EXAMINATION OF A MODIFIED DUAL PATHWAY MODEL OF DISORDERED EATING IN YOUTH WITH TYPE 1 DIABETES

by

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(Under the Direction of Sarah Nowaczyk)

ABSTRACT

Disordered eating behavior (DEB) may be more prevalent in adolescents with type 1 diabetes (T1D) than their non-diabetic peers, and consists of binge eating, dietary restriction, purging, and/or extreme weight concerns. Co-occurring DEB and T1D may decrease glycemic control and can place individuals at greater risk for poor health outcomes. Given the risk for health complications in this population, it is important to consider variables that distinguish DEB from normative eating behavior in the context of a diabetic care regimen. Further, there are several aspects of the T1D disease process and treatment regimen which may place youth at higher risk for DEB, and which need to be accounted for when conceptualizing DEB in this population. Insulin treatment, subsequent weight gain, and disruptions to hunger and satiety regulation are hypothesized mechanisms through which the treatment of T1D may increase vulnerability to DEB as proposed by the Modified Dual Pathway model. These factors, as well as body image dissatisfaction, dietary restraint, and depression were examined in conjunction with bulimic symptoms in a sample of 101 youth (43 were transitioning to insulin pump, and thus experienced with the treatment, and 58 were newly diagnosed). The current study
lends support for the Modified Dual Pathway Model by demonstrating the unique relationship between hunger and satiety dysregulation and DEB, even when controlling for other known risk factors for DEB. Thus, the current study begins to address gaps in the literature regarding risk for DEB in youth with T1D by testing this expanded model. The Modified Dual Pathway Model incorporates psychosocial factors as well as physiological disease and treatment related factors in a more accurate and comprehensive conceptualization of risk for DEB in youth with T1D.

INDEX WORDS: Disordered Eating Behavior, Type 1 Diabetes
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B. A., University of Kentucky, 2005
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A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2014
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December, 2014
DEDICATION

I would like to dedicate my dissertation to my family, my partner, friends, mentors, and colleagues. Thank you for your unending academic, emotional, financial, and social support during my graduate school years. A special thanks to my father who knew first-hand about the rigors of academia and always had helpful words of wisdom along the way. “The best dissertation is a done dissertation.” Thanks, Dad.
ACKNOWLEDGEMENTS

I would like to express my sincerest gratitude to Dr. Sarah Fischer for her invaluable mentorship over the years. I was fortunate enough to begin collaborating with her during my undergraduate years at UK where she encouraged me to take on my first independent research project and mentored me throughout the process. I would later on to be her graduate student at UGA and similarly, Dr. Fischer has always encouraged me to pursue more than I thought possible. Her confidence in my abilities and unending support of my work have shaped my professional development and allowed for me to be where I am today. I am incredibly fortunate to have had the opportunity to learn from her, and to have her as a mentor, colleague, and friend going forward. I would like to thank Dr. Anne Shaffer who mentored me throughout graduate school, as my major professor my last year of graduate school, and while on internship. She provided encouragement, support, and professional guidance during a period filled with both personal and professional challenges. I am thankful for her sharp insights, tenacity, and for always pushing me to think more critically about research ideas. I would like to thank Dr. Deborah Young Hyman for her guidance on this project, and for encouraging me to pursue this area of research. I was fortunate to be able to collaborate with her during my time in graduate school, and without her support the current study would not have been possible. Finally I would like to thank Dr. Ronald Blount for his input on my project, as well as his mentorship and support during my graduate training.
# TABLE OF CONTENTS

ACKNOWLEDGEMENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER

<table>
<thead>
<tr>
<th>Number</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>INTRODUCTION</td>
</tr>
<tr>
<td>2</td>
<td>METHOD</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Need for Integration of Pediatric Medical and Psychosocial Models of Risk for BN</td>
</tr>
<tr>
<td>2</td>
<td>Stice’s Dual Pathway Model</td>
</tr>
<tr>
<td>3</td>
<td>Type 1 Diabetes Treatment Regimen</td>
</tr>
<tr>
<td>4</td>
<td>Prevalence of Eating Pathology in Youth with T1D</td>
</tr>
<tr>
<td>5</td>
<td>Health Consequences of Co-Occurring T1D and DEB</td>
</tr>
<tr>
<td>6</td>
<td>Factors in T1D that Influence DEB</td>
</tr>
<tr>
<td>7</td>
<td>Integrating Psychosocial Risk Theories and Disease Models to Explain DEB in T1D</td>
</tr>
<tr>
<td>8</td>
<td>Applying the Dual Pathway Model to T1D</td>
</tr>
<tr>
<td>9</td>
<td>The Current Study</td>
</tr>
<tr>
<td>10</td>
<td>Statistical Approach</td>
</tr>
<tr>
<td>Sections</td>
<td>Pages</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Power Analysis</td>
<td>27</td>
</tr>
<tr>
<td>Procedure</td>
<td>28</td>
</tr>
<tr>
<td>Measures</td>
<td>29</td>
</tr>
<tr>
<td>RESULTS</td>
<td>32</td>
</tr>
<tr>
<td>Descriptive Analyses</td>
<td>32</td>
</tr>
<tr>
<td>Multiple Regression Analyses</td>
<td>35</td>
</tr>
<tr>
<td>DISCUSSION</td>
<td>38</td>
</tr>
<tr>
<td>Clinical Implications</td>
<td>44</td>
</tr>
<tr>
<td>Strengths, Limitations, and Future Directions</td>
<td>46</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>49</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table 1: Descriptive Statistics.................................................................63
Table 2: Correlations of Combined Group Variables...............................64
Table 3: Correlations of Newly Diagnosed Group Variables......................65
Table 4: Correlations of Pump Transitioning Group Variables...................66
Table 5: Hierarchical Multiple Regression Analysis: Combined Group Predicting Bulimic Symptoms.................................................................67
Table 6: Hierarchical Multiple Regression Analysis: Newly Diagnosed Group Predicting Bulimic Symptoms.................................................................68
Table 7: Hierarchical Multiple Regression Analysis: Pump Transitioning Group Predicting Bulimic Symptoms.................................................................69
LIST OF FIGURES

Figure 1: Stice’s Original Dual Pathway Model………………………………61
Figure 2: Modified Dual Pathway Model…………………………………….62
CHAPTER 1

INTRODUCTION

Eating disorders affect an estimated 2.8% of adolescents, with subclinical eating disorder symptoms affecting a greater proportion (Beato-Fernandez, Rodriguez-Cano, Belmonte-Lario, & Delgado, 2004; Patton, Coffey, & Sawyer, 2003; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). Eating pathology onsets as early as middle childhood, with mean age of onset occurring in adolescence and peaking at age 16 (Favarro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009; Stice, Killen, Hayward, & Taylor, 1998). Disordered eating symptoms such as weight and shape concerns, excessive dieting, binge eating, and purging can increase in severity over time, and are associated with a number of poor outcomes for adolescents (Neumark-Sztainer, Ackard, Utter, Story, & Sockalosky, 2002; Neumark-Sztainer, Wall, Guo, Story, Haines, & Eisenberg, 2006).

Disordered eating behavior (DEB) may be significantly higher in adolescents with T1D as compared to their non-diabetic peers (Nielsen, 2002) and has been shown to be related to poor health outcomes in adolescents with T1D (Silverstein, 2005). DEB has been linked to a number of significant health complications in T1D stemming from poor glycemic control (DCCT, 1994; EDIC, 2003; Jones, Lawson, Daneman, Olmsted, & Rodin, 2000; Neilson, 2002; Rydall, Rodin, Olmsted, Devenyi, & Daneman, 1997). These include neuropathy, retinopathy and nephropathy. Even a one percentage point increase in A1c (a standard indicator of glycemic control) can result in a significant
increase in risk of developing retinopathy (DCCT, 1994), demonstrating the potential deleterious effects that DEB can have on glycemic control.

