsistencies among the clinical samples and the one study of a non-clinical sample, there is a need for further research to clarify the link between extraversion and eating disorders. Furthermore, while low sociability has been linked to bulimia nervosa, we know little concerning the relation between sociability and anorexia nervosa, a distinct type of eating pathology that may have its own unique set of personality correlates. Finally, although previous studies have established independent relations between disordered eating, neuroticism, and extraversion, the present study appears to be the first to examine the interaction of the variables neuroticism and extraversion in eating problems in a non-clinical, university-aged population. Indeed, we sought to extend previous studies, which have examined personality dimensions independently, by looking at the interaction of personality traits in accounting for unique variance. Specifically, we examined whether extraversion and problem eating might be intensified for those individuals who are higher on neuroticism as compared to those who show a disposition towards lower neuroticism. We chose to examine neuroticism as a moderator given Claridge and Davis's (2001) argument that neuroticism acts as an "emotional amplifier" by inflating pre-existing personality traits from adaptive, healthy, behaviors to unhealthy behaviors (p. 396). They argue that neuroticism contributes to the dynamics of abnormal behavior through its role as a moderator.

Overall there were three goals of the present study. The first goal was to clarify research on the role extraversion plays in disordered eating in a *non-clinical* sample. Second, we were interested in identifying whether the personality dimensions extraversion and neuroticism would be similarly connected to both anorexia nervosa and bulimia nervosa given that most studies have linked these personality dimensions to bulimia nervosa alone. Third, we sought to examine whether neuroticism moderates the association between extraversion and disordered eating. To this end, we tested the interaction of personality traits in relation to disordered eating rather than focusing solely on individual personality characteristics.

We recruited a group of first year undergraduate women and had them complete the Eysenck Personality Inventory (EPQ) measures of Neuroticism and Extraversion along with the Eating Disorder Inventory (EDI) and the Eating Attitudes Test (EAT-26), which are widely used to index symptoms of eating problems. Based on previous findings we predicted pathological eating would be related uniquely to: (1) high levels of neuroticism, and (2) low levels of extraversion (i.e., introversion). Also, we predicted that the combination of neuroticism and extraversion would account for unique variance in eating problems, above and beyond the individual contributions of each trait.

2. Method

2.1. Participants

Participants were 196 first year female undergraduate students (M=19.5 years, SD=1.5 years) recruited from on-campus residences at a medium size university located in southern Ontario. A

majority of the participants were Caucasian (64.3%), followed by South Asian (14%) and Chinese (8%). The sample was restricted to women because of the disproportionate representation of women among all types of eating disorders (Murray, 2003). Participants were recruited on a voluntary basis in the main lobby of two of the university's main on-campus residences, and were offered a can of soft drink in return for filling out a short questionnaire. All procedures were approved by the University's Research Ethics Board.

2.2. Measures

2.2.1. The Eysenck Personality Questionnaire - Revised (EPQ-R)

Participants were asked to complete the 48-item short-scale version of the EPQ-R, which measures four major personality dimensions: Neuroticism (N), Extraversion (E), Psychoticism (P), and Social Desirability/Lie Scale (L) (Eysenck, Eysenck, & Barrett, 1985). Of particular interest to the present study were the N and E dimensions. Sample items from each scale include: "Does your mood often go up and down?" or "Are you an irritable person?" (N); "Are you a talkative person?" or "Are you rather lively?" (E). Participants are asked to rate each item as "yes" or "no", (coded as 1 and 0) depending on how applicable the statement was to them. Accordingly, subscale scores ranged from 0 to 12. The reliabilities for individual subscales have previously been demonstrated to yield good test–retest and internal consistency (Eysenck & Eysenck, 1991; Eysenck et al., 1985). In the present investigation, internal consistency was observed to be very good with alpha=.80 for the Neuroticism subscale and alpha=.83 for Extraversion.

2.2.2. Eating Disorder Inventory (EDI-2)

The EDI measures behavioral and symptomatic patterns of anorexia nervosa and bulimia nervosa. Two subscales of the EDI were included in the questionnaire packet: *Bulimia* (B; 7 items) and *Drive for Thinness* (DFT; 7 items). Sample items from each scale include: "I eat when I am upset" and "I have thought of trying to vomit in order to lose weight" (B); "I think about dieting" and "I am terrified of gaining weight" (DFT). Respondents are asked to rate items on a 0 (rarely, never) to 3 (always) metric. Item scores contribute to only one subscale, and subscale scores are computed by summing all items in a particular subscale (Garner, 1991). The EDI has established internal consistency, criterion-related validity, and convergent and discriminant validity for all subscales (Garner, Olmstead, & Polivy, 1983). Internal consistency of these scales in the present study was observed to be very good (Bulimia, α=.75; Drive for Thinness, α=.86).

2.2.3. Eating Attitudes Test (EAT-26)

Disordered eating was further assessed through the 26-item EAT-26 (Garner & Garfinkel, 1979). Sample items from the EAT-26 are: "I vomit after I have eaten," "I engage in dieting behaviour," "I like my stomach to be empty," and "I am preoccupied with a desire to be thinner." Items are scored on a 0 (rarely, never) to 3 (always) metric based on the frequency with which participants engage in behaviors related to food and dieting. The EAT-26 has been shown to be a reliable and valid assessment of clinical symptoms associated with anorexia nervosa (primarily) and bulimia nervosa (Garner et al., 1983). Coefficient alpha for the EAT-26 was observed to be excellent (.90) in the present sample.

3. Results

3.1. Descriptive statistics

Descriptive statistics are presented in Tables 1 and 2. The sample population was comparable to normative standardized data (using female controls) reported by Garner and Olmstead (1984). However, the mean scores in the current study for the EDI-Bulimia subscale were higher than normative data (2.7 compared to 1.7; $t(195)=3.88$, $p<.0001$).

Four participants (2%) obtained raw scores on the EDI-Bulimia that exceeded the clinical cutoff score of 14 (Garner, Olmstead, Polivy, & Garfinkel, 1984). Fifteen participants (8%) scored higher than the clinical cutoff on the EDI-DFT and thirty participants (15.5%) scored higher than the clinical cutoff (20) for the EAT-26.

3.2. Moderator analyses

In order to examine the hypothesis that the combination of neuroticism and extraversion would be related to problem eating, we first centered the data and then conducted separate hierarchical multiple regression analyses for each outcome (Aiken & West, 1991; Baron & Kenny, 1986). Specifically, in Step 1 of each regression, Neuroticism and Extraversion were entered to predict variability in scores on the Bulimia and Drive for Thinness subscales of the EDI as well as a total score on the EAT-26. For each regression, the interaction between Neuroticism and Extraversion was entered on Step 2 to investigate the hypothesis that neuroticism moderates the relation between extraversion and disordered eating. At each step $R^2\Delta$ was calculated, with statistically significant increments in explained variance for the interaction term in Step 2 providing evidence for the moderator effect (Aiken & West, 1991). For each of the three measures of disordered eating, statistically significant simple effects were subsequently investigated by dichotomizing the moderator variable into high and low based on a ± 1 standard deviation split. This statistical procedure, recommended by Aiken and West (1991), facilitates the interpretation of moderator effects in that it allows one to examine how extraversion (X) relates to disordered eating (Y) with varying levels of neuroticism (Z). The relation between the predictor variables and the criterion variable are estimated in the form of unstandardized beta coefficients at each level of the moderator variable (see Aiken & West, 1991). Results of these analyses are described below.

Table 1
Descriptive statistics for EDI-bulimia, EDI-drive for thinness, and EAT-26

Measures	Mean	SD	% Clinical
EDI			
Bulimia	2.7(1.7)	3.5(3.1)	2.0
Drive for thinness	4.6(5.1)	5.2(5.5)	8.0
EAT-26	9.7(9.9)	11.2(9.2)	15.5

EDI=Eating Disorder Inventory, EAT-26=Eating Attitudes Test.
$N=196$; Clinical cases based on cutoff scores used by Garner et al., 1984. The numbers in brackets represent normative data from Garner & Olmstead, 1984.

Table 2
Zero-order correlations for EPQ-neuroticism, EPQ-extraversion, EDI-bulimia, EDI-drive for thinness, and EAT-26 (N=196)

	1	2	3	4
1. Neuroticism	–			
2. Extraversion	−.309**	–		
3. Bulimia	.276**	−.189**	–	
4. Drive for thinness	.366**	−.227**	.567**	–
5. EAT-26	.288**	−.244**	.661**	.784**

EAT-26=Eating Attitudes Test, EPQ=Eysenck Personality Questionnaire.
*p<.05 level (two-tailed). **p<.01 level (two-tailed).

3.3. Bulimia scale

Results from the first hierarchical regression revealed that when the Bulimia subscale of the EDI was considered, 11.1% of the variability could be accounted for by the predictors Neuroticism and Extraversion at Step 1. At Step 2, we entered Neuroticism (b=.296, p<.0001) and Extraversion (b=−.047, p=.57) along with the interaction of Neuroticism and Extraversion (b=−.074, p<.001), and found that the interaction accounted for an additional 4.8%, suggesting significant moderation effects ($F\Delta$=10.49, p<.001). Follow-up tests revealed that the negative relation observed between Extraversion and EDI-Bulimia became stronger as the level of Neuroticism increased (b's for low and high Neuroticism=.16, p=.13, and −.24, p<.003, respectively). In other words, the relation between Bulimia and Extraversion depended on the level of Neuroticism such that EDI-Bulimia was *only* associated with low Extraversion when high levels of Neuroticism were present. In contrast, Extraversion and EDI-Bulimia were not related for those women scoring low in Neuroticism.

3.4. Drive for thinness scale

Results from the second hierarchical regression showed that when the Drive for Thinness subscale of the EDI was considered, 15.5% of the variability could be accounted for by Neuroticism and Extraversion at Step 1. At Step 2, we entered Neuroticism (b=.531, p<.0001) and Extraversion (b=−.128, p<.315) along with the interaction of Neuroticism and Extraversion (b=−.119, p<.001) and found an additional 5.1% was explained by the interaction term, again suggesting significant moderation effects ($F\Delta$=11.77, p<.001). Follow-up tests revealed that the negative relation observed between Extraversion and Drive for Thinness was stronger at higher levels of Neuroticism (b's for low and high Neuroticism=.15, p=18, and −.30, p<.001, respectively). Specifically, Drive for Thinness was *only* statistically significantly associated with low Extraversion when high levels of Neuroticism were present. As seen with respect to Extraversion and EDI-Bulimia, Extraversion and Drive for Thinness were not related for those women scoring low on Neuroticism.

3.5. EAT-26 total

Results from the third hierarchical regression showed that when the EAT-26 measure was considered, 12.2% of the variability could be accounted for by Neuroticism and Extraversion at Step 1. At Step 2, we entered Neuroticism (b=.894, p<.0001) and Extraversion (b=−.366, p<.185) along with the

interaction of Neuroticism and Extraversion ($b = -.241$, $p < .002$) and found that an additional 4.7% was explained by the interaction term again suggesting significant moderation effects ($F\Delta = 10.30$, $p < .001$). When we examined Neuroticism as a moderator, we once more noted a relation between *low* Extraversion and more problem eating (as measured by the EAT-26) for those women who were high, but not low, in Neuroticism (b's for low and high Neuroticism = .11, $p = .32$, and $-.31$, $p < .0001$, respectively).

4. Discussion

We examined whether a combination of neuroticism and extraversion would be related to eating problems in a non-clinical sample of first year female undergraduate students. As hypothesized, we found that high neuroticism was related to problematic eating patterns. Specifically, women high on neuroticism were more prone to problem eating as indexed by three different disordered eating measures (EDI bulimia, EDI drive for thinness, and the EAT-26). These basic findings are consistent with prior studies using clinical (Becker, DeViva, & Zayfert, 2004) and non-clinical samples (Cervera et al., 2003; Gual et al., 2002). We also extended these findings by examining the interaction between neuroticism and extroversion in relation to disordered eating. Claridge and Davis (2001) have suggested that because neuroticism is consistently present in most psychopathologies (including eating disorders), it alone does very little to differentiate between abnormal populations. These authors further argue that what is often overlooked in the literature is a combination of neuroticism and other personality dimensions, or rather, dimensions specific to the disorder itself. What we found was that an interaction of neuroticism and extraversion significantly accounted for some symptoms of eating problems above the trait of neuroticism alone. Undergraduate women who scored high on neuroticism and low on extraversion were at greatest risk for symptoms of eating problems across each of the eating disorder measures. In contrast, we found extraversion to be unrelated to eating problems among women who scored *low* on neuroticism.

These findings highlight two strengths of the present study. First, our results converge across three eating disorder measures; the EDI-Bulimia, the EDI-DFT and the EAT-26. Although the most appropriate method for deriving a diagnosis of anorexia nervosa or bulimia nervosa is the structured clinical interview, the EDI and the EAT-26 are both economical measures designed to identify individuals with sub-clinical eating problems, as they are at the greatest risk of developing clinical eating disorders (Garner, 1991). Second, the interaction of neuroticism and extraversion was related to symptoms common to *both* anorexia nervosa and bulimia nervosa. The EDI-DFT subscale is designed to tap into dimensions associated with anorexia nervosa, while the EDI-Bulimia reports symptoms of bulimia nervosa. The EAT-26 primarily captures symptoms common to anorexia nervosa. Many researchers studying eating disorders have argued that anorexia nervosa and bulimia nervosa are the result of differing personality dimensions. For example, Westen and Harnden-Fischer (2001) described patients with anorexia as constricted and over-controlled and patients with bulimia as emotionally dysregulated and under-controlled. In addition, the criteria of a clinical diagnosis of anorexia nervosa differ considerably from bulimia nervosa. Pearlstein (2002) stated in her review that characteristics of clinical patients were heterogeneous and although some overlap exists such as perfectionism, risk factors cannot be transmitted across subtypes of eating pathology. However, our results suggest both types of disordered eating can be predicted by a combination of neurotic and introverted personality traits.

The results of this study suggest that a combination of personality traits make a more robust predictor of psychopathology than consideration of one trait alone. Prior studies have suggested a link between personality traits and psychopathology, yet few researchers have examined how the interaction of personality traits might explain additional variance in understanding psychopathology above and beyond any one trait. Recent evidence suggests that there is an advantage to considering the interaction of personality traits in understanding problem behaviour and risk factors for psychopathology. For example, a number of studies have shown that a combination of fundamental traits such as shyness and sociability might help to explain antisocial and problem behaviour in children (Schmidt, 2003), adolescents (Page, 1990), and young adults (Santesso, Schmidt, & Fox, 2004) beyond the trait of shyness or sociability alone. Similarly, the present study found a combination of theoretically linked variables, neuroticism and extraversion, to be related to problematic eating behaviours.

4.1. Limitations

The present study is not without limitations. Although our university-based sample was comparable in size and composition to other research studies in this area, future research should consider the strength of using a larger, more generalized sample, such as a community-based participant pool. Second, the correlational nature of this research design precludes any statements of causal relations. Nevertheless, it is a preliminary and necessary first step in determining whether the interaction of neurotic and introverted personality traits can predict eating disorders in prospective research studies. In addition, an important direction for future research would be to examine the risky combination of neuroticism and extraversion across different psychopathologies in order to determine specificity. Given the parallel findings by Schmidt and his colleagues (2003, 2004) concerning the link between problem behaviours and the constructs shyness and sociability, it may be that neurotic and introverted personalities represent a dangerous combination of personality dimensions for a range of psychopathologies, including, but not limited to, disordered eating.

4.2. Conclusion

The findings from this cross-sectional study revealed that an interaction of neuroticism and extraversion increased the vulnerability to symptoms of problem eating above anxiety alone. Symptoms of abnormal eating such as dieting and body dissatisfaction are high risk factors for developing an eating disorder (Forman-Hoffman, 2004). Given that an interaction of vulnerable personality traits predicts abnormal eating, preventative research should focus on the interaction of personality variables in identifying at-risk populations. Whereas neurotic and introverted personality traits may increase susceptibility to disordered eating, this is but one combination of many risk factors of problem eating. A thorough understanding of the etiology of eating disorders can only be achieved by examining the role of personality in relation to the biological, socio-cultural, and familial factors of eating pathology.

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References

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.

Baron, R. M., & Kenny, D. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 6*, 1173–1182.

Becker, C. B., DeViva, J. C., & Zayfert, C. (2004). Eating disorder symptoms among female anxiety disorder patients in clinical practice: The importance of anxiety comorbidity assessment. *Journal of Anxiety Disorders, 18*, 255–274.

Brookings, J. B., & Wilson, J. F. (1994). Personality and family-environment predictors of self-reported eating attitudes and behaviors. *Journal of Personality Assessment, 63*, 313–326.

Bruce, K. R., Steiger, H., Koerner, N. M., Israel, M., & Young, S. N. (2004). Bulimia nervosa with co-morbid avoidant personality disorder: Behavioural characteristics and serotonergic function. *Psychological Medicine, 34*, 113–124.

Bulik, C. M., Sullivan, P. F., Joyce, P. R., & Carter, F. A. (1995). Temperament, character, and personality disorder in bulimia nervosa. *The Journal of Nervous and Mental Disease, 183*, 593–598.

Bulik, C. M., Sullivan, P. F., & Kendler, K. S. (2000). An empirical study of the classification of eating disorders. *American Journal of Psychiatry, 157*, 886–895.

Casper, R. C. (1990). Personality features of women with good outcome from restricting anorexia nervosa. *Psychosomatic Medicine, 52*, 156–170.

Cervera, -S., Lahortiga, F., Martinez-Gonzalez, M., Gual, P., Irala-Estevez, J., & Alonso, Y. (2003). Neuroticism and low self-esteem as risk factors for incident eating disorders in a prospective cohort study. *International Journal of Eating Disorders, 33*, 271–280.

Claridge, G., & Davis, C. (2001). What's the use of neuroticism? *Personality and Individual Differences, 31*, 383–400.

Costa Jr., P. T., & McCrae, R. R. (1985). *The NEO personality inventory manual*. Odessa, FL: Psychological Assessment Resources.

Davis, C. (1997). Normal and neurotic perfectionism in eating disorders: An interactive model. *International Journal of Eating Disorders, 22*, 421–426.

De Silva, P., & Eysenck, S. (1987). Personality and addictiveness in anorexic and bulimic patients. *Personality and Individual Differences, 8*, 749–751.

Eysenck, H. J., & Eysenck, S. B. G. (1991). *Eysenck Personality Scales (EPS Adult)*. London: Hodder and Stroughton.

Eysenck, S. B. G., Eysenck, H. J., & Barrett, P. (1985). A revised version of the psychoticism scale. *Personality and Individual Differences, 6*, 21–29.

Feldman, J., & Eysenck, S. (1986). Addictive personality traits in bulimic patients. *Personality and Individual Differences, 7*, 923–926.

Forman-Hoffman, V. (2004). High prevalence of abnormal eating and weight control practices among high-school students. *Eating Behaviors, 5*, 325–336.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., et al. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *American Journal of Psychiatry, 152*, 1052–1058.

Garner, D. M. (1991). *Eating disorder inventory – 2: Professional manual*. Florida: Psychological Assessment Resources Inc.

Garner, D. M., & Garfinkel, P. E. (1979). The eating attitudes test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9*, 273–279.

Garner, D. M., & Olmstead, M. P. (1984). *Manual for the eating disorder inventory*. Odessa, FL: Psychological Assessment Resources.

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15–34.

Garner, D. M., Olmstead, M. P., Polivy, J., & Garfinkel, P. E. (1984). Comparison between weight-preoccupied women and anorexia nervosa. *Psychosomatic Medicine, 46*, 255–266.

Gual, P., Perez-Gaspar, M., Martinez-Gonzalez, M., Lahortiga, F., de-Irala-Estevez, J., & Cervera-Enguix, S. (2002). Self-esteem, personality, and eating disorders: Baseline assessment of a prospective population-based cohort. *International Journal of Eating Disorders, 31*, 261–273.

Hayaki, J., Friedman, M. A., Whisman, M. A., Delinsky, S. S., & Brownwell, K. (2003). Sociotropy and bulimic symptoms in clinical and non-clinical samples. *International Journal of Eating Disorders, 34*, 172–176.

Heaven, P. Cl., Mulligan, K., Merrilees, R., Woods, T., & Fairooz, Y. (2001). Neuroticism and conscientiousness as predictors of emotional, external, and restrained eating behaviors. *International Journal of Eating Disorders, 30*, 161–166.

Hoek, H. W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders, 34*, 383–396.

Kendler, K. S., MacClean, C., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1991). The genetic epidemiology of bulimia nervosa. *The American Journal of Psychiatry, 148*, 1627–1637.

Kleifield, E. I., Sunday, S., Hurt, S., & Halmi, K. A. (1993). Psychometric validation of the tridimensional personality questionnaire: Application to subgroups of eating disorders. *Comprehensive Psychiatry, 34*, 249–253.

Kleifield, E. I., Sunday, S., Hurt, S., & Halmi, K. A. (1994). The tridimensional personality questionnaire: An exploration of personality traits in eating disorders. *Journal of Psychiatry Research, 28*, 413–423.

Murray, T. (2003). Wait not, want not: Factors contributing to the development of anorexia nervosa and bulimia nervosa. *The Family Journal: The Counseling and Therapy Journal for Couples and Families, 11*, 276–280.

Page, R. M. (1990). Shyness and sociability: A dangerous combination for illicit drug use in adolescent males? *Adolescence, 25*, 803–806.

Pearlstein, T. (2002). Eating disorders and comorbidity. *Archives of Women's Mental Health, 4*, 67–78.

Santesso, D. L., Schmidt, L. A., & Fox, N. A. (2004). Are shyness and sociability still a dangerous combination for substance use? Evidence from a US and Canadian sample. *Personality and Individual Differences, 37*, 5–17.

Scarano, G. M., & Kalodner-Martin, C. R. (1994). A description of the continuum of eating disorders: Implications for intervention and research. *Journal of Counseling and Development, 72*, 356–362.

Schmidt, L. A. (2003). Shyness and sociability: A dangerous combination for preschoolers. *International Society for the Study of Behavioural Development, Newsletter, 1*, 6–8.

Strober, M. (1980). Personality and symptomatological features in young, nonchronic anorexia nervosa patients. *Journal of Psychosomatic Research, 24*, 353–359.

Sullivan, P. F., Bulik, C. M., & Kendler, K. S. (1998). The epidemiology of bulimia nervosa: Symptoms, syndromes and diagnostic thresholds. *Psychological Medicine, 28*, 599–610.

Wade, T., Tiggemann, M., Heath, A. C., Abraham, S., & Martin, N. G. (1995). EPQ-R personality correlates of bulimia nervosa in an Australian twin population. *Personality and Individual Differences, 18*, 283–285.

Westen, D., & Harden-Fischer, J. (2001). Personality profiles in eating disorders: Rethinking the distinction between axis I and axis II. *American Journal of Psychiatry, 158*, 547–562.

Zaider, T. I., Johnson, J. G., & Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *International Journal of Eating Disorders, 28*, 58–67.

Zerbe, K. J. (1995). *The body betrayed: A deeper understanding of women, eating disorders and treatment*. Carlsbad, CA: Gurze Book.

CHAPTER 3

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EATING BEHAVIORS

Shyness, sociability, and eating problems in a non-clinical sample of female undergraduates ☆

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Abstract

Previous empirical studies have shown that the personality trait of shyness, either alone or in combination with varying levels of sociability (i.e., a socially-conflicted profile—high shyness with high sociability) to be a reliable predictor of various psychopathologies, including substance abuse and mood disorders. Extending these findings to other forms of dysregulated behaviours, we examined multiple measures of eating problems in relation to self-reported shyness and sociability in a sample of 520 undergraduate females ($M = 20.7$ years). Analyses revealed a consistent significant main effect for shyness across all measures of disordered eating. These findings extend earlier work on shyness to another form of psychopathology (i.e., eating problems) not previously examined in a non-clinical sample.
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Keywords: Disordered eating; Non-clinical; Females; Shyness; Sociability; Socially-conflicted

Shyness, the discomfort and/or inhibition experienced in interpersonal situations, is a personality trait that has been empirically shown over the past several decades to be a reliable predictor of both poor physical and mental health among children and adolescents (Hirshfield et al., 1992; Kagan, Reznick, & Snidman, 1987, 1988; Page, 1990), young adults (Bell, Jasnoski, Kagan, & King, 1990; Reznick, Hegeman, Kaufman, Woods, & Jacobs, 1992; Schmidt & Fox, 1995), and even the elderly (Bell et al., 1993). Among adults, a number of psychiatric disorders are correlated with shyness, including anxiety disorders (e.g., social phobia), mood disorders (e.g., depression), and personality disorders (e.g., avoidant personality disorder; Biedel & Turner, 1999; Cox, MacPherson, & Enns, 2005; Heiser, Turner, & Biedel, 2003).

More recent evidence suggests that there is an advantage to considering the interaction of personality traits in understanding psychopathology. For example, some have argued that a combination of the traits shyness and sociability might help to explain antisocial and problem behaviour in children (Schmidt, 2003), adolescents (Page, 1990), and young adults (Santesso, Schmidt, & Fox, 2004) in addition to the trait of shyness or sociability alone. Specifically, these studies

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have noted that individuals who are classified as high on both shyness and sociability are more likely to engage in risky behaviours such as substance use and abuse, and to experience problems with social adjustment. Shyness and sociability are known to be orthogonal personality traits in normal (Bruch, Gorsky, Collins, & Berger, 1989; Cheek & Buss, 1981) and atypical (Jetha, Schmidt, & Goldberg, 2007) populations and each of these traits has distinct behavioural (Cheek & Buss, 1981) and psychophysiological correlates (Schmidt, 1999; Schmidt & Fox, 1994).

The notion that shyness and sociability are orthogonal traits was first discussed by Cheek and Buss (1981), who noted that some people withdraw from social situations for different reasons. Cheek and Buss argued that some people may withdraw because they are anxious in social situations (i.e., shy) and others may withdraw because they prefer to be alone (i.e., introverted). After describing on a conceptual level that being high on shyness is not the same as being low on sociability, they then sought to prove that the two dimensions were orthogonal at an empirical level by developing measures to index the two traits. Cheek and Buss first created brief self-report measures to index shyness and sociability separately. They found that both shyness and sociability were only modestly related ($r = -.30$), suggesting that the two traits were orthogonal. Cheek and Buss then had undergraduates selected for high and low shyness and sociability interact in unfamiliar dyads. The high-shy/high-social group displayed more anxiety during the interaction than undergraduates in the other three groups.

Schmidt (1999, 2003) has argued that the origins of the shy and social personality style may be linked to underlying differences in approach–avoidance tendencies, resulting in a predisposition to psychological conflict in social situations or anticipation of such situations. Engagement in antisocial and problem behaviours in shy and social individuals may be the result of failed attempts to cope with their stress. Individuals who are both anxious and sociable, are defined by Schmidt as "socially-conflicted". Shy and sociable people exhibit higher overall frontal activation in both the left and right frontal lobes measured using resting EEG recordings (Schmidt, 1999) and were distinguishable from high shy and low sociable people who exhibited less left frontal EEG activity. This pattern of resting frontal activation may reflect a predisposition toward an approach–avoidance conflict elicited during social interaction or anticipation of social interaction. Schmidt and his colleagues have subsequently argued that the socially-conflicted style may reflect less lateralization of psychological processes (Spere, Schmidt, Riniolo, & Fox, 2005).

While intuitively contradictory, the socially-conflicted person is characterized by a strong desire to be social and interact in a variety of social settings, yet their shyness make social interactions uncomfortable and anxiety provoking. The experience of anxiety leads to withdrawal from social situations, or for some, maladaptive coping mechanisms such as abuse of alcohol and other drugs to ease the experience of social interactions. For instance, Page (1990) reports that shy adolescent males use drugs and alcohol as a coping mechanism during anxiety-prone social interactions.

The socially-conflicted personality may also exist with a greater propensity among certain subtypes of eating disorders, such as anorexia nervosa binge/purge subtype and bulimia nervosa purging subtype, due to the greater difficulty with impulse regulation, sensation-seeking and risk-taking behaviors among both the socially-conflicted (see Santesso et al., 2004, for example) and among individuals with eating disorders characterized by bingeing and purging (see Cassin & von Ranson, 2005). While no studies to date have investigated the relation between social-conflictedness and eating problems, previous research has shown a link between the construct shyness (although not sociability) and eating pathology in clinical samples (Bulik, Sullivan, & Joyce, 1999; Bulik, Sullivan, Weltzin, & Kaye, 1995; Fairburn, Welch, Doll, Davies, & O'Connor, 1997; Lehoux, Steiger, & Jabalpurlawa, 2000; Slopien, Rybakowski, & Rajewski, 2004; Troop & Bifulco, 2002; Vitousek & Manke, 1994; Youssef et al., 2004). Importantly, many of these previous studies examined and measured shyness through the related concept "harm avoidance" (Klump et al., 2004), which is a dimension of temperament assessed through Cloninger's Temperament and Character Inventory (Cloninger, Przybeck, Svrakic, & Wetzel, 1994), and is characterized by extreme shyness, fearfulness, and doubt (Klump et al., 2004). Thus, studies explicitly measuring "shyness" in the eating disorders are still needed.

Since shyness is an important personality variable in clinical syndromes of eating disorders, we hypothesized that non-clinical women demonstrating elevated levels of disordered eating may also exhibit shyness, as some research has suggested a genetic endophenotype associated with the vulnerability to disordered eating (Bachner-Melman, Zohar, & Ebstein, 2006). Although some personality traits, such as impulsivity, have been shown to appear concurrent with eating disorders and remit upon remission of behavioural symptomology (Bachner-Melman et al., 2006; Cassin & von Ranson, 2005), other personality traits have been shown to exist prior to the onset of eating disorder illnesses and to persist following recovery (Sullivan, Bulik, Fear, & Pickering, 1998; Klump et al., 2004). If personality traits of clinical interest can be observed in a population-based sample or in a high-risk-sample, this would suggest that these characteristics exist

prior to illness onset. Ideally research should examine at-risk samples using a prospective research design, however preliminary evidence of personality vulnerabilities among individuals with disordered eating using cross-sectional research is useful in identifying personality traits of clinical interest. To test this hypothesis, we examined the interaction of shyness with sociability in a large non-clinical sample of university women, a group at high-risk for disordered eating. University and college students may be at higher risk for disordered eating behaviours due to the stress associated with the transition from home to college (see Taylor et al., 2006) as well as the fact that eating disorders are most prevalent in the 15 to 19 age group (APA, 1994).

Although the present study appears to be the first to investigate the impact of shyness on eating problems in a non-clinical sample of university women, as well as the first study to examine the interaction of the personality variables shyness and sociability in accounting for unique variance in disordered eating, Miller, Schmidt, Vaillancourt, McDougall, and Laliberté (2006) recently reported an interaction of neuroticism and introversion in predicting disordered eating in a sample of first-year female university students living in campus residences ($N = 196$). Although recognized as a distinct construct, neuroticism shares some conceptual and empirical overlap with shyness and both seem to be related to a variety of psychopathologies (Cox et al., 2005). Both Heiser et al. (2003) and Schmidt and Fox (1995) found that individuals with extreme shyness scored significantly higher on measures of neuroticism than less shy individuals in non-patient populations. Still, shyness and neuroticism are distinct in that shyness seems to represent a broader and more heterogeneous personality trait, one that is not consistently association with psychopathology (Heiser et al., 2003). Neuroticism, however, denotes an enduring tendency to experience negative emotionality and distress (Cox et al., 2005). Therefore, while preliminary research supports the role of neuroticism in relation to disordered eating in a non-clinical population, it remains unclear how shyness is related to problem eating and more specifically, how the interaction of shyness and sociability may contribute to elevated levels of problem eating.