DEB is characterized by dieting, binge eating (the consumption of objectively large amounts of food with associated cognitions regarding loss of control), purging, fasting, and excessive use of physical activity (Podar, Hannus, & Allik, 1999; Rosen, 2003). DEB in the T1D population includes these behaviors as well as insulin restriction or omission (APA, 2000). Weight gain typical of pubertal development can be exacerbated in adolescent girls (and boys) with T1D because of rebound adiposity following initiation of insulin treatment (Helgeson, Siminerio, Escobar, & Becker, 2009).

Weight gain secondary to treatment may yield increased body dissatisfaction, an identified precursor to dietary restriction and binge eating in the non-diabetic adolescent population (Stice, 2002). Additionally, the T1D treatment regimen necessitates a preoccupation with food intake in order to count carbohydrates and accurately dose insulin. Thus, the adolescent T1D population appears to have multiple risk factors for the development of DEB, but the mechanisms by which this occurs have not been studied within the context of treatment of the disease. Given the medical risks associated with co-occurring DEB and T1D, it is important to identify risk factors for the development of disordered eating in this population that could help inform intervention efforts.

**Need for Integration of Pediatric Medical and Psychosocial Models of Risk for BN**

An important issue to consider when integrating literature on risk for DEB in youth with T1D includes how psychological and pediatric models may complement each other. Pediatric medical models and psychosocial models have separately introduced theories explaining the increased risk for eating pathology in T1D. Pediatric medical
models focus on the physiological nature of the disease, and caution against over-pathologizing adolescents with chronic illnesses (misinterpreting behaviors associated with the disease and treatment regimen as disordered, e.g., forgetting an insulin dose). Psychosocial risk models tend to exclude disease related variables such as insulin dosing and consideration of the disease context, and emphasize prototypical symptoms seen in the non-T1D population such as dietary restriction and binge eating, thus failing to acknowledge the effects of the treatment regimen and disruptions in hunger and satiety. As a result, the extant literature on eating pathology in T1D lacks integrative models to explain the role of psychosocial factors on DEB in the context of the illness.

Stice’s Dual Pathway model (Stice & Agras, 1998), extensively researched in adolescents without T1D, can be modified and extended to include disease-related variables relevant to pediatric populations. In this way, incorporating both medical and psychosocial risk factors in the development of eating pathology could provide a more comprehensive account specifically relevant to this population. Stice's model posits that internalization of the thin ideal and pressure for thinness lead to body image dissatisfaction. Body image dissatisfaction can promote both dieting and negative affect, which can each potentiate risk for binge eating. Further, negative affect and dieting can directly influence one another (Allen, Byrne, & McLean, 2012; Gleaves & Eberenz, 1995; Killen et al., 1994; Stice, 2001; Tylka & Subich, 2004). In applying this model to T1D, it may be that a combination of food preoccupation associated with the treatment regimen, body image dissatisfaction as a result of rebound adiposity, and elevated levels of depression promote disordered eating. It is also important to consider disease-related
variables such as the T1D insulin regimen, hunger and satiety dysregulation that occurs in youth with T1D, and glycemic control in this overall model.

**Stice’s Dual Pathway Model**

Adolescence is a time of risk for the onset of disordered eating behaviors such as extreme dieting, binge eating, and purging (Stice & Agras, 1998). A number of theoretical models explain reasons for this increased risk for the onset of disordered eating behaviors during this developmental period. Stice proposed a novel model as a framework for explaining the development and maintenance of binge eating and associated eating disorder symptoms in the general population (Stice, Shaw, & Nemeroff, 1998). According to Stice’s Dual Pathway model, body image dissatisfaction develops as a result of exposure to sociocultural pressure for thinness and elevated body mass. Stice posits that repeated messages that one is not thin enough may lead to internalization of an ideal thin physique, resulting in dissatisfaction of one’s own body shape and weight because this ideal is so unrealistic to achieve (Stice et al., 1998). This dissatisfaction with one’s physique related to the thin ideal may be even greater in adolescent girls who are concurrently undergoing physiological changes related to pubertal development including a substantial weight gain (Stice, 2001).

In the general population body image dissatisfaction can in turn influence binge eating via two pathways: the dietary restraint pathway and the negative affect pathway. The dietary restraint pathway posits that dieting as a result of body image dissatisfaction could promote binge eating as a result of caloric deprivation as well as the abstinence-violation effect (Polivy et al., 2005). Thoughts associated with breaking food rules increase negative mood, disinhibit attempts to control what one eats, and lead to binge
eating (Grilo & Shiffman, 1994). Additionally, dietary restriction can increase negative affect due to the effects of caloric deprivation on mood, as well as the result of failures at both dieting and weight loss (Lowe & Timko, 2004; Stice, 2001).

Negative affect is another pathway to bulimic behavior because eating is frequently thought of as a way to cope with distress via distraction (Heatherton & Baumeister, 1991). Consistent with this hypothesis, prospective studies have linked negative affect to later increases in BN symptoms (Stice, 2001; Stice, Akutagawa, Gaggar, & Agras, 2000) and onset of binge eating among female adolescents (Stice & Agras, 1998; Stice, Killen, Hayward, & Taylor, 1998). The following sections review empirical literature supporting the role of thin ideal internalization, body image dissatisfaction, dieting, and negative affect in risk for DEB.

**Internalization of the Thin Ideal**

A very thin ideal standard of beauty is a putative risk factor for body image dissatisfaction and subsequent eating pathology in girls and women (see Figure 1; Stice, 2002; Tylka & Subich, 2004). Researchers have examined potential mechanisms whereby sociocultural pressure for thinness yields decreased body image satisfaction. Rodin and colleagues (1985) first postulated that internalization of the thin ideal is one such mechanism that leads to body image dissatisfaction via awareness and knowledge of the social implications of this thin ideal. The extent to which women have associated thinness with reinforcement such as success, happiness, increased attractiveness to others, increased self-esteem, and respect from others if they can be thinner translates to the strength of their expectancies, and subsequently, their level of drive for thinness (Smith et al., 2007). Individuals then adopt this thin ideal to the point that it influences their
attitudes and behaviors (Cafri, Yamamiya, Brannick, & Thompson, 2005; Striegel-Moore et al., 2004). The media promotes an unrealistic standard of thinness that most women cannot achieve, which contributes to more women feeling negatively towards their bodies and greater pressure to be thin (Stice et al., 1996). Thin-ideal internalization and emphasis on appearance may contribute to body image dissatisfaction because of the difficulty in achieving this ideal (Fairburn, 1997; Stice, 2002; Stice & Shaw, 2002). This becomes a cycle because the more pressure there is to be thin, the more women may experience a disturbed body image (Stice et al., 1996; Stice & Shaw, 2002; Tylka & Subich, 2004).

**Body Image Dissatisfaction**

Body image dissatisfaction is one of the most consistent and robust risk and maintenance factors for eating pathology (Stice, 2002). The Dual Pathway Model proposes that internalization of the thin ideal leads to body image dissatisfaction, in part because this physical ideal is extremely difficult for most individuals to achieve. Over-evaluation of the importance of body weight and shape seems to play an important role in the risk of developing disordered eating behavior that includes binge eating (Stice, Presnell, & Spangler, 2002). This finding has been replicated in both college and community samples (e.g., Ricciardelli, Tate & Williams, 1997). Higher levels of body image dissatisfaction in women entering college has been shown to relate to worsening patterns of eating pathology across the college years (Cooley & Toray, 2001). It is theorized that body image dissatisfaction promotes dieting and negative affect, placing individuals at high risk for DEB (Allen, Byrne, & McLean, 2011; Gleaves & Eberenz, 1994; Killen et al., 1994; Stice, 2001; Tylka & Subich, 2004).
**Dietary Restraint**

Body image dissatisfaction as a result of sociocultural pressure for thinness can lead to dieting efforts to lose weight (Stice, 2002). Studies indicate that dietary restriction increases risk for DEB in non-diabetic populations (Fairburn, Cooper, & Shafran, 2003). Dietary restriction is thought to increase risk for binge eating; specifically, because of caloric deprivation and the abstinence violation effect (Fairburn et al., 2003; Polivy, Coleman, & Herman, 2005; Polivy & Herman, 1985). The abstinence violation effect is characterized by a cognitive/affective reaction to a lapse (Marlatt & Gordon, 1985). The lapse is defined as an initial violation of an abstinence rule (e.g., dietary rule). Several studies have demonstrated the relationship between the physiological susceptibility that dietary restriction has on subsequent binge eating (Polivy, 1996; Polivy et al., 1994; Stice et al., 2008). Research on processes related to abstinence violation and eating behavior suggest that the imposition of strict dietary rules, and then breaking these rules, can lead to dysregulated eating (Polivy & Herman, 1985; Polivy et al., 2005).