To summarize, the purpose of this study was to 1) extend prior research on shyness and sociability by examining whether the socially-conflicted personality style might also explain disordered eating in a non-clinical sample of young

Table 1
Participant characteristics (N=520)

Category	Frequency
Age (Mean/SD)	20.7/2.40 (range 17–38)
Smoker	11.4%
History of eating disorder	8.3%
History of psychiatric illness*	20.2%
Currently dieting	17%
BMI category**	
Underweight (<18.5)	9.5%
Normal (18.5–24.9)	66.8%
Overweight (25.0–29.9)	16.5%
Obese (30.0 >)	7%
Ethnicity	
Caucasian	64.3%
Chinese	7.7%
South Asian	13.8%
Black	2.6%
Arab/West Asian	1.5%
Filipino	0.5%
Korean	2.6%
Other/Mix	7.1%
Program/discipline	
Social science	30.6%
Science	29.1%
Humanities	19.6%
Business	10.2%
Engineering	6.1%
Health sciences	3.6%
Undecided	0.5%

Note. *Psychiatric illness = all mental illness other than eating disorders. **BMI category based on self-reported weight and height.

women and 2) to examine the role of shyness in a non-clinical sample of women scoring high on measures of disordered eating. Based on the literature reviewed herein, we predicted a significant main effect for shyness, with high shyness related to high levels of disordered eating. We also predicted a significant shyness by sociability interaction, with high shyness and high sociability related to high levels of disordered eating compared with other levels of shyness and sociability.

1. Methods

1.1. Participants

Participants were 520 female undergraduate students (M = 20.7 years, SD = 2.4 years) recruited from the student center of a medium size research intensive university located in southern Ontario, Canada. Ethnic composition of the sample, along with other sample characteristics are listed in Table 1. Participants were recruited on a voluntary basis in the atrium of the university's student center and were offered $5.00 compensation for filling out a 20 min questionnaire. All procedures were approved by the university's research ethics board.

1.2. Measures

1.2.1. Cheek and Buss Shyness and Sociability Scale (Cheek, 1983; Cheek & Buss, 1981)

Shyness was measured using the 13-item revised Cheek and Buss Shyness Scale (see Bruch, Gorsky et al., 1989; Cheek, 1983). Sociability was measured using the five-item Cheek and Buss Sociability Scale. Each item is rated on a 0 (not at all characteristic) to 4 (extremely characteristic) metric with a maximum possible score of 52 for shyness and 20 for sociability. Sample items from each subscale include, "I feel tense when I'm with people I don't know well" or "I have trouble looking someone right in the eye" (Shyness); "I like to be with new people" or "I find people more stimulating than anything else" (Sociability). Internal reliability of the subscales in the present study were very good (Shyness, α = .88; Sociability, α = .88). See Table 2 for the scale intercorrelations along with means and standard deviations.

1.2.2. Eating Disorder Inventory (EDI-2)

The EDI-2 (Garner, Olmstead, & Polivy, 1983) measures behavioural and symptomatic patterns of anorexia nervosa and bulimia nervosa. Three of the eight subscales of the EDI-2 were included in the questionnaire packet: Bulimia (B; 7 items), Drive for Thinness (DFT; 7 items), and Body Dissatisfaction (BD; 9 items). Sample items from each scale include: "I eat when I am upset" or "I have thought of trying to vomit in order to lose weight" (B); "I think about dieting" or "I am terrified of gaining weight" (DFT); "I think that my stomach is too big" or "I think my hips are too big" (BD). Respondents are asked to rate items on a 0 (rarely, never) to 3 (always) metric. Item scores contribute to only one subscale and subscale scores are computed by summing all items (Garner, 1991). The EDI has established internal consistency, criterion-related validity, and convergent and discriminant validity for all subscales (Garner et al., 1983). Internal consistency of these scales in the present study was observed to be very good (Bulimia, α = .74; Drive for Thinness, α = .84; Body Dissatisfaction, α = .88).

1.2.3. Eating Attitudes Test (EAT-26)

Disordered eating was further assessed through the 26-item EAT-26 (Garner & Garfinkel, 1979). Sample items from the EAT-26 are: "I vomit after I have eaten," "I engage in dieting behaviour," "I like my stomach to be empty," or "I am preoccupied with a desire to be thinner." Items are scored on a 0 (rarely, never) to 3 (always) based on the frequency participants engage in behaviours related to food and dieting and then summed to reveal a total score. Total scores above 20 are indicative of high-risk. The EAT-26 has been

Table 2
Means, standard deviations, and interscale correlations (N = 520)

Scale	EDI-Bulimia	EDI-DFT	EDI-BD	EAT-Total	Shyness	Sociability
EDI-Bulimia	1	.496**	.314**	.562**	.190**	−.100*
EDI-DFT	–	1	.604**	.794**	.129**	−.059
EDI-BD	–	–	1	.470**	.140**	−.098*
EAT-Total	–	–	–	1	.099*	−.040
Shyness	–	–	–	–	1	−.567**
Sociability	–	–	–	–	–	1
Means (Standard Deviations)	2.22(3.17)	4.50(4.99)	9.61(7.00)	9.43(9.30)	18.08(10.10)	13.10(4.46)

Note: EDI = Eating Disorder Inventory, DFT = Drive for Thinness, BD = Body Dissatisfaction, EAT = Eating Attitudes Test. **p < .01, *p < .05.

shown to be a reliable and valid assessment of clinical symptoms associated with anorexia nervosa (primarily) and bulimia nervosa (Garner et al., 1983). Coefficient alpha for the EAT-26 was observed to be very good (.86) in the present sample.

1.3. Data analysis

We performed separate hierarchical regression analyses in order to examine shyness and sociability separately along with their interaction in predicting the following eating disordered behaviours: EDI-Bulimia, EDI-DFT, EDI-BD and the EAT-Total. In the first step of the equation, the variables shyness and sociability were entered simultaneously, and in the second step we entered the interaction term shyness by sociability. As recommended by Aiken and West (1991), data were centered prior to analysis.

2. Results

2.1. EDI data

2.1.1. EDI-Bulimia

Table 3A presents the results of the hierarchical regression analysis for the EDI Bulimia subscale. The model presented in Table 3A accounted for approximately 4% of the variability in predicting Bulimia [$F(2,518) = 9.175, p < 0.0001$]. In the first step, we found Shyness, [$t(1) = 3.750, p < 0.0001$], but not Sociability, [$t(2) = 0.217, p = 0.828$], was a statistically significant predictor of Bulimia. Increased Shyness predicted Bulimic tendencies. In the second step of the analysis, we found that the interaction of Shyness by Sociability was not a statistically significant predictor of Bulimia [$t(3) = -1.179, p = 0.239$].

2.1.2. EDI-DFT

Table 3B presents the results of the hierarchical regression analysis for the EDI-DFT subscale. In predicting DFT, the model accounted for approximately 2% of the variability [$F(2, 518) = 4.442, p < 0.05$]. In Step 1, we again found Shyness [$t(1) = 2.658, p < 0.01$], but not Sociability, significantly predicted DFT [$t(2) = 0.395, p = 0.693$], indicating that higher levels of Shyness predict increased levels of DFT. Step 2 of this model's analysis revealed that while Shyness remained a statistically significant predictor [$t(1) = 2.556, p < 0.01$], the interaction of Shyness by Sociability did not significantly predict DFT [$t(3) = -1.108, p = 0.268$].

2.1.3. EDI-BD

Table 3C presents the results of the hierarchical regression analysis for the EDI-BD subscale. In predicting BD, the model accounted for approximately 2% of the variability [$F(2, 518) = 5.289, p < 0.005$]. In Step 1, we again found Shyness [$t(1) = 2.345, p < 0.01$], but not Sociability, significantly predicted BD [$t(2) = -0.528, p = 0.598$], indicating that higher levels of Shyness predicted increased levels of BD. Step 2 of this model's analysis revealed that while Shyness remained a statistically significant predictor [$t(1) = 2.214, p < 0.05$], the interaction of Shyness by Sociability did not predict BD [$t(3) = -1.486, p = 0.138$].

2.2. Eating Attitudes Test (EAT) data

Table 3D presents the results of the regression analysis for the EAT measure. The model presented in Table 3D accounted for 1% of the variability in predicting scores in the clinical range on the EAT [$F(2,183) = 2.647, p = 0.072$]. In Step 1 of the regression analysis, we found that Shyness [$t(1) = 2.111, p < 0.05$], but not Sociability [$t(2) = 0.441, p = 0.659$], was a statistically significant predictor of clinical scores on the EAT. High levels of reported Shyness predicted clinical scores on the EAT. Lastly, in Step 2 of the EAT regression analysis, we found that an interaction of Shyness by Sociability failed to reveal a statistically significant effect in predicting scores in the clinical range on the EAT [$t(3) = -1.179, p = 0.239$].

Table 3
Summary of hierarchical regression analysis for shyness and sociability in predicting EDI-Bulimia, EDI-Drive for thinness, EDI-Body Dissatisfaction, and the EAT-Total (N=520)

Variable	(A) EDI-Bulimia				(B) EDI-DFT				(C) EDI-Body Dissatisfaction				(D) EAT-TOTAL			
	B	SEB	R^2	ΔR^2	B	SEB	R^2	ΔR^2	B	SEB	R^2	ΔR^2	B	SEB	R^2	ΔR^2
Step 1																
Shyness	.062*	.016	.036		.069*	.026	.017		.086*	.037	.020		.103*	.049	.010	
Sociability	.008	.037	.036		.023	.059	.017		−.044	.083	.020		.049	.110	.010	
Step 2																
Shyness X Sociability	−.003	.003	.038	.002	−.005	.005	.019	.002	−.010	.007	.024	.004	−.010	.009	013	.003

Note: *p<0.05; B=beta; SEB=standard error of beta; EDI=Eating Disorder Inventory; EAT=Eating Attitudes Test.

3. Discussion

As predicted, we found that shyness reliably predicted patterns of disordered eating in our sample across multiple measures of eating problems. The present results were specific to shyness and not sociability. Specifically, we found that shyness predicted a clinically significant score on the Bulimia, Drive for Thinness, and Body Dissatisfaction subscales of the Eating Disorder Inventory and a total score on the Eating Attitudes Test. These results are in line with and extend prior work using a conceptually and empirically related construct to shyness; neuroticism, which was found to significantly increase the risk of eating problems in a previous non-clinical sample (Miller et al., 2006). The present results also extend earlier research indicating that shyness may be a risk factor for psychopathology among young adults (Bell et al., 1990; Reznick et al., 1992; Schmidt & Fox, 1995). Furthermore, our results are in line with clinical studies of eating disorders indicating shyness (and its related constructs, e.g., harm avoidance) is a robust predictor of eating disorders.

Contrary to our predictions, the interaction of shyness and sociability did not predict unique variance in problem eating across any of the criterion variables in the current sample of undergraduate women. The socially-conflicted personality does not appear to characterize those individuals who engage in aberrant eating behaviours such as bingeing and purging, although these results require replication. One reason for this may be the relatively low levels of disordered eating in the sample (i.e., the frequency of disordered eating may be too low for the interaction of shyness and sociability to show statistical significance). Nevertheless, while the results of the present study do not support the hypothesis of a "socially-conflicted" personality in disordered eating, the results do support and extend past empirical and theoretical literatures linking shyness to various psychopathologies in young adulthood.

Despite several small R^2 statistics in our models (R^2 values ranging from 1% to 4%), the associated effect sizes ranged from small to moderate (Cohen's *d* (1988) ranged from .23 (EAT-Total) to .40 (Bulimia)). In addition, we would argue that explaining even a small percentage of the variance in disordered eating is important, especially in a non-clinical population where base rates of disordered eating are extremely low. It is important to highlight that the etiology of disordered eating is multifaceted including direct influence from parents (such as harsh criticism; Keery, Boutelle, van der Berg, & Thompson, 2005), indirect influence from parents and peers (modeling of body dissatisfaction and dieting behaviours; Stice, 2002), genetic vulnerability (personality traits; Cassin & von Ranson, 2005), societal and media images portraying the "thin is beauty" ideal, as well as the critical incidents of childhood including teasing and bullying from peers, and physical and/or sexual abuse (Johnson, Cohen, Kasen, & Brook, 2002; Polivy & Herman, 2002). The co-existence of so many factors in predicting disordered eating precludes anything but a small effect size in such a diverse non-clinical sample. One predictor such as shyness can only account for a small portion of the variance when considering such a multifaceted model for disordered eating.

In addition, we would argue that effect size and statistical significance are the least important attribute of the current study (see Lykken, 1968). Lykken (1968) among others, has argued that effect size in social, clinical and personality research must expect small effect sizes as an artifact of the large level of ambient noise present in correlational research. What must be considered as support for results of such studies should be the usefulness of the theory and the corroboration of this theory through replication. While this study is the first to find evidence that shyness may be correlated to risk for pathological eating in a non-clinical sample, it does support previous research suggesting the trait of shyness may have broad reaching consequences for a range of psychopathologies.

The design of this study does not allow for causal assumptions to be drawn regarding the direction of the relation between shyness and disordered eating, therefore it is impossible to conclude that shyness increases the propensity to develop eating pathology. However, shyness is considered a stable personality trait that often appears from a very early age and persists beyond childhood (Henderson & Zimbardo, 1998) despite some research evidence supporting the role of situational shyness, or shyness appearing in adulthood for people who were not previously shy but for whom severe experiences of rejection or failure have led to decreased self-esteem (Henderson & Zimbardo, 1998). Longitudinal models addressing the direction of the relation between shyness and eating pathology are needed. As well, future research should control for present levels of anxiety and depression in participants as these variables may have influenced the relation between shyness and eating behaviors.

This study utilized a large sample of university females and although this limits our results to similar-aged university students, the purpose of using a university sample was to draw upon a population known to be at high-risk for eating problems. However, extending these findings to other populations will be important for future research. For instance, given the findings by Schmidt (2003) and Spere et al. (2005) concerning the link between psychopathology and the constructs of shyness and sociability, it may be useful to examine the possibility of the socially-conflicted personality

among clinical populations of eating disordered individuals where finding a statistically significant interaction may be more likely given the heightened level of psychopathology among these individuals.

References

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions.* Newbury Park, CA: Sage.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bachner-Melman, R., Zohar, A. H., & Ebstein, R. P. (2006). An examination of cognitive versus behavioral components of recovery from anorexia nervosa. *The Journal of Nervous and Mental Disease, 194*, 697–703.

Bell, I. R., Jasnoski, M. L., Kagan, J., & King, D. S. (1990). Is allergic rhinitis more frequent in young adults with extreme shyness? A preliminary survey. *Psychosomatic Medicine, 52*, 517–525.

Bell, I. R., Martino, G. M., Meredith, K. E., Schwartz, G. E., Siani, M. M., & Morrow, F. D. (1993). Vascular disease risk factors, urinary free cortisol, and health histories in older adults: Shyness and gender interactions. *Biological Psychology, 35*, 37–49.

Biedel, D. C., & Turner, S. M. (1999). The natural course of shyness and related syndromes. In L. A. Schmidt & J. Schulkin (Eds.), *Extreme fear, shyness and social phobia: Origins, biological mechanisms and clinical outcomes* (pp. 202–223). New York: Oxford University Press.

Bruch, M. A., Gorsky, J. M., Collins, T. M., & Berger, P. A. (1989). Shyness and sociability re-examined: A multicomponent analysis. *Journal of Personality and Social Psychology, 57*, 904–915.

Bulik, C. M., Sullivan, P. F., & Joyce, P. R. (1999). Temperament, character and suicide attempts in anorexia nervosa, bulimia nervosa and major depression. *Acta Psychiatrica Scandinavia, 100*, 27–32.

Bulik, C. M., Sullivan, P. F., Weltzin, T. E., & Kaye, W. H. (1995). Temperament in eating disorders. *International Journal of Eating Disorders, 17*, 251–261.

Cassin, S. E., & von Ranson, K. M. (2005). Personality and eating disorders: A decade in review. *Clinical Psychology Review, 25*, 895–916.

Cheek, J. M. (1983). The revised Cheek and Buss shyness scale. Unpublished manuscript. Wellesley College.

Cheek, J. M., & Buss, A. H. (1981). Shyness and sociability. *Journal of Personality and Social Psychology, 41*, 330–339.

Cloninger, C. R., Przybeck, T. R., Svrakic, D. M., & Wetzel, R. D. (1994). *The Temperament and Character Inventory (TCI): A guide to its development and use.* Center for Psychobiology of Personality, Washington University: St. Louis, MI.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

Cox, B. J., MacPherson, P. S. R., & Enns, M. W. (2005). Psychiatric correlates of childhood shyness in a nationally representative sample. *Behaviour Research and Therapy, 43*, 1019–1027.

Fairburn, C. G., Welch, S. L., Doll, H. A., Davies, B. A., & O'Connor, M. E. (1997). Risk factors for bulimia nervosa: A community-based case-control study. *Archives of General Psychiatry, 54*, 509–517.

Garner, D. M. (1991). *Eating Disorder Inventory-2: Professional manual.* U.S: Psychological Assessment Resources Inc.

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9*, 273–279.

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15–34.

Heiser, N. A., Turner, S. M., & Biedel, D. C. (2003). Shyness: Relationship to social phobia and other psychiatric disorders. *Behaviour research and Therapy, 41*, 209–221.

Henderson, L., & Zimbardo, P. (1998). Shyness. *Encyclopedia of Mental Health*. San Diego, CA: Academic Press.

Hirshfield, D. R., Rosenbaum, J. F., Biederman, J., Bolduc, E. A., Faraone, S. V., Snidman, N., et al. (1992). Stable behavioural inhibition and its association with anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 103–111.

Jetha, M. K., Schmidt, L. A., & Goldberg, J. O. (2007). Stability of shyness, sociability, and social dysfunction in schizophrenia: A preliminary investigation of the influence of social skills training in a community-based outpatient sample. *European Journal of Psychiatry, 21*, 189–198.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry, 159*, 394–400.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioural inhibition in children. *Child Development, 58*, 1459–1473.

Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological basis of childhood shyness. *Science, 240*, 167–171.

Keery, H., Boutelle, K., van der Berg, P., & Thompson, J. K. (2005). The impact of appearance-based teasing by family members. *Journal of Adolescent Health, 37*, 120–127.

Klump, K. L., Strober, M., Bulik, C. M., Thornton, L., Johnson, C., Devlin, B., et al. (2004). Personality characteristics of women before and after recovery from an eating disorder. *Psychological Medicine, 34*, 1407–1418.

Lehoux, P. M., Steiger, H., & Jabalpurlawa, S. (2000). State/trait distinction in bulimic syndromes. *International Journal of Eating Disorders, 27*, 36–42.

Lykken, D. T. (1968). Statistical significance in psychological research. *Psychological Bulletin, 70*, 151–159.

Miller, J. L., Schmidt, L. A., Vaillancourt, T., McDougall, P., & Laliberté, M. (2006). Neuroticism and introversion: A risky combination for disordered eating among a non-clinical sample of undergraduate women. *Eating Behaviors, 7*, 69–78.

Page, R. M. (1990). Shyness and sociability: A dangerous combination for illicit drug use in adolescent males? *Adolescence, 25*, 803–806.

Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology, 53*, 187–213.

Reznick, J. S., Hegeman, I. N., Kaufman, E. R., Woods, S. W., & Jacobs, M. (1992). Retrospective and concurrent self-report of behaviour inhibition and their relation to adult mental health. *Development and Psychopathology, 4*, 301–321.

Santesso, D. L., Schmidt, L. A., & Fox, N. A. (2004). Are shyness and sociability still a dangerous combination for substance use? Evidence from a US and Canadian sample. *Personality and Individual Differences, 37*, 5–17.

Schmidt, L. A. (1999). Frontal brain electrical activity in shyness and sociability. *Psychological Science, 10*, 316–320.

Schmidt, L. A. (2003). Shyness and sociability: A dangerous combination for preschoolers. International Society for the Study of Behavioural Development. *Newsletter, 1*, 6–8.

Schmidt, L. A., & Fox, N. A. (1994). Patterns of cortical electrophysiology and autonomic activity in adults' shyness and sociability. *Biological Psychology, 38*, 183–198.

Schmidt, L. A., & Fox, N. A. (1995). Individual differences in young adults' shyness and sociability: Personality and health correlates. *Personality and Individual Differences, 19*, 455–462.

Slopien, A., Rybakowski, F., & Rajewski, A. (2004). Evaluation of temperament and personality in bulimia nervosa. *Psychiatria Polska, 38*, 85–93.

Spere, K. A., Schmidt, L. A., Riniolo, T. C., & Fox, N. A. (2005). Is a lack of cerebral hemisphere dominance a risk factor for social "conflictedness"? Mixed-handedness in shyness and sociability. *Personality and Individual Differences, 39*, 271–281.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin, 128*, 825–848.

Sullivan, P., Bulik, C., Fear, J., & Pickering, A. (1998). Outcome of anorexia nervosa: A case-controlled study. *American Journal of Psychiatry, 155*, 939–946.

Taylor, C. B., Bryson, S., Luce, K. H., Cunning, D., Doyle, A. C., Abascal, L. B., et al. (2006). Prevention of eating disorders in at-risk college-age women. *Archives of General Psychiatry, 63*, 881–888.

Troop, N. A., & Bifulco, A. (2002). Childhood social arena and cognitive sets in eating disorders. *British Journal of Clinical Psychology, 41*, 205–211.

Vitousek, K., & Manke, F. (1994). Personality variables and disorders in anorexia nervosa and bulimia nervosa. *Journal of Abnormal Psychology, 103*, 137–147.

Youssef, G., Plancherel, B., Laget, J., Corcos, M., Flament, M. F., & Halfon, O. (2004). Personality trait risk factors for attempted suicide among young women with eating disorders. *European Psychiatry, 19*, 131–139.

CHAPTER 4

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The Eating Disorder Continuum Revisited: "Eating Disorder Thoughts" versus "Eating Disorder Behaviours".

Abstract

Background: The purpose of this paper is to call attention to the need for researchers to examine independently the contributions of psychological and behavioural symptoms of eating disorders when examining non-clinical or at-risk populations. **Method:** We begin the paper with a brief review of the eating disorder continuum hypothesis and introduce an alternative model for conceptualizing eating disorders where we suggest it is the *clinical features* of eating disorders which are categorical (the behaviours; "I vomit after eating") *and* dimensional (the thoughts; "I hate my body"). We evaluate three areas of research that offer support for this hypothesis. **Results**: 1) eating disorder thoughts are so prevalent that they poorly discriminate between non-clinical and clinical groups; but behaviours are excellent markers of clinical groups 2) empirical studies of the latent factor structure of eating disorders show dimensional models when using psychological indicators, but categorical models when using behavioural indicators; 3) recovery rates for eating disorders are highest when defined by the absence of behavioural symptoms and lowest when defined by psychological symptoms. **Conclusion**: Because the psychological features of an eating disorder are not specific to clinical populations but in fact occur at an alarming rate within the general population of adolescent and adult females, we propose that behavioural criteria be identified separately from psychological criteria when establishing risk for eating disorders.

Introduction

Do eating disorders exist on a continuum? This question has been debated theoretically and empirically in both the clinical and scientific domains for over four decades (see Gleaves, Brown, & Warren, 2004; Polivy & Herman, 1987; Williamson, Gleaves, & Stewart, 2005). This debate is not specific to eating disorders, rather it resonates throughout much of the past and current scientific inquiries into the nature of all psychopathology (Adams & Cassidy, 1993; Gangestad & Snyder, 1985; Krueger & Piasecki, 2002; Watson & Clark, 2006; Widiger & Samuel, 2005). Nearly one hundred years ago German psychiatrist Emil Kraepelin (1913) argued that while personality dimensions were the basic underlying constructs necessary for development of any psychological disorder, mental illness itself was qualitatively distinct from ordinary human behaviour.

Kraepelin's fundamental theories on the etiology and diagnosis of psychiatric disorders in the 1900's formed the foundation for the classification system later devised by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM) first published in 1952. With the inclusion of eating disorders as a diagnostic category in the third edition of the DSM (American Psychiatric Association, 1980), controversy sparked over whether eating disorders should be considered along a continuum. Many theorists argued that eating disorders were best conceptualized as existing along a continuum of severity, ranging from 'healthy normals' (no weight preoccupation) to mildly weight-preoccupied individuals, to the more severe clinical manifestations evidenced by those with anorexia nervosa, bulimia nervosa and more

recently, binge eating disorder[1] (Button & Whitehouse, 1981; Fries, 1977; Garner, Garner, & Rosen, 1993; Garner & Garfinkel, 1980; Garner, Olmstead, & Garfinkel, 1983; Nylander, 1971; Polivy & Herman, 1987; Rodin, Silberstein, & Striegel-Moore, 1984; Striegel-Moore, Silberstein, & Rodin, 1986). Some researchers and clinicians disputed the notion of an eating disorder continuum and argued for fundamental differences in clinical eating disorders compared to the mild syndromes of weight preoccupation (Bruch, 1973; Crisp, 1965; Selvini-Palazzoli, 1978; Katzman & Wolchik, 1984; Prather & Williamson, 1988; Ruderman & Besbeas, 1992).

Over the last two decades, fewer research studies have been put forth in support of a discontinuous model of eating disorders. It seemed the field was making a shift towards a more continuous approach to understanding normal and abnormal eating behaviours. However, recent research studies using advanced statistical methods (i.e., taxometrics) have re-introduced the notion of a categorical model of eating disorders. These studies do not contradict the prevailing theoretical perspective of an eating disorder continuum, but rather, they provide evidence for simultaneously employing a dimensional and a categorical model (Lowe et al., 1996; Gleaves, Lowe, Green, Cororve, & Williams, 2000; Gleaves, Lowe, Snow, Green, & Murphy-Eberenz, 2000) as the two are not mutually exclusive (Wilfley, Bishop, Wilson, & Agras, 2007).

The aim of this review is to offer support towards the notion of a combined categorical-dimensional model of eating disorders by demonstrating that it is the

[1] Binge eating disorder (BED) is not currently recognized as a distinct clinical category in the DSM-IV-TR; rather it is included in the Eating Disorder Not-Otherwise Specified category. However, clinically, BED is treated as a distinct clinical entity much like anorexia and bulimia nervosa.

psychological symptoms of eating disorders that are dimensional and the behavioural symptoms that are categorical. We hypothesize, and endeavor to demonstrate through existing research, a continuum of eating disorder thoughts (e.g., "I am terrified of gaining weight", "I feel dissatisfied with the shape of my body") and a discontinuum of eating disorder behaviours (severe and chronic restriction, vomiting, laxative abuse, bingeing, excessive exercise etc.). This discussion will highlight a critical issue for researchers interested in detection of eating disorders in community or population-based samples: if the psychological features of an eating disorder are not specific to clinical groups, and they can occur without the behaviours, then the psychological symptoms should not be used in isolation to identify clinical cases in large screening studies.

Towards this aim, we discuss three areas of eating disorder research that will a) provide evidence in support of our hypothesis of a dimension of eating disorder thoughts and a category of eating disorder behaviours and b) demonstrate the importance of distinguishing between psychological and behavioural symptoms in the assessment and measurement of eating disorders.

First, we discuss research indicating that many females in non-clinical populations have the psychological symptoms of eating disorders and often at levels that closely approximate weight and shape concerns in clinical eating disorder groups. Yet the behavioural components of eating disorders are not necessarily present. In other words, it is possible to have elevated levels on the psychological criteria for an eating disorder with none of the behavioural features. In contrast, we will demonstrate that while low levels of eating disorder behaviours may be present in non-clinical populations, they do not occur

without psychological symptoms – suggesting that these groups are in fact an undiagnosed sub-clinical or clinical population.

Second, we review recent studies that have tested the latent constructs of eating disorders using taxometric methods and draw attention to the fact that both categorical and dimensional models of eating disorders seem to fit the data, but these results vary depending on whether the targeted indicators are behavioural symptoms or psychological symptoms and also whether the composition of the sample is clinical or non-clinical. Here we will discuss the critical need for re-designing existing measures of eating disorder symptoms in order to clearly differentiate between thoughts and behaviours, given that most of the screening instruments currently in use have thoughts and behaviours mixed within the same subscale.

Third, we examine the literature on treatment and recovery, where we see clinicians and researchers already distinguishing between psychological and behavioural symptoms in defining recovery from an eating disorder and in predicting who will recover, the length of time to recovery and who will relapse. The relevance of this literature to our theoretical hypothesis is to highlight the distinct roles of psychological and behavioural symptoms in an eating disorder, as well as to highlight the usefulness of measuring eating disorder thoughts and eating disorder behaviours separately from one another as they may be related to different outcomes and individual etiological variables.

We conclude this review with a summary of the implications for research in the prevention and detection of eating disorders and offer recommendations for improving detection and diagnosis in epidemiological studies.

The 'Normative Discontent'

In 2004, Cash, Morrow, Hrabosky and Perry published a critical and systematic review of body image among college women and men using 22 studies – all published between 1983 and 2001. Using data from non-clinical samples they found 29% of white women, 17% of black women and 16% of white men reported *extreme* body dissatisfaction. Body dissatisfaction, which entails an intense disparagement towards specific body regions, as well as a general dissatisfaction with ones shape and weight (American Psychiatric Association, 2000) is increasingly common among non-clinical populations (Feingold & Mazzella, 1998; Tiggemann, 2004) and is often associated with significant psychopathology (Polivy & Herman, 2002). In conjunction with the overvaluation of shape/weight and fear of fat, body dissatisfaction has been described as the most prominent risk and maintenance factor in pathological eating (Attie & Brooks-Gunn, 1989; Fairburn, Cooper, Doll, & Welch, 1999; Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997; Killen et al., 1996; Killen, Taylor, Hammer, & Litt, 1993; Polivy & Herman, 2002; Stice & Shaw, 2002; Striegel-Moore et al., 1986; Striegel-Moore, Silberstein, Frensch, & Rodin, 1989; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999; Wiederman & Pryor, 2000); the most robust predictor of bulimic symptoms (Killen, Taylor, Hayward, & Wilson, 1994; Stice, 2001, 2002); and the core psychopathology of both bulimia and anorexia nervosa (Beumont, 2002; Cooper, Taylor, Cooper, & Fairburn, 1987; Fairburn, Cooper, & Shafran, 2003; Garner, 2002; Garfinkel, 2002; Rosen, 1992; Thompson, 1990). Yet despite the prominent role of these cognitions in clinical eating disorders, their increasing prevalence in non-clinical populations has

threatened the specificity of using these symptoms to detect eating disorders in epidemiological studies.

In the years following Cash et al's (2004) review of body dissatisfaction, a number of large epidemiological studies have demonstrated a similar level of discontent with weight and shape among females of varying ages. Approximately 46% of girls and 26% of boys are dissatisfied with their bodies according to recent statistics from Project EAT (Eating Among Teens), which to date is one of the largest (N= 4746), most comprehensive and ethnically diverse epidemiological studies of middle and high school students' eating pathology (Ackard, Fulkerson, Neumark-Sztainer, 2007). In a review of epidemiological studies of partial syndrome eating disorders from 1980 to 2003, Chamay-Weber, Narring, and Michaud (2005) found 46% to 80% of adolescent girls in the United States reported intense body dissatisfaction. McVey, Tweed, and Blackmore (2004) sampled 2279 females, 10-14 years of age, from 42 Canadian schools and found 31.3% of the sample felt "too fat" and 29.3% stated they were currently trying to lose weight. Two epidemiological studies on the prevalence of disordered eating using the same national Canadian sample (N=36 984), found 26% of women aged 15-24 responded "yes" to having a *strong fear* of being too fat in the past 12 months (Piran & Gadalla, 2006). Extending this age range to women aged 15-65 years Park and Beaudet (2007) found one in five women (19%) responded "yes" to the same question of having a strong fear of being too fat in the past 12 months and this fear was associated with negative self-esteem, body image preoccupation and food obsession (Park & Beaudet, 2007).

High weight preoccupation, however, is not necessarily associated with eating disorder behaviours. For example, Ackard et al. (2007) reported that 26.7% of girls and 20.4% of boys from Project EAT had severe body disparagement without any accompanying eating disorder behaviours. Bulik, Sullivan, and Kendler (2000) identified a subgroup of women who experienced preoccupied thoughts about weight and shape, but who had not engaged in behaviours associated with a clinical diagnosis of an eating disorder. Cooper and Goodyer (1997) found significant weight and shape concerns among 14.5% of 11-12 year olds, 14.9% of 13-14 year olds, and 18.9% of 15-16 year olds. However, only the 15-16 year olds showed any significant presence of behavioural pathology. Garner, Olmsted, Polivy, and Garfinkel (1984) compared patients with anorexia nervosa to weight-preoccupied and not-weight-preoccupied women drawn from a female college sample and a female sample of ballet students. The weight-preoccupied group displayed similar mean levels of body dissatisfaction and weight preoccupation as the patients with anorexia nervosa. However, only a portion of the weight-preoccupied women were engaged in clinical eating disorder behaviours. A cluster analysis of the weight-preoccupied group revealed one group of women who were elevated on all subscales of the EDI and in fact scored as high as or higher than the anorexia nervosa group on dieting and weight preoccupation, but the second cluster was elevated only on scores of body dissatisfaction, drive for thinness and perfectionism. This weight-preoccupied-only group had low scores on all other EDI subscales including the bulimia subscale which contains items pertaining to binge eating and purging (core eating disorder behaviours).

Although these results by Garner et al. (1984) suggest that eating disorder behaviours were low or absent in this second cluster of weight-preoccupied women, it is important to note that binge eating and purging do not represent all eating disorder behaviours and these women may have been elevated on other eating behaviours, such as caloric restriction leading to weight loss. Indeed, both clusters of weight-preoccupied women scored high on the drive for thinness subscale which includes eating behaviours related to dieting and/or restriction of food intake. However, it is important to keep in mind that measures of dieting do not necessarily represent or capture the caloric restriction that leads to weight loss in anorexia nervosa. The relation between dieting and restriction is addressed in more detail at the end of this section.

Garner et al. (1984) concluded that their results supported the notion that some weight-preoccupied women resemble individuals with clinical eating disorders, and are likely a subclinical variant of a clinical syndrome, but that not all weight preoccupied women resemble eating disorder populations. In fact, Garner and colleagues (1984) were the first to point out that the pursuit of thinness is not necessarily associated with psychopathology. We suggest that the main difference between weight-preoccupied women who do and who do not resemble an eating disorder population is in whether or not they engage in clinical eating disorder behaviours. This is difficult to establish by examining existing research because measures of eating disorder symptoms have never clearly delineated thoughts from behaviours. Even the studies by Garner et al. (1984) do not distinguish between behavioural and psychological factors since they measured

symptoms using the Eating Disorder Inventory, where thought and behaviour items are mixed within the same subscale.

While high weight preoccupation in a non-clinical population is not necessarily associated with eating disorder behaviours, it can be. Eating disorder behaviours do occur in non-clinical populations, albeit at low frequencies. For instance, quoting the same research studies as above, statistics from Project EAT note 9.4% of girls and 13.5% of boys engage in recurrent purging behaviours such as vomiting, laxative abuse or excessive exercise (Ackard et al., 2007). Chamay-Weber et al. (2005) report purging behaviours (vomiting, laxatives, and diuretics) between 5% and 16%; and McVey et al. (2004) found 3.9% of their sample endorsed binge eating and 1.5% endorsed self-induced vomiting.

Despite the presence of eating disorder behaviours in non-clinical populations, we still maintain that clinical eating behaviours are discrete. We would argue that engaging in any level or frequency of eating disorder behaviours moves you from the non-clinical domain, to the sub-clinical/clinical domain. For behaviours to be on a continuum with normalcy there needs to be evidence showing the existence of eating disorder behaviours in non-clinical populations that are *not* associated with clinically significant distress and psychopathology. One way to show this would be to provide evidence that there is some "safe" level of eating disorder behaviours that is not associated with psychopathology such as impairment in self-esteem, depression, anxiety, etc. Or provide evidence that eating disorder behaviours can occur without the accompanying eating disorder thoughts, similar to what we have shown with weight preoccupied groups who do not engage in

any behavioural symptomology. There is at-present no empirical studies that have directly tested for the existence of "behaviour-only" groups and compared the level of psychological impairment associated with a range of eating disorder behaviours, the main reason being that we are the first to propose assessing and measuring eating disorder thoughts separately from eating disorder behaviours.

According to Stice, Ziemba, Margolis, and Flick (1996) the discontinuity perspective of eating disorders would be supported if research were to show that the same variables that distinguish non-clinical groups from sub-clinical groups fails to distinguish between sub-clinical and clinical populations. There is ample research evidence to support behaviours failing to distinguish sub-clinical and clinical populations. Sub-clinical and clinical groups both engage in eating disorder behaviours, and even though they often differ in the frequency with which they engage in these behaviours, current research does not support evidence of any meaningful distinction between those engaged in behaviours once a week versus twice a week (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Garfinkel et al., 1995; Kendler et al., 1991; Mond, Hay, Rodgers, Owen, & Mitchell, 2006). In fact, the accompanying pathology of those exhibiting sub-clinical symptoms of eating disorders has been shown to resemble the pathology observed in individuals with a full-blown eating disorder (Fairburn et al., 2007; Garfinkel et al., 1995; Zaider, Johnson, & Cockell 2000). And, the preponderance of those meeting criteria for a diagnosis of "eating disorder not-otherwise-specified" (EDNOS; approximately 60% of cases) suggests that the frequency level of eating disorder behaviours is less critical than the actual presence of eating disorder behaviours (see Fairburn & Bohn, 2005; Fairburn

& Cooper, 2007; Wade, Crosby, & Martin, 2006; Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007).

As noted above, an important caveat to our hypothesis of a discontinuous model of eating disorder behaviours is that not all behaviours related to eating pathology are discrete. We have argued that the behaviours outlined in the DSM criteria for eating disorders are the behaviours that are discrete— severe restriction, binge eating, and compensatory or non-compensatory strategies. Still, there are other behaviours that are not part of the diagnostic criteria for an eating disorder that are often found in both non-clinical and clinical eating disorder populations, such as body checking behaviours (weighing oneself, pinching body parts, glancing at one's body in reflective surfaces, etc.), excessive exercise and dieting which may not be discrete. Dieting is a prime example of this as rates of dieting among adolescents and young adults in non-clinical populations are alarmingly high (Neumark-Sztainer & Story, 1998). 'Dieting' is considered a clinically relevant behaviour for two reasons; its role as a risk factor and its role as a behavioural feature of an eating disorder, and often the distinction between the two becomes blurred by inconsistent definitions of what constitutes a 'diet'.

Both prospective and cross-sectional research indicates eating disorders commonly begin with behaviours that 'resemble' normal dieting (Fairburn, Cooper, Doll, & Davies, 2005; Fairburn & Harrison, 2003; Hsu, 1990; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). In longitudinal studies, dieting is most clearly demonstrable as a variable risk factor and maintenance factor to the onset of bulimia nervosa and anorexia nervosa binge-purge subtype because of its purported relation to binge eating (Fairburn,

1995; Jacobi et al., 2004; Polivy & Herman, 1985). The temporal precedence between dietary restraint and the onset of binge eating supports this assumption (Fairburn et al., 2005; Patton, Johnson-Sabine, Wood, Mann & Wakeling, 1990; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999).

For the restricting type of anorexia, dieting is presumed to be a forerunner to the more severe caloric restriction and starvation that produces the abnormally low body weight exhibited by those with anorexia. But dieting is not synonymous with restriction nor is it sufficient in producing the abnormally low body weight typical of anorexia nervosa. If dieting were equivalent to the restriction characteristic of an eating disorder, rates of anorexia nervosa would be higher, especially given the prevalence of dieting. In reality, anorexia restricting type is the rarest form of eating disorder (Hoek, 2006). In addition, the majority of dieters do not go on to develop an eating disorder, offering support to the role of dieting as a risk factor, rather than a symptom/feature of an eating disorder (Patton et al., 1990; Patton et al., 1999).

The relation between dieting and restriction in the eating disorders depends on how dieting is defined. Chronic dieting resulting in consistent caloric restriction accompanied by weight loss is consistent with clinical features of anorexia nervosa, and thus, in this sense, dieting appears to be interchangeable with restriction. Research by Lowe et al. (1996) indicates that dieting is related to eating disorder behaviours (e.g., binge eating) only when accompanied by food restriction and weight loss. In contrast, dieting practices accompanied by 'normative' body dissatisfaction may lead to emotional distress, but are not indicative of eating pathology. The DSM definition of anorexia

nervosa does not describe which eating behaviours are considered abnormal or clinically relevant in terms of what leads to the 'refusal to maintain minimally normal body weight'. Weight loss is referred to loosely as the result of a reduction in total food intake and this reduction is often preceded by the exclusion or restriction of certain foods from one's diet (APA, 2000).

Restriction in anorexia nervosa, by definition, always includes caloric deprivation and weight loss – in comparison, dieting can mean cognitive or behavioural deprivation, with or without weight loss. Dieting is most often defined and interpreted as a method of weight control, yet it is not always associated with weight loss (Brownell & Rodin, 1994; Garner & Wooley, 1991). In fact dieting is a notoriously ineffective means of achieving weight loss according to some researchers because 95% of those who lose weight will regain the weight within a few years and many will gain more than they originally lost (Grodstein, Levine, Spencer, Colditz, & Stampfer, 1996; National Institutes of Health Technology Assessment Conference Panel, 1993). The term "dieting" is also used in reference to cognitive restraint or the 'desire to lose weight' (e.g., see Neumark-Sztainer & Story, 1998) and for this reason it seems clear why numerous research studies have found self-reported dieting to be negatively related to actual reductions in caloric intake or weight loss (Lowe, 1993). Lowe (1993) has argued that none of the existing dietary restraint measures (e.g., Restraint Scale, Herman & Polivy, 1980) actually identify individuals in a state of energy deficit. In support of this are studies by Laessle and colleagues (1989a, 1989b), Lowe et al. (1996), Neumark-Sztainer, Jeffery, and French (1997), and more recently Stice, Cooper, Schoeller, Tappe and Lowe (2007), who have

demonstrated through various experimental, survey and observational research studies that self-reported dieting behaviours do not correspond well to actual caloric intake or to weight loss. In other words, dieters (who are defined by scores on measures of dietary restraint) are often not eating any less than non-dieters in the majority of these studies. Herman and Polivy (1984) have suggested that dieters may be best characterized by their attempts to lose weight than by actual weight loss.

Restriction in anorexia nervosa is unhealthy and pathological – but dieting is not necessarily unhealthy or pathological. Dieting is a heterogeneous term that includes both healthy (eating less fat, eating more fruits and vegetables, exercising) and unhealthy behaviours (skipping meals, fasting; Neumark-Sztainer & Story, 1998). According to Lowe and Timko (2004), dieting is neither beneficial nor harmful, but simply ineffective for most individuals in the long-term. Dieting may have beneficial effects if the purpose of the diet is to counteract a predisposition to overeat or gain weight (Lowe & Timko, 2004). Studies have found long-term low-calorie diets to be effective in decreasing binge eating in obese individuals with and without an eating disorder with length of follow-up varying from 3 to 24 months post-treatment (Epstein et al., 2001; Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998; Nauta et al., 2000; Telch & Agras, 1993; Wadden et al., 1994; Yanovski & Sebring, 1994; or for a review see Stice, 2002). Even in a normal weight population, Presnell and Stice (2003) found that when college students lost a small amount of weight in a weight-loss program, levels of bulimic symptoms were reduced – not increased after a 3 month follow-up. Lowe and Timko state that the merits

of "dieting" are inconclusive without first clearly defining what is meant by the term, specifying the purpose of the diet and towards which population it is intended.

These differences between the interpretation of and consequences to dieting versus restriction suggest the two are not synonymous with on another and that despite any overlap in behaviours across those who engage in normative dieting and those who engage in clinical restriction, dieting should not be used as a proxy for restriction unless the instruments used to measure dieting take into account cognitive versus behavioural restraint in conjunction with abnormally low BMI or measured weight loss. If the purpose is to detect restrictors rather than dieters, it is necessary to separate out the small percentage of successful dieters (5%) from the unsuccessful dieters (95%; Grodstein et al., 1996).

Modeling the Latent Constructs of Eating Disorder Symptoms

Most research on the continuous-discontinuous debate of eating disorders has come from cross-sectional investigations using cluster analysis (Garner et al., 1983; Hay, Fairburn, & Doll, 1996; Stice & Agras, 1999; Westen & Harnden-Fischer, 2001; Williamson, Gleaves, & Savin, 1992), latent class analysis (Bulik et al., 2000; Duncan et al., 2007; Keel et al., 2004; Striegel-Moore et al., 2005; Sullivan, Bulik, & Kendler, 1998a, 1998b; Sullivan & Kendler, 1998; Wade et al., 2006), discriminant function analysis (Franko & Omori, 1999; Golder, Srikameswaran, Schroeder, Livesley, & Birmingham, 1999; Lowe et al., 1996; Stice, Killen, Hayward, & Taylor, 1998; Stice et al., 1996; Tylka & Subich, 1999), factor analysis (Gleaves & Eberenz, 1993, 1995; Gleaves, Williamson, & Barker, 1993; Tobin, Johnson, Steinberg, & Staats, 1991;

Vanderheyden, Fekken, & Boland, 1988; Williamson et al., 2002) and most recently, a series of taxometric investigations (see Williamson et al., 2005). These studies have been mixed in their findings of continuous and discontinuous models of eating disorders (for a review of these studies see Gleaves et al., 2004; Williamson et al., 2005; Wonderlich et al., 2007). The problem with latent class, discriminant function, factor and cluster analysis techniques is the a priori assumption that the data are categorical or dimensional rather than testing ad hoc for a dimensional or categorical structure (Williamson et al., 2005).

Taxometric analyses specifically test for dimensional versus categorical constructs in a dataset (Waller & Meehl, 1998; Meehl, 1992). A taxon is an underlying entity that drives the relations between common indicators of a disorder, similar to a latent variable in factor analysis; however, a taxon indicates a discrete category while a latent factor could be either dimensional or discrete. A taxon, with a certain degree of error, can separate those with clinical eating disorders, those who are symptomatic but not clinical, and those who are healthy, and thus the utility of this analytic technique has spurred a number of taxometric investigations in the field of eating disorders over the last five years.

Williamson et al. (2002) examined three latent features of eating disorders derived through a series of exploratory and confirmatory factor analyses; binge eating, fear of fatness/compensatory behaviours, and drive for thinness. These latent scores were then used as indicators in a series of taxometric analyses comparing individuals with clinical eating disorders to obese persons without an eating disorder and to a normal weight

comparison group. Binge eating disorder and bulimia nervosa were judged to be qualitatively distinct (taxonic) from both the normal-weight comparison group and the obese comparison group. In contrast, taxometric analyses did not reveal strong support for anorexia nervosa being qualitatively distinct from normalcy (i.e. not taxonic; Williamson et al., 2002). Excluding non-eating disorder groups, Williamson et al. went on to compare the categorical versus dimensional characteristics within the eating disorders (i.e., anorexia nervosa, bulimia nervosa, and binge eating disorder) and concluded that while there was mixed support for a categorical model of eating disorder subtypes, some of the *features* appeared categorical and some appeared dimensional. Taken together, the taxometric results reported by Williamson et al. support 'bingeing and purging' disorders being taxonic (i.e., categorical), especially when using a mixed sample (clinical and non-clinical), while anorexia nervosa appears to be more dimensional. These findings are consistent with the three existing taxometric studies conducted on eating disorder populations to date (Gleaves, Lowe, Green et al, 2000; Gleaves, Lowe, Snow et al., 200l; Tylka & Subich, 2003).

How might we interpret these findings that certain features of eating disorders are discrete from non-clinical populations while others are continuous and certain features within the eating disorders are discrete while others are continuous? If we examine these results from a framework of 'continuous eating disorder thoughts' and 'discontinuous eating disorder behaviours' we can make clearer the inconsistencies reported across taxometric studies. Bingeing and purging disorders (bulimia nervosa and anorexia nervosa binge-purge subtype) may appear taxonic especially in mixed samples because

they are defined by the presence of behaviours, which incidentally occur at exceedingly low base rates in non-clinical populations. Bingeing and purging are extreme behaviours that are specific to eating disorder populations; thus when comparing clinical and non-clinical groups, rates of bingeing and purging will be very high in clinical populations and very low in normal populations, increasing the likelihood of detecting a taxon.

In contrast, restricting behaviours (anorexia-restricting subtype) are often measured using dieting items (e.g., Drive for Thinness subscale of the EDI) which are common among non-clinical populations (Chamay-Weber et al., 2005; Neumark-Sztainer, 2005). In general, when restriction is measured in terms of dieting there will be more people in both clinical and non-clinical populations who will endorse items such as ("I eat diet foods") than items related to bingeing or purging ("I vomit after I have eaten"). The range of restricting behaviours will reflect a continuum in taxometric analyses compared to bingeing and purging where the limited variability will result in stronger evidence for a taxon. Williamson et al. (2005) proposed a three-dimensional model that argued for binge eating being taxonic while "fear of fatness/compensatory strategies", and "drive for thinness" were continuous. The appearance of a continuum of 'ear of fatness/compensatory strategies' may be an artifact of including restricting items that range from somewhat normative behaviours (skipping breakfast, periods of fasting, eating diet foods) to more extreme behaviours (prolonged periods of starvation and chronic lowering of caloric intake) in taxometric studies. We suspect that if Williamson et al. were to re-specify their model by only including extreme restricting items that reflect weight loss, they may find restricting behaviours to appear taxonic. In addition,

the continuous nature of this "fear of fatness-compensatory strategies" may be confounded by the combination of thought and behavioural items within the same latent factor. For example, latent factors for "fear of fatness" have typically combined items pertaining to thoughts, (preoccupation with weight and shape) with items of behaviours (caloric limitation) within the same latent class (e.g., Keel et al., 2004). Therefore conclusions regarding the continuous nature of restricting behaviours may be premature.

Taxometric analyses on non-eating disordered populations can also be understood within a thought-behaviour framework. Tylka and Subich (2003) examined the latent structure of eating disorders by performing taxometric analyses on a sample of 532 college women. They included items representative of non-behavioural features of eating disorders such as body dissatisfaction, and found five distinct factors, all indicative of a dimensional solution. Specifically, taxometric analyses revealed no presence of a taxon in the data, although importantly, Tylka and Subich did not include *any* behavioural indicators of eating disorders in their model. These authors argue that much of the opposing findings in the literature surrounding the existence of a continuous versus a discontinuous model of eating disorders results from the fact that different indicators are used by different researchers. While some researchers use behavioural indicators (binge eating, purging, restricting) with non-behavioural indicators (interoceptive awareness, maturity fears or body dissatisfaction), other researchers use one but not the other. When researchers utilize more behavioural indictors of eating disorders than non-behavioural indicators, results support categorical models of eating disorders (Lowe et al., 1996; Gleaves, Lowe, Green et al., 2000; Gleaves, Lowe, Snow, et al., 2000), whereas studies

that use more non-behavioural indicators find more evidence for dimensional models of eating disorders (Tylka & Subich, 1999, 2002, 2003).

Tylka and Subich (2003) argue that bingeing and purging should not be included in research aimed at uncovering the latent structure of eating disorders because these indicators may either inflate the notion of a categorical model or mask the presence of dimensional models of eating disorders. These authors suggest that one reason for this inflation may be that some researchers classify females in their sample as clinical or non-clinical based on bingeing and purging behaviours only to turn around and use these *same* behaviours as the criterion variable. Evidence in support of a taxon in the data may be artificial because of confounding indicators with the criterion (Tylka & Subich, 2003). However, while Tylka and Subich (2003) suggest excluding behavioural symptoms of eating disorders, behavioural indicators are fundamental attributes of individuals with eating disorders and must be considered in the continuum debate. Evidence for both categorical *and* dimensional models in taxometric studies are not conflicting findings; they are in fact telling us something critical about the nature of eating disorders.

Psychological versus behavioural symptoms in recovery

A number of researchers have found the rate and timing of eating disorder recovery to vary depending on the presence or absence of psychological criteria in the definition of recovery (Clausen, 2004; Cogley & Keel, 2003; Couturier & Lock, 2006; Crisp, Hsu, Harding, & Hartshorn, 1980; Eckert, Halmi, Marchi, Grove, & Crosby, 1995; Fennig, Fennig, & Roe, 2002; Herzog, Schellber, & Deter, 1997; Saccoman, Savoini, Naselli, Cirrincione, & Matricardi, 1989; Strober, Freeman, & Morrell, 1997). In general,

psychological recovery occurs more slowly, extending years beyond the cessation of behavioural symptoms and as a result, rates of recovery vary depending on the length of remission studied and the variables included in definitions. Saccoman et al. (1989) found that when only the physical aspects of an eating disorder (restricting, bingeing, purging) were considered, 79% of those with anorexia in their study had recovered. Yet when the psychological criteria were examined, this recovery rate fell to 48%. Strober et al. (1997), report recovery rates based on normal weight and regular menstruation to be 86% occurring on average 57.4 months from illness onset. But in this same study, recovery based on psychological criteria was only 76%; occurring an average of 79.1 months from illness onset. Similarly, Fennig et al. (2002) found the psychosocial recovery to take on average two years longer than the physical (i.e., behavioural) recovery from an eating disorder.

Bachner-Melman, Zohar, and Ebstein (2006) compared women behaviourally but not cognitively (i.e., lack of body image distortion or fear of weight gain) recovered from anorexia nervosa to a group of women recovered both cognitively and behaviourally and found that the symptoms and personality profile of the behaviourally-recovered group showed residual features of anorexia whereas the cognitively- and behaviourally-recovered were indistinguishable from controls. It seems that while behavioural recovery is critical for initial symptom remission, psychological recovery is necessary for longer term maintenance of recovery from eating disorders (Couturier & Lock, 2006). In a similar study, Cogley and Keel (2003) evaluated the concurrent validity of requiring remission of 'undue influence of weight and shape on self-evaluation' in defining

recovery from bulimia nervosa. Three groups were compared: 31 women fully recovered from bulimia, 28 women behaviourally recovered only and 59 matched controls. Participants completed measures of mood, anxiety, psychosocial functioning and body dissatisfaction. Results showed no differences between matched controls and the fully recovered individuals with bulimia on any measures, while the behaviourally-recovered only group showed significant pathology across all reported measures.

Of particular importance in the distinction between behavioural and psychological recovery are the findings by researchers that body dissatisfaction and overvaluation are the *best* predictors of relapse. For instance, Fairburn, Peveler, Jones, Hope, and Doll (1993) and Freeman, Beach, Davis, and Solyom (1985) found the residual level of shape concern that remained at the end of "successful" treatment was the strongest predictor of relapse among patients with bulimia nervosa. These findings are disconcerting given the high rate of shape concern remaining among individuals who have completed treatment successfully; as many as 30-50% according to some estimates (Farrell, Shafran, Lee, & Fairburn, 2005; Probst, Vandereycken, Coppenolle, & Pieters, 1999). Keel, Dorer, Franko, Jackson, and Herzog (2005) found similar results in their prospective study of predictors of relapse. Specifically, Keel et al. report that body image disturbance was the best predictor of relapse at nine years post-treatment across both bulimia and anorexia, and this was across a range of relapse predictors including behavioural, psychological and Axis I and II comorbid conditions.

Aside from empirical support for the distinction between psychological and behavioural symptoms in recovery, another important perspective on recovery that is

often neglected is the view of the patient. Indeed, the patient's perspective on recovery is almost always absent from definitions of recovery (Jarman & Walsh, 1999). But a patient's own experience with an eating disorder and the factors they feel are significant to their improvement are essential components of recovery and to understanding the efficacy of various treatments. Interestingly when patients' perspectives have been included in definitions of recovery what is revealed is the importance patients place on *psychological* recovery, not behavioural recovery. Only rarely is recovery mentioned in association with behavioural eating disorder symptoms (see Keski-Rahkonen & Tozzi, 2005; Noordenbos, 1992; Noordenbos & Seubring, 2006).

The overvaluation of shape and weight appears among individuals in non-clinical populations, clinical populations, and recovered or partially recovered populations. In contrast, behaviours are absent in non-clinical groups, acutely present in clinical groups and absent or remitted in recovered or partially recovered groups. This ongoing battle for psychological recovery across the course of the eating disorder offers support to our hypothesis of a continuum of disordered thinking, while disordered behaviours are more specific to the active phase of the illness where individuals are still meeting diagnostic criteria for an eating disorder.

Clinical and Research Implications

If we scrutinize the many studies conducted over the last few decades that have found evidence in favour of either discrete or continuous models of eating disorders, we see that the competing results of most studies are accurate when we consider the sample composition and the features of eating disorders that were examined. Tylka and Subich

(2003) argued that many studies find evidence for categorical models when using behavioural features of eating disorders and more dimensional models emerge when using non-behavioural features; we would agree since it is precisely the behavioural features that appear categorical, not the disorder itself. When researchers examine clinical populations, or mixed samples, there will be more evidence for categorical models because the clinical groups will have more of the disordered behaviours, whereas a truly non-clinical population will mostly be comprised of a continuum of disordered thinkers who do not engage in disordered behaviours.

It is important to highlight that we are not suggesting eating disorder behaviours are the only difference between clinical and non-clinical populations, rather we would argue in a similar vein to Crisp (1965) and Bruch (1973), as well as Polivy and Herman (1987), that those with eating disorders have fundamental differences in personality, such as ego deficits or psychopathology such as comorbid mood disorder. Engaging in bingeing, purging, or restricting behaviours can be painful and difficult, both mentally and physically, and individuals who engage in these aberrant behaviours likely have underlying personality features that are very different from those who do not engage in these behaviours. For instance, impulsivity, characteristic of some individuals with bulimia (Bulik, Sullivan, Joyce, & Carter, 1995; Kleifield, Sunday, Hurt, & Halmi, 1993) may facilitate the disinhibited eating of a binge. Impulsivity is also associated with substance abuse, and the use of alcohol or other drugs among individuals with eating disorders has been linked to frequency of binges and purges (Davis & Claridge, 1998; de Silva & Eysenck, 1987; Feldman & Eysenck, 1986). Concurrent mood disorder may

facilitate restricting or bingeing behaviours as a result of changes to brain neurochemistry which can disrupt appetite and/or feelings of satiety (Halmi & Sunday, 1991; Moreno & Judd, 2001). Additionally, neuroticism has been linked to symptoms of eating disorders (Cervera et al., 2003; Davis & Claridge, 1998; Miller, Schmidt, Vaillancourt, McDougall, & Laliberté, 2006), with the suggestion that fear of weight gain may enable the highly neurotic individual to restrict food intake more easily than those low in neuroticism. Herman and Polivy's (1984) boundary model of eating behaviour postulates that both anorexics and bulimics have a lowered hunger boundary that allows them to tolerate greater degrees of food deprivation than normal. This 'hunger boundary' may explain the restricting anorexics' ability to maintain consumption at such stringent levels (Polivy and Herman, 1987). Although, the degree to which this hunger boundary is imposed by environmental factors, a genetic predisposition, or both is uncertain.

Not only may individuals who engage in disordered behaviours differ from disordered thinkers in terms of personality and psychiatric profile, but there will be heterogeneity within this group as well. Individuals who purge may differ from those who restrict because underlying personality features may drive the choice of a particular coping mechanism (the behaviour) in dealing with the shared psychopathology (overvaluation of shape and weight) of eating disorders. What this would imply is that divergent findings concerning shared and non-shared personality features between bulimia nervosa and anorexia nervosa reflect at once the similarity in underlying psychopathology (overvaluation of shape and weight) as well as differences in chosen behavioural manifestation across eating disorder subtypes. In support of this assumption

are latent class and cluster analysis studies which have identified three to four clusters of personality profiles among eating disorder individuals that are largely *independent* of eating disorder diagnosis, and that differ based on etiologic variables such as family background and eating disorder *symptoms* (see Wonderlich et al., 2007).

From an epidemiological perspective, researchers interested in the measurement of eating disorder symptoms are in need of improved screening instruments that can help sort groups based on those in need of treatment, those who would benefit from prevention (high-risk), and those that need neither intervention nor prevention as this would improve the efficiency of research protocols and avoid unnecessary diagnostic follow-ups. However, creating more efficient screening tools that can sort categories into clinical/sub-clinical, high-risk and no-risk requires an understanding of whether these groups are in fact discrete (taxonic) or whether they fall along a continuum. For instance, if eating disorder thoughts are continuous, the psychometric goal is to choose items that disperse the scores widely and discriminate all regions of the dimension (Meehl, 1992; Ruscio & Ruscio, 2002). When dealing with behaviours, if they are in fact categorical, the goal will be to sort items at a best cut-off so as to minimize "in/out" misclassifications (Meehl, 1992). Thus, the development of eating disorder assessment instruments should be based on the nature of the construct being measured. Most epidemiological research to date has been based on the assumption that eating disorders exist on a continuum. If eating disorder thoughts are continuous, but behaviours are discrete then screening for eating disorders using a cut-off of 20 out of 26 items on the Eating Attitudes Test is an arbitrary criteria and one that has and will continue to result in a number of false positives in

screening for clinical syndromes. If our theoretical hypothesis is correct then screening instruments should essentially be used to look for a relatively high cut point on psychological symptoms of eating disorders, and separately, determine the presence or absence of clinically relevant eating disorder behaviours.

Future Research:

Do thoughts predict behaviours? Rizvi, Stice and Agras (1999) measured rates of bulimic behaviours and eating disorder attitudes over two 6-year periods and found the behaviours decreased while eating disorder attitudes increased. However, Rizvi et al. (1999) measured eating attitudes by examining mean scores on the EDI subscales where thought and behaviour items are mixed and therefore what the change in eating attitudes refers to is not clear.

What is the role of the emotional or affective component of disordered thoughts? Some researchers have found that body dissatisfaction and weight control behaviours are related through the mediating role of emotional distress or negative affect associated with their body dissatisfaction (Sim & Zeman, 2005; Stice, Presnell, & Spangler, 2002). A similar triad between body dissatisfaction, negative affect and subsequent weight control efforts has been modeled using the dual pathway model of bulimic pathology (see Stice, 2001), although not within the context of a continuum of eating disorder thoughts and a discontinuum of eating behaviours.

What is the absolute threshold for feeling fat and hating your body before one actually engages in eating disorder behaviours? We would predict that the developmental course from disordered thoughts to disordered thoughts-and-behaviours represents a

critical phase in the development of eating disorders and holds potential for informing us of what protects and what harms at this crucial juncture.

If eating disorder thoughts are continuous and eating disorder behaviours discrete, we expect future research studies investigating this hypothesis to find distinct latent factors for the thoughts and behaviours related to eating disorders when examining non-clinical populations. However, among clinical groups, we predict eating disorder thoughts will be indistinguishable from eating disorder behaviours because of the high occurrence of both thoughts and behaviours among clinical eating disorder groups. We would also expect to see evidence of taxonicity in behavioural factors but not psychological factors, particularly if restricting behaviours are measured in terms of weight loss in order to account for the increasing normality of dietary restraint among the general population. Moreover, we would expect to find similar patterns of relationships in clinical and non-clinical populations when it comes to the variables associated with eating disorder thoughts (e.g., neuroticism, perfectionism). In contrast we would not expect to see similar relationships across clinical and non-clinical groups among variables associated with eating disorder behaviours (e.g., impulsivity).

Conclusion

The goal of this review was to introduce a new framework for understanding the inconsistent findings surrounding the continuous or discontinuous nature of eating disorders. Regardless of whether future research supports or refutes the notion of taxonicity in disordered behaviours and/or continuity in disordered thoughts, the purpose of this review was to highlight the importance and benefit of examining the role of

psychological symptoms in the eating disorders independently from the behavioural symptoms.

The argument over the continuity and discontinuity of eating disorders and psychopathology more generally, is akin to the historic debate concerning the relative contribution of genetics versus environment to human personality and behaviour. For hundreds of years scientists have argued vehemently their perspective of the nature-nurture controversy, each side finding evidence in support of their particular theoretical biases. It is only more recently, specifically in the latter half of the 19th century, that modern scientists have begun to concede this nature-nurture debate by adopting a more holistic interactionist approach, admitting the crucial, even if disproportionate role of both genes *and* environment in human development. Despite great strides made in the last half century in our understanding of the underlying nature of eating disorders, the results of these studies and the literature reviewed herein suggest a need to re-think the continuum-discontinuum perspective of eating disorder to one that will encompass the multifaceted nature of eating and weight-related pathologies.