In several longitudinal studies of adolescents, dietary restraint has demonstrated significant associations with the development of disordered eating behaviors such as binge eating and purging (Allen et al., 2011; Stice, 2001; Stice & Agras, 1998). In a longitudinal study examining eating behaviors of youth ages 9-14 over three years, dieting was prospectively associated with binge eating in boys and girls (Field, Austin, Taylor, et al., 2003). Over the course of a recent five year study youth who were engaging in dietary restraint were 2-3 times more likely to develop binge eating problems, and depression significantly moderated this relationship (Goldschmidt, Wall,
Loth, Le Grange, & Neumark-Sztainer, 2012). Finally, a recent ecological momentary assessment study found that dietary restraint significantly increased participant’s odds of engaging in binge eating on that day and the following day in a sample of young women with BN (Zunker, Peterson, Crosby, Cao, Engel, Mitchell, & Wonderlich, 2011). Dietary restriction is a salient risk factor for binge eating and is also associated with negative affect.

**Negative Affect**

In the non-diabetic population, major Depressive Disorder is one of the most common co-occurring disorders with DSM-IV Bulimia Nervosa and DEB (Bushnell, Wells, McKenzie, Hornblow, Oakley-Brown, & Joyce, 2009). Co-occurring depression in individuals with eating disorders has been associated with lower rates of recovery and higher rates of treatment drop out (Agras, Crow, Halmi, Mitchell, Wilson, & Kraemer, 2000). Negative affect and temperamental emotionality have been found to predict future onset of DEB (Stice & Agras, 1998; Stice, Pressnell, & Spangler, 2002) and increases in these symptoms (Cooley & Toray, 2001; Stice, 2001). It is hypothesized that negative affect is a risk factor for binge eating due to affect regulation functions. Studies have demonstrated that negative affect precedes binge eating events, and individuals cite that binge eating serves as a distraction from aversive emotional states (Heatherton & Baumeister, 1991; Stice et al., 2002). Therefore, binge eating is emotionally regulating because of negative reinforcement. During negative mood states, an individual engages in binge eating, resulting in decreases in negative mood, which is reinforcing because it serves to remove negative thoughts and emotions, and increases the likelihood of binge
eating again (Smyth et al., 2007). If weight and shape concerns persist, binge eating behaviors may then lead to efforts to compensate for binging and to prevent weight gain.

There is ample empirical support for risk factors for DEB delineated in Stice’s Dual Pathway Model. The model posits that individuals may engage in disordered eating behaviors because of dieting, negative affect, or an interaction of these two factors (Stice, 2001). However, these studies have all been conducted on populations without chronic illnesses, such as T1D. Prior to considering how these factors might interact with the unique experiences of youth with T1D, it is important to understand the course of diabetes and associated treatment regimen.

**Type 1 Diabetes Treatment Regimen**

Type 1 diabetes is an autoimmune disease caused in part by T-cell mediated destruction of pancreatic beta cells (Jahromi & Eisenbarth, 2007). The incidence of T1D has steadily increased over the last decade with a 3.2% increase in incidence since 1990 (The DiaMond Project Group, 2006). The epidemiology of T1D suggests that shifting gene by environment interactions are potentially triggering and/or accelerating the autoimmune destruction of the beta cells leading to complete insulin deficiency (Vehik & Dabelea, 2010). Long-term health complications associated with T1D include problems with eyesight including retinopathy, kidneys, peripheral and autonomic nervous systems, and increased risk for mortality (Nathan, 1993). Although the exact pathogenesis of these complications is not completely delineated, hyperglycemia (i.e., high blood sugar) plays a central role.

The goal of treatment for T1D is to maintain tight glycemic control and to avoid hypoglycemia (low blood sugar) and hyperglycemia (high blood sugar). Exogenous
insulin replacement is the foundation of treatment by which these goals are achieved (Silverstein, Klingensmith, Copeland, et al., 2005). The standard for measuring glycemic control is hemoglobin A1c, a form of hemoglobin that assesses average plasma glucose concentration over a 3 month period of time. The target HbA1c for youth between the ages of 8-18 is 7.5%-8.0% (Silverstein et al., 2005).

The T1D regimen requires patients to adhere to a complex treatment plan, including coordinating the amount and timing of insulin administration with results of frequent blood glucose monitoring (i.e., ideally 6-11 times per day), type and amount of dietary intake, and frequency and intensity of physical activity (ADA, 2011). Adherence to this regimen has been associated with better glycemic control and confers risk reduction for poor health outcomes associated with T1D including microvascular complications (DCCT, 1993; EDIC, 2003; Hood, Peterson, Rohan, & Drotar, 2009). However, nonadherence rates to the demands of the T1D regimen are high, and severe hypoglycemic events and poor glycemic control has consistently been reported in youth with T1D (DCCT, 1994; Morris et al., 1997; Wysocki, Greco, & Buckloh, 2003).

Specifically, adherence to the T1D regimen appears to decrease substantially during the early adolescent years (DCCT, 1994; Weissberg-Benchell, et al., 1995). Adequate adherence and metabolic control may become more difficult due to decreased insulin sensitivity (Moran, Jacobs, Steinberger, Hong, Prineas, Luepker, & Sinaiko, 1999). Other factors such as family conflict and decreased parental involvement in diabetes care may also contribute to the decreases in adherence and metabolic control during adolescence (Anderson, Vangsness, Connell, Butler, Goebel-Fabbri, & Laffel, 2002; Delamater, 2000). One study examined the most frequently cited reasons for
nonadherence in T1D and they included being too busy, skipped meals, stress/emotional problems, travelling, and public embarrassment (Peyrot, Barnett, Meneghini, & Schumm-Draeger, 2012).

There are significant consequences of nonadherence during this stage of development supported by the mounting evidence that the pre-pubertal and pubertal years have a significant impact on microvascular complications in diabetes (diabetic nephropathy, neuropathy, and retinopathy; DCCT, 1993; Kostraba et al., 1989). However, for adolescents who maintain strict glycemic control, there is still risk for hypoglycemic episodes and associated weight gain (Boland, Grey, Oesterle, Fredrickson, & Tamborlane, 1999; DCCT, 1993; DCCT, 1994; Domargard et al., 1999) which can prompt body image dissatisfaction and weight control behaviors (Bryden et al., 1999; DCCT, 1994). Therefore, adherence to the type 1 diabetes regimen could be compromised by efforts to avoid weight gain. Studies suggest that there is a higher prevalence of eating pathology in youth with T1D because of these factors.

**Prevalence of Eating Pathology in Youth with T1D**

Previous research has suggested that adolescent females with T1D are at an increased risk for DEB compared to their non-diabetic peers (Affenito et al., 2009; Alice Hsu, 2009; Bryden et al., 1999; Colton et al., 2004; Crow, Keel, & Kendall, 1998; Mannucci et al., 2005; Rodin et al., 2002; Ryan, Gallanagh, Livingstone, Gaillard, & Ritz, 2008). One study found that adolescents with T1D are twice as likely to meet DSM-IV diagnostic criteria for an eating disorder than age-matched adolescents without T1D (Jones et al., 2000). In another prospective study, researchers conducted a 5-year follow up study examining the prevalence of disordered eating behavior and diagnosable eating
disorders in youth with T1D. They found that out of their sample of 98 youths with T1D, 49% were engaging in disordered eating behavior including dietary restraint (43%), binge eating (6.1%), self-induced vomiting (3.1%), insulin omission (3.1%), and intense, excessive exercise for weight control (25.5%; Colton et al., 2007).

Although previous studies have found an increased prevalence of eating pathology in youth with T1D as compared to their non-diabetic peers, a recent review of the extant literature produced mixed results, indicating that prevalence rates are not known when compared to non-diabetic age and weight matched samples (Young-Hyman & Davis, 2010). This is consistent with a recent study which found that both male and female adolescents with type 1 diabetes were less likely to engage in disordered eating behaviors and reported having more body image satisfaction than their peers without diabetes (Ackard et al., 2008). One explanation for the discrepant findings in the literature is differences in assessment and conceptualization of eating pathology in the T1D population.

**Health Consequences of Co-Occurring T1D and DEB**

The health consequences of eating pathology in the context of T1D are significant. The Diabetes Complications and Control Trial (1993) found that disordered eating behavior at baseline was more highly predictive of retinopathy at the four-month follow up than duration of diabetes (DCCT, 1993). Many studies report that the presence of disordered eating pathology is associated with worsened glycemic control, increased risk for diabetes-related complications, (Jones, et al., 2000; Neilsen, 2002; Neumark-Sztainer et al., 2002; Rydall et al., 1997; Takii et al., 1999) and increased episodes of ketoacidosis (Polonsky et al., 1994). Two separate studies have documented that
individuals with T1D engaging in DEB have poorer glycemic control with hemoglobin A1c levels as much as 2% higher than non-eating disordered individuals with T1D (Colton et al., 2007; Jones et al., 2000). Many of these negative health outcomes appear to be related to insulin restriction and omission.

Insulin omission may serve as an inappropriate compensatory mechanism (Cantwell & Steel, 1996; Nielsen, 2002) in patients’ attempts to control weight gain and to regulate hunger. Rates of insulin restriction in individuals with T1D have been shown to be as high as 30% (Goebel-Fabbri, Fikkan, Franko, Pearson, Anderson, & Weinger, 2008). Severe, long-term insulin misuse appears to be the most closely associated eating disorder symptom with retinopathy and nephropathy (Takki et al., 2008). In one study following young women with T1D for 4 years, they found some level of retinopathy in 86% of participants with highly disordered eating, compared to 43% of the moderately disordered group and 24% of the non-disordered group (Rydall et al., 1997). In an 11-year follow up study of young women with T1D, the researchers noted that insulin restrictors (for weight loss purposes) had significantly higher HbA1c (9.6 vs. 8.3) than appropriate insulin users. Further, insulin restrictors at baseline were more likely to evidence nephropathy (25% vs. 10%), foot problems (25% vs. 12%), and had a 3.2 times higher relative risk for mortality over the course of the 11-year study (Goebel-Fabbri et al., 2008).

The insulin regimen aims to replicate the body’s natural insulin production, yet the process is not without flaws. If overinsulinization occurs, the effect is excessive hunger with the potential for dysregulated eating (Young Hyman & Davis, 2010). If under-insulinization occurs following food intake, the effect is purging of calories via
glucosuria (Polonsky et al., 1994). Therefore it is critical for practitioners to understand the physiology of insulin because it can be misused and affords a discrete method of purging. Improper dosing of insulin related to difficulties in treatment adherence and intentional misuse for weight loss purposes both lead to very similar negative health outcomes. It is important for providers to disentangle the reasons for insulin misuse as it could inform different interventions.

Given that a high percentage of youth with T1D endorse DEB symptoms, it is essential to identify risk for disordered eating in this population and to accurately distinguish disordered eating from non-pathological adjustment to the diabetes care regimen and regulation of glycemic control. In conjunction with traditional assessment measures of eating disorder symptoms, assessment of treatment regimen and adjustments to illness, hunger, and satiety can improve the accuracy at which disordered eating behavior is identified in individuals with T1D.

**Factors in T1D that Influence DEB**

**Disruptions in Hunger and Satiety**

Individuals with T1D suffer from disruptions in perceptions of hunger and satiety secondary to hormonal dysregulation and use of exogenous insulin (DeWitt & Hirsch, 2003). Specifically, with loss of beta cell function, the body ceases to produce insulin and amylin, which results in dysregulation of hunger and satiety mechanisms (Mack, Wilson, Athanacio, Reynolds, Laugero, Guss, Vu, Roth, & Parkes, 2007; Lutz, 2005). Further, non-physiologic exogenous dosing of insulin and attendant fluctuations in blood sugar can result in variations in hunger beyond that would be expected by food intake only (Engström, Kroon, Arvidsson, Segnestam, Snellman, & Aman, 1999). Thus,
dysregulated eating may be a result of excessive hunger related to insulin dosing, lack of satiety hormones, and fluctuations in blood glucose.

Satiety can be disrupted in youth with T1D as a consequence of the treatment regimen and the necessity for timed meals and subsequent dosing of insulin that are not necessarily based on actual hunger cues. Amylin mediates several satiety mechanisms via its effects on the area postrema, an area of the brainstem that integrates hormonal and metabolic signals to regulate food intake (Mack et al., 2007). Amylin facilitates an important role in the regulation of hunger and subsequent weight in concert with insulin, glucagon, leptin, and cholecystokinin (Lutz, 2005). Therefore, satiety can be disrupted in youth with T1D from both learning (i.e., eating in response to timing of meals as necessitated by the treatment regimen as opposed to hunger) and hormonal disruption associated with the disease which can contribute to binge eating. Additionally, hypoglycemia caused by low sugar in the blood can potentially promote binge eating as the body attempts to restore glucose balance. One potential difficulty in accurately assessing disordered eating behavior in youth with T1D is that assessment tools fail to consider these disruptions to hunger and satiety. Consequently, questions assessing DSM-IV-TR criteria for eating disorders such as “do you lose control over your eating” may have the tendency to overpathologize youth with T1D. Thus, it is important to simultaneously consider diabetes treatment factors that might overpathologize youth with T1D, and how other treatment related factors such as manipulation of insulin may be unique forms of disordered eating in this population.

Insulin treatment, attendant weight gain, and externally imposed food preoccupation are hypothesized mechanisms through which the treatment of T1D may
increase vulnerability to DEB. Treatment of T1D is complex and demands monitoring of one’s eating, blood glucose levels, activities and responsive insulin adjustments. Weight re-gain at onset of insulin treatment (which often accompanies achievement of good glycemic control) could play a role in the development of body dissatisfaction in adolescents with T1D (Olmstead, Colton, Daneman, Rydall, & Rodin, 2008). Insulin misuse could be a natural outcome of concerns about weight in the context of diabetes care. For example, based on blood sugar level, an adolescent with T1D is instructed to bolus insulin according to the combination of blood sugar level and “prescribed” breakfast carbohydrates. If the adolescent still feels hungry they might chose to bolus less insulin, thus reducing hunger. As insulin reduction or omission is a DSM-IV TR symptom of DEB, it is not surprising that manipulation of regimen based behaviors in this population can be mis-identified as pathology resembling an eating disorder (Crow et al, 1998). Thus, it is very important to understand the motivation behind misusing insulin.

The insulin pump is one method of insulin administration that could be manipulated. Short acting insulin is infused subcutaneously from a wearable pump at one or more basal rates, with extra dosing administered at mealtimes (Pickup et al., 1978). The insulin pump provides a readily available means of purging via insulin omission that also affords the individual discretion. However, it is important to differentiate insulin misuse as a form of purging related to body dissatisfaction, from under-dosing or over-dosing insulin due to feelings of hunger associated with loss of amylin, disruptions in normal perceptions of hunger and satiety secondary to exogenous insulin use, or a desire not to gain weight secondary to treatment. Current models of risk for eating pathology in
youth with T1D do not fully capture psychosocial variables and these important diseased related factors.