References

Ackard, D. M., Fulkerson, J. A., & Neumark-Sztainer, D. (2007). Prevalence and utility of DSM-IV eating disorder diagnostic criteria among youth. *International Journal of Eating Disorders, 40*, 409-417.

Adams, H. E., & Cassidy, J. F. (1993). The classification of abnormal behaviour. In P.B. Sutker and H.E. Adams (Eds.), *Comprehensive Handbook of Psychopathology, 2nd ed.* (pp. 3-25). New York, Plenum.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders (1st ed.)*. Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd *ed.*). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th *ed.*, text revision). Washington, DC: Author.

Attie, I., & Brooks-Gunn, J. (1989). Development of eating problems in adolescent girls: A longitudinal study. *Developmental Psychology, 25*, 70-79.

Bachner-Melman, R., Zohar, A. H., & Ebstein, R. P. (2006). An examination of cognitive versus behavioural components of recovery from anorexia nervosa. *The Journal of Nervous and Mental Disease, 194*, 697-703.

Beumont, P. (2002). Clinical presentation of anorexia nervosa and bulimia nervosa. In C. G. Fairburn and K. D. Brownell (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook, 2nd ed.* (pp. 162-170). New York, Guilford Press.

Brownell, K. D., & Rodin, J. (1994). The dieting maelstrom: Is it possible and advisable to lose weight? *American Psychologist, 49*, 781-791.

Bruch, H. (1973). *Eating Disorders*. New York: Basic Books.

Bulik, C. M., Sullivan, P. F., Joyce, P. R., & Carter, F. A. (1995). Temperament, character, and personality disorder in bulimia nervosa. *Journal of Nervous and Mental Disease, 183*, 593-598.

Bulik, C. M., Sullivan, P. F., & Kendler, K. S. (2000). An empirical study of the classification of eating disorders. *American Journal of Psychiatry, 157*, 886-895.

Button, E. J., & Whitehouse, A. (1981). Subclinical anorexia nervosa. *Psychological Medicine, 11*, 509-516.

Cash, T. F., Morrow, J. A., Hrabosky, J. I., & Perry, A. A. (2004). How has body image changed? A cross-sectional investigation of college women and men from 1983 to 2001. *Journal of Consulting and Clinical Psychology, 72*, 1081-1089.

Cervera, S., Lahortiga, F., Martínez-González, M. A., Gual, P., Irala-Estévez, J. D., & Alonso, Y. (2003). Neuroticism and low self-esteem as risk factors for incident eating disorders in a prospective cohort study. *International Journal of Eating Disorders, 33*, 271-280.

Chamay-Weber, C., Narring, F., & Michaud, P. (2005). Partial eating disorders among adolescents: A review. *Journal of Adolescent Health, 37*, 417-427.

Clausen, L. (2004). Time course of symptom remission in eating disorders. *International Journal of Eating Disorder, 36*, 296-306.

Cogley, C. B., & Keel, P. K. (2003). Requiring remission of undue influence of weight and shape on self-evaluation in the definition of recovery for bulimia nervosa. *International Journal of Eating Disorders, 34*, 200–210.

Cooper, P. J., & Goodyer, I., (1997). Prevalence of weight and shape concerns in girls aged 11-16 years. *British Journal of Psychiatry, 171*, 42-44.

Cooper, P. J., Taylor, M. J., Cooper, Z., & Fairburn, C. G. (1987). The development and validation of the body shape questionnaire. *International Journal of Eating Disorders, 6*, 485-494.

Couturier, J., & Lock, J. (2006). What is recovery in adolescent anorexia nervosa? *International Journal of Eating Disorders, 39*, 550-555.

Crisp, A. H. (1965). Some aspects of the evolution, presentation and follow-up of anorexia nervosa. *Proceedings of the Royal Society of Medicine, 11*, 509-516.

Crisp, A. H., Hsu, L. K., Harding, B., & Hartshorn, J. (1980). Clinical features of anorexia nervosa: A study of a consecutive series of 102 female patients. *Journal of Psychosomatic Research, 24*, 179-191.

Crow, S. J., Agras, W. S., Halmi, K., Mitchell, J. E., & Kraemer, H. C. (2002). Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: A multicenter study. *International Journal of Eating Disorders, 32*, 309-318.

Davis, C., & Claridge, G. (1998). The eating disorders as addiction: A psychobiological perspective. *Addictive Behaviors, 23*, 463-475.

de Silva, P., & Eysenck, S. (1987). Personality and addictiveness in anorexic and bulimic patients. *Personality and Individual Differences, 8*, 749-751.

Duncan, A. E., Bucholz, K. K., Neuman, R. J., Agrawal, A., Madden, P. A. F., & Heath, A. (2007). Clustering of eating disorder symptoms in a general population female twin sample: A latent class analysis. *Psychological Medicine, 37*, 1097-1107.

Eckert, E. D., Halmi, K. A., Marchi, P., Grove, W., & Crosby, R. (1995). Ten-year follow-up of anorexia nervosa: Clinical course and outcome. *Psychological Medicine, 25*, 143-156.

Epstein, L. H., Paluch, R. A., Saelens, B. E., Ernst, M. M., & Wilfley, D. E. (2001). Changes in eating disorder symptoms with pediatric obesity treatment. *Journal of Pediatrics, 139*, 58-65.

Fairburn, C. G. (1995). *Overcoming Binge Eating.* New York, Guilford Press.

Fairburn, C. G., & Bohn, K. (2005). Eating disorder NOS (EDNOS): An example of the troublesome "not otherwise specified" (NOS) category in DSM-IV. *Behavior Research and Therapy, 43*, 691-701.

Fairburn, C. G., & Cooper, Z. (2007). Thinking afresh about the classification of eating disorders. *International Journal of Eating Disorders, 40*, S107-S110.

Fairburn, C. G., Cooper, Z., Bohn, K., O'Connor, M. E., Doll, H. A., & Palmer, R. L. (2007). The severity and status of eating disorder NOS: Implications for DSM-V. *Behaviour Research and Therapy, 45*, 1705-1715.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: A prospective, population-based study. *American Journal of Psychiatry, 162*, 2249-2255.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Welch, S. L. (1999). Risk factors for anorexia nervosa: Three integrated case-control comparisons. *Archives of General Psychiatry, 56*, 468-476.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic theory and treatment. *Behavior Research and Therapy, 41*, 509-528.

Fairburn, C. G., Doll, H. A., & Welch, S. L., Hay, P. J., Davies, B. A., & O'Connor, M. E. (1998). Risk factors for binge eating disorder: A community-based case-control study. *Archives of General Psychiatry, 55*, 425-432.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *The Lancet, 361*, 407-416.

Fairburn, C. G., Welch, S. L., Doll, H. A., Davies, B. A., & O'Connor, M. E. (1997). Risk factors for bulimia nervosa: A community-based case-control study. *Archives of General Psychiatry, 54*, 509-517.

Fairburn, C. G., Peveler, R. C., Jones, R., Hope, R. A., & Doll, H. A. (1993). Predictors of 12 month outcome in bulimia nervosa and the influence of attitudes to shape and weight. *Journal of Consulting and Clinical Psychology, 61*, 696-698.

Farrell, C., Shafran, R., Lee, M., & Fairburn, C. G. (2005). Testing a brief cognitive-behavioural intervention to improve extreme shape concern: A case series. *Behavioral and Cognitive Psychotherapy, 33*, 189-200.

Feingold, A., & Mazzella, R. (1998). Gender differences in body image are increasing. *Psychological Science, 9*, 190-195.

Feldman, J., & Eysenck, S. B. (1986). Addictive personality traits in bulimic patients. *Personality and Individual Differences, 7*, 923-926.

Fennig, S., Fennig, S., & Roe, D. (2002). Physical recovery in anorexia nervosa: Is this the sole purpose of a child and adolescent medical-psychiatric unit? *General Hospital Psychiatry, 24*, 87-92.

Franko, D. L., & Omori, M. (1999). Subclinical eating disorders in adolescent women: A test of the continuity hypothesis and its psychological correlates. *Journal of Adolescence, 22*, 389-396.

Freeman, R. J., Beach, B., Davis, R., & Solyom, L. (1985). The prediction of relapse in bulimia nervosa. *Journal of Psychiatry Research, 19*, 349-353.

Fries, H. (1977). Studies on secondary amenorrhea, anorectic behaviour and body image perception: Importance for the early recognition of anorexia nervosa. In R. Vigersky (Ed.), *Anorexia nervosa* (pp. 163-176). New York: Raven.

Gangestad, S., & Snyder, M. (1985). "To carve nature at its joints": On the existence of discrete classes in personality. *Psychological Review, 92*, 317-349.

Garfinkel, P. E. (2002). Classification and diagnosis of eating disorders. In C. G. Fairburn and K. D. Brownell (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook, 2nd ed.* (pp. 155-161). New York, Guilford Press.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., et al. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *American Journal of Psychiatry, 152*, 1052-1058.

Garner, D. M. (2002). Measurement of eating disorder psychopathology. In C. G. Fairburn and K. D. Brownell (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook, 2nd ed.* (pp. 141-146). New York, Guilford Press.

Garner, D. M., & Garfinkel, P. E. (1980). Socio-cultural factors in the development of anorexia nervosa. *Psychological Medicine, 10*, 647-656.

Garner, D. M., Garner, M. V., & Rosen, L. W. (1993). Anorexia nervosa "restrictors" who purge: Implications for subtyping anorexia nervosa. *International Journal of Eating Disorders, 13*, 171-185.

Garner, D. M., Olmstead, M. P., & Garfinkel, P. E. (1983). Does anorexia nervosa exist on a continuum? Subgroups of weight-preoccupied women and their relationship to anorexia nervosa. *International Journal of Eating Disorders, 2*, 11-20.

Garner, D. M., Olmstead, M. P., Polivy, J., & Garfinkel, P. E. (1984). Comparison between weight-preoccupied women and anorexia nervosa. *Psychosomatic Medicine, 46*, 255-266.

Garner, D. M., & Wooley, S. (1991). Confronting the failure of behavioural and dietary treatments for obesity. *Clinical Psychology Review, 11*, 729-780.

Gleaves, D. H., Brown, J. D., & Warren, C. S. (2004). The continuity/discontinuity models of eating disorders: A review of the literature and implications for assessment, treatment, and prevention. *Behavior Modification, 28*, 739-762.

Gleaves, D. H., & Eberenz, K. (1993). The psychopathology of anorexia nervosa: A factor analytic investigation. *Journal of Psychopathology and Behavioral Assessment, 15*, 141-152.

Gleaves, D. H., & Eberenz, K. P. (1995). Validating a multidimensional model of the psychopathology of bulimia nervosa. *Journal of Clinical Psychology, 51*, 181-189.

Gleaves, D. H., Lowe, M. R., Green, B. A., Cororve, M. B., & Williams, T. L. (2000). Do anorexia and bulimia nervosa occur on a continuum? A taxometric analysis. *Behavior Therapy, 31*, 195-219.

Gleaves, D. H., Lowe, M. R., Snow, A. C., Green, B. A., & Murphy-Eberenz, K. P. (2000). Continuity and discontinuity models of bulimia nervosa: A taxometric investigation. *Journal of Abnormal Psychology, 109*, 56-68.

Gleaves, D. H., Williamson, D. A., & Barker, S. E. (1993). Confirmatory factor analysis of a multidimensional model of bulimia nervosa. *Journal of Abnormal Psychology, 102*, 173-176.

Goldner, E. M., Srikameswaran, S., Schroeder, M. L., Livesley, W. J., & Birmingham, C. L. (1999). Dimensional assessment of personality pathology in patients with eating disorders. *Psychiatry Research, 85*, 151-159.

Goodrick, G. K., Poston, W. S., Kimball, K. T., Reeves, R. S., & Foreyt, J. P. (1998). Nondieting versus dieting treatments for overweight binge-eating women. *Journal of Consulting and Clinical Psychology, 66*, 363-368.

Grodstein, F., Levine, R., Spencer, T., Colditz, G. A., Stampfer, M. J. (1996). Three-year follow-up of participants in a commercial weight loss program: Can you keep it off? *Archives of Internal Medicine, 156,* 1302-1306.

Halmi, K. A., & Sunday, S. R. (1991). Temporal patterns of hunger and fullness ratings and related cognitions in anorexia and bulimia. *Appetite, 16,* 219-237.

Hay, P. J., Fairburn, C. G., & Doll, H. A. (1996). The classification of bulimic eating disorders: A community-based cluster analysis study. *Psychological Medicine, 26,* 801-812.

Herman, C. P., & Polivy, J. (1980). Restrained eating. In A. J. Stunkard (Ed.), *Obesity* (pp. 208-225). Philadelphia: W. B. Saunders.

Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. In A. J. Stunkard & E. Stellar (Eds.), *Eating and its disorders* (pp. 141-156). New York: Raven Press.

Herzog, W., Schellberg, D., Deter, H. C. (1997). First recovery in anorexia nervosa patients in the long-term course: A discrete-time survival analysis. *Journal of Consulting and Clinical Psychology, 65,* 169-177.

Hsu, L. K. G. (1990). *Eating Disorders*. New York, Guilford.

Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Applications of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin, 130,* 19-65.

Jarman, M., & Walsh, S. (1999). Evaluating recovery from anorexia nervosa and bulimia nervosa: Integrating lessons learned from research and clinical practice. *Clinical Psychological Review, 19*, 773-788.

Katzman, M.A., & Wolchik, S.A. (1984). Bulimia and binge eating in college women: A comparison of personality and behavioural characteristics. *Journal of Consulting and Clinical Psychology, 52*, 423–428.

Keel, P. K., Dorer, D. J., Franko, D. L., Jackson, S. C., & Herzog, D. B. (2005). Postremission predictors of relapse in women with eatiung disorder. *American Journal of Psychiatry, 16*, 2263-2268.

Keel, P. K., Fichter, M., Quadflieg, N., Bulik, C. M., Baxter, M. G., Thornton, L., et al. (2004). Application of a latent class analysis to empirically define eating disorder phenotypes. *Archives of General Psychiatry, 61*, 192-200.

Kendler, K. S., MacLean, C., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1991). The genetic epidemiology of bulimia nervosa. *American Journal of Psychiatry, 148*, 1627-1637.

Keski-Rahkonen, A., & Tozzi, F. (2005). The process of recovery in eating disorder sufferers' own words: An internet-based study. *International Journal of Eating Disorders, 37*, S80-S86.

Killen, J. D., Taylor, C. B., Hammer, L. D., & Litt, I. (1993). An attempt to modify unhealthful eating attitudes and weight regulation practices of young adolescent girls. *International Journal of Eating Disorders, 13*, 369-384.

Killen, J. D., Taylor, C. B., Hayward, C., & Wilson, D. M. (1994). Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: A three-year prospective analysis. *International Journal of Eating Disorders, 16*, 227-238.

Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Wilson, D. M., Hammer, L., et al. (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study. *Journal of Consulting and Clinical Psychology, 64*, 936-940.

Kleifield, E. I., Sunday, S., Hurt, S. W., & Halmi, K. A. (1993). Psychometric validation of the tridimensional personality questionnaire: Application to subgroups of eating disorders. *Comprehensive Psychiatry, 34*, 249-253.

Kraepelin, E. (1913). *General Paresis*. (monog. series, no. 14.). Nervous and Mental Disease Publishing Company: New York

Krueger, R. F., & Piasecki, T. M. (2002). Toward a dimensional and psychometrically-informed approach to conceptualizing psychopathology. *Behavior Research and Therapy, 40*, 485-500.

Laessle, R. G., Tuschl, R. J., Kotthaus, B. C., & Pirke, K. M. (1989a). A comparison of the validity of three scales for the assessment of dietary restraint. *Journal of Abnormal Psychology, 98*, 504-507.

Laessle, R. G., Tuschl, R. J., Waadt, S., & Pirke, K. M. (1989b). The specific psychopathology of bulimia nervosa: A comparison with restrained and

unrestrained (normal) eaters. *Journal of Consulting and Clinical Psychology, 57*, 772-775.

Lowe, M. R. (1993). The effects of dieting on eating behaviour: A three-factor model. *Psychological Bulletin, 114*, 100-121.

Lowe, M. R., Gleaves, D. H., DiSimone-Weiss, R. T., Furgueson, C., Gayda, C. A., & Kolsky, P. A., et al. (1996). Restraint, dieting, and the continuum model of bulimia nervosa. *Journal of Abnormal Psychology, 105*, 508-517.

Lowe, M. R., & Timko, C. A. (2004). Dieting: Really harmful, merely ineffective or actually helpful? *British Journal of Nutrition, 92*, S19-S22.

McVey, G., Tweed, S., & Blackmore, E. (2004). Dieting among preadolescent and young adolescent females. *Canadian Medical Association Journal, 170,* 1559-61.

Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality, 60*, 117-174.

Miller, J. L., Schmidt, L. A., Vaillancourt, T., McDougall, P., & Laliberte, M. (2006). Neuroticism and introversion: A risky combination for disordered eating among a non-clinical sample of undergraduate women. *Eating Behaviors, 7*, 69-78.

Mond, J. J., Hay, P. J., Rodgers, B., Owen, C., & Mitchell, J. (2006). Correlates of the use of purging and non-purging methods of weight control in a community sample of women. *Australian and New Zealand Journal of Psychiatry, 40*, 136-142.

Moreno, M. A, & Judd, R. (2001). Eating disorders: Bulimia. *eMedicine Journal, 2*, 1-14.

National Institutes of Health Technology Assessment Conference Panel. (1993). Methods for voluntary weight loss and control. *Annals of Internal Medicine, 199*, 764-770.

Nauta, H., Hospers, H., Kok, G., & Jansen, A. (2000). A comparison between a cognitive and a behavioural treatment for obese binge eaters and obese non-binge eaters. *Behavior Therapy, 31*, 441-461.

Neumark-Sztainer, D. (2005). *I'm, Like, SO Fat!* New York: The Guilford Press.

Neumark-Sztainer, D., Jeffrey, R. W., & French, S. A. (1997). Self-reported dieting: How should we ask? What does it mean? Associations between dieting and reported energy intake. *International Journal of Eating Disorders, 22*, 437-449.

Neumark-Sztainer, D., Story, M. (1998). Dieting and binge eating among adolescents: What do they really mean? *Journal of the American Dietetic Association, 98*, 446-450.

Noordenbos, G. (1992). Important factors in the process of recovery according to patients with anorexia nervosa. In W. Herzog, H.C. Deter, & W. Vandereycken (Eds.), *The course of eating disorders: Long-term follow-up studies of anorexia and bulimia nervosa* (pp.304-322). London: Springer-Verlag.

Noordenbos, G., & Seubring, A. (2006). Criteria for recovery from eating disorders according to patients and therapists. *Eating Disorders, 14*, 41-54.

Nylander, J. (1971). The feeling of being fat and dieting in a school population: Epidemiologic interview investigation. *Acta Sociomedica Scandinavica, 3*, 17-26.

Park, J., Beaudet, M. P. (2007). Eating attitudes and their correlates among Canadian women concerned about their weight. *European Eating Disorders Review, 15,* 311-320.

Patton, G. C., Johnson-Sabine, E., Wood, K., Mann, A. H., & Wakeling, A. (1990). Abnormal eating attitudes in London schoolgirls – A prospective epidemiological study: Outcome at twelve month follow-up. *Psychological Medicine, 20*, 383-394.

Patton, G. C., Selzer, R., Coffey, C., Carlin, J. B., & Wolfe, R. (1999). Onset of adolescent eating disorders: Population based cohort study over 3 years. *British Medical Journal, 318*, 765-768.

Piran, N., & Gadalla, T. (2006). Eating disorders and substance abuse in Canadian women: A national study. *Addiction, 102*, 105-113.

Polivy, J., & Herman, C. P. (1985). Dieting and binging. *American Psychologist, 40*, 193-210.

Polivy, J., & Herman, C. P. (1987). Diagnosis and treatment of normal eating. *Journal of Consulting and Clinical Psychology, Special Issue: Eating Disorders, 55*, 635-644.

Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology, 53*, 187-213.

Prather, R. C., & Williamson, D. A. (1988). Psychopathology associated with bulimia, binge eating, and obesity. *International Journal of Eating Disorders, 7*, 177–184.

Presnell, K., & Stice, E. (2003). An experimental test of the effect of weight-loss dieting on bulimic pathology: Tipping the scales in a different direction. *Journal of Abnormal Psychology, 112*, 166-170.

Probst, M., Vandereycken, W., Coppenolle, H. V., & Pieters, G. (1999). Body experience in eating disorders before and after treatment: A follow-up study. *European Psychiatry, 14*, 333-340.

Rizvi, S. L., Stice, E., & Agras, W. S. (1999). Natural history of disordered eating attitudes and behaviours over a 6-year period. *International Journal of Eating Disorders, 26*, 406-413.

Rodin, J., Silberstein, L., & Striegel-Moore, R. (1984). Women and weight: A normative discontent. *Nebraska Symposium on Motivation, 32*, 267-307.

Rosen, J. C. (1992). Body image disorder: Definition, development, and contribution to eating disorders. In J. H. Crowther, D. L. Tennenbaum, S. E. Hobfoll, & M. A. P. Stephens (Eds.), *The etiology of bulimia: The individual and familial context (pp.* 157-177). Washington, DC: Hemisphere.

Ruderman, A. J., & Besbeas, M. (1992). Psychological characteristics of dieters and bulimics. *Journal of Abnormal Psychology 101,* 383–390.

Ruscio, J., & Ruscio, A. M. (2002). A structure-based approach to psychological measurement: Matching measurement models to latent structure. *Assessment, 9*, 4-16.

Saccoman, L., Savoini, M., Naselli, A., Cirrincione, M., & Matricardi, A. (1989). Anorexia nervosa in adolescents: Clinical aspects of the diagnosis and at follow-up. *Minerva-Pediatrica, 41*, 5-10.

Selvini Palazzoli, M. (1978). *Self-starvation: From individualization to family therapy in the treatment of anorexia nervosa.* New York: Aronson.

Sim, L., & Zeman, J. (2005). Emotion Regulation Factors as Mediators Between Body Dissatisfaction and Bulimic Symptoms in Early Adolescent Girls. *The Journal of Early Adolescence, 25,* 478-496.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin, 128*, 825-848.

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology, 110*, 124-135.

Stice, E., & Agras, W. S. (1999). Subtyping bulimic women along dietary restraint and negative affect dimensions. *Journal of Consulting and Clinical Psychology, 67*, 460-469.

Stice, E., Cooper, J. A., Schoeller, D. A., Tappe, K., & Lowe, M. R. (2007). Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioural data suggest not. *Psychological Assessment, 19*, 449–458.

Stice, E., Killen, J. D., Hayward, C., & Taylor, C. B. (1998). Support for the continuity hypothesis of bulimic pathology. *Journal of Consulting and Clinical Psychology, 66*, 784-790.

Stice, E., Presnell, K., Spangler, D. (2002). Risk factors for binge eating onset in adolescent girls: a 2-year prospective investigation. *Health Psychology, 21*, 131-138.

Stice, E., & Shaw, H. E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *Journal of Psychosomatic Research, 53*, 985-993.

Stice, E., Ziemba, C., Margolis, J., & Flick, P. (1996). The dual pathway model differentiates bulimics, subclinical bulimics, and controls: Testing the continuity hypothesis. *Behavior Therapy, 27*, 531-549.

Striegel-Moore, R., Franko, D. L., Thompson, D., Barton, B., Schreiber, G. B., & Daniels, S. R. (2005). An empirical study of the typology of bulimia nervosa and its spectrum variants. *Psychological Medicine, 35*, 1563-1572.

Striegel-Moore, R. H., Silberstein, L. R., Frensch, P., & Rodin, J. (1989). A prospective study of disordered eating among college students. *International Journal of Eating Disorders, 8*, 499-509.

Striegel-Moore, R. H., Silberstein, L. R., & Rodin, J. (1986). Toward an understanding of risk factors for bulimia. *American Psychologist, 41*, 246-263.

Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders, 22*, 339-360.

Sullivan, P. F., Bulik, C. M., & Kendler, K. S. (1998a). The epidemiology and classification of bulimia nervosa. *Psychological Medicine, 28*, 599-610.

Sullivan, P. F., Bulik, C. M., & Kendler, K. S. (1998b). Genetic epidemiology of bingeing and vomiting. *British Journal of Psychiatry, 173*, 75-79.

Sullivan, P. F., & Kendler, K. S. (1998). Typology of common psychiatric syndromes: An empirical study. *British Journal of Psychiatry, 173*, 312-319.

Telch, C. F., & Agras, W. S. (1993). The effects of a very low calorie diet on binge eating. *Behavior Therapy, 24*, 177-193.

Thompson, J. K. (1990). *Body image disturbance: Assessment and Treatment*. New York: Pergamon Press.

Thompson, J. K., Heinberg, L. J., Altabe, M., & Tantleff-Dunn, S. (1999). *Exacting beauty: Theory, assessment, and treatment of body image disturbance*. American Psychological Association, Washington, DC: US.

Tiggemann, M. (2004). Body image across the adult life span: Stability and change. *Body Image, 1*, 29-41.

Tobin, D. L., Johnson, C., Steinberg, S., & Staats, M. (1991). Multifactorial assessment of bulimia nervosa. *Journal of Abnormal Psychology, 100*, 14-21.

Tylka, T. L., & Subich, L. M. (2003). Revisiting the latent structure of eating disorders: Taxometric analyses with nonbehavioural indicators. *Journal of Counseling Psychology, 50*, 276-286.

Tylka, T. L., & Subich, L. M. (2002). A preliminary investigation of the eating disorder continuum with men. *Journal of Counseling Psychology, 49*, 273-279.

Tylka, T. L., & Subich, L. M. (1999). Exploring the construct validity of the eating disorder continuum. *Journal of Counseling Psychology, 46*, 268-276.

Vanderheyden, D. A., Fekken, G. C., & Boland, F. J. (1988). Critical variables associated with bingeing and bulimia in a university population: A factor analytic study. *International Journal of Eating Disorders, 7*, 321-329.

Wade, T. D., Crosby, R. D., & Martin, N. G. (2006). Use of latent profile analysis to identify eating disorder phenotypes in an adult Australian twin cohort. *Archives of General Psychiatry, 63*, 1377-1384.

Wadden, T. A., Foster, G. D., & Letizia, K. A. (1994). One-year behavioural treatment of obesity: Comparisons of moderate and severe caloric restriction and the effects of weight maintenance therapy. *Journal of Consulting and Clinical Psychology, 62*, 165-171.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua.* Sage Publications, Inc, Thousand Oaks, CA: US.

Watson, D., & Clark, L. A. (2006). Clinical diagnosis at the crossroads. *Clinical Psychology: Science and Practice, 13*, 210-215.

Westen, D., & Harnden-Fischer, J. (2001). Personality profiles in eating disorders: Rethinking the distinction between axis I and axis II. *American Journal of Psychiatry, 158*, 547-562.

Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the diagnostic and statistical manual of mental disorders--fifth edition. *Journal of Abnormal Psychology. Special Issue: Toward a Dimensionally Based Taxonomy of Psychopathology, 114*, 494-504.

Wiederman, M. W., & Pryor, T. L. (2000). Body dissatisfaction, bulimia, and depression among women: The mediating role of drive for thinness. *International Journal of Eating Disorders, 27*, 90-95.

Wilfley D. E., Bishop, M. E., Wilson, G. T., Agras, W. S. (2007). Classification of eating disorders: toward DSM-V. *International Journal of Eating Disorders, 40*, S123–129.

Williamson, D. A., Gleaves, D. H., & Savin, S. S. (1992). Empirical classification of eating disorder not otherwise specified: Support for DSM-IV changes. *Journal of Psychopathology and Behavioral Assessment, 14*, 201-216.

Williamson, D. A., Gleaves, D. H., & Stewart, T. M. (2005). Categorical versus dimensional models of eating disorders: An examination of the evidence. *International Journal of Eating Disorders, 37*, 1-10.

Williamson, D. A., Womble, L. G., Smeets, M. A. M., Netemeyer, R. G., Thaw, J. M., & Kutlesic, V., et al. (2002). Latent structure of eating disorder symptoms: A factor

analytic and taxometric investigation. *American Journal of Psychiatry, 159*, 412-418.

Wonderlich, S. A., Joiner, T. E., Keel, P. K., Williamson, D. A., & Crosby, R. D. (2007). Eating disorder diagnoses. *American Psychologist, 62*, 167-180.

Yanovski, S., & Sebring, N. (1994). Recorded food intake of obese women with binge eating disorder before and after weight loss. *International Journal of Eating Disorders, 15*, 135-150.

Zaider, T. I., Johnson, G., Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *International Journal of Eating Disorders, 28,* 58-67.

CHAPTER 5

Miller, J.L., Vaillancourt, T., & Hanna, S. (submitted June 20, 2008). Second-Order Confirmatory Factor Analysis of "Eating-Disorder-Thoughts" versus "Eating-Disorder-Behaviours": Implications for Assessment and Detection of Eating Disorders in Epidemiological Studies.

Abstract

Objective: A theoretical model of eating disorders that distinguishes between eating disorder thoughts, "I feel fat" and eating disorder acts, "I vomit after eating" may be useful for the measurement and detection of eating disorders in epidemiological studies. **Method**: This theoretical model was tested with a large non-clinical sample (N=1816) of female university students using confirmatory factor analysis to determine whether the core clinical features of eating disorders (e.g., fear of fat, dissatisfaction with body shape and weight, binging, purging and restricting) could be separated based on a thought–behavioural distinction and hierarchically organized into a second-order confirmatory factor structure. Based on these factors, composite groups based on high, moderate or low levels of thoughts and behaviours were created. Mean differences in risk for depression across composite groups were examined using ANOVA. **Results**: A second-order model of "eating disorder thoughts" and "eating disorder behaviours" was supported by the data, based on model fit, factor loadings, and model parsimony. Results of the ANOVA indicated that mean scores on depression were highest for groups with any level of disordered behaviour combined with moderate to high levels of thoughts. **Conclusion**: Detection of eating disorders in non-clinical populations may be enhanced by measuring on a high threshold of thoughts, or on moderate levels of thoughts combined with the presence of any level of eating disorder behaviour.

Introduction

Previous examinations of the latent structure of eating disorders have been based on the assumption that eating disorders exist on a continuum (Garner, Olmsted, & Polivy, 1983). However, in a recent review of the continuum theory of eating disorders, Miller and Vaillancourt (2008) suggest that viewing eating disorders based on a separation of the symptoms into "thoughts" and "behaviours" may simplify the way we understand the differences between clinical, sub-clinical, and non-clinical eating syndromes.

Individuals with sub-clinical or clinical eating disorder diagnoses have both thought and behavioural disturbances related to food, eating, and weight. Individuals in non-clinical populations share a startling degree of psychological disturbance in their fear of weight gain and body dissatisfaction, and for some, a degree of psychological disturbance that approximates clinical levels (Polivy & Herman, 1987). What is not shared across clinical and non-clinical populations are the behavioural indicators of an eating disorder – the severe caloric restriction, compensatory strategies and binge eating. While individuals in a non-clinical population may engage in "thoughts about behaviours" (e.g., "I have thought of vomiting to try and lose weight", "I think about bingeing") the majority of these individuals will not follow through on these thoughts, and those that do act on these disordered thoughts are in fact part of a sub-clinical or clinical group (Miller & Vaillancourt, 2008). Miller and Vaillancourt have hypothesized that this suggests a continuum of eating disorder thoughts, and a discontinuum of eating disorder behaviours.