**Potential for Misdiagnosis of DEB in Youth with T1D Due to Physiological and Disease Related Factors**

While the co-occurrence of DEB and T1D appears elevated, recent reviews raise questions regarding the reasons for this co-occurrence (Criego, Crow, Goebel-Fabbri, Kendall, & Parkin, 2009; Young-Hyman & Davis, 2010). For example, traditional self-report measures of eating disorder symptoms such as the Eating Disorder Inventory-III and the Eating Disorder Examination Questionnaire were all normed on non-diabetic samples (Garner, 2004; Fairburn & Beglin, 1994). Measures of DEB validated on non-diabetic populations may not yield accurate assessments of eating pathology in youth with T1D. For individuals with T1D, endorsement of symptoms such as food preoccupation and dietary restriction are embedded within the diabetes treatment regimen (Criego et al., 2009). These behaviors may not be indicative of disordered eating behavior, which is thought to be motivated by internalization of distorted thoughts about the importance of weight and shape (Striegel-Moore & Bulik, 2007).

Additionally, questionnaires which assess insulin omission in the context of eating pathology do not assess motivations for this omission (e.g., to avoid hypoglycemia, forgetting, non-adherence). Several studies have indicated that insulin restriction is associated with multiple factors such as non-adherence, embarrassment, and interference with daily activities, as well as depression (Ciechanowski, Katon, Russo, & Hirsch, 2003; Gonzalez et al., 2007; Peyrot, Rubin, Kruger, & Travis, 2010). Methodologies must be tailored to assess motivations for insulin misuse.
Integrating Psychosocial Risk Theories and Disease Models to Explain DEB in the T1D Population

There is very little integration of psychosocial risk theories (traditionally tested in non-diabetic populations) and disease based models explaining risk for eating pathology in the T1D population. While there are several well-developed models of the etiology of DEB in the non-diabetic population, the application of these models to youth with T1D may be problematic. Disease-related variables, which influence hunger and eating behavior, are not accounted for. The Dual Pathway Model for the development of bulimic symptoms incorporates both negative affect and dietary restraint in the etiology of DEB in adolescent youth (Stice, 1998) and has the potential to guide hypotheses regarding the development of DEB in the T1D population. Integration of these two concepts (a model of pathologic behavior and a disease model) is important in youth with T1D to achieve a comprehensive conceptualization for DEB risk. Importantly, the integration of disease-related variables into a model to correctly identify eating pathology is an important step towards better understanding risk for DEB in diabetic youth. Development of an integrated model can be informative for other populations at high psychosocial and physiological risk for the development of DEB (e.g., Cystic Fibrosis).

Applying the Dual Pathway Model to T1D

Although the Dual Pathway Model of risk seems particularly salient for youth with T1D, Stice’s original model does not account for hunger and satiety disruptions, or other disease-related factors. It seems that concurrently having diabetes and engaging in treatment potentiates risk for bulimia above and beyond risk that an individual without diabetes might experience. The issue is not simply the presence of two co-morbid conditions, but rather that the intersection of both bulimic symptoms and T1D is a
product of a variety of factors that are not sufficiently captured by traditional eating disorder risk models. Thus, the proposed model of risk for DEB integrates treatment, psychological, and physiological variables.

**Initiation of Insulin, Weight Gain, and Body Dissatisfaction**

Several aspects of diabetes treatment need to be integrated into the Dual Pathway Model. Specifically, initiation of insulin treatment for T1D can exacerbate weight gain in children and adolescents (see Figure 2; Russell-Jones & Khan, 2007) which may increase their vulnerability for body image dissatisfaction. Additionally, overweight status appears to be a risk factor for the development of eating pathology in youth with T1D (Engstrom et al., 1999; Herpertz et al., 2001; Peveler et al., 1992). Despite evidence that both girls and boys experience rebound adiposity during insulin initiation (e.g., Boland et al., 1999) adolescent girls appear to have more Leptin resistance which may explain why girls with T1D gain substantially more weight during puberty than their male counterparts (Ahmed, Ong, Preece, & Dunger, 1999). It follows then that adolescent girls with T1D may be vulnerable to weight gain related to physiological sequelae associated with the disease, which may promote body image dissatisfaction. Further, adolescents with T1D are simultaneously managing the physical changes that their bodies incur as a result of pubertal development, a process that can also increase body fat and promote subsequent body image dissatisfaction (Tremblay & Lariviere, 2009; Verrotti et al., 1999). Given that these adolescents are undergoing pubertal development concurrently, this process of weight loss and weight gain upon insulin treatment can exacerbate body image dissatisfaction. Thus, this integrated model suggests that rebound weight gain secondary to treatment initiation increases risk for DEB via body image dissatisfaction.
**Dietary Regimen and Dietary Restraint**

Youth with T1D are charged with intensive dietary management requiring monitoring of carbohydrate intake and matching carbohydrate intake to insulin levels (Bantle et al., 2008). Adherence to carbohydrate recommendations is associated with good glycemic control while non-adherence is related to hyper and hypoglycemic events (Mehta et al., 2008; Patton et al., 2007). Additionally, it is recommended that youth with T1D adhere to a “healthy diet” and incorporate fruits, vegetables, and whole grains while limiting high carbohydrate, high fat foods (ADA, 2003). The Dual Pathway Model proposes that body dissatisfaction as a result of thin ideal internalization during pubertal development then leads to dieting behavior (Stice et al., 1998). One modification of the proposed model is that both weight gain and externally imposed changes in eating behaviors, which may have similarities to self-imposed diets in a non-diabetic population, occur at the same time.

As described previously, dietary restraint can lead to disordered eating (in non-diabetic populations) in several ways. It may increase risk for binge eating, or it may increase risk for excessive restriction of eating through positive reinforcement of weight loss (Stice, 2002). The proposed model suggests that the body dissatisfaction associated with treatment-related weight gain increases risk for excessive restriction, particularly because these adolescents have externally imposed food preoccupation from their treatment regimen. Evidence supporting this hypothesis includes cross sectional and longitudinal studies that have demonstrated associations between weight gain, body image dissatisfaction, and food preoccupation in youth with T1D (Bryden et al., 1999; Peveler et al., 1992).
Further, it is unknown if failing to adhere to one’s diet when managing T1D represents a similar risk for abstinence violation driven binge eating, as breaking dietary rules does in non-diabetic populations (Herman & Polivy, 1980). Alternatively, cognitive and affective consequences of failure to adhere to diet regimens may promote binge eating via affect regulation mechanisms. For example, an adolescent may experience feelings of guilt and frustration regarding failure to adhere to treatment guidelines, which may promote emotionally driven eating. In this case, the adolescent may not have been attempting to manage diet for weight loss purposes, but rather, may be experiencing difficulty with adherence (Young-Hyman & Davis, 2010). Further, loss of control over eating in youth with T1D may be due to factors beyond negative affect and dietary restriction.

**Physiological and Psychological Pathways to Binge Eating**

One disordered eating behavior in T1D youth may be increased restriction of food intake, driven by body dissatisfaction, and masked by externally imposed dietary regimens. Another may be insulin restriction, again influenced by body dissatisfaction. Binge eating behavior is another disordered eating behavior that may occur via unique pathways in T1D youth. Insulin treatment is associated with weight gain and subsequently, higher doses of insulin are required to control blood glucose which can lead to excessive hunger, hypoglycemia, and increased caloric intake (Rezek, 1976). It is postulated that episodes of overeating in response to excessive insulin and hypoglycemia are related to disruptions to appetite regulation in the hypothalamus (Engstrom et al., 1999). In short, overinsulinization leads to excessive hunger (Larger, 2005), which could potentially drive episodes of loss-of-control over eating. The proposed model suggests
that treatment imposed dietary regimen enhances the risk for binge eating through AVE mechanisms, affect regulation mechanisms, and disruptions in hunger and satiety caused by overinsulinization.