In Miller and Vaillancourt's (2008) review they discuss three lines of evidence that offer support for the notion that behaviours may be discrete while thoughts are on a continuum. First, clinical eating disorder behaviours (severe caloric restriction, bingeing and purging) occur at very low base rates in the general population, yet eating disorder thoughts such as fear of weight gain, fear of fat, and body dissatisfaction are rampant. For example, in a review of epidemiological studies of partial syndrome eating disorders from 1980 to 2003, Chamay-Weber, Narring, and Michaud (2005) found 46% to 80% of adolescent girls in the United States report intense body dissatisfaction. Using a national Canadian sample (N=36 984), Piran and Gadalla (2006) found 26% of females aged 15-24 responded "yes" to having a *strong fear* of being too fat in the past 12 months. Yet a high prevalence of eating disorder thoughts in epidemiological samples is not always accompanied by eating disorder behaviours (Miller & Vaillancourt, 2008). According to Miller and Vaillancourt's theory, although it is possible to have a weight-preoccupied population that does not engage in clinically significant eating disorder behaviours (i.e., the 'normative discontent' – Rodin, Silberstein, & Striegel-Moore, 1984), once behavioural symptoms are introduced, a sub-clinical population is now present. That is, while behavioural symptoms are necessarily associated with eating disorder thoughts, and with pathology, eating disorder thoughts can occur independent of the behaviours and independent of pathology.

Second, when empirical tests of the continuum hypothesis rely on behavioural indictors (vomiting, bingeing) to examine the latent structure of eating disorders, a categorical model of eating disorders is more often supported (Gleaves, Lowe, Green,

Cororve, & Williams, 2000; Gleaves, Lowe, Snow, Green, & Murphy-Eberenz, 2000; Lowe et al., 1996). Conversely, studies that use non-behavioural indicators (fear of weight gain, body dissatisfaction) find more support for a dimensional model of eating disorders (Mintz & Betz, 1988; Tylka & Subich, 1999, 2002, 2003).

Third, the distinction between eating disorder behaviours and eating disorder thoughts is also evident in the treatment literature, where recovery status is already defined by the presence/absence of psychological and behavioural symptoms separately (Miller & Vaillancourt, 2008). For instance, the rate and timing of recovery in the eating disorders varies depending on the presence of psychological symptoms, specifically, psychological recovery occurs more slowly, extending years beyond the cessation of behavioural symptoms (Strober, Freeman, & Morrell, 1997). Psychological and behavioural symptoms are differentially related to outcome, relapse, and timing of recovery, highlighting the importance of considering psychological and behavioural symptoms separately (Miller & Vaillancourt, 2008).

The current study consisted of two parts, both of which address primary assumptions of the theoretical hypothesis of continuous thoughts and discontinuous behaviours. In the first part of the study, the question of distinct latent constructs for eating disorder thoughts and eating disorder behaviours is addressed by modeling a theoretically-driven, two-factor second-order confirmatory factor analysis (CFA) model of eating disorder symptoms - one that is based on the theoretical notion that the pathology inherent in eating disorders can be reduced to an underlying dimension of thoughts and a discrete class of behaviours. Although CFA cannot directly test for

continuity in the latent constructs (Williamson, Gleaves, & Stewart, 2005), developing a stable measurement model of thoughts and behaviours is a necessary first step before testing for continuity in these latent factors.

This theoretical CFA model differs from previous models examining the latent structure of eating disorders, not only in the number of factors and the composition of factors, but also in the underlying theoretical factor structure. Of the nine confirmatory factor studies published in the last 20 years none have articulated a thought-behaviour distinction among eating disorders, or within disordered eating symptomology (Espelage et al., 2003; Gleaves, Williamson, & Barker, 1993; Gleaves & Eberenz, 1993, 1995; Price Foundation Collaborative Group, 2001; Tobin, Johnson, Steinberg, Staats, & Dennis, 1991; Vanderheyden, Fekken, & Boland, 1988; Varnado, Williamson, & Netemeyer, 1995; Williamson et al., 2002). In fact, although similar first-order latent factors have previously been modeled within a confirmatory factor structure (i.e., fear of fat; restrictive eating, bingeing, purging), none of these studies have separated within the scales the thoughts associated with dieting, "I wish I ate less, I would like my stomach to be empty" from actual behaviours "I avoid eating when I am hungry. I skip meals entirely."

Towards this aim, multiple items from the Eating Disorder Inventory-2 (EDI-2) and the Eating Attitudes Test-26 (EAT-26) were used to determine if two distinct (but related) latent factors could be found based on a division of thoughts and behaviours in a large sample of university women who represent a range of variability in terms of normal and pathological eating and thought disturbances.

Using a different analytic approach, the second part of the study also assesses the theory that what is shared across clinical and non-clinical populations are the thoughts indicators of an eating disorder and not the behavioural indicators. Specifically, using the factor scores derived from the CFA part of the study, composite groups were formed based on the degree to which thoughts and behaviours were endorsed in the sample: high, moderate and low thoughts and high, moderate and low behaviours. Based upon the proposed hypothesis, it was expected that individuals with high levels of "eating disorder thoughts" (e.g., body dissatisfaction, fear of fat) would not necessarily experience high levels of behaviours (restricting, purging, bingeing), but that high levels of behaviours would be inextricably linked to high levels of thoughts. In other words, while a robust group of individuals high on eating disorder thoughts, with or without eating disorder behaviours was expected, a group of individuals high on behaviours and low on thoughts was not anticipated.

In addition, considering the hypothesis that it is the presence of behaviours that discriminates non-clinical from sub-clinical/clinical, it was also expected that the risk of comorbid psychopathology (e.g., concurrent depression) would be highly differentiated by the presence of eating disorder behaviours. Indeed, while the combination of high thoughts and high behaviours was expected to be most strongly related to concurrent depression, it was also expected that even minimal endorsement of eating disorder behaviours in conjunction with eating disorder thoughts would be associated with significant risk for depression.

In sum, two distinct latent constructs with robust measurement properties based on a split between thought and behavioural indicators of eating disorder symptoms were expected. As well, on the basis of the theoretical hypothesis that eating disorder behaviours are specific to sub-clinical/clinical populations it was also predicted that the presence of behaviours within subgroups would be the marker for risk of depression.

Methods

Participants

Participants were 1816 female undergraduate students aged 20.3 years on average (*SD* = 2.5). The ethnic background of the participants was predominantly White/Caucasian (55.9%), followed by South Asian (15.5%), and Chinese (11.6%). The remaining 17% of participants were evenly distributed amongst Arab, Black, Latin-American, and Aboriginal ethnic groups. The Body Mass Index of participants (based on self-reported height and weight) was as follows: 8.8% were underweight (BMI<18.5); 70.5% were normal weight (BMI >19.0 and <24.9); 15.8% were overweight (BMI >25.0 and <29.9); and 4.8% were obese (BMI >30.0). The mean BMI for all participants was 22.62 (SD = 3.93; **min/max** = 15.12 to 49.20).

Procedures

Data were collected from a medium-sized southern Ontario university. Participants were recruited from the university's on-campus student residences and the university's student centre and were compensated $5.00 for participating in the study. All procedures were approved by the university's research ethics board. To facilitate data collection for such a large sample, data were collected on three separate occasions from

three independent samples over the course of a year. Potential differences between the three samples were examined using a one-way ANOVA, with the three samples entered as three levels of the independent variable (Group) and the EAT-26, EDI-bulimia, EDI-drive for thinness, EDI-body dissatisfaction subscale scores entered as the dependent variables. There were no statistically significant differences between the three samples on mean scores of the Eating Attitudes Test (total subscale score) or the three subscales of the Eating Disorder Inventory (Bulimia, Drive for thinness, Body Dissatisfaction) and thus the three samples were collated.[2]

Measures

The Eating Disorder Inventory (EDI-2): The EDI-2 (Garner et al., 1983), measures behavioural and symptomatic patterns of anorexia nervosa and bulimia nervosa. Three subscales of the Eating Disorder Inventory were included in the current study; Drive for Thinness (DFT; 7 items), Bulimia (B; 7 items), and Body Dissatisfaction (BD; 9 items). Sample items from each scale include: "I eat when I am upset" or "I have thought of trying to vomit in order to lose weight" (B); "I think about dieting" or "I am terrified of gaining weight" (DFT); "I think that my thighs are too large" or "I think that my stomach is too big" (BD). Respondents are asked to rate items based on the following metric: Always, Usually, Often, Sometimes, Rarely, or Never. Responses are then weighted and assigned values as follows: Always =3; Usually = 2; Often = 1; Sometimes = 0; Rarely = 0; and Never = 0. Item scores contribute to only one subscale each, and subscale scores are computed by summing all items in a particular subscale (Garner,

[2] Data for the ANOVA are not shown here, but available upon request.

1991). The EDI has established internal consistency, criterion-related validity, convergent, and discriminant validity for all subscales (Garner et al., 1983). Internal consistency scores, means and standard deviations for the Bulimia, Drive for thinness and Body dissatisfaction subscales along with normed comparisons from Garner (1991) are presented in Table 1.

Eating Attitudes Test (EAT-26): Disordered eating was further assessed through the EAT-26 (Garner, Olmsted, Bohr, & Garfinkel, 1982). Sample items from the EAT-26 are: "I vomit after I have eaten," "I engage in dieting behaviours," "I like my stomach to be empty," or "I am preoccupied with a desire to be thinner." Items were scored using the same metric as items from the EDI-2; 0 (rarely, never) to 3 (always) scale based on the frequency with which participants engage in behaviours related to food and dieting. The EAT-26 has been shown to be a reliable and valid assessment of clinical symptoms associated with anorexia nervosa (primarily) and bulimia nervosa (Garner et al., 1983). The internal consistency, mean, and standard deviation for the EAT-26 are presented in Table 1.

Beck Depression Inventory-II (BDI-II): Depressive symptoms were measured using the 21-item BDI-II (Beck, Steer, & Brown, 1996), however, the suicide item was removed from the scale in compliance with the institutional ethics review board, thus our depression total scores are based on 20 of the 21 original items. Items are scored on a 0 - 3 metric, with higher scores indicating more pathology and a total possible score of 60. Following conventional standards, an individual score of 19 or greater on the BDI-II is indicative of moderate to severe depression (Beck et al., 1996). However, the depression

score in this study was based on 20 items and therefore a cut-off score of 16 or greater was used as a marker for high depression risk. In addition, 9% of the sample had missing BDI scores, however none of the variables collected in the study differentiate participants who did and did not complete the BDI and thus the missing data were considered random. The Cronbach alpha for the 20-item depression scale was .92, with a mean of 13.5 and a standard deviation of 10.0.

Sample Characteristics: 7.3% of the sample reported a history of an eating disorder and 19.3% reported a history of mood or anxiety disorder. Regarding current psychopathology, 37.2% of the sample scored 16 or greater on the BDI-II indicating clinical risk for depression (see Beck et al., 1996). On the EAT-26, 10.5% scored above the recommended cut-off score of 20 indicating significant risk of an eating disorder (see Garner et al., 1982). Furthermore, 15.6% indicated they were currently dieting to lose weight and 65.5% reported having dieted one or more times in the last 5 years.

Testing for distinct latent constructs for eating disorder thoughts and behaviours

Model Building – Analytic Strategy: The selection of items for the measurement model was driven by the assumption that symptoms of eating disorders can be reduced to two underlying dimensions of disordered eating thoughts and disordered eating behaviours. The EDI-2 and the EAT-26 were used because they are the two most widely recognized and most commonly used measures in clinical and non-clinical settings (Garner, 2002) and because both scales contain unique components despite some overlap in items. While both instruments contain items that reflect eating disorder thoughts and behaviours, the thought-items and behaviour-items are mixed within subscales (e.g., "I

have thought of vomiting to try and lose weight" and "I stuff myself with food" from the EDI-bulimia subscale; "I think about dieting" and "I eat sweets and carbohydrates without feeling nervous" from the EDI-drive for thinness subscale). Thus, the first task was to separate out the thoughts from the behaviours. Accordingly, all 23 items of the EDI-2 Bulimia, Drive for Thinness and Body Dissatisfaction subscales as well as the 26 items of the Eating Attitudes Test were separated into eating disorder thoughts (29 items); eating disorder behaviours (17 items); or ambiguous (3 items).

Thought-items were categorized based on perceptions, emotions, and attitudes whereas behaviour-items were chosen based on actions and/or impulses. One item in particular was difficult to categorize; "I have the impulse to vomit after meals" which may reflect a thought process while at the same time may reflect a physiological arousal and a physical sensation that makes it more behavioural than cognitive. This item was placed in the behaviour grouping as it was an important symptom of eating pathology, as well as one of the few indicators for purging behaviours that were available across both measures (i.e., neither the EDI-2 nor the EAT-26 ask questions concerning use of diuretics, laxatives, diet pills, or other compensatory strategies).

Next, meaningful symptom categories were formed that were closely based on the core behaviours and thoughts of both anorexia nervosa and bulimia nervosa as outlined in the DSM-IV-TR: *Fear of fat/fear of weight gain, body dissatisfaction, restriction, compensatory strategy-purging, and bingeing.* Each of the 29 thought items, the 17 behaviour items and the 3 ambiguous items were placed into one of five symptom categories, or a sixth group, "other" for items that did not fit any of the five main

symptom categories. Although these symptom categories did not map onto DSM-IV-TR criteria per se, the categories were matched as closely as possible to DSM criteria, based on the available items from the EDI-2 and the EAT-26. For example, one of the subgroups was "body dissatisfaction" however the DSM-IV-TR criteria for an eating disorder is not just body dissatisfaction, but rather the undue influence of this body dissatisfaction on one's self-concept and self-esteem (American Psychiatric Association, 1994).

This sorting procedure resulted in the placement of six items in the *fear of fat/fear of weight gain* category, nine items in the *body dissatisfaction* category, six items in the *restricting* category, three items in the *purging* category, five items in the *bingeing* category, and the remaining 20 items were placed in the "*other*" category.

To test for reliability of these categorizations, 15 mental health professionals (11 clinical psychologists – 6 of whom are experts in eating disorders; and 4 psychiatrists) were enlisted to independently rate and code the 49 items of the EDI-2 and the EAT-26. The raters were asked to code each statement first based on whether it best represented a "Thought", a "Behaviour" or "Ambiguous", and second to categorize all items into 1 of 6 symptom categories (*Fear of fat/fear of weight gain, body dissatisfaction, restricting, purging, bingeing* or *other*). The inter-rater reliability was calculated using Fleiss' kappa coefficient (Fleiss, 1971). Fleiss' kappa is an extension of Cohen's kappa and Scott's pi statistic, which are measures of inter-rater reliability, useful when assessing agreement between two raters. However, Fleiss' kappa works for any number of raters giving categorical ratings to a fixed number of items. Similar to Cohen's kappa, Fleiss' kappa is

a chance-corrected measure of agreement—expressing the extent to which the observed amount of agreement among raters exceeds that which would be expected by chance alone. Agreement based on our first categorization of items into "Thoughts", "Behaviours", or "Ambiguous" was $K = .72$; where number of raters (n) was 15, number of items (N) was 49, and total categories (k) was 3. Agreement based on our second categorization of items into the symptom categories *fear of fat/fear of weight gain, body dissatisfaction, restricting, purging, bingeing* or *other* was $K = .75$; where number of raters (n) was 15, number of items (N) was 49, and total categories (k) was 6. According to Landis and Koch (1977), kappa values ranging between .61 - .80 reflect substantial agreement between raters. In addition, the percent agreement across categories ranged from 80% to 95%.

Exclusion Criteria for CFA: The items "I am preoccupied with the desire to be thinner", and "I have gone on eating binges where I felt that I could not stop" were found on both the EDI and EAT. The EDI items were kept while the EAT items were dropped as this was reflective of the order in which the self-report questionnaires were completed. Four positive (reversed scored) items of the body dissatisfaction category ("I think that my stomach is just the right size", "I like the shape of my buttocks", "I think my thighs are just the right size", "I think my hips are just the right size") were also excluded for model parsimony as these items were complimentary opposites of the negative body shape items already in the model. These exclusions changed *fear of fat/fear of weight gain* from a six-item to a five-item category; *body dissatisfaction* from a nine-item to a five-item category; and *bingeing* from a five-item to a four-item category.

Three additional items "I think about dieting", "I think about bingeing" and "I have thought of trying to vomit in order to lose weight" were also dropped as these items represented thoughts *about* behaviours and because there were not enough items to form both thought and behaviour latent constructs for restricting, bingeing and purging. Although the recommended number of indicators for a single latent factor is three to four items (Anderson & Gerbing, 1988; Kline, 2005), the *purging* factor had only two items because of the poor representation of purging behaviours across the EDI-2 and the EAT-26. Still, two-indicator latent factor may be assessed provided that the sufficient conditions for identification are met which include 1) the matrix of error variance/covariance is diagonal; 2) all latent variables are given a scale and in the case of a two-indicator latent variable this is accomplished by setting the factor variance equal to one; 3) the factor complexity of each observed variable is 1.00; and 4) all latent factors are free to covary (Bollen, 1989). The model met all of these conditions. Moreover, Garson (2008) has argued that the prime consideration in selecting indicators is whether they are theoretically sound and reliably measured (Garson, 2008). Purging behaviours are one of the three core eating disorder behaviours and have the highest sensitivity and specificity of all eating disorder symptoms (Keski-Rahkonen et al., 2006). Therefore due to the importance of this symptom in defining eating pathology and because of the high face validity of the items the two-item *purging* latent factor was retained. The final items retained for testing the CFA model are found in Table 2.

Model Assumptions: Multivariate tests of normality were evaluated using AMOS 16.0 Normality Check. Three items violated assumptions of normality. Responses to the

items EAT-38, "I vomit after I have eaten", EAT-54, "I have the impulse to vomit after meals", and EDI-13, "I have gone on eating binges where I feel I may not be able to stop", clustered around low frequencies (see Table 2). All three items were positively skewed (values between 3.0 and 7.0) and items EAT-38 and EAT-54 showed moderate kurtosis (absolute values between 10 and 20 = moderate kurtosis: Kline, 2005) whereas item EDI-13 was less problematic with a kurtosis index <10. The fact that these items were not normally distributed is not surprising given that they reflect aberrant eating disorder behaviours (vomiting, bingeing), which occur at very low frequencies in non-clinical populations. Transforming these items was deemed inappropriate as these behaviours cluster around the tail end of the distribution not because of sampling or measurement error but rather because they are reflective of the true population variance (e.g., bulimia has a prevalence rate of less than 1% in the general population; Hoek, 2006). Thus, these items were retained in their original form (as were all the other items) despite their non-normal distributions because of their high face validity.

Missing Value Analysis: Missing values were replaced using mean replacement to facilitate the CFA analyses. Our CFA was based on the covariance matrix, which requires complete data (no empty cells). Otherwise, the means and intercepts must be estimated and estimation with the covariance matrix is preferred. The percent missing across the EDI and EAT variables was less than 1% and percent missing across the BDI items was less than 1% for all items except item #20, (loss of interest in sex), where there was 4% missing.

Goodness of Fit Criteria: Three global fit indices were examined to assess overall model fit: the Comparative Fit Index (CFI), the Tucker-Lewis Index (TLI), and the Root Mean Square Error of Approximation (RMSEA), with special attention given to the RMSEA. Hu and Bentler (1998) suggest that RMSEA values of .05 and lower reflect close fitting models but values between .08 and .05 are adequate. RMSEA also provides a significance test of close fit (PCLOSE) which tests the hypothesis that RMSEA is no greater than .05. A non-significant value is interpreted as evidence of good fit (Joreskog & Sorbom, 1996). Values over .90 for both the CFI and TLI are suggested as adequate fit although Hu and Bentler (1998) state that .95 or greater is ideal (Bollen, 1989; Kline, 2005). When comparing nested models the sequential chi-square difference test (Steiger, Shapiro, & Browne, 1985) and the Akaike's Information Criterion (AIC; Akaike, 1987) were used. It is important to note however that using goodness of fit indices to accept or reject models has been the subject of much debate within the field of Structure Equation Modeling. Although there are general rules for acceptance of model fit (ex., that CFI should be at least .90), Bollen (1989) reminds readers that these cut-offs are arbitrary and that evaluation of model fit must encompass more salient criterion such as comparing the fit of one model to the fit of another, strength of the factor loadings, and having theoretically driven models.

All CFA analyses were conducted on the covariance matrix using AMOS 16.0 with Maximum Likelihood Estimation (Analysis of Moment Structures; Amos Development Corporation, 2006).

Testing for distinct sub-groups of individuals on the basis of thoughts and behaviours

Using the factor scores derived from the CFA, nine composite groups based on the levels (high, moderate, or low) of the second-order factors "Eating Disorder Thoughts" and "Eating Disorder behaviours" were created. Five risk profiles were then computed and used to test for group differences in depression scores using a one-way analysis of variance. First, the latent factors "Thoughts" and "Behaviours" were trichotomized: high, moderate and low thoughts and high, moderate and low behaviours.

The cut-off points selected to convert each latent factor into a trichotomous variable were based on the weighted responses used to score the EDI-2 and EAT-26, developed by Garner (1991). The latent factor "Thoughts" which consisted of 10 indicators (see figure 1) was trichotomized into *high-thoughts* for scores greater than or equal to 40; *moderate-thoughts* for scores between 21-39; and *low-thoughts* for scores less than 20. A score of 40 or greater represents the minimum score possible for individuals who answered "always", "usually" and "often" (i.e., Garner's symptomatic response options) for each of the 10 items in the 'thought' subscale. For the purposes of this study Garner's original asymptomatic response options ("sometimes, rarely, or never") were split into moderately symptomatic (response option = "sometimes") and asymptomatic (response options "rarely/never") in order to test whether minimal endorsement of behaviours would reflect pathology. A score between 21 and 39 represents individuals who answered "sometimes", to all or some of the 10 items in the 'thought' subscale, and a score below 20 indicates individuals who responded "never" or "rarely" to each of the 10 Thought items. The latent factor 'behaviours' was trichotomized in the same manner, again, with 10 indicators (see figure 1), which meant

the cut-off criteria for *high-behaviours* were scores greater than or equal to 40; *moderate-behaviours* were scores between 21 and 39; and *low-behaviours* were scores less than 20 on the 'behaviour' subscale score.

Results

Confirmatory Factor Analysis

The theoretical model was assessed in steps in which the measurement components (first-order factor model) were examined first followed by the structural model (second-order factor model). The first model consisted of five latent factors: *Body Dissatisfaction* with five indicators; *Fear of Fat* with five indicators; *Restrict* with five indicators; *Binge* with three indicators and *Purge* with two indicators. All latent factors were free to correlate and all parameters freely estimated. A covariance between the residual errors for "I am terrified of gaining weight" and "I am terrified of being overweight" from the latent factor "Fear of fat" was added as these items are theoretically intertwined, given that those who are terrified of *any* amount of weight gain will also be terrified of being overweight. Based on goodness of fit indices, the model fit was quite good, $\chi^2(159) = 886.69$, CFI = .94, RMSEA = .050 (95% CI = .047 - .050, PCLOSE = .50), TLI = 0.93. Factor loadings ranged from .54 to .88 and are presented in Table 2.

Next the second-order factors, "Eating disorder thoughts" and "Eating disorder behaviours" were added in (see Figure 1). Model fit for the second-order CFA model was good, based on evaluation of the goodness of fit indices, $\chi^2(164) = 976.984$, CFI = .94, RMSEA = .053 (95% CI = .047 - .059, PCLOSE = .117), TLI = 0.93. Due to the high correlation between the two second-order factors ($r = .79$), the possibility that the model

may be better represented by one underlying factor rather than two was tested even though a positive correlation was expected due to the dependency of thoughts and behaviours among a subset of individuals in the sample representing a sub-clinical or clinical group. To test this assumption the two second-order factors (Thoughts and Behaviours) were collapsed into one factor (Eating Disorder Symptoms). Statistically significant declines in the first-order factor loadings along with a decline in model fit compared to the two-factor second-order CFA, $\chi^2(165) = 1093.5$, CFI = .92, RMSEA = .056 (95% CI = .053 - .059, PCLOSE = .001), TLI = 0.91 was found. This decline was statistically significant based on $\Delta\chi^2$ of 116.5 with 1 degree of freedom at $p < .001$. The RMSEA PCLOSE fit for this alternative model was statistically significant indicating that the model was not a close fit. Additionally, the AIC for both the 'one' versus 'two' second-order CFA models was evaluated. The AIC for the 'one-factor' second-order CFA Model was 1183.5 and the 'two-factor' second-order CFA Model was 1068.9. Importantly, because the AIC "rewards" models for parsimony, the fact that the more *complex* model (i.e., model with two second-order factors) has a smaller AIC value than the competing model with fewer parameters (the one-factor, second-order CFA) suggests that the two-factor hierarchical CFA (Eating Disorder Thoughts and Eating Disorder Behaviours) is in fact the better fitting model.

Based on all of these criteria combined the best fitting model was determined to be the hypothesized two-factor second-order CFA. Factor loadings for Restrict, Binge, Purge, Fear of Fat, and Body Dissatisfaction onto the Thought and Behaviour latent constructs are shown in Table 3.

Distinct sub-groups

The cross-tabulations for the trichotomized latent factors 'thoughts' and 'behaviours' are displayed in Table 4. *Low-thoughts* had a mean score of 17.41 (SD = 2.32), *moderate thoughts* had a mean score of 29.61 (SD = 5.25), and *high-thoughts* had a mean score of 46.14 (SD = 4.58). *Low-behaviours* had a mean score of 15.73 (SD = 2.75), *moderate behaviours* had a mean score of 26.18 (SD = 4.59), and *high-behaviours* had a mean score of 44.52 (SD = 3.89). Three cells had observed frequencies less than 1% (low thoughts/high behaviours; low thoughts/moderate behaviours; moderate thoughts/high behaviours) and were not included in any further analyses. Based on the remaining six cells, five risk profiles were created: Group 1 (asymptomatic) = low thoughts/low behaviours (13.5%); Group 2 (normative discontent) = moderate thoughts/low behaviours (41.6%); Group 3 (high-risk thinkers) = high thoughts/low behaviours (3.4%); Group 4 (sub-clinical) = moderate or high thoughts/moderate behaviours (38.8%); Group 5 (clinical) = high thoughts/high behaviours (2%).

A one-way analysis of variance was conducted to test for mean differences between the five risk profiles on the continuous dependent variable depression. The results of the ANOVA were statistically significant, $F(4,1603) = 67.72$, $p < .001$, indicating that the groups differed with respect to their mean depression scores. The variance accounted for in the model, as assessed by the partial Eta-squared (η^2) was 14.5%. Means and standard deviations on the depression inventory for each risk group are displayed in Figure 2. Group 3 (high risk thinkers), 4 (sub-clinical), and 5 (clinical), all had mean scores above the cut-off of 16 on the BDI-II, indicating significant clinical

risk for depression. Post-hoc comparisons using Tukey's HSD ($p < .05$) were conducted to evaluate pairwise differences among the mean scores of depression among our five risk profiles. Groups 1 and 2 (asymptomatic and normative discontent) did not differ significantly in their mean scores of depression ($p = .53$). Group 3 (high risk thinkers) and Group 4 (sub-clinical) did not differ significantly in their mean scores of depression ($p = .81$), but were both statistically significantly different from Groups 1 and 2 on mean depression scores. Group 5 (clinical) was statistically significantly different from Groups 1 to 4.

Discussion

The purpose of this study was to test a theoretical model of eating disorder symptoms that proposes a continuum of psychological symptoms and a discontinuum of behavioural symptoms. This theoretical model has implications for the method with which researchers and clinicians measure and establish risk of eating pathology in epidemiological surveys.

In the current study, a latent model of eating disorder thoughts and eating disorder behaviours was first examined as preliminary support for the proposed theoretical model. The results of the confirmatory factor analysis suggest that symptoms of eating disorders can in fact be distinguished based on a separation of "Eating Disorder Thoughts" and "Eating Disorder Behaviours". While both 'thoughts' and 'behaviours' showed considerable overlap, evidenced by the high inter-factor correlation between our two second-order factors, model fit and model parsimony clearly favored the two-factor model as opposed to a one-factor model. The results of the CFA present the first

measurement model of eating disorders where thoughts are measured independently from behaviours.

Of the first-order latent factors, *fear of fat* (standardized coefficient = .98) contributed more variance to the second-order latent factor "Eating Disorder Thoughts" than *body dissatisfaction* (standardized coefficient = .73). Given the high degree of comorbid anxiety among clinical eating disorders both at treatment (Godart, Perdereau, Jeammet, & Flament, 2003; Godart et al., 2003) and at follow-up (Steinhausen, 2002), as well as the correlation between anxiety and symptoms of disordered eating in non-clinical populations (John, Meyer, Rumf, & Hapke, 2006; Zaider, Johnson, & Cockell, 2000) the intensity of 'fear of fat' should be examined as a potential mediating variable between those with eating disorder thoughts and those with both thoughts and behaviours. The first-order factor *restricting* (standardized coefficient = .92) contributed the most variance to the second-order latent factor "Eating Disorder Behaviours". This is likely a reflection of the type of items that make up the *restricting* latent variable (e.g., "I eat diet foods"), as dieting behaviours have been shown to be more common than bingeing or vomiting behaviours in non-patient samples (Ackard, Fulkerson, Neumark-Sztainer, 2007), a pattern that was evident in the current study as well (see Table 2). Dieting is not a recognized diagnostic criterion for an eating disorder, but rather is considered relevant because it is associated with the onset of more severe caloric restriction that leads to the abnormally low body weight seen in anorexia nervosa (Miller & Vaillancourt, 2008). In the present study, a lower base rate of restricting behaviours would have been expected

had there been items reflecting chronic caloric deprivation, as opposed to items measuring dieting.

Support for the notion that behaviours are specific to sub-clinical/clinical populations was also provided by examining the frequency with which females in the present study endorsed problematic thoughts versus problematic behaviours. Examining the individual frequencies of each item retained in the CFA model revealed that behaviours occurred more infrequently than thoughts (see Table 2) which was expected given that a non-clinical sample was used. Eating disorder behaviours ranged from a low of 3% ("I vomit after I have eaten") to a high of 27% ("I stuff myself with food"). Eating disorder thoughts ranged from a low of 21% ("I think my buttocks are too large") to a high of 54% ("I think my stomach is too big"). A striking 46% of the sample reported feeling "*terrified* of gaining weight". These statistics are troubling given that the sample used in this study was non-clinical and comprised predominantly of normal weight women. Fear of being fat and fear of gaining weight are one of the fundamental diagnostic features of an eating disorder (APA, 1994). That nearly 50% of the females in this non-clinical sample endorsed a strong fear of being fat leads us to question the specificity of this item in terms of its use as a screening instrument. If the purpose of a screening study is to identify caseness in a non-clinical population, then using eating disorder thoughts will result in high rates of false positives. Alternatively, eating disorder behaviours will have higher specificity. Thus, the likelihood of detecting an individual who has an eating disorder when examining the level of eating disorder thoughts is low; but when the individual is engaged in eating disorder behaviours the likelihood of

detection is high. In the present study, sensitivity and specificity of the thought or behaviour items from the factor analysis were not formally assessed. Nevertheless, results from a study by Keski-Rahkonen et al. (2006), lend support to this argument, where the very best items for sensitivity and specificity were found when using purging behaviours, and the worst were found when using the Body Dissatisfaction subscale of the EDI-2, which incidentally, is comprised of only thought items.

It is important to note, however, that screens developed for the purpose of detection of clinical syndromes may not be appropriate for use in identifying at-risk individuals because of differences in target population, as well as research objectives (i.e., detection vs. prevention; Jacobi, Abascal, & Taylor, 2004; Offord, Kraemer, Kazdin, Jensen, & Harrington, 1998). Accordingly, if the purpose of the screen is to identify at-risk populations then eating disorder thoughts may be appropriate for use as a prognostic test, given that in the current study, high levels of eating disorder thoughts even without the eating disorder behaviours were still related to clinically significant scores on a measure of depression.

Using the factors derived from the CFA, risk composite groups based on the level of endorsement of thought and behavioural items were created and the resultant cross-tabulations are perhaps the most noteworthy finding of the study. Most of the females in this study (41.6%) were categorized as having no (or few) eating disorder behaviours combined with moderate levels of eating disorder thoughts (Group 2 – normative discontent; see Table 4). This 'normative discontent' group had twice the number of women as Group 1, the non-clinical group (13.5%). This suggests that in the present

study it was more common to have eating disorder thoughts than it was to not have them. Group 2 was termed "normative discontent" because it captured the essence of the weight preoccupation described by Rodin and colleagues two and a half decades ago, "For an overwhelming number of women in our society, being a woman means feeling too fat" (pg. 267; 1984). These authors critically noted the social phenomenon occurring in the 1970's and 80's, where worry and obsession over weight and dieting had reached proportions where it was considered normative to feel dissatisfied with your body. The results of this study support the notion of a normative discontent given that overall there were more women who had disordered thoughts (86%), than did not (14%).

As predicted, high levels of eating disorder thoughts were present either with or without eating disorder behaviours; whereas high levels of eating disorder behaviours were only associated with high levels of thoughts (see Table 4). Having moderate levels of eating disorder behaviours was associated with both moderate and high levels of eating disorder thoughts (23.0% and 15.8%). In other words, behaviours appeared *only* in conjunction with eating disorder thoughts. The number of individuals who were engaged in behaviours in the absence of eating disorder thoughts was less than 1%. Given that diagnostic criteria for an eating disorder entails the presence of both the psychological and the behavioural symptoms of eating disorders, these findings are consistent with the theoretical notion that behaviours are specific to clinical populations, while thoughts occur across both clinical and non-clinical populations, offering further support for the theory of discrete behaviours and a continuum of thoughts.

When the risk of depression across the five risk groups was examined several interesting yet predictable patterns were noted. First, as hypothesized, high levels of thoughts combined with high levels of behaviours was associated with the highest risk for depression. This group represents 2% of the sample and most likely reflects an undiagnosed clinical population. Second, being high on eating disorder thoughts without the accompanying eating disorder behaviours was also associated with clinically elevated scores on the depression inventory implying that there may be a threshold above which thoughts alone are associated with psychopathology. Figure 2 illustrates the 'dose response' relation between increasing levels of eating disorder thoughts and higher mean scores on depression, when eating behaviours are held constant. When behaviours are low or absent; only groups with high levels of eating disorder thoughts reach clinically significant levels of depression. Whereas the presence of even moderate levels of behaviours always signified clinically significant mean scores on depression.

Limitations

Although the results provide support for a model of eating disorders based on a behaviour-thought division, there are some considerations to this investigation that are important for future research. The first is that the items from the EDI-2 and EAT-26 were not originally constructed to distinguish between eating disorder thoughts and eating disorder behaviours. Items that reflect thoughts about behaviours need to be constructed in such a way as to take into account individuals who have the thoughts but not the behaviours (e.g., "I have thought of vomiting to lose weight, *but haven't.*"). Although it makes sense that thoughts about behaviours would be strongly correlated to actual

behaviours among some individuals (specifically among sub-clinical/clinical groups), the individual items need to be phrased to take into consideration those individuals who entertain the thoughts without following through on them. For instance, we would predict that while thoughts of vomiting (e.g., I have thought of vomiting to try and lose weight) would be highly correlated to actual vomiting (e.g., "I vomit after I have eaten") in clinical populations, there would be very small correlations between thoughts of vomiting and actual vomiting in non-clinical populations. This assumption could not be tested in the current study because it requires re-formatting or adding items to the existing EDI-2 and/or EAT-26 scales.

Moreover, in using the existing items from the EDI-2 and EAT-26, other methodological problems were encountered. For example, binge/purge/restrict 'thoughts' were excluded from the model because there were not enough of these types of items to form additional latent categories. The inclusion of items tapping thoughts about restricting behaviours such as "I wish I could not eat when I am hungry." "I wish I could suppress my appetite" "I am happy when I have no appetite" "I wish I ate less" or "I wouldn't mind having the flu if it meant I would lose weight", are the types of items we predict to be highly endorsed by weight-preoccupied women who are not at present engaged in eating disorder behaviours. These types of items would have likely strengthened the CFA model, and at the same time, would indicate a level of thought pathology that has not been previously examined in non-clinical populations.

Despite the problems with item representation in the EDI-2 and the EAT-26, an a priori decision was made to use the items from these existing scales as they have been

empirically shown to be valid indicators of eating-related thoughts and behaviours. Also, by using items from the EDI-2 and the EAT-26, we have made it possible for other researchers to test this theoretical model and replicate these findings given that these instruments are routinely collected in both clinical and non-clinical eating disorder samples. An important next step in testing this model however will be to examine items from other eating disorder instruments as well as to generate new items that more clearly distinguish thoughts from behaviours.

The CFA model was driven by the theoretical assumption that eating disorders can be reduced to two underlying latent constructs representing eating disorder thoughts and disordered actions. This is not to suggest that the individual items used in the CFA models are the best items or the only items that reflect eating disorder thoughts and behaviours. On the contrary, a number of important elements are missing from the items used in the models including more specific DSM criteria such as severe caloric restriction instead of 'dieting' and the undue influence of weight and shape on self-esteem instead of 'body dissatisfaction'. Future studies will likely demonstrate that the composition and number of first-order factors will vary depending on the measures used to tap eating disorder symptoms and the populations being studied. However, if eating disorder items are separated into thoughts and behaviours it is expected that the two higher order factors will remain consistent in future CFA models, at least in non-clinical and mixed samples. According to the tenets of this theoretical model, eating disorder thoughts will not be distinct from eating disorder behaviours in clinical groups because both thoughts and

behaviours are required for a clinical diagnosis and this would result in a near perfect relation between the two constructs.

The present results require replication using more diverse community samples, as this study sample was comprised of female university students, most of whom were Caucasian (56%). In addition, despite evidence indicating university and college students are at increased risk of eating disorders (Schwitzer, Bergholz, Dore, & Salimi, 1998), large epidemiological samples are ideal for research on symptoms of eating disorders because of increased generalizability and because of the low prevalence of eating disorder behaviours making large and diverse samples a necessity.

Implications and Future Directions

Having found a stable measurement structure of eating disorder thoughts and behaviours, the next steps will be to replicate this research using different indicators for thoughts and behaviours, and to then test for the dimensional and categorical nature of the thought and behaviour latent constructs. These questions can be addressed through more advanced statistical modeling such as taxometric analyses. These studies will be a critical avenue of future research for those interested in testing the theoretical model proposed by Miller and Vaillancourt (2008).

Measurement of eating pathology in epidemiological research has historically relied on the notion that eating disorders exist on a continuum, and as such, cumulative risk has been indicative of greater perceived risk for an eating disorder. Yet if eating disorder thoughts are continuous and eating disorder behaviours are discrete then assessments of risk should be determined separately for thoughts versus behaviours. If

the purpose of measuring eating pathology is to detect cases of eating disorders, we recommend screening instruments focus on the presence or absence of behavioural symptomology and setting a high threshold on psychological symptoms. Determining the exact threshold of eating disorder thoughts will vary with the type of measurement items, but we would suggest setting a high threshold on thoughts given the level of normative discontent in non-clinical populations. Finally, if the purpose of measuring eating pathology is to identify at-risk populations who are in need of preventative treatment, then screening instruments may need to focus on those who score high on psychological symptoms of eating disorders, regardless of the presence of behavioural criteria.

The prevalence of body dissatisfaction and fear of weight gain among non-clinical populations has biased the specificity of psychological symptoms in identifying prodromal eating disorder populations. Indeed, the notion of a "normative discontent" concerning weight and shape among females has been the rationale upon which we have based this theoretical model. Given the results of this research study, we suggest that behavioural criteria be identified separately from psychological criteria when establishing risk for eating disorders. In order to accomplish this task, we must re-define our criteria for risk and construct new scales of eating disorder symptoms that examine behaviours separately from thoughts. This study is the first empirical paper to demonstrate a methodology with which to accomplish this task.

References

Ackard, D. M., Fulkerson, J. A., & Neumark-Sztainer, D. (2007). Prevalence and utility of DSM-IV eating disorder diagnostic criteria among youth. *International Journal of Eating Disorders, 40*, 409-417.

Akaike, H. (1987). Factor Analysis and AIC. Psychometrika, 52, 317-332.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th *ed.*). Washington, DC: Author.

Anderson, J. C., & Gerbing, D. W. (1988). Structural equation modeling in practice: A review and recommended two-step approach. *Psychological Bulletin, 103*, 411-423.

Arbuckle, J. L. (2007). Amos (Version 16.0) [Computer Program]. Chicago: SPSS.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II, Beck Depression Inventory: Manual*. San Antonio (TX): Psychological Corporation, 1996.

Bollen, K. A. (1989). *Structural equations with latent variables*. John Wiley & Sons, Oxford: England.

Chamay-Weber, C., Narring, F., & Michaud, P. (2005). Partial eating disorders among adolescents: A review. *Journal of Adolescent Health, 37*, 417-427.

Espelage, D. L., Mazzeo, S. E., Aggen, S. H., Quittner, A. L., Sherman, R., & Thompson, R. (2003). Examining the construct validity of the eating disorder inventory. *Psychological Assessment, 15*, 71-80.

Fleiss, J. (1971). Measuring nominal scale agreement among many raters. *Psychological Bulletin, 76*, 378.

Garner, D.M. (2002). Measurement of eating disorder psychopathology. In C. G. Fairburn and K. D. Brownell (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook, 2nd ed.* (pp. 141-146). New York, Guilford Press.

Garner, D.M. (1991). *The Eating Disorder Inventory-2: Professional Manual.* Florida: Psychological Assessment Resources Inc.

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: Psychometric features and clinical correlates. *Psychological medicine, 12*, 871-878.

Garner, D. M., Olmsted, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15-34.

Garson, D. (2008). *Structural Equation Modeling*, Retrieved May 26, 2008 from http://www2.chass.ncsu.edu/garson/pa765/structur.htm

Gleaves, D. H., & Eberenz, K. P. (1995). Validating a multidimensional model of the psychopathology of bulimia nervosa. *Journal of Clinical Psychology, 51*, 181-189.

Gleaves, D. H., & Eberenz, K. (1993). The psychopathology of anorexia nervosa: A factor analytic investigation. *Journal of Psychopathology and Behavioral Assessment, 15*, 141-152.

Gleaves, D. H., Lowe, M. R., Green, B. A., Cororve, M. B., & Williams, T. L. (2000). Do anorexia and bulimia nervosa occur on a continuum? A taxometric analysis. *Behavior Therapy, 31*, 195-219.

Gleaves, D. H., Lowe, M. R., Snow, A. C., Green, B. A., & Murphy-Eberenz, K. P. (2000). Continuity and discontinuity models of bulimia nervosa: A taxometric investigation. *Journal of Abnormal Psychology, 109*, 56-68.

Gleaves, D. H., Williamson, D. A., & Barker, S. E. (1993). Confirmatory factor analysis of a multidimensional model of bulimia nervosa. *Journal of Abnormal Psychology, 102*, 173-176.

Godart, N. T., Flament, M. F., Curt, F., Perdereau, F., Lang, F., & Venisse, J. L., et al. (2003). Anxiety disorders in subjects seeking treatment for eating disorders: A DSM-IV controlled study. *Psychiatry Research, 117*, 245-258.

Godart, N. T., Perdereau, F., Jeammet, P., & Flament, M. F. (2003). Comorbidité et chronologie d' apparition des troubles anxieux dans les troubles du comportement alimentaire. / comorbidity and time of occurrence of anxiety disorders in eating disorders. *Annales Médico-Psychologiques, 161*, 498-503.

Hoek, H. W. (2006). Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry, 19*, 389-394.

Hu, L., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods, 3*, 424-453.

Jacobi, C., Abascal, L., & Taylor, C. B. (2004). Screening for eating disorders and high-risk behaviour: Caution. *International Journal of Eating Disorders, 36*, 280-295.

John, U., Meyer, C., Rumpf, H., & Hapke, U. (2006). Psychiatric comorbidity including nicotine dependence among individuals with eating disorder criteria in an adult general population sample. *Psychiatry Research, 141*, 71-79.

Joreskog, K. G., & Sorbom, D. (1996). *Structural Equation Modeling.* Workshop presented for the NORC Social Science Research Professional Development Training Sessions, Chicago.

Keski-Rahkonen, A., Sihvola, E., Raevuori, A., Kaukoranta, J., Bulik, C. M., Hoek, H. W., et al. (2006). Reliability of self-reported eating disorders: Optimizing population screening. *International Journal of Eating Disorders, 39*, 754-762.

Kline, R. B. (2005). *Principles and practice of structural equation modeling (2nd ed.).* Guilford Press, New York, NY: US.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics, 33,* 150-174.

Lowe, M. R., Gleaves, D. H., DiSimone-Weiss, R. T., Furgueson, C., Gayda, C. A., & Kolsky, P. A., et al. (1996). Restraint, dieting, and the continuum model of bulimia nervosa. *Journal of Abnormal Psychology, 105*, 508-517.

Miller, J. L., & Vaillancourt, T. (2008). The eating disorder continuum revisited: "Eating disorder thoughts" versus "eating disorder behaviours". Manuscript submitted for publication.

Mintz, L. B., & Betz, N. E. (1988). Prevalence and correlates of eating disordered behaviours among undergraduate women. *Journal of Counseling Psychology, 35*, 463-471.

Offord, D. R., Kraemer, H. C., Kazdin, A. E., Jensen, P. S., & Harrington, R. (1998). Lowering the burden of suffering from child psychiatric disorder: Trade-offs among clinical, targeted, and universal interventions. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 686-694.

Piran, N., & Gadalla, T. (2006). Eating disorders and substance abuse in Canadian women: A national study. *Addiction, 102*, 105-113.

Polivy, J., & Herman, C. P. (1987). Diagnosis and treatment of normal eating. *Journal of Consulting and Clinical Psychology, Special Issue: Eating Disorders, 55*, 635-644.

Price Foundation Collaborative Group, Geneva, Switzerland. (2001). Deriving behavioural phenotypes in an international, multi-centre study of eating disorders. *Psychological medicine, 31*, 635-645.

Rodin, J., Silberstein, L., & Striegel-Moore, R. (1984). Women and weight: A normative discontent. *Nebraska Symposium on Motivation, 32*, 267-307.

Schwitzer, A. M., Bergholz, K., Dore, T., & Salimi, L. (1998). Eating disorders among college women: Prevention, education, and treatment responses. *Journal of American College Health, 46*, 199.

Steiger, J. H., Shapiro, A., & Browne, M. W. (1985). On the multivariate asymptotic distribution of sequential Chi-square statistics. *Psychometrika, 50*, 253-264.

Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry, 159*, 1284-1293.

Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders, 22*, 339-360.

Tobin, D. L., Johnson, C., Steinberg, S., Staats, M., & Dennis, A. B. (1991). Multifactorial assessment of bulimia nervosa. *Journal of abnormal psychology, 100*, 14-21.

Tylka, T. L., & Subich, L. M. (2003). Revisiting the latent structure of eating disorders: Taxometric analyses with nonbehavioural indicators. *Journal of Counseling Psychology, 50*, 276-286.

Tylka, T. L., & Subich, L. M. (2002). A preliminary investigation of the eating disorder continuum with men. *Journal of Counseling Psychology, 49*, 273-279.

Tylka, T. L., & Subich, L. M. (1999). Exploring the construct validity of the eating disorder continuum. *Journal of Counseling Psychology, 46*, 268-276.

Vanderheyden, D. A., Fekken, G. C., & Boland, F. J. (1988). Critical variables associated with bingeing and bulimia in a university population: A factor analytic study. *International Journal of Eating Disorders, 7*, 321-329.

Varnado, P.J., Williamson, D.A., & Netemeyer, R. (1995). Confirmatory factor analysis of eating disorder symptoms in college women. *Journal of Psychopathology and Behavioral Assessment, 17*, 69-79.

Williamson, D. A., Gleaves, D. H., & Stewart, T. M. (2005). Categorical versus dimensional models of eating disorders: An examination of the evidence. *International Journal of Eating Disorders, 37*, 1-10.

Williamson, D. A., Womble, L. G., Smeets, M. A. M., Netemeyer, R. G., Thaw, J. M., & Kutlesic, V., et al. (2002). Latent structure of eating disorder symptoms: A factor analytic and taxometric investigation. *American Journal of Psychiatry, 159*, 412-418.

Zaider, T. I., Johnson, J. G., & Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *International Journal of Eating Disorders, 28*, 58-67.

Table I: Means, Standard Deviations and Reliabilities for the Original EDI Subscales with Normed Comparison Groups.

Measure	Current Sample (*N*=1816)	Comparison Sample Non-ED (*N*=205)	Comparison Sample ED (*N*=889)
EDI – Bulimia	2.16(3.10) α = .75	1.2(1.90) --	10.5(5.50) α = .86
EDI – DFT	4.30(5.04) α = .87	5.5(5.50) --	14.5(5.60) α = .83
EDI – BD	9.13(6.96) α = .88	12.2(8.30) --	16.6(8.30) α = .92
EAT-26	9.02(9.85) α = .90	--	--

Note: Means are presented first with standard deviations presented in parentheses. Reliability coefficients for the non-ED group are not available. The Non-ED group is a non-patient normed comparison sample of females (M=18-25 years) in their first and second years of college (Garner, 1991). The ED sample data (*M*=24 years) represents patient data from two eating disorder programs used in the initial validation of the Eating Disorder Inventory (see Garner 1991). DFT= Drive for thinness; BD=Body Dissatisfaction; EAT-26=Eating Attitudes Test-26.

Table II: Eating Disorder Inventory (EDI-2) and Eating Attitudes Test (EAT-26) Item Descriptions, Factor Loadings, and Frequencies (Percentages).

Item Descriptions	Latent Factor	Factor Loadings	Item Frequencies
EAT 45: I avoid foods with sugar in them.	R	.55(.56)	7.9%
EAT 31: I avoid eating when I am hungry	R	.54(.55)	6.3%
EAT 46: I eat diet foods.	R	.59(.61)	14.8%
EAT 52: I engage in dieting behaviour.	R	.74(.76)	19.9%
EAT 36: I particularly avoid food with high carb content	R	.67(.68)	14.3%
EDI 4: I stuff myself with food	B	.60(.60)	26.7%
EDI 13: gone on eating binges where felt I could not stop	B	.68(.72)	10.2%
EDI 21: eat moderately in front of others, stuff myself when they're gone.	B	.61(.59)	11.0%
EAT 38: I vomit after I have eaten.	P	.70(.70)	3.4%
EAT 54: I have the impulse to vomit after meals.	P	.88(.90)	4.7%
EDI 10: I am terrified of gaining weight.	F	.70(.76)	45.5%
EDI 16: I am preoccupied with the desire to be thinner.	F	.80(.81)	31.6%
EDI 22: If I gain a pound, I worry I will keep gaining.	F	.74(.75)	23.7%
EAT 43: preoccupied with thought of having fat on my body.	F	.75(.77)	21.6%
EAT 30: I am terrified about being overweight	F	.68(.71)	43.5%
EDI 2: I think that my stomach is too big.	BD	.61(.62)	53.8%
EDI 6: I think that my thighs are too large.	BD	.69(.70)	50.9%
EDI 11: I feel dissatisfied with the shape of my body	BD	.67(.69)	49.2%
EDI 20: I think my hips are too big	BD	.76(.77)	31.1%
EDI 26: I think my buttocks are too large.	BD	.67(.68)	20.9%

Note: All factor loadings are presented as standardized coefficients and are statistically significant, $p < .001$. First-order factor loadings are presented first with the second-order factor loadings presented second in parentheses. Item frequencies were derived by collapsing across "often", "usually" or "always" response categories. R = Restrict; B = Binge; P = Purge; F = Fear of fat; BD = Body Dissatisfaction.

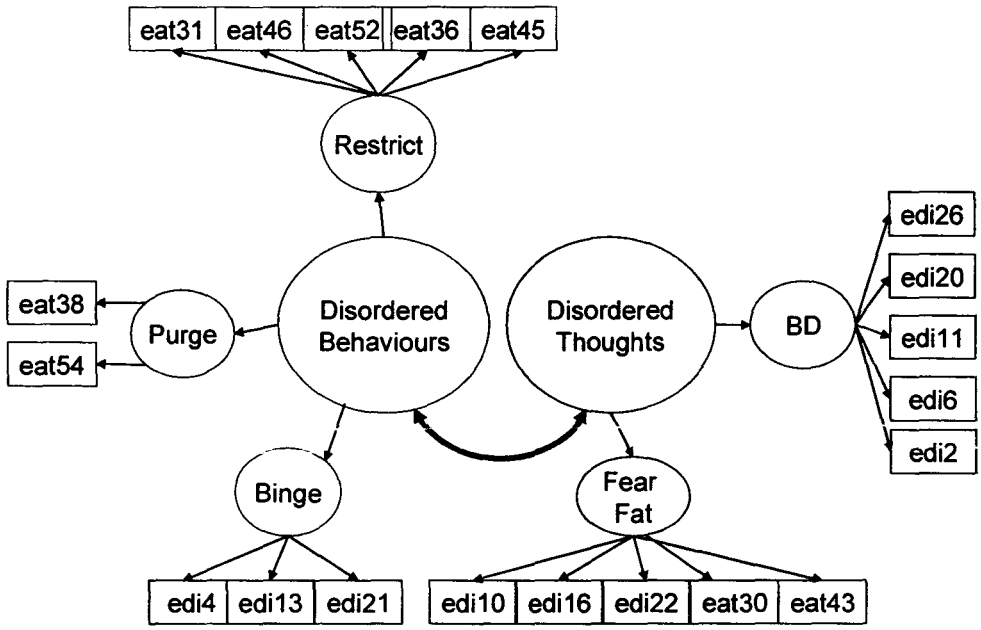
Table III: Latent Factor Correlations, Internal Consistency Coefficients, and Second-Order Factor Loadings ($N = 1816$)

	1	2	3	4	5
1. Restrict	*				
2. Binge	.51	*			
3. Purge	.58	.42	*		
4. Fear of Fat	.76	.57	.43	*	
5. Body Dissatisfaction	.53	.48	.33	.75	*
Means	1.07	.87	.14	3.36	4.33
Standard Deviations	2.06	1.60	.66	4.18	4.11
Minimum/Maximums	0-15	0-9	0-6	0-15	0-15
Cronbach's Alpha	.70	.68	.73	.89	.86
Factor Loadings	.92	.64	.60	.98	.73

Table IV: Cross-tabulations and Standardized Residuals for Thought/Behaviour Groupings.

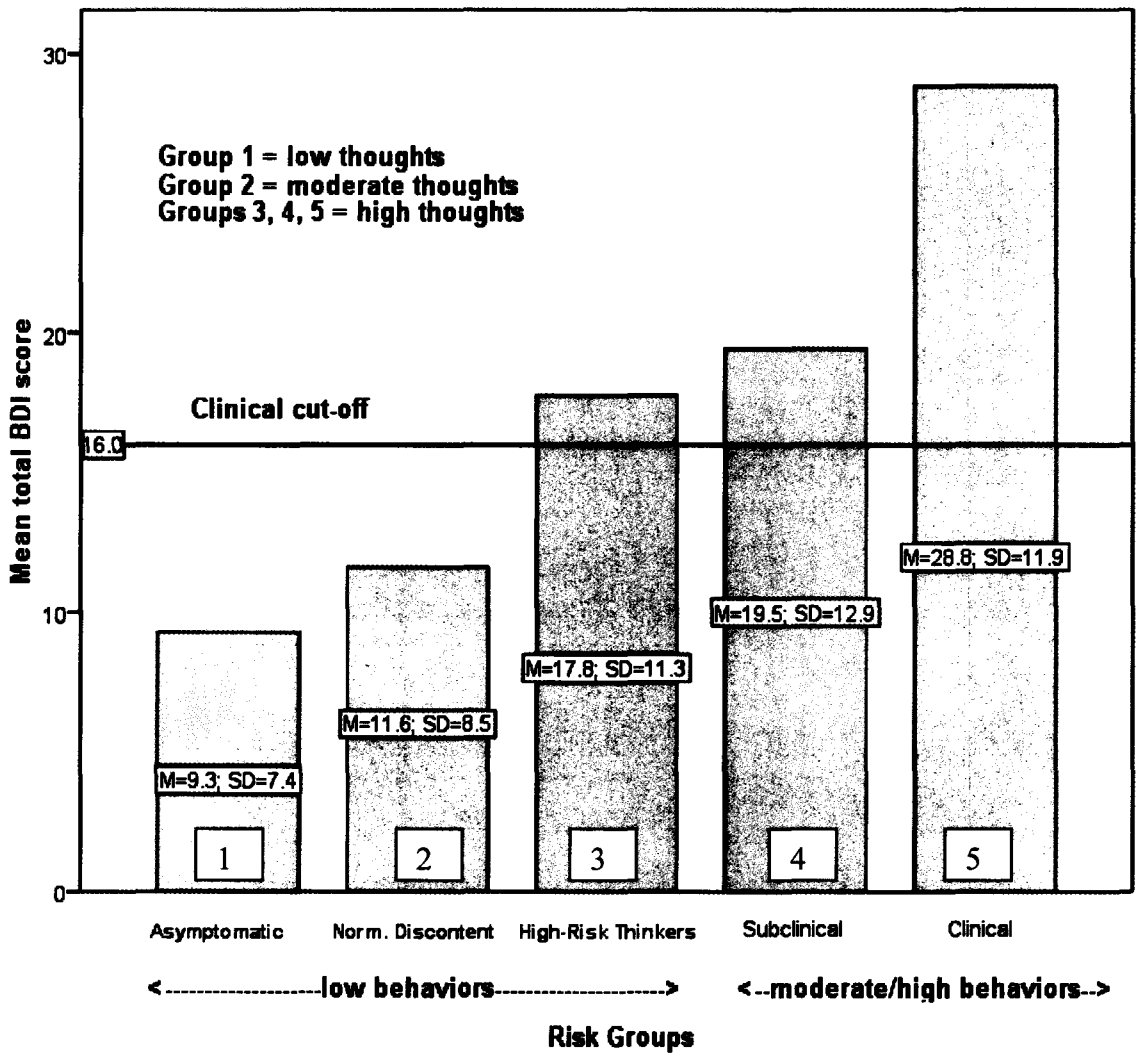
		Thoughts			
		Low	Moderate	High	Total
Behaviours	Low				
	Count	245	756	61	1062
	% Total	13.5%	41.6%	3.4%	
	Std. Residual	9.0	2.4	11.3	
	Moderate				
	Count	9	418	287	714
	% Total	0.50%	23.0%	15.8%	
	Std. Residual	-10.4	-2.1	12.1	
	High				
	Count	0	4	36	40
	% Total	0%	0.22%	2.0%	
	Std. Residual	-2.7	-3.9	8.0	
	Total Count	254	1178	384	1816

Figure 1. Second-Order Confirmatory Factor Model of Disordered Eating Symptoms.



Numbers correspond to the items in Table II. BD = Body Dissatisfaction

Figure 2. Mean Depression Scores by Risk Group



CHAPTER 6

Miller, J.L. & Vaillancourt, T. (unsubmitted manuscript). Psychiatric Comorbidity of Eating Disorder Thoughts and Eating Disorder Behaviours in a National Canadian Sample

The analyses presented in this chapter are based on Statistics Canada's Canadian Community Health Survey, Cycle 1.2, Mental Health and Well-being (2002), Public Use Microdata file, which contains anonymized data collected. All computation on these microdata were prepared by Jessie Miller of McMaster University and the responsibility for the use and interpretation of these data is entirely that of the authors.

Introduction

Both eating disorders (anorexia, bulimia and binge eating disorder) and disordered eating (unhealthy weight control strategies, body dissatisfaction) have been linked concurrently and prospectively to comorbid psychiatric illnesses (Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Lecrubier, & Jeammet, 2000; Johnson, Cohen, Kotler, Kasen, & Brook, 2002; Kaye et al., 2004; Pearlstein, 2002; Stice & Bearman, 2001; Zaider, Johnson, & Cockell, 2000). In clinical samples, the most commonly reported comorbid Axis I disorders include major depression, dsythymia, obsessive-compulsive disorder, social phobia, generalized anxiety disorder and substance use disorders (Godart et al., 2003; Pearlstein, 2002).

Among non-clinical populations, disordered eating is most often correlated to mood and anxiety disorders (Becker, DeViva, Zayfert, 2004; Wildes, Simons, & Marcus, 2005). However, disordered eating is an umbrella term used to describe a cluster of behaviours and attitudes related to weight concerns, and thus, which aspects of disordered eating are related to these comorbid conditions is rarely specified. Zaider et al., (2000) reported a correlation between eating disorder symptoms and mood and anxiety disorders in an adolescent community sample, yet they never defined the term 'eating disorder symptoms' in their paper. Troop, Serpell, and Treasure (2001) examined the degree to which specific eating disorder symptoms would be related to specific symptoms of depression. The authors used the Beck Depression Inventory to assess symptoms of depression and items from the Short Evaluation for Eating Disorders (SEED, Kordy & Treasure, 1997) to assess eating disorder symptoms. Using exploratory factor analysis,

Troop et al. extracted three factors from the SEED: 1) dietary restriction which included items such as fear of weight gain (thoughts), use of diet foods (behaviours), and frequency of exercise (behaviours); 2) bulimia which consisted of binge eating and vomiting (behaviours); and 3) body mass index. The dietary restraint and bulimia factors significantly predicted cognitive symptoms of depression (pessimism, low self-esteem), however, the dietary restraint factor consisted of items pertaining to both psychological (fear of fat) and behavioural symptoms of an eating disorder (dietary restraint), making it difficult to conclude whether the relation existed between depression and dietary restraint, depression and fear of being fat, or a combination of the two symptoms together.

Many studies that have examined psychopathological correlates of disordered eating use the Eating Attitudes Test (EAT; Garner, Olmsted, Bohr, & Garfinkel, 1982) or the Eating Disorder Inventory (EDI; Garner, Olmsted, & Polivy, 1983) to measure the level of behavioural and attitudinal disturbance (e.g., Bas, Hulya, Karabudak, & Kiziltan, 2004; Fisher, Schneider, Pegler, & Napolitano, 1991; Nelson, Hughes, Katz, & Searight, 1999; Silberstein, Striegel-Moore, Timko, & Rodin, 1988; Wichstrom, 1995). Although both of these instruments have multiple subscales where eating disorder behaviours and dysfunctional eating thoughts are combined together to produce a total score of risk on some factor pertaining to eating pathology, the behavioural, cognitive and affective components of an eating disorder are not normally distributed in non-clinical populations (Miller & Vaillancourt, 2008). Thus, measuring them as if they are equivalent in their occurrence and in their relation to psychopathology may be premature. If the research

question can be satisfied with a more general association between disordered eating and psychopathology then the current use of screening instruments such as the EAT and the EDI may be sufficient in providing an index of cumulative risk. Alternatively, if the purpose of the research is to determine which aspects of disordered eating are related to psychopathology in order to prevent or detect future eating disorder syndromes then the measures used must be more specific to the variables under examination.

In recent years there have been a number of empirical studies that have examined disordered eating and comorbid psychopathology by separating the symptomology into more specific features; the cognitive and behavioural symptoms (Darby, Hay, Mond, Rodgers, & Owen, 2007; Forbush, Heatherton, Keel; Fulkerson, Sherwood, Perry, Neumark-Sztainer, & Story, 2004; Miller, Vaillancourt, & Hanna, 2008; Neumark-Sztainer, Story, Hannah, Perry & Irving, 2002; Tomori & Rus-Makovec, 2000; Wildes et al., 2005). In these studies, the psychological components of eating disorders, such as weight concerns (body dissatisfaction, weight disparagement), were assessed separately from the behavioural features (dieting with weight loss, use of diet pills, or laxatives, bingeing, and vomiting) in determining associated psychopathological characteristics.