**The Unique Role of Depression in DEB Risk in Youth with T1D**

Dietary restraint can promote depressive symptoms due to potential failed efforts at dieting (Stice et al., 1998; Stice, 2000). This is especially salient in youth with T1D who already have increased rates of depressive symptoms, and whom are overweight (Kanner, Hamrin, & Grey, 2003). The proposed model suggests that these factors interact to produce eating pathology in the following ways: For youth who have depressive symptoms prior to onset of T1D, the effects of receiving the diagnosis, demands of managing the treatment regimen, and weight gain associated with insulin therapy may exacerbate depression even more. Furthermore, for youth with pre-existing body image concerns, the effects of weight gain could worsen body image, which can increase negative affect. In an integrated model, it is proposed that negative affect, or exacerbation of pre-morbid depression related to weight and shape concerns, and the effects of a chronic illness diagnosis may increase risk for binge eating via negative reinforcement or increased risk for manipulation of insulin/dietary regimen for weight loss.

**The Current Study**

While it is clear that the co-occurrence of eating disorders and T1D is potentially life-threatening and can lead to long term complications, it is less clear how many individuals with T1D actually have eating disorders once regimen and physiologically driven eating behaviors are taken into account (Young-Hyman & Davis, 2010), and what
the prevalence is of diabetes specific DEB. This is especially true in an adolescent population in which weight concerns, dieting, and the development of disordered eating behavior are more prevalent (Daneman, Olmsted, Rydall, et al., 1998) and for whom control of diabetes and weight becomes more difficult. Furthermore, these models may need to be adjusted to consider the interaction of disease-related processes with psychosocial processes impacting the development of DEB. Thus, the current study will begin to address several gaps in the literature regarding risk for DEB in youth with T1D by testing an integrated psychosocial and disease based model of risk in the prediction of DEB in a T1D sample. Validation of risk models for eating pathology in these chronic illness groups can then inform important interventions.

**Statistical Approach**

All analyses were conducted in the context of a larger prospective study of the relationships between implementation of diabetes treatment, (insulin dose and dosing methods and monitoring of blood glucose) glycemic control and weight concerns. The study sample is a cohort of newly diagnosed patients and patients transitioning to use of the insulin pump, between 10 and 17 years of age. The larger study examines multiple outcomes in newly diagnosed (New) and pump transitioning (Pump) children and adolescents over the course of one year. However, the current study will analyze data obtained from baseline assessments only, focusing specifically on bulimic behaviors.

The purpose of the current study is to compare two different models for DEB risk in T1D youth. First, we examined the relationships between disordered eating behavior and the Dual Pathway Model; (body dissatisfaction, dietary restraint, and depression) in the entire sample. Second, we examined the amount of variance accounted for in DEB by
variables delineated by our proposed model integrating disease related variables with psychosocial risk; (pump vs. new diagnosis treatment regimen, dietary restraint, body dissatisfaction, depression, hunger and satiety indicators, and HbA1C measurements). The rationale for having two groups: newly diagnosed and pump transitioning is to isolate factors specifically associated with the T1D disease processes and treatment regimen. The newly diagnosed group is assessed within 10 days of diagnosis, capturing behavior associated with disease onset and before significant experience with management of the regimen. The pump transitioning group consists of youth who have been living with T1D and the insulin regimen, before actual use of the insulin pump. Thus, comparison of these groups allows for examination of risk for bulimic symptoms at different time points in disease course and with differences in experience with disease management. It is hypothesized that a model that integrates disease-related variables with psychosocial risk will account for the most variance in the development of DEB in youth with T1D.

**Hypotheses**

1. At baseline, body dissatisfaction, dietary restraint and depression will be significantly positively associated with DEB in the entire sample and disruption in perceived hunger and satiety in the pump group will be positively associated with DEB.

2. Newly diagnosed youth will report significantly less body dissatisfaction than youth in the pump-transitioning group since they were just diagnosed, had not yet begun insulin treatment, nor experienced consequent weight rebound.
3. Group differences in depression will be examined. A priori differences between groups in endorsement of depressive symptoms or clinically significant depression was not hypothesized because previous findings have demonstrated higher rates of depression in youth with T1D. However, the newly diagnosed group may also have elevated levels of depressive symptoms given the effects of recently being diagnosed with a chronic illness.

4. Pump transitioning youth will have increased dietary restriction compared to newly diagnosed youth as measured by the EDI, above and beyond treatment regimen. This is consistent with predictions from the modified model, in which weight gain associated with insulin treatment is related to body image dissatisfaction and subsequent dietary restraint in order to lose weight.

5. Pump transitioning youth will exhibit higher levels of binge eating due to hypothesized mechanisms of action explained in the modified Dual Pathway Model including increased risk for weight gain, dietary regimen, body image dissatisfaction, dietary restraint, and negative affect.

6. When controlling for body dissatisfaction, dietary restraint, and depression, glycemic control, and uncontrollable hunger secondary to insulin treatment and blood glucose will account for a statistically significant variance in DEB, consistent with predictions from the proposed model.

Tests of Exploratory Aims

As female youth are typically at higher risk for DEB, but both male and female youth report DEB in diabetic samples, the potential moderating effects of gender were examined. Additionally, as youth being treated with insulin pump have been living with
T1D for longer periods of time and are purported to have higher rates of depressive symptoms, it was hypothesized that the relationship of depressive symptoms to DEB may be different in this group than those newly diagnosed. This interaction effect was examined in the combined sample.
CHAPTER 2

METHOD

Participants

One hundred and one eligible children/adolescents were recruited who had either recently received an initial diagnosis of T1D or were transitioning to an insulin pump regimen were recruited. There were 58 participants who were newly diagnosed with T1D and 43 participants transitioning to an insulin pump with an average length of illness of 4.3 years (sd=3.5 years). 75.2% of the sample is Caucasian, 21.8% African-American, and 3% identified as another ethnicity. All 101 (54.4% female) eligible participants were between the ages of 10 and 17 (mean age 12.8 years +/- 2.1) and had a diagnosis of diabetes mellitus. The participation of an eligible caregiver (person with whom the child/adolescent spends at least 50% of their time with and is responsible for overseeing diabetes management) was also required. Exclusionary criteria included having a co-occurring chronic illness that would affect weight, (e.g. gastrointestinal or adrenal disorder) a severe psychiatric illness that would significantly affect the individual’s ability to follow the diabetes treatment regimen, a disorder that affects metabolism, and if female, pregnancy.

Power Analysis.

A power analysis, given the number of predictors and anticipated effect size, indicated that the total sample size need to detect significant variance in DEB with 80% power was 81 participants. The sample size of 101 participants was thus adequate.
Procedure

Endocrinologists referred study participants at three different sites: Georgia Regents University (GRU) in Augusta, GA, Emory University in Atlanta, GA, and Joslin Diabetes Center in Boston, MA. These three sites were selected to ensure three distinct geographic sites would increase the diversity of the sample in terms of practice patterns, variation in regiment prescription, and culture regarding eating, physical activity, body size, and weight. Once eligible participants were identified, they were referred by physicians and staff members from the medical team to a research assistant from the study. Of the total sample, 40 participants were recruited from GRU, 31 from Emory, and 30 from Joslin.

The parent study consisted of four assessments over 12 months. The baseline assessment was conducted with the child and primary caregiver either during diabetes education (during a home visit, in a hospital, or clinic) shortly after diagnosis or when the child/family was being educated prior to initiation of insulin pump therapy. Patients transitioning to pump were approached for recruitment after the pump was approved by insurance, but prior to actual insulin administration utilizing the pump. Assessments occurred at 1, 6, and 12 months following baseline for the adolescents and at baseline, 6, and 12 months for the primary caregivers. All subjects, both adolescents and caregivers, were compensated with a $25 gift card for each assessment completed. For completion of all assessments over the course of the year, a family could receive up to $175. The current study utilizes data from assessments conducted at baseline.
Measures

Demographic Information Form

Parents completed a Demographic Information Form at the initial visit to obtain date of birth, gender, parent’s age, parent occupation, and composition of the family. Information about the child’s medical history was collected from the parent.