For most of these studies, the specific eating behaviours and weight concerns were reported as frequencies in relation to some form of psychopathology such as depression, low self-esteem, perfectionism, substance use, or physical activity. However, one of these studies compared and examined the interaction of psychological and behavioural symptoms in order to determine the relative contribution of each to risk of comorbid psychopathology. Specifically, Miller et al. (2008) used confirmatory factor analysis to

demonstrate independent latent factors for 'eating disorder thoughts' and 'eating disorder behaviours' and then derived five risk groups based on the level of thought and behaviour symptoms. The rationale for measuring thoughts separate from behaviours when assessing eating disorders was based on a theoretical model put forth by Miller and Vaillancourt (2008), that argues that eating disorders have both continuous and discontinuous features; the psychological features are continuous (e.g., fear of weight gain) and are shared between non-clinical and sub-clinical/clinical populations, but the behavioural features (vomiting, binge eating, fasting and severe caloric restriction) are not continuous and are specific to sub-clinical/clinical populations. In order to test a theoretical model of discrete behaviours and continuous thoughts it was necessary to first establish distinct latent constructs for eating disorder thoughts and behaviours (Miller et al., 2008). In addition, Miller and Vaillancourt (2008) have hypothesized that if behaviours are specific to clinical and sub-clinical populations, the behavioural symptoms should be more closely associated with other forms of psychopathology commonly found in eating disorder populations, namely, mood and anxiety disorders.

Using the factor scores derived from their CFA, Miller et al. (2008) formed risk groups based on high, moderate or minimal levels of eating disorder thoughts (fear of fat, body dissatisfaction) and behaviours (bingeing, vomiting, and restricting). Their results showed that varying the level of eating disorder thoughts and eating disorder behaviours differentially predicted risk for depression using scores on the Beck Depression Inventory (BDI). Although 42% of their sample endorsed significant levels of eating disorder thoughts (fear of becoming fat) without eating disorder behaviours, only 3% of this group

scored above the clinical cut-off on the BDI. In contrast, 41% of their sample engaged in a combination of moderate-to-high levels of both eating disorder behaviours and thoughts and all 41% met clinical criteria on the BDI. Their results showed that eating disorder thoughts can occur without accompanying eating disorder behaviours and that these thoughts were not always associated with depression. In contrast, behaviours were always associated with thoughts and always associated with depression.

Miller et al's (2008) study was the first to demonstrate a risk of psychopathology (i.e., depression) based on the interaction of varying levels of eating disorder thoughts and behaviours. The strengths of their study include theory-driven a-priori research hypotheses, use of multiple statistical analyses to address the research hypotheses and the relatively large sample size (N=1816). Despite these strengths, Miller et al. were limited in the conclusions that could be drawn from their results because it was the first study of its kind and because the sample was not random but rather was a convenience sample comprised of female university students in their late teens and early 20's who were predominantly Caucasian. In addition, risk for depression was based on a self-report questionnaire with a clinical cut-off suggestive of a depressive disorder; however the BDI cannot be used to assess clinical depression.

In the current study, we were interested in testing whether we could replicate the results reported by Miller et al. (2008) by first examining a confirmatory factor structure of eating disorder symptoms based on a separation of thoughts and behaviours and second establishing risk groups based on varying levels of thoughts and behaviours to determine risk for psychopathology. In addition to replicating their results, we sought to

extend the findings by Miller et al. by using data from a national epidemiological survey of mental health rather than a university-based sample and by using as our measure of psychopathology, three *clinically* diagnosed psychiatric disorders rather than a single continuous score on a self-report measure of depression.

Method

Data Set

This research was based on secondary data analyses on sample data drawn from the Canadian Community Health Survey 1.2 Mental Health and Well-Being Supplement conducted in 2002 (CCHS, Cycle 1.2; Statistics Canada, 2002). The CCHS 1.2 represents the first national estimate of the prevalence of major psychiatric conditions in Canada (Streiner, Cairney, & Lesage, 2005). The survey used a cross-sectional multistage stratified cluster design based on the Canadian Labour Force Survey in which the dwelling is the final sampling unit. The survey targeted persons aged 15 years and older living in private dwellings in the ten provinces during the period of May 2002 through to December 2002. The three territories were excluded as well as those residing on Indian Reserves, Crown lands, residents of institutions, full-time members of the Canadian Armed Forces and residents of some remote areas. In total, the survey represents approximately 98% of the population aged 15 and older across the ten provinces. The breakdown of participants by province has been detailed elsewhere (see Gravel & Béland, 2005).

Sample Size and Allocation

One person aged 15 years or older was randomly selected from the sampled households with an over selection procedure in place for those aged 15 to 24 and aged 65 or older (for details see Gravel & Béland, 2005). Approximately 48 000 households were selected from the area frame yielding a target sample of 38 492. Taking into account vacant dwellings and non-response, the total sample was 36 984. The survey was conducted primarily in English and French (the official languages of Canada) however to reduce the impact of language as a barrier to participation, Statistics Canada recruited interviewers with varied language skills. An official translation of key terms was provided to these interviewers in the languages of Chinese and Punjabi, the two most prevalent non-official languages (Gravel & Béland, 2005).

Study Participants

Of the total respondents (36 984), 20 211 were female and 16 773 were male. Two screener questions were used to determine whether respondents would complete the 26 item Eating Attitudes Test (EAT-26). Respondents who answered yes to the first screener "Was there ever a time in your life when you had a strong fear or a great deal of concern about being too fat or overweight?"(lifetime criterion), were asked the second screener question, "During the past 12 months did you have a strong fear or a great deal of concern about being too fat or overweight?" A total of 9182 respondents endorsed the lifetime criterion and 5116 endorsed the past 12 months criterion. The EAT-26 was completed only by participants endorsing the past 12 months criterion and not the lifetime criterion.

Of the 5116 respondents who answered the EAT-26, 3792 respondents were female. In the present study, only female respondents were included in subsequent analyses. We excluded 32 females who were currently pregnant and restricted our sample to adolescence and early adulthood, thus the remaining sample (N=1627) by age group was: 15 to 19 years, n=403; 20 to 24 years, n=395; 25 to 29 years, n=374; and 30 to 34 years, n=455.

Sample Characteristics: Ethnicity was not recorded in the CCHS, only country of birth was recorded and it was only a two-item response category: "Canada" (89.2%) or "Other" (10.6%). The Body Mass Index of participants[3] (based on self-reported height and weight) by age groups is shown in Table 1. In this sub-sample 58 respondents gave their height but refused to give their weight to the interviewer and thus BMI could not be computed on these participants. As well, there were two respondents who replied "don't know" when asked about their weight. Accordingly, BMI data was available for 1567 respondents (60 missing). Participants were also asked to give an opinion on their weight status, "Do you consider yourself: overweight, underweight, or just about right." The responses by age group are also presented in Table 1.

Regarding psychopathology, 23.8% of the study sample met lifetime criteria for major depression episode, and 14.4% received a current diagnosis of major depression.

[3] Although the BMI number is calculated the same way for adolescents and adults, the criteria used to interpret the meaning of the BMI number for adolescents is different from that which is used with adults. For adolescents, BMI is an age and sex specific percentile that takes into account body fat changes with age and different amounts of body fat between males and females. For adults, BMI is interpreted through categories that do not take into account sex or age (Centers for Disease Control and Prevention, 2008). Therefore the BMI categories for the 15 to 19 year old age group should be interpreted with caution.

For Social phobia, 20.1% met lifetime criteria, and 10.5% had a current diagnosis of social phobia. For Substance dependence (any type), 7.3% of respondents had a current diagnosis. On the EAT-26, 15.4% of participants scored above the recommended cut-off score of 20, indicating significant risk of an eating disorder (see Garner et al., 1982). Of this 15.4%, 3.4% were in the 15 to 19 year old group, 4.0% were in the 20 to 24 year old group, 3.4% were in the 25 to 29 year old group, and 4.5% were in the 30 to 34 year old group. The means and standard deviations on the Eating Attitudes Test by age group are presented in Table 2. There were no statistically significant differences between any of the age groups on their EAT score, using independent sample t-tests.

Procedures

Participants were interviewed over the telephone using a Computer-Assisted Personal Interviewing method (CAPI: Statistics Canada, 2002). One advantage of this interview technique over other collection methods is that it allows immediate feedback to the respondent and the interviewer is able to correct inconsistencies immediately (Statistics Canada, 2002). For instance, based on responses recorded for question 1a, any secondary responses that are not consistent with the previous one will show an invalid entry on the computer screen to the interviewer. In addition, questions that are not applicable to the respondent are skipped automatically (Statistics Canada, 2002).

Interview questions for the disorders in the mental health supplement were based on the World Mental Health 2000 Project (WMH); an international initiative responsible for the development of the Composite International Diagnostic Interview (WMH-CIDI; Gravel & Béland, 2005). The CIDI is a structured interview capable of generating

lifetime prevalence and past 12-month diagnoses of mental disorders as defined by both the ICD-10 and the DSM-IV (Gravel & Béland, 2005). The CIDI has elsewhere been shown to have both internal and external reliability and validity across various populations (Wittchen, 1994). For the purpose of Cycle 1.2, mental disorders were coded partially to the DSM-IV only and some modifications to the content of the disorder modules were made to shorten the length of the survey and clarify confusing concepts (Gravel & Béland, 2005).

Problems of interview length, response burden and quality of the data were serious considerations in the design of Cycle 1.2. To address these concerns, an advisory committee from Statistics Canada Population Health Surveys in consultation with a Mental Health Expert Group selected a "priority" area of mental disorders. The impact of this selection was a reduction in the total disorders followed through for diagnostic assessment. The target disorders were major depression, mania, panic disorder, social phobia, and agoraphobia. However, mental health problems and correlates were also included in the design including suicidal ideation, gambling, substance dependence and eating problems and these questionnaires were based on instruments used previously by Statistics Canada surveys such as the National Population Health Survey (NPHS), CCHS Cycle 1.1, and the Health Population Survey (HPS).

Interviewers first collected data on basic economic, social, demographic, occupational and environmental factors associated with health (CCHS, user guide, see Statistics Canada, 2002). Next, survey participants were screened for evidence of the five target psychiatric disorders. If respondents met criteria for a disorder based on the

screening, the interviewer then followed with the diagnostic assessment (CIDI). Following the major disorders, participants were administered questions pertaining to mental health problems (e.g., disordered eating). To facilitate data analysis, over 200 derived variables were created. In some circumstances derived variables were computed based on collapsing response categories (e.g., The Eating Attitudes Index Score) and in other instances several variables were combined to create a new variable (lifetime algorithm for Major Depressive Disorder). For the five target psychiatric disorders the derived variables were based on DSM-IV criteria although substance dependence was coded to reflect DSM-III-R criteria. The derived variables were reviewed and tested by the CCHS analysts along with several mental health experts including the Ontario Mental Health Survey team and the WMH 2000 Project team. Feedback from the authors of the instruments was also used (Gravel & Béland, 2005). The derived variables used as outcome measures in the current analyses include lifetime algorithms and past 12 months algorithms for all five target psychiatric disorders, as well as the 12 months algorithm for substance dependence disorder. For complete details on the criteria used to create the algorithms for the target psychiatric disorders please see the CCHS Cycle 1.2 Derived Variables Document (Statistics Canada, 2002).

Measures

Eating Problems: Disordered eating, a risk factor for clinical eating disorders, was assessed through the Eating Attitudes Test Revised (EAT-26). The EAT-26 was developed by Garner & Garfinkel (1979) and is the most widely used tool for screening features, symptoms, and concerns characteristic of eating disorders. Although the EAT-

26 does not yield a specific diagnosis, this standardized scale provides an assessment of risk that is highly correlated with a clinical diagnosis of an eating disorder (Garner et al., 1983). The CCHS 1.2 added two screener questions to assess past 12 months and lifetime concerns with eating attitudes and behaviours. As mentioned above, those who responded positively to the screener were then administered all of the items of the EAT-26. Respondents were asked to rate 26 statements based on the following metric: Always, Usually, Often, Sometimes, Rarely, or Never. Responses were then weighted and assigned values as follows: Always =3; Usually = 2; Often = 1; Sometimes, Rarely, Never = 0 (see Garner et al., 1982). A clinical cut-off of 20 or more has been established as an indicator of significant clinical risk of an eating disorder. Sample items included, "I vomit after I have eaten," "I engage in dieting behaviours," "I like my stomach to be empty," or "I am preoccupied with a desire to be thinner."

Major Depression: The criteria for a diagnosis of major depressive disorder included: 1) a period of two weeks or more with persistent depressed mood and loss of interest or pleasure in normal activities, 2) accompanied by five of the following symptoms such as decreased energy, changes in sleep and appetite, impaired concentration, and feelings of guilt, hopelessness, or suicidal thoughts. A sample item from the interview includes: "At any time in the past 12 months, did you have an episode lasting 2 weeks or longer when you felt and also had episodes of being sad, empty or depressed."

Social Phobia: The criteria for a diagnosis of social phobia was computed based on persistent, irrational fear of situations in which the person may be closely watched and

judged by others, as in public speaking, eating, or using public facilities. Symptoms include avoidance or anxiety related to the situations and physical symptoms such as rapid heart rate, nausea, dry mouth, sweating, chest pain, faintness, dizziness, and panic attacks. A sample item from the interview includes: "In the past 12 months how often did you avoid situations like - entering a room when others are already present?"

Substance Dependence: A diagnosis of substance (any alcohol or illicit drugs) dependence disorder was compiled based on having at least one dependency for *any* alcohol or illicit drug in the past 12 months. Sample items included, "During the past 12 months, have you had any emotional or psychological problems because of alcohol use, such as feeling uninterested in things, depressed or suspicious of people? During the past 12 months, have you had such a strong desire or urge to drink alcohol that you could not resist it or could not think of anything else? During the past 12 months, did you ever find that you had to drink more alcohol than usual to get the same effect or that the same amount of alcohol had less effect on you than usual? (alcohol dependence). During the past 12 months, did you ever have periods of several days or more when you spent so much time using drugs or recovering from the effects of using drugs that you had little time for anything else? During the past 12 months, did you ever continue to use drugs when you knew you had a serious physical or emotional problem that might have been caused by or made worse by your use? (illicit drug use).

Statistical Analyses:

Confirmatory Factor Analysis of thoughts and behaviours: Using confirmatory factor analysis we modeled three latent variables and one observed variable based on

items from the EAT-26. Selection of items for the latent factors was based on the model by Miller et al. (2008), where eating disorder thoughts and eating disorder behaviours were modeled in a second-order confirmatory factor analysis of eating disorder symptoms with five first-order latent factors; *Fear of fat/fear of weight gain, body dissatisfaction, restricting/dieting, compensatory strategies, and bingeing.* Miller et al. used a combination of items from the Eating Disorder Inventory (EDI-2; Garner et al., 1983) and the Eating Attitudes Test (EAT-26; Garner et al., 1982) and divided items based on whether they were Thoughts/Behaviours/Ambiguous and then placed them in one of six possible categories that closely resembled DSM-IV criteria for an eating disorder. The authors subsequently verified their classifications by enlisting 15 mental health professionals, each of whom independently classified items as Thoughts/Behaviours/Ambiguous, and then placed them in one of the six DSM categories defined by the authors. There was substantial agreement across the 15 mental health professionals based on inter-rater agreement (Fleiss' Kappa coefficient) of the theoretically driven factors. Thus, the items within each factor for this current study are items that have gone through a rigorous item selection process and have demonstrated excellent construct validity as reported by Miller et al. (2008).

In their original model, Miller et al. used a combination of items from the EDI-2 and the EAT-26, however only items from the EAT-26 were available in the CCHS sample and as a result, the entire second-order CFA model by Miller et al. could not be replicated. Instead, we replicated two of the five latent factors, *restrict* and *purge* in their original form, with a third latent factor *fear of fat*, comprised of 3 of the 5 original

indicators. Our fourth variable, *bingeing*, was modeled as observed and not latent, because the EAT-26 contains only 1 of the 3 indicators used in the CFA by Miller et al.

We examined three global fit indices to assess overall model fit: the Comparative Fit Index (CFI), the Tucker-Lewis Index (TLI), and the Root Mean Square Error of Approximation (RMSEA). Hu and Bentler (1998) suggest that RMSEA values of .05 and lower reflect close fitting models but values between .08 and .05 are adequate. RMSEA also provides a significance test of close fit (PCLOSE) which tests the hypothesis that RMSEA is no greater than .05. A non-significant value is interpreted as evidence of good fit (Joreskog & Sorbom, 1996). Values over .90 for both the CFI and TLI are suggested as adequate fit although Hu and Bentler (1998) state that .95 or greater is ideal (Bollen, 1989; Kline, 2005). Model invariance was tested by constraining the factor loadings to be equal and then using a sequential chi-square difference test to examine change in model fit. All CFA analyses were conducted using AMOS 16.0 using Maximum Likelihood Estimation (Analysis of Moment Structures; Arbuckle, 2007).

Testing for distinct sub-groups of thoughts and behaviours: Following procedures described in Miller et al. (2008) and using the factor scores derived from our CFA to create total scores for thoughts and total scores for behaviours, six risk profiles were computed and used to test for group differences in psychiatric comorbidity to depression, social phobia and substance dependence disorders using logistic regressions. The thought subscale was comprised of the three items from the *fear of fat* latent factor and the behaviour subscale was comprised of the five items from the *restrict* latent factor, the two items from the *purge* latent factor and the one *binge* item. Thoughts and

Behaviours were then trichotomized: high, moderate and low thoughts and high, moderate and low behaviours.

The cut-off points selected to convert each latent factor into a trichotomous variable were based on the weighted responses used to score the EAT-26 (Garner et al., 1982), developed by Garner (1991). When the EAT-26 is administered, respondents are asked to rate items based on the following metric: Always, Usually, Often, Sometimes, Rarely, or Never. Garner has suggested that the responses 'always, usually, and often' reflect symptomatic responses and that the responses 'sometimes, rarely, and never' reflect non-symptomatic responses. This weighting system is based on the scoring system developed for the Eating Disorder Inventory by Garner (1991).

The Thought subscale was trichotomized into *high-thoughts* for scores greater than or equal to 12; *moderate-thoughts* for scores between 7-11; and *low-thoughts* for scores less than 6. A score greater than or equal to 12 represents the minimum score possible for individuals who answered 'always', 'usually' and 'often' (i.e., Garner's symptomatic response options) to each of the three thought items. Following methods by Miller et al. (2008), we divided the asymptomatic response options (sometimes, rarely and never) into moderate thoughts (sometimes) and low thoughts (rarely and never). A score between 7 and 11 represents individuals who answered 'sometimes' to all or some of the three thought items, and a score below 6 represents individuals who endorsed the response options 'rarely' or 'never' for each of the three thought items.

Our Behaviour subscale was trichotomized in the same manner, this time however we had nine items, which meant our cut-off criteria for *high-behaviours* were scores

greater than or equal to 32, moderate-behaviours were scores between 17 and 31, and our criteria for *low-behaviours* were scores less than or equal to 16.

Results

Our CFA model consisted of three unobserved and one observed variables: *Fear of Fat* with three indicators; *Restrict* with five indicators; *Purge* with two indicators; and *Binge* with one indicator (observed variable). To examine potential age effects in eating disorder items, we conducted a multiple group comparison CFA with our groups representing the four age categories in our sample (15 to 19 yrs; 20 to 24 yrs; 25 to 29 yrs; 30 to 34 yrs).

For the baseline model, all latent factors were free to correlate and all parameters freely estimated. Model fit was good, $\chi^2(156) = 547.64$, CFI = .93, RMSEA = .037 (95% CI = .033 – .040; PCLOSE = 1.00), TLI = 0.89. Factor loadings ranged from a low of .50 ('I avoid eating when I am hungry') to a high of .89 ('I vomit after I have eaten') for the 15 to 19 year old group; a low of .52 ('I particularly avoid food with a high carbohydrate content') to a high of .99 ('I have the impulse to vomit after meals') for the 20 to 24 year old group; a low of .35 ('I avoid eating when I am hungry') to a high of .94 ('I have the impulse to vomit after meals') for the 25 to 29 year old group; and a low of .37 ('I avoid eating when I am hungry') to a high of .97 ('I have the impulse to vomit after meals') for the 30 to 34 year old group.

Next we constrained the factor loadings across the four groups to be equal to test for measurement invariance. Model fit was still good, $\chi^2(177) = 588.05$, CFI = .93, RMSEA = .03 (95% CI = .032 – .038; PCLOSE = 1.00), TLI = 0.90. We compared the

change in model fit using the chi square difference test and the results showed the models were not statistically significantly different in fit; $\Delta\chi^2$ of 40.4 with 21 degrees of freedom at $p < .005$, indicating support for model invariance across participants ages 15 to 34 years.

Distinct sub-groups

Having shown model invariance across age, we combined the four age groups for the cross-tabulations and logistic regression analyses. The cross-tabulations for the trichotomized 'thoughts' and 'behaviours' are displayed in Table 3. *Low-thoughts* had a mean score of 4.77 (SD = 1.09), *moderate thoughts* had a mean score of 8.99 (SD = 1.36), and *high-thoughts* had a mean score of 14.86 (SD = 2.19). *Low-behaviours* had a mean score of 12.39 (SD = 2.55), *moderate behaviours* had a mean score of 20.84 (SD = 3.29), and *high-behaviours* had a mean score of 33.71 (SD = 2.74). Two cells had observed frequencies of 0% (low thoughts/high behaviours; moderate thoughts/high behaviours) and were not included in any further analyses. Based on the remaining seven cells, six risk profiles were created: Group 1 (asymptomatic) = low thoughts/low behaviours (17.4%); Group 2 (normative discontent) = moderate thoughts/low behaviours (27.6%); Group 3 (Dieters)[4] = moderate behaviours/low thoughts (2.6%); Group 4 (high-risk thinkers) = high thoughts/low behaviours (13.7%); Group 5 (sub-clinical) = moderate or high thoughts/moderate behaviours (37.3%); Group 6 (clinical) = high thoughts/high behaviours (1.3%).

[4] Post-hoc analysis of the mean differences in the three behaviour variables – purge, binge and restrict – revealed that the only variable that distinguished this group from groups 1, 2 and 4, was the restrict latent variable. Of the five items in the restrict variable the highest observed means were for the items, "I engage in dieting behaviours" and "I eat diet foods" hence the label "Dieters" for this group.

Next, we used logistic regression to examine the strength of association between each thought/behaviour group in predicting concurrent diagnoses (present/absent) of Major Depressive Disorder, Social Phobia and Substance Dependence. The logistic regression analysis was carried out using SPSS for Windows version 16.0. The predictor variable in this model was "Group" (6 levels) and Group 1 (asymptomatic) was always the indicator variable.

Results of the logistic regressions indicated that all three models provided a statistically significant improvement over the constant-only model [Depression: χ^2 (5) = 48.32, $p < .001$; Social Phobia: χ^2 (5) = 31.56, $p < .001$; Substance Dependence: χ^2 (5) = 12.65, $p < .027$]. A statistically non-significant Homer and Lemeshow test across all models indicated good model fit (close match between predicted and observed probabilities). Table 4 presents the regression coefficients (β), the Wald statistics, significance level, odds ratio [Exp(β)], and the 95% confidence intervals (CI) for the odds ratios of each group.

Compared to our asymptomatic group (1), individuals in the normative discontent (2) and dieter (3) groups were no more likely to be diagnosed with major depression, social phobia or substance dependence disorder. In contrast, there was an incremental likelihood of having major depression, social phobia or substance dependence disorder across groups 3 (high risk thinkers), 4 (sub-clinical), and 5 (clinical) compared to the reference group - asymptomatic individuals (see Table 4).

Discussion

The purpose of this study was to replicate and extend the findings by Miller et al. (2008) who used confirmatory factor analysis (CFA) to model a theoretical latent structure of eating disorder symptoms, with thoughts and behaviours representing two second-order latent variables, and from these latent factors created extreme groups based on low, moderate and high levels of thoughts and behaviours, in order to predict psychopathology.

The results of the current study offer further support for the theoretical confirmatory factor model of thoughts and behaviours by Miller et al. (2008). We were able to replicate three of the five first order factors developed by Miller et al. (fear of fat, purging, restricting), but do to lack of items representing eating behaviours and thoughts we were unable to model the other two latent factors (body dissatisfaction, and binge eating) . The CCHS used the Eating Attitudes Test (EAT) to examine symptoms of eating pathology in their national survey. While the EAT is widely used as a screening instrument in epidemiological studies because it reflects symptoms and concerns characteristic of an eating disorder, the representation of 'thought' and 'behaviour' items was insufficient to test the complete model developed by Miller et al. For instance, none of the 26 items from the EAT capture concerns over body shape, and only one item addresses binge eating behaviours. Still, despite the limited selection of items to model thoughts and behaviours, the fact that we were able to achieve good model fit using only half the items used by Miller et al. only adds strength to the concurrent validity of a thought/behaviour structural model.

The results of our cross-tabulations showed more equal distribution across groups than what was reported by Miller et al. (2008) For example, Miller et al. reported 42% of their sample to be comprised of individuals with no (or few) eating disorder behaviours with moderate levels of disordered thoughts – the 'normative discontent' group. In our study, 28% of individuals were categorized as part of a normative discontent. This difference may be the result of the quantity and quality of our eating disorder thought items. We had fewer items and in particular, we had no items representing body image dissatisfaction. The thought items used by Miller et al. in their CFA included both body dissatisfaction ("I feel dissatisfied with the shape of my body") and fear of fat items ("I am terrified of gaining weight") and frequency analyses revealed body dissatisfaction to be the most highly endorsed items, (e.g. "I think my stomach is too big" – 54% endorsement; Miller et al., 2008). If body dissatisfaction is more normative than fear of fat, then it makes sense for our normative discontent group to be smaller than the normative discontent group reported by Miller et al. since our model did not include items measuring body dissatisfaction. Interestingly, Miller et al. found the first-order latent factor 'fear of fat' contributed more to the second-order factor 'eating disorder thoughts' than did the first-order latent factor 'body dissatisfaction'.

It is notable that despite the fact that our thought subscale was comprised of only three items (I am terrified of being overweight, I am preoccupied with the thought of having fat on my body, and I am preoccupied with the desire to be thinner), our results were consistent with results reported by Miller et al., who used 10 thought items. The major difference in items was we had no measure of body dissatisfaction. Fear of fat may

hold more promise as an indicator of pathological eating thoughts than body dissatisfaction, at least when using items similar in content to those from the EDI and the EAT.

Similar to results by Miller et al. (2008), more individuals fell into the normative discontent group (28% - moderate thoughts/low behaviours) than the asymptomatic group (17% - low thoughts/low behaviours) and although the difference between the two groups was not as large as the difference reported by Miller et al., it was still more common to have eating disorder thoughts than it was to not have them. Furthermore, when we consider the total number of individuals who endorsed eating disorder thoughts (moderate to high levels) we see that more females had disordered thoughts (80%; N=1299) than did not have them (20%; N=326) and this is consistent with results reported by Miller et al.

Again, in accordance with results reported by Miller et al. (2008), high levels of eating disorder thoughts were present either with or without eating disorder behaviours; whereas high levels of eating disorder behaviours were only associated with high levels of thoughts (see Table 3). In fact, not a single individual in this sample could be described as being engaged in extreme levels of eating disorder behaviours without eating disordered thoughts (high behaviours/low thoughts = 0%). Nor could we find a group of individuals who were extreme in their behaviours with moderate levels of thoughts. These findings would seem to imply that the pathology of eating disorder behaviours (vomiting, binge eating) is inevitably linked to equally pathological thoughts.

One notable difference in our cross-tab results compared to results by Miller et al. (2008) was with the moderate behaviour/low thought group (Group 3 – Dieters). The percent of individuals in this cell was 0.5% in Miller et al's study and while still a low percentage in our sample (3%), it was significant enough to warrant including it as a risk profile in our logistic regressions. This group was named 'Dieters' because post-hoc analyses of mean differences across the three behaviour variables – purge, binge and restrict – revealed that restriction was the only distinguishing variable that set this group apart from the others.

The results of our logistic regression analyses mapped perfectly onto the results of the analysis of variance reported by Miller et al. (2008). High levels of thoughts and high levels of behaviours (Group 6 – Clinical) had the highest odds ratio for current diagnoses of major depression, social phobia and substance dependence. Both the high-risk thinkers (Group 4) and the sub-clinical group (Group 5) were statistically significantly more likely to have a diagnosis of major depression, social phobia or substance dependence than the reference group (Group 1 – asymptomatic) and consistent with what was reported by Miller et al., there was no statistically significant risk for concurrent psychopathology among the normative discontent group (Group 2).

The dieter group (Group 3) was not associated with increased risk of concurrent mood, anxiety, or substance disorder, and in fact, was the only group that had a negative odds ratio associated with any of the dependent variables, although importantly this relation was not statistically significant. While a 'dieter' group was not one of the original risk profiles identified by Miller et al. (2008), the existence of this group and the

fact that it was not related to psychopathology has interesting implications for the theoretical model by Miller and Vaillancourt (2008). Miller and Vaillancourt have argued that behaviours are inextricably linked to thoughts and that even moderate levels of eating behaviours will be associated with eating disorder thoughts. The findings by Miller et al. (2008) supported this hypothesis in that they were unable to find a significant group of individuals who engaged in behaviours without thoughts. However, in our study, approximately 3% of the sample could be classified as having moderate eating disorder behaviours and few to no eating disorder thoughts. Yet Miller and Vaillancourt have also noted that dieting per se is not an eating disorder behaviour and the link between dieting and restricting food intake varies considerably depending on how dieting is defined and interpreted (Neumark-Sztainer, Jeffrey, & French, 1997).

Dieting items are often included in measures of restriction because dieting can imply caloric restriction and weight loss although in reality, dieting is rarely accompanied by weight loss, or at least not permanently and is only weakly correlated with actual reductions in caloric intake (Stice, Cooper, Schoeller, Tappe, & Lowe, 2007). Moreover dieting behaviours are considerably more prevalent in non-clinical populations (Ackard, Fulkerson, Neumark-Sztainer, 2007; Miller et al., 2008) and as illustrated in this study, not always related to psychopathology. Thus, the finding that certain behaviours can exist without a high level of thought disturbance does not stand in contrast to what Miller and Vaillancourt have proposed. It simply illustrates the importance of being clear in how we define abnormal eating behaviours.

Limitations and Future Directions

The results of our study supported findings by Miller et al. (2008) that varying the level of eating disorder thoughts and eating disorder behaviours differentially predicts risk for psychopathology. Our study also offered some support for a measurement model of thoughts and behaviours. More importantly we replicated Miller et al's results by using a more representative population and by having more reliable measures of psychopathology for our dependent variables.

The drawbacks of this study are similar to those noted by Miller et al. in that the items representing eating disorder thoughts and behaviours were imperfect and few in quantity. Although we would point out that the limited selection of items did not impact our ability to replicate a structural model of thoughts and behaviours, generating new items that clearly distinguish between symptoms would add strength to future replications of this measurement model.

We conclude this paper by restating the implications specified by Miller et al. (2008) who suggest behavioural criteria be identified separately from psychological criteria when establishing risk for eating disorders or comorbid psychopathology and that to accomplish this we must re-evaluate the instruments by which we assess eating symptomology.

References

Ackard, D. M., Fulkerson, J. A., & Neumark-Sztainer, D. (2007). Prevalence and utility of DSM-IV eating disorder diagnostic criteria among youth. *International Journal of Eating Disorders, 40*, 409-417.

Arbuckle, J. L. (2007). *Amos* (Version 16.0) [Computer Program]. Chicago: SPSS.

Bas, M., Hulya Asci, F., Karabudak, E., & Kiziltan, G. (2004). Eating attitudes and their psychological correlates among Turkish adolescents. *Adolescence, 39*, 593-599.

Becker, C. B., DeViva, J. C., & Zayfert, C. (2004). Eating disorder symptoms among female anxiety disorder patients in clinical practice: The importance of anxiety comorbidity assessment. *Journal of Anxiety Disorders, 18*, 255-274.

Bollen, K. A. (1989). *Structural equations with latent variables*. John Wiley & Sons, Oxford: England.

Bulik, C. M., Sullivan, P. F., Fear, J. L., & Joyce, P. R. (1997). Eating disorders and antecedent anxiety disorders: A controlled study. *Acta psychiatrica Scandinavica, 96*, 101-107.

Centers for Disease Control and Prevention. (2008). BMI - Body Mass Index. Retrieved July 1, 2008 from http://www.cdc.gov/nccdphp/dnpa/bmi/index.htm

Darby, A., Hay, P., Mond, J., Rodgers, B., & Owen, C. (2007). Disordered eating behaviours and cognitions in young women with obesity: relationship with psychological status. *International Journal of Obesity, 31*, 876-882.

Fisher, M., Schneider, M., Pegler, C., & Napolitano, B. (1991). Eating attitudes, health-risk behaviours, self-esteem, and anxiety among adolescent females in a suburban high school. *Journal of Adolescent Health, 12*, 377-384.

Forbush, K., Heatherton, T. F., & Keel, P. K. (2007). Relationships between perfectionism and specific disordered eating behaviours. *International Journal of Eating Disorders, 40*, 37-41.

Fulkerson, J. A., Sherwood, N. E., Perry, C. L., Neumark-Sztainer, D., & Story, M. (2004). Depressive symptoms and adolescent eating and health behaviours: A multifaceted view in a population-based sample. *Preventive Medicine, 38*, 865-875.

Garner, D.M. (1991). *The Eating Disorder Inventory-2: Professional Manual.* Florida: Psychological Assessment Resources Inc.

Garner, D. M., & Garfinkel, P. E. (1979). The eating attitudes test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9*, 273-279.

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: Psychometric features and clinical correlates. *Psychological medicine, 12*, 871-878.

Garner, D. M., Olmsted, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15-34.

Godart, N. T., Flament, M. F., Lecrubier, Y., & Jeammet, P. (2000). Anxiety disorders in anorexia nervosa and bulimia nervosa: Comorbidity and chronology of appearance. *European Psychiatry, 15*, 38-45.

Godart, N. T., Flament, M. F., Curt, F., Perdereau, F., Lang, F., Venisse, J. L., et al. (2003). Anxiety disorders in subjects seeking treatment for eating disorders: A DSM-IV controlled study. *Psychiatry research, 117,* 245-258.

Gravel, R., & Béland, Y. (2005). The Canadian community health survey: Mental health and well-being. *Canadian Journal of Psychiatry, 50*, 573-579.

Hu, L., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods, 3*, 424-453.

Johnson, J. G., Cohen, P., Kotler, L., Kasen, S., & Brook, J. S. (2002). Psychiatric disorders associated with risk for the development of eating disorders during adolescence and early adulthood. *Journal of Consulting and Clinical Psychology, 70*, 1119-1128.

Joreskog, K. G., & Sorbom, D. (1996). *Structural Equation Modeling.* Workshop presented for the NORC Social Science Research Professional Development Training Sessions, Chicago.

Kaye, W. H., Bulik, C. M., Thornton, L., Barbarich, N., Masters, K., Fichter, M. M. et al. (2004). Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *American Journal of Psychiatry, 161*, 2215-2221.

Kline, R. B. (2005). *Principles and practice of structural equation modeling (2nd ed.).* Guilford Press, New York, NY: US.

Kordy, H., & Treasure, J. L. (1997). Effectiveness and efficiency of psychotherapy treatment programmes: The European collaborative longitudinal study on eating disorders (ECLOSE-ED). In P. Bria, A. Ciocca, & S. De Rissio (Eds.), *Psychotherapeutic issues in eating disorders: Models, methods and results* (pp. 1-89). Rome: Socula Editrice Universo.

Miller, J. L., & Vaillancourt, T. (2008). The eating disorder continuum revisited: "Eating disorder thoughts" versus "eating disorder behaviours". Manuscript submitted for publication.

Miller, J. L., Vaillancourt, T., & Hanna, S. (2008). Second-order confirmatory factor analysis of "Eating-disorder-thoughts" versus "Eating-disorder-behaviours": Implications for assessment and detection of eating disorders in epidemiological studies. Manuscript submitted for publication.

Nelson, W. L., Hughes, H. M., Katz, B., & Searight, H. R. (1999). Anorexic eating attitudes and behaviours of male and female college students. *Adolescence, 34,* 621-633.

Neumark-Sztainer, D., Jeffrey, R. W., & French, S. A. (1997). Self-reported dieting: How should we ask? What does it mean? Associations between dieting and reported energy intake. *International Journal of Eating Disorders, 22,* 437-449.

Neumark-Sztainer, D., Story, M., Hannan, P. J., Perry, C. L., & Irving, L. M. (2002). Weight-related concerns and behaviours among overweight and nonoverweight

adolescents: Implications for preventing weight-related disorders. *Archives of Pediatrics Adolescent Medicine, 156*, 171-178.

Pearlstein, T. (2002). Eating disorders and comorbidity. *Archives of Womens Mental Health, 4*, 67-78

Silberstein, L. R., Striegel-Moore, R. H., Timko, C., & Rodin, J. (1988). Behavioral and psychological implications of body dissatisfaction: Do men and women differ? *Sex Roles, 19*, 219-232.

Statistics Canada. (2002). *Canadian Community Health Survey, Cycle 1.2, Mental Health and Well-being.* Public Use Microdata File, on CD-ROM (Catalogue number 82M0021XCB).

Stice, E., & Bearman, S. K. (2001). Body-Image and eating disturbances prospectively predict increases in depressive symptoms in adolescent girls: A growth curve analysis. *Developmental Psychology, 37*, 597-607.

Stice, E., Cooper, J. A., Schoeller, D. A., Tappe, K., & Lowe, M. R. (2007). Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioural data suggest not. *Psychological Assessment, 19,* 449–458.

Streiner, D. L. Cairney, J., & Lesage, A. (2005). Psychiatric epidemiology in Canada and the CCHS study. *Canadian Journal of Psychiatry, 50*, 571-572.

Tomori, M., & Rus-Makovec, M. (2000). Eating behaviour, depression, and self-esteem in high school students. *Journal of Adolescent Health, 26*, 361-367.

Troop, N. A., Serpell, L., & Treasure, J. (2001). Specificity in the relationship between depressive and eating disorder symptoms in remitted and nonremitted women. *International Journal of Eating Disorders, 30*, 306-311.

Wichstrom, L. (1995). Social, psychological and physical correlates of eating problems. A study of the general adolescent population in Norway. *Psychological Medicine, 25*, 567-580.

Wildes, J. E., Simons, A. D., & Marcus, M. D. (2005). Bulimic symptoms, cognitions, and body dissatisfaction in women with major depressive disorder. *International Journal of Eating Disorders, 38*, 9-17.

Wittchen, H. U. (1994). Reliability and validity studies of the WHO--composite international diagnostic interview (CIDI): A critical review. *Journal of Psychiatric Research, 28*, 57.

Zaider, T. I., Johnson, G., Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *International Journal of Eating Disorders, 28*, 58-67.

Table I: Body Mass Index and Weight Opinion by Age Group

	BMI Category			
Age Group	*underweight*	*normal*	*overweight*	*obese*
15 to 19 year olds [1]M=24.3; SD=4.6	4.0%	61.0%	22.3%	10.4%
20 to 24 year olds [2]M=26.1; SD=5.0	2.0%	45.6%	28.6%	19.5%
25 to 29 year olds [3]M=28.2; SD=6.1	1.6%	35.0%	25.9%	33.7%
30 to 34 year olds [4]M=28.1; SD=5.8	1.1%	34.5%	28.8%	31.2%
Total Sample (1567)	2.2%	43.9%	26.5%	23.8%
	Respondent's opinion of own weight			
Age Group	*underweight*	*just right*	*overweight*	
15 to 19 year olds	0.7%	42.9%	54.3%	
20 to 24 year olds	0.8%	31.6%	63.3%	
25 to 29 year olds	1.3%	22.2%	73.0%	
30 to 34 year olds	1.1%	19.3%	75.2%	
Total Sample (1567)	1.0%	28.8%	66.6%	

Note: BMI are based on self-reported height and weight. Underweight is a BMI<18.5; normal weight is a BMI >=18.5 and <= 24.9; overweight is a BMI >= 25.0 and <=29.9; and obese is a BMI >30.0. Means and standard deviations for BMI by age group are presented beneath each age category: [1]N=394; [2]N=378; [3]N=360; [4]N=435

Table II: Means and Standard Deviations for the Eating Attitudes Test (total score)

Age Group	Means	SDs	*N*
15 to 19 year olds	10.61	6.99	403
20 to 24 year olds	11.27	8.83	394
25 to 29 year olds	11.87	11.35	371
30 to 34 year olds	11.22	11.27	450
Total Sample	11.23	9.75	1618

Note: No statistically significant differences between means across any age group, based on independent samples t-tests, $p < .05$

Table III: Cross-tabulations and Standardized Residuals for Thought/Behaviour Groupings.

		Thoughts			
		Low	Moderate	High	Total
Behaviours	Low				
	Count	283	449	222	954
	% Total	17.4%	27.6%	13.7%	
	Std. Residual	6.6	1.7	-6.8	
	Moderate				
	Count	43	257	350	650
	% Total	2.6%	15.8%	21.5%	
	Std. Residual	-7.7	-1.5	7.3	
	High				
	Count	0	0	21	21
	% Total	0.0%	0.0%	2.0%	
	Std. Residual	-2.1	-3.0	4.8	
	Total Count	326	706	593	1625

Table IV: Logistic Regression Results for Predicting Psychiatric Disorders using Level of Eating Disorder Thoughts and Eating Disorder Behaviours as Categorical Independent Variables (N=1627).

Major Depression – 12 months		β	SE β	Wald's χ^2	*df*	*p*	Odds ratio	95% CI	
								Lower	Upper
Group	**Description**								
1. Asymptomatic*	low behaviours/low thoughts	----	----	47.16	5	.00	----	----	----
2. Normative Discontent	low behaviours/mod thoughts	0.35	.28	1.62	1	.20	1.42	0.83	2.43
3. Dieters	mod behaviours/low thoughts	0.49	.53	0.87	1	.35	1.64	0.58	4.59
4. High Risk Thinkers	low behaviours/high thoughts	0.63	.30	4.35	1	.03	1.88	1.04	3.39
5. Sub-clinical	mod behaviours/mod or high thoughts	1.14	.25	21.13	1	.00	3.13	1.93	5.10
6. Clinical	high behaviours/high thoughts	2.43	.49	24.26	1	.00	11.30	4.31	29.65
Social Phobia – 12 months		β	SE β	Wald's χ^2	*df*	*p*	Odds ratio	95% CI	
								Lower	Upper
Group	**Description**								
1. Asymptomatic*	low behaviours/low thoughts	----	----	28.54	5	.00	----	----	----
2. Normative Discontent	low behaviours/mod thoughts	0.43	0.33	1.71	1	.19	1.54	0.81	2.93
3. Dieters	mod behaviours/low thoughts	-0.04	0.78	0.00	1	.96	0.96	0.21	4.37
4. High Risk Thinkers	low behaviours/high thoughts	0.91	0.35	6.85	1	.01	2.48	1.26	4.89
5. Sub-clinical	mod behaviours/mod or high thoughts	1.22	0.30	17.02	1	.00	3.40	1.90	6.09
6. Clinical	high behaviours/high thoughts	1.51	0.62	5.90	1	.02	4.50	1.34	15.17
Substance Dependence – 12 months		β	SE β	Wald's χ^2	*df*	*p*	Odds ratio	95% CI	
								Lower	Upper
Group	**Description**								
1. Asymptomatic*	low behaviours/low thoughts	----	----	11.94	5	.03	----	----	----
2. Normative Discontent	low behaviours/mod thoughts	0.25	0.36	0.46	1	.50	1.28	0.63	2.60
3. Dieters	mod behaviours/low thoughts	0.87	0.60	2.06	1	.15	2.38	0.73	7.75
4. High Risk Thinkers	low behaviours/high thoughts	0.91	0.37	6.02	1	.01	2.48	1.20	5.14
5. Sub-clinical	mod behaviours/mod or high thoughts	0.78	0.33	5.59	1	.02	2.17	1.14	4.13
6. Clinical	high behaviours/high thoughts	1.33	0.69	3.69	1	.05	3.76	0.97	14.55

Note: *denotes reference group for logistic regressions

CHAPTER 7

GENERAL DISCUSSION

Psychiatric illnesses occurring in early life are often especially poignant in that they affect children at a time in their lives when they have relatively little ability to cope. Some problems of childhood and adolescence prevent children from achieving their full potential, both physically and emotionally. Eating disorders are a prime example of this. The onset of eating disorders occurs predominantly during adolescence, anorexia nervosa has an earlier onset, 14 to 18 years, than bulimia nervosa which typically begins in late adolescence or early adulthood (APA, 2000). Although less common, eating disorders do occur among prepubescent children, even those as young as 5 years (Neumark-Sztainer, 2005).

Eating disorders in youth have both immediate and long-term physical health ramifications that can be attributed to the severity of the behavioural manifestations. Malnutrition in early life is hazardous to the brain because ongoing neural connectivity is critical to the development of a mature adult brain (Spreen, Risser, & Edgell, 1995). Some of the physical consequences of an eating disorder include heart irregularities, electrolyte imbalances leading to cardiac arrest, osteoporosis, dehydration, fainting spells and fractures, stunted growth (height), fertility problems, cognitive impairment, decreased brain size and death (APA, 2000; Katzman, 2005; Lambe, Katzman, Mikulis, Kennedy, & Zipursky, 1997). More children and adolescents die from anorexia nervosa than from all other psychiatric illnesses combined (Sullivan, 1995). The prognosis of an eating disorder is grim; for those who do recover the process is slow, an average of 6 to 8

years from illness onset (Strober, Freeman, & Morrell, 1997), and two-thirds of those with anorexia will remain chronically ill. Among the factors that predict successful recovery, age of onset and duration of illness are two important variables. A more protracted course is indicative of a poorer outcome (Fahy & Russell, 1993; Steinhausen, 2002), and early onset (before puberty) is negatively related to recovery (Russell, 1992). Still, it is more common for eating disorders to occur in pubertal or post-pubertal adolescents and in such cases earlier age of onset is associated with a better outcome (Steinhausen, 2002). For these reasons, prevention and early detection is critical.

Prevention efforts require prospective studies where high-risk populations are identified and followed over time to examine differences between those who develop an eating disorder and those who do not. This information can provide us with clues to the etiology of eating disorders. Prospective research is a long-term, labour intensive and costly research methodology. However it is the most important research design in prevention research because of the ability to draw more causal inferences about risk factors in the onset of an eating disorder. Choosing a target sample in a prospective study requires knowledge of characteristics that increase vulnerability of an eating disorder. This is where cross-sectional studies are useful. Cross-sectional research allows us to test relationships among variables in an easy and efficient manner, and while the results of cross-sectional studies do not allow us to predict who is at-risk for an eating disorder, it provides us with direction in where to start looking. The studies presented in Chapter 2 and Chapter 3 of this thesis demonstrated higher levels of eating pathology among both shy and neurotic females. As both these studies were cross-sectional we cannot infer

either shyness or neuroticism play a casual role in the onset of eating disorders. What we can offer and what these studies have contributed to the literature on risk assessment in the eating disorders is direction in selecting target samples in future prospective research studies.

Detection of undiagnosed cases of eating disorders in community settings requires epidemiological research; however the usefulness of such studies is highly dependent on valid measures and clearly defined indicators of risk. The most rigorous method for identifying cases of eating disorders in those who do not seek medical treatment voluntarily is to employ a two-stage screening study. A two-stage screening study is considered the gold-standard in epidemiological research on eating disorders (Jacobi, Abascal, & Taylor, 2004). The two-stage screen typically involves administering a questionnaire, such as the Eating Attitudes Test (EAT-26) to a large sample of at-risk individuals (e.g., adolescent females) and identifying the subset of individuals who score above a pre-determined threshold on the questionnaire (e.g., 20 or above on the EAT-26) to return for the second stage of the screen: the clinical interview. The EAT is the most widely used screen for detecting eating disorders in epidemiological studies, although its primary use has been for the detection of cases of anorexia nervosa, and not bulimia nervosa (Jacobi et al., 2004). The EAT has high sensitivity and high specificity according to three existing validation studies noted by Jacobi et al. in their review of screening instruments (Button & Whitehouse, 1981; Garner & Garfinkel, 1979, 1980; Mann et al., 1983). Sensitivity refers to how good a test is at correctly identifying people who have a disorder whereas specificity refers to how good a test is at correctly identifying people

who do not have a disorder. Jacobi et al. (2004) recommend sensitivity and specificity be at least 90% in populations where the prevalence of the disorder is below 1% because low prevalence disorders lead to low positive predictive value (PPV). PPV represents the probability that someone with a positive screen actually has the disorder. High sensitivity and specificity do not guarantee high PPV. Say for example that a screening instrument for eating disorders has a specificity of 89% and sensitivity of 100% in a population where the prevalence of an eating disorder is 1%, then the PPV is 8.4% because;

$$PPV = \quad p*Se/(1-Sp) + p\,(Se + Sp - 1)$$

Where p = the prevalence of the disorder in the population being sampled. Thus despite having high sensitivity and specificity, the predictive value of a sensitive test can be quite poor (Jacobi et al., 2004).

To illustrate this point, consider the five following two-stage epidemiological studies of eating disorders. Supposing that each clinical interview takes a minimum of 30 minutes to complete and costs approximately $100 per interview, take a look at the amount of resources spent using measures purported to have both high sensitivity and high specificity (example of resource costs are based on Jacobi et al., 2004). Rosenvinge, Borgen, and Borresen (1999) screened 15-year-old adolescents using a score >10 on the drive for thinness subscale of the EDI as a threshold. Of the 678 students who were sampled, 73 screened positive – 14 were subsequently diagnosed with an eating disorder. It took 37 hours and $7300 to detect 14 cases in this study. Nasser (1994) screened Egyptian secondary school girls using the EAT. Of the 351 students who were sampled, 40 screened positive – 3 eating disorders were detected. In this study, it took 20 hours and

$4000 to detect 3 cases. Nobakht and Dezhkam (2000), surveyed Iranian females ages 15-18 years using the EAT. Of the 3100 students sampled, 749 screened positive – 128 eating disorders were detected. It took 375 hours and $74 900 to detect these 128 cases. Rojo et al. (2003) surveyed 12-18 year old Spanish students using the EAT. Of the 544 sampled, 58 screened positive – 15 eating disorders were detected. In this study, it took 29 hours and $5800 to detect 15 cases. Finally, Childress, Brewerton, Hodges, and Jarrell (1993) surveyed children grades 5 to 8 using the Kids' Eating Disorders Survey (KEDS). Of the 3175 students surveyed, 175 screened positive – 18 subsequently were diagnosed with an eating disorder. It took 88 hours and $17 500 to detect these 18 cases.

Relying on screening measures such as the EAT or EDI for detecting undiagnosed eating disorders will be costly and time consuming because both measures contain far more normative weight concern items and dieting items than clinically significant eating disorder behaviours. The high cost and time consuming nature of epidemiological studies may negatively impact the number of studies that are conducted, thereby reducing the chances of detecting undiagnosed cases of eating disorders. Importantly, the resources spent on screening for eating disorders using a measure with poor specificity takes away resources that could be spent on treatment or prevention programs aimed at improving body satisfaction and size acceptance (e.g., "Healthy schools – Healthy Kids", McVey, Tweed, Blackmore, 2007).

Throughout this thesis I have argued that eating disorder thoughts have poor specificity despite having high sensitivity. The thoughts will always identify all true cases of eating disorders, as long as the criteria for an eating disorder continues to be partially

defined by disturbances in body image and fears of weight gain; but the thoughts will also identify far too many non-cases because thoughts are normative. Given the level of normative discontent concerning weight and shape in our culture it would seem prudent to examine other measures of eating pathology that have higher specificity.

There is something to be said for being over inclusive in screening for eating disorders, given that early detection of progressing pathological behaviour leads to early intervention of potential full syndrome eating disorders (Ackard et al., 2007). However, there is a more efficient way to have both high sensitivity and high specificity and also detect sub-threshold cases, while still reducing the high costs of epidemiological studies and that is by examining the eating disorder thoughts separately from the behaviours.

Future Directions

If the thoughts are the risk factors and the behaviours are the process factors, what is it about the behaviours that make something clinical? The studies published in Chapters 2 and 3 would imply that there is a personality factor related to those who engage in these disinhibited behaviours, but again with cross-sectional research we are limited in the conclusions that can be drawn from such studies. Temperamental dispositions combined with environmental pressures to have a thin physique may increase the risk of an eating disorder through a diathesis-stress model of pathology. Longitudinal studies examining the personality profiles of individuals with eating disorders prior to the onset of the illness are needed in order to further test this relation.

It may be beneficial to focus future research on understanding potential executive functioning deficits associated with the behaviours of an eating disorder. For example,

reward deficiency syndrome has been put forth as an explanation for other compulsive, impulsive, and addictive disorders based on a common genetic deficiency in the dopamine D_2 receptor (Blum et al., 1996). Dopamine is not the only neurotransmitter implicated in the reward system, serotonin is also known to be involved (Blum et al., 1996) and there is already evidence to support the role of serotonin imbalances in the eating disorders, bulimia nervosa in particular (e.g., Steiger, Israel, Gauvin, Kin, & Young, 2003).

In order to distinguish between 'normative discontent' and clinical eating disorder thoughts there needs to be a shift in the way we think about and measure fear of weight gain or body disturbance. Measuring body dissatisfaction is not equivalent to measuring the over-evaluation of weight and shape on an individuals' self-concept (Masheb, Grilo, Burke-Martindale, & Rothschild, 2006). Measures of weight concern need to place more emphasis on the level of impairment as this is a fundamental part of how we define what is abnormal. Asking how often someone feels dissatisfied with the shape of their body is not a good indicator of the presence of distress, disability or dysfunction that characterizes an eating disorder. If it were, the prevalence of eating disorders would be epidemic.

An interesting avenue of future research would be to examine sub-groups of individuals who engage in disordered thinking. Weight fears and body disparagement may be lower on a thought continuum compared to thoughts about behaviours ("I have thought of trying to vomit in order to lose weight"), in terms of risk for engaging in disordered behaviours. Moreover, it may be that thoughts about disordered behaviours

and the frequency of these thoughts are what mark the transition from non-clinical to clinical.

In Conclusion

At the beginning of this thesis I asked a question: "How do we define eating disorders?" Without an accurate definition of what is abnormal about an eating disorder we cannot design valid and reliable measures to be used in the assessment of risk and detection of cases. To define what is abnormal, we must consider what is normal within a given culture. If weight discontent is normative in our culture then it is problematic to use these 'normal' features to screen for an abnormal syndrome.

A final remark concerning the 'normative discontent'. The term 'normative discontent' refers to the high prevalence of weight concern in the general population, In other words, normative refers to the fact that these concerns are common, or statistically frequent. However, this is not equivalent to saying it is acceptable or tolerable for there to be an epidemic of weight discontent. Rather the term 'normative discontent' simply reflects the unfortunate level of unrest that exists in the mind of a society that has become obsessed with being thin.

REFERENCES

Ackard, D. M., Fulkerson, J. A., & Neumark-Sztainer, D. (2007). Prevalence and utility of DSM-IV eating disorder diagnostic criteria among youth. *International Journal of Eating Disorders, 40*, 409-417.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th *ed.*). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th *ed.*, text revision). Washington, DC: Author.

Bulik, C. M., Sullivan, P. F., Carter, F. A., McIntosh, V. V., & Joyce, P. R. (1999). Predictors of rapid and sustained response to cognitive-behavioural therapy for bulimia nervosa. *International Journal of Eating Disorders, 26*, 137-144.

Button, E. J., & Whitehouse, A. (1981). Subclinical anorexia nervosa. *Psychological Medicine, 11*, 509-516.

Blum, K., Sheridan, P. J., Wood, R. C., Braverman, E. R., Chen, T. J. H., Cull, J. G., et al. (1996). The D_2 dopamine receptor gene as a determinant of reward deficiency syndrome. *Journal of the Royal Society of Medicine, 89*, 396-400.

Cash, T. F., Morrow, J. A., Hrabosky, J. I., & Perry, A. A. (2004). How has body image changed? A cross-sectional investigation of college women and men from 1983 to 2001. *Journal of Consulting and Clinical Psychology, 72*, 1081-1089.

Chamay-Weber, C., Narring, F., & Michaud, P. (2005). Partial eating disorders among adolescents: A review. *Journal of Adolescent Health, 37*, 417-427.

Childress, A. C., Brewerton, T. D., Hodges, E. L., Jarrell, M. P. (1993). The Kids' Eating Disorder Survey (KEDS): A study of middle school students. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 843-850.

Crowther, J.H., Wolf, E.M., & Sherwood, N. (1992). Epidemiology of bulimia nervosa. In M. Crowther, D. L. Tennenbaum, S. E. Hobfoll, & M. A. P. Stephens (Eds.), *The Etiology of Bulimia Nervosa: The Individual and Familial Context* (pp. 1-26). Washington, DC: Taylor & Francis.

Fahy, T. A., & Russell, G. F. M. (1993). Outcome and prognostic variables in bulimia nervosa. *International Journal of Eating Disorders, 14*, 135-145.

Fairburn, C. G., & Cooper, P. J. (1982). Self-induced vomiting and bulimia nervosa: An undetected problem. *British Medical Journal, 284*, 1153-1155.

Fairburn, C. G., & Cooper, Z. (2007). Thinking afresh about the classification of eating disorders. *International Journal of Eating Disorders, 40*, S107-S110.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic theory and treatment. *Behavior Research and Therapy, 41*, 509-528.

Fairburn, C. G., Hay, P. J., & Welch, S. L. (1993). Binge eating and bulimia nervosa: Distribution and determinants. In C. G. Fairburn & G. T. Wilson, (Eds.), *Binge Eating: Nature, Assessment, and Treatment* (pp. 123-143). New York: Guilford.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *The Lancet, 361*, 407-416.

Fairburn, C. G., & Walsh, B. T. (2002). Atypical eating disorders not otherwise specified. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook, 2nd ed.* (pp 171-177). New York: Guilford Press.

Fisher, M., Golden, N. H., Katzman, D. K., Kreipe, R. E., Rees, J., Schebendach, J., et al. (1995). Eating disorders in adolescents: A background paper. *Journal of Adolescent Health, 16,* 420-437.

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9*, 273-279.

Garner, D. M., & Garfinkel, P. E. (1980). Socio-cultural factors in the development of anorexia nervosa. *Psychological Medicine, 10*, 647-656.

Gordon, R. A. (1990). *Anorexia and Bulimia: Anatomy of a Social Epidemic.* New York: Blackwell.

Hoek, H. W. (1995). The distribution of eating disorders. In K. D. Brownell & C. G. Fairburn (Eds.), *Eating Disorders and Obesity: A Comprehensive Handbook* (pp. 207-211). New York: Guilford.

Hoek, H. W. (2006). Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry, 19*, 389-394.

Hoek, H. W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders, 34*, 383-396.

Jacobi, C., Abascal, L., & Taylor, C. B. (2004). Screening for eating disorders and high-risk behaviour: Caution. *International Journal of Eating Disorders, 36*, 280-295.

Katzman, D. K. (2005). Medical complications in adolescents with anorexia nervosa, *International Journal of Eating Disorders, 37*, 552-559.

Lambe, E. K. Katzman, D. K., Mikulis, D. J., Kennedy, S. H., & Zipursky, R. B. (1997). Cerebral gray matter volume deficits after weight recovery from anorexia nervosa. *Archives of General Psychiatry, 54*, 537-542.

Lucas, A. R., Beard, C. M., O'Fallon, W. M., & Kurland, L. T. (1991). 50-year trends in the incidence of anorexia-nervosa in Rochester, Mn – a population-based study. *American Journal of Psychiatry, 148*, 917-922.

Mann, A. H., Wakeling, A., Wood, K., Monck, E., Dobbs, R., & Szmuckler, G. (1983). Screening for abnormal eating attitudes and psychiatric morbidity in an unselected population of 15-year-old schoolgirls. *Psychological Medicine, 13*, 573-580.

Masheb, R. M., Grilo, C. M., Burke-Martindale, C. H., & Rothschild, B. S. (2006). Evaluating oneself by shape and weight is not the same as being dissatisfied about shape and weight: A longitudinal examination in severely obese gastric bypass patients. *International Journal of Eating Disorders, 39*, 716-720.

McVey, G., Tweed, S., & Blackmore, E. (2004). Dieting among preadolescent and young female adolescents. *Canadian Medical Association journal, 170*, 1559-1561.

McVey, G., Tweed, S., & Blackmore, E. (2007). Healthy schools-healthy kids: A controlled evaluation of a comprehensive eating disorder prevention program. *Body Image, 4*, 115-136.

Nasser, M. (1994). Screening for abnormal eating attitudes in a population of Egyptian secondary school girls. *Social Psychiatry and Psychiatric Epidemiology, 29*, 25-30.

Neumark-Sztainer, D. (2005). *I'm, Like, SO Fat!* New York: The Guilford Press.

Nobakht, M., & Dezhkam, M. (2000). An epidemiological study of eating disorders in Iran. *International Journal of Eating Disorders, 28*, 265-271.

Polivy, J., & Herman, C. P. (1987). Diagnosis and treatment of normal eating. *Journal of Consulting and Clinical Psychology, Special Issue: Eating Disorders, 55*, 635-644.

Puhl, R. M., & Latner, J. D. (2007). Stigma, obesity, and the health of the nation's children. *Psychological Bulletin, 133*, 557-580.

Rodin, J., Silberstein, L., & Striegel-Moore, R. (1984). Women and weight: A normative discontent. *Nebraska Symposium on Motivation, 32*, 267-307.

Rojo, L., Livianos, L., Conesa, L., Garcia, A., Dominguez, A., Rodrigo, G., et al. (2003). Epidemiology and risk factors of eating disorders: A two-stage epidemiologic study in a Spanish population aged 12-18 years. *International Journal of Eating Disorders, 34*, 281-291.

Rosenvinge, J. H., Borgen, J. S., Borresen, R. (1999). The prevalence and psychological correlates of anorexia nervosa, bulimia nervosa and binge eating among 15-year-old students: A controlled Epidemiological Study. *European Eating Disorders Review, 7*, 382-391.

Russell, G. F. M. (1992). Anorexia nervosa of early onset and its impact on puberty. In P. J. Cooper, & A. Stein (Eds.), *Feeding problems and Eating Disorders in Children and Adolescents*, (pp 85-112). Chur, Switzerland, Harwood Academic Publishers.

Schmidt, L. A. (1999). Frontal brain electrical activity in shyness and sociability. *Psychological Science, 10*, 316-320.

Schmidt, L. A. (2003). Shyness and sociability: A dangerous combination for preschoolers. *International Society for the Study of Behavioral Development, Newsletter, 1*, 6-8.

Shisslak, C. M., Crago, M., & Estes, L. S. (1995). The spectrum of eating disturbances. *International Journal of Eating Disorders, 18*, 209-219.

Smolak, L. (2006). Body image. In J. Worell, & C. D. Goodheart (Eds.), *Handbook of girls' and women's psychological health: Gender and well-being across the lifespan.* Oxford series in clinical psychology (pp. 69-76). New York, NY: Oxford University Press

Spreen, O., Risser, A. H., & Edgell, D. (1995). *Developmental Neuropsychology*. New York: Oxford University Press.

Steiger, H., Israel, M., Gauvin, L., Ng, Y. K., & Young, S. N. (2003). Implications of compulsive and impulsive traits for serotonin status in women with bulimia nervosa. *Psychiatry Research, 120*, 219-229.

Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. *The American Journal of Psychiatry, 159*, 1284-1293.

Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders, 22*, 339-360.

Sullivan, P. F. (1995). Mortality in anorexia-nervosa. *American Journal of Psychiatry, 152*, 1073-1074.

Wilfley D. E., Bishop, M. E., Wilson, G. T., & Agras, W. S. (2007). Classification of eating disorders: Toward DSM-V. *International Journal of Eating Disorders, 40*, S123–129.