Eating Disorder Inventory-III (EDI-III)

The EDI is a widely used self-report measure of eating disorder symptoms and consists of 8 standardized subscales representing dimensions that are clinically relevant to eating disorders including the bulimia and body image dissatisfaction subscales. Acceptable convergent validity for the EDI-III with other valid and reliable measures of eating pathology has been established in prior studies (Cumella, 2006). Internal consistency reliability estimates for the 18 clinical and composite scales were reported separately for the U.S. adult, international adult, and U.S. adolescent samples and with rare exception, ranged above .80 (Garner et al., 1983). Internal consistency was acceptable for the bulimia subscale ($\sigma=.83$) and body image dissatisfaction subscale ($\sigma=.80$) in the current sample.

A1c

Hemoglobin A1c laboratory assay values were obtained from study participant charts. Methods for obtaining these assays were consistent with standards established by DCCT (Little, 2003).

BMI

Height and weight were measured at the baseline assessment, or obtained from the participant’s chart review. Three measurements of height and weight were obtained,
and then averaged if conducted by a research assistant. Sex and age adjusted BMI (BMI z-score standard deviation [z-BMI] based on the Center for Disease Control [CDC] growth charts) was calculated (Kuczmarski & Flegal, 2000).

**Duration of Diabetes**

Duration (or time since diagnosis) of diabetes may affect eating disorder symptoms, and was obtained from chart review for each study participant.

**Dietary Restraint (Youth Risk Behavior Survey [YRBS], CDC, 2009)**

The YRBS is a cross-sectional school-based survey conducted biennially by the Center for Disease Control and Prevention to youth behaviors that influence health. For the current study, an item from this survey was utilized to assess for dietary restraint; “During the past 30 days, did you eat less food, fewer calories, or foods low in fat to lose weight or to keep from gaining weight?”

**The Children’s Depression Inventory (CDI; Kovacs, 1992)**

The CDI is used to assess cognitive, affective, and behavioral signs of depression in children and adolescents age 6-17 years old. The CDI self-report measure consists of 27 items and for the purposes of this study, a total score was utilized. A raw score of 13 or higher is indicative of clinical levels of depressive symptoms (Kovacs, 1992). Internal consistency for the CDI ranges from .71 to .89 and the test retest coefficients range from .74-.83. In the current study, reliability for the CDI was adequate, σ=.80.

**The Diabetes Treatment and Satiety Scale (DTSS) (Young-Hyman; manuscript under review).**

The DTSS is used to assess hunger, satiety, and fullness in the context of food intake, insulin regimen, and blood sugar. The six subscales include Uncontrollable
Hunger, Remain Hungry, Low Blood Sugar, Feeling Full, Satiety, and High Blood Sugar. Alphas for the six subscales are as follows: Uncontrollable Hunger ($\alpha=.76$), Remain Hungry ($\alpha=.75$), Low Blood Sugar ($\alpha=.78$), Feeling Full ($\alpha=.78$), Satiety ($\alpha=.43$) and High Blood Sugar ($\alpha=.47$). The DTSS will be used to assess eating behaviors that are associated with disease variables. In the current study, the Uncontrollable Hunger subscale was utilized, as it was predicted that this subscale would be associated with endorsement of DEB.
CHAPTER 3
RESULTS

Descriptive Statistics

All analyses were conducted using IBM SPSS Statistics 21, Release Version 21.0 (Armonk, NY: IBM Corp). Consistent with recommendations put forth by Hawthorne and Elliot (2005), missing data were replaced using the mean of the items at the level of the individual. Descriptive statistics for the entire sample as well as each group (pump transitioning and newly diagnosed) can be found in Table 1. Independent samples t-tests were conducted to determine if there were significant differences between sites where study participants were recruited. Study participants from Georgia Regents University (GRU) had significantly higher z-BMI scores, A1c, and endorsed higher levels of bulimic symptoms ($t = 2.76, p < .01$) compared to study participants recruited from Joslin Diabetes Center (Joslin). There were no significant differences between Joslin, GRU, or Emory on depression, body image dissatisfaction, or dietary restraint. Participants from GRU reported higher scores on DTSS uncontrollable hunger as compared to those study participants from Joslin ($t = 2.31, p < .05$). Additionally, participants from Emory reported higher DTSS uncontrollable hunger as compared to Joslin study participants ($t = 2.33, p < .05$). Given these significant differences, site was controlled for in all regression models.

Comparison of mean group differences revealed that the pump group had significantly higher mean z-BMI scores ($t = 3.12, p < .05$). Further, there were
significantly more white youths in the pump transitioning group \((X^2 (100) = 9.59, p < .01\) (51.3\% vs. 16.0\%) as compared to the newly diagnosed group. Given these differences in the pump transitioning group versus the newly diagnosed group, both race and z-BMI score were statistically controlled for in all regression models.

In addition to group (pump vs. new) differences, several significant sex differences emerged, thus sex was controlled for in the first step of each regression model. In the combined group (pump and newly diagnosed), female participants had higher A1c values (10.6\% vs 9.1\%, \(t = -3.03, p < .01\)). Females also had higher A1c values in the newly diagnosed group (12.13\% vs 10.41\%, \(t = -2.75, p < .01\)). Females in the pump transitioning group had significantly higher scores on body image dissatisfaction \((t = -2.19, p < .05)\). There were no significant sex differences within the pump groups in terms of age, A1c, z-BMI percentile, depressive symptoms, dietary restraint, DTSS, or bulimic symptoms.

Bivariate correlations between major variables of interest in the combined group, newly diagnosed group, and the pump transitioning group were conducted to test hypothesis 1, and are presented in Tables 2, 3, and 4, respectively. For the combined sample, there were significant positive associations between depressive symptoms, bulimic symptoms, and body image dissatisfaction. Dietary restraint was significantly positively associated with body image dissatisfaction and z-BMI percentile only. For the combined sample, A1c was negatively associated with z-BMI percentile and length of illness. Finally, bulimic symptoms were correlated with age, indicating that older study participants endorsed higher levels of bulimic symptoms.
Bivariate correlations in the pump group revealed that of the DTSS subscales, only the DTSS Uncontrollable Hunger subscale was found to be significantly associated with bulimic symptoms. The pump group was the only group administered the DTSS since they were the only participants living with the treatment regimen. Additionally, in the pump group, higher z-BMI score, body image dissatisfaction, and depressive symptoms were all associated with higher bulimic symptoms. A1c was significantly associated with higher body image dissatisfaction, and with higher scores on DTSS Remain Hungry (remaining hungry after eating) but not bulimic symptoms. Finally, body image dissatisfaction was significantly associated with depressive symptoms in the pump group.

In the newly diagnosed group, higher age, depressive symptoms, and body image dissatisfaction were all significantly associated with bulimic symptoms. Further, dieting was associated with higher z-BMI scores, and body image dissatisfaction and depressive symptoms were positively significantly associated.

To examine hypotheses 2-5 (group differences between body image dissatisfaction, depressive symptoms, dietary restraint, and binge eating), independent samples t-tests were conducted. Results indicated that the newly diagnosed youth did not differ significantly from the pump transitioning youth on dietary restraint, depressive symptoms, body image dissatisfaction, or bulimic symptoms. There were also no significant group differences between binge eating, although this finding approached significance with the pump group endorsing higher levels of binge eating ($t = -1.77, p = .08$). Of note, mean scores on the CDI, body image dissatisfaction, and bulimia were not in the clinical range for the combined sample. On the CDI, 12% (raw scores $\geq 12$) of the
sample reached scores in the clinical range, 3% (raw scores ≥ 22) of the sample reached the clinical range on body image dissatisfaction, and 15% (raw scores ≥ 5) of the sample reached the clinical range on the bulimia subscale.

**Multiple Regression Analyses**

**Combined sample**

Hierarchical multiple regression was used to test the contributions of disease management experience (New vs. Pump, and length of illness), and the Dual Pathway Model (i.e., body image dissatisfaction, depressive symptoms, dietary restraint) to symptoms of bulimia in the combined sample (see Table 5). Participant sex, race, age, z-BMI score, A1c, length of illness, and site were entered in the first step. Given the relationship of A1c to other predictor variables, it was important to include it in all models, thus participants without A1c values were excluded for regression analyses, as this data cannot be imputed. Group membership, depressive symptoms, dietary restraint, and body image dissatisfaction were entered in the second step. Two-way interactions (group X depressive symptoms, group X body image dissatisfaction, and depressive symptoms X body image dissatisfaction) were entered in the third step of the model. In order to reduce multicollinearity impacting interaction terms, both body image dissatisfaction scores and depression scores were centered (Aiken & West, 1991).

In the combined group model using all youth with A1c data (total $R^2 = 65\%, p > .01$) an examination of the beta weights revealed that z-BMI score ($\beta = -.20, p < .05$), depression scores ($\beta = .49, p < .01$), body image dissatisfaction ($\beta = .31, p < .01$), and in the final step of the model, the two way interaction: Depression X body image dissatisfaction ($\beta = .40, p < .01$) made significant contributions to variance in bulimia
scores. There were no main effects for group or dietary restraint. Further, the two-way interactions between group X body image, and group X depression were both non-significant. Consistent with the Dual Pathway Model (Stice, 2001), the significant two-way interaction indicated that a combination of high body image dissatisfaction and high depressive symptoms accounted for unique variance in bulimic symptoms.

**Newly diagnosed group**

Hierarchical linear regression analyses were conducted to test variables consistent with the Dual Pathway Model in the newly diagnosed group (see Table 6). Participant sex, race, age, z-BMI percentile, A1c, length of illness, and site were entered in the first step. In the second step, depressive symptoms, dietary restraint, and body image dissatisfaction were entered. Finally, a two-way interaction between depressive symptoms and body image dissatisfaction was entered in the third step.

Results revealed that there were significant main effects for z-BMI score ($\beta = - .42, p < .01$), depressive symptoms ($\beta = .54, p < .01$), and body image dissatisfaction ($\beta = .36, p < .01$) in the second step of the model. There were no significant main effects for dietary restraint. In the third step of the model, a significant two-way interaction ($\beta = .34, p < .05$) indicated that a combination of both high depressive symptoms and high body image dissatisfaction resulted in the most bulimic symptoms, consistent with the Dual Pathway Model.

**Pump transitioning group**

Hierarchical multiple regression was utilized to test the effects of disruptions in hunger and satiety on bulimic symptoms when controlling for variables drawn from the Dual Pathway Model (i.e., body image dissatisfaction, dietary restraint, and depression;
See Table 7). Participant sex, race, age, z-BMI score, A1c, length of illness, and site were entered in the first step. In the second step, depressive symptoms, dietary restraint, body image dissatisfaction, and DTSS uncontrollable hunger were entered. Finally, all two-way interactions were entered in the third step including between depressive symptoms X DTSS uncontrollable hunger, depressive symptoms X body image dissatisfaction, and DTSS uncontrollable hunger X body image dissatisfaction.

In the model using all youth with A1c data (total $R^2 = 79\%, \ p >.01$) an examination of the beta weights revealed that in the second step of the model there were main effects for site, ($\beta = -.27, \ p < .05$) depression, ($\beta = .36, \ p <.05$) and DTSS uncontrollable hunger ($\beta = .27, \ p <.05$). There were no main effects of z-BMI score, body image dissatisfaction, dietary restraint, length of illness, or A1c. However, in the third step of the model there was a significant two way interaction: depression X DTSS uncontrollable hunger ($\beta = .42, \ p <.01$) that made significant contributions to variance in bulimia scores. A probing of the interaction revealed that a combination of high depression scores and high DTSS uncontrollable hunger scores resulted in the most bulimic symptoms. This finding was consistent with hypothesis 6, in that DTSS uncontrollable hunger was significantly associated with endorsement of bulimic symptoms even when controlling for body image dissatisfaction, depression, dietary restraint, and weight. This finding lends support for the Modified Dual Pathway model in that depressive symptoms and hunger dysregulation both contribute to DEB.
CHAPTER 4
DISCUSSION

Previous research on youth with T1D suggests that this population is at a higher risk for disordered eating behavior than their non-diabetic peers (e.g., Affenito et al., 2009; Alice Hsu, 2009). However, upon closer examination, the prevalence of DEB in youth with T1D seems variable and highly dependent upon type of measurement used to assess DEB (Young, Eiser, Johnson, Brierley, Epton, Elliott, & Heller, 2012). One explanation for variability in the prevalence of DEB in youth with T1D is that traditional measures of eating pathology do not account for factors unique to the T1D disease sequelae and treatment regimen (Young-Hyman & Davis, 2010). Indeed, in a recent study, the EDE-Q and EDI-III were administered to youth with T1D and DEB and age-matched peers with DEB and no T1D (Powers, Richter, Ackard, Critchley, Meier, & Criego, 2013). The results indicated that items on the EDE-Q and (to a lesser extent) EDI-III could be influence by having T1D. Taken together, the current state of the literature on DEB in youth with T1D suggests that it is inaccurate to conceptualize risk for DEB in this population utilizing traditional risk factors for eating disorders alone. Doing so without accounting for disease and treatment related factors has the potential to pathologize behaviors which may actually be normative, adaptive responses to living with T1D. Further, inaccurate conceptualization of DEB in this population could misinform treatment efforts.
Youth with T1D have to calculate the amount of carbohydrates they consume at meals and snacks, and check blood glucose levels to support decisions about insulin dosing. This necessary focus on food intake to manage T1D could be interpreted as maladaptive food preoccupation in traditional measures of eating pathology. Further, dosing of exogenous insulin can lead to excessive hunger, which may contribute to dysregulated eating, particularly in the case of overinsulinization (Engstrom et al., 1999). Insulin omission may be in response to excessive hunger, nonadherence, or for purposes of intentional weight manipulation. It is critical that motivation behind insulin omission is determined, as this would differentially guide assessment and treatment intervention (Goebel-Fabbri, 2009). These aspects of the disease course and treatment regimen may increase risk for DEB. Thus, to accurately assess the pathways by which pathological eating symptoms occur in youth with T1D, it is important to both elucidate traditional eating disorder risk factors, as well account for hunger and satiety disruption as proposed by the Modified Dual Pathway Model.

The current study examined traditional eating disorder risk factors delineated by the Dual Pathway Model (i.e., body image dissatisfaction, dietary restraint, and depression; Stice, 2001) in conjunction with disease related factors (e.g., A1c, length of illness) and disruptions in hunger and satiety that may also contribute to risk for DEB, which are incorporated in the Modified Dual Pathway Model. The current study consisted of youth who were newly diagnosed with T1D and were naïve to the treatment regimen, as well as youth transitioning to an insulin pump who had experience living with the disease and treatment regimen. It was predicted that the Modified Dual Pathway Model would be represented in the pump transitioning group and that traditional risk factors
representing the Dual Pathway Model would be significantly associated with DEB in the newly diagnosed group. Group differences related to disease process and difficulty controlling glycemia and weight were accounted for by controlling for length of illness in all analyses.

In the combined sample, results indicated that lower z-BMI percentile, body image dissatisfaction, and depressive symptoms all contributed to unique variance in bulimic symptoms. These results are consistent with the Dual Pathway Model (Stice, 2001). However, the direction of the effect for z-BMI score was unexpected in that lower z-BMI scores accounted for significant unique variance in bulimic symptoms. This effect for z-BMI may be due to differences in weight status between the groups (pump vs. newly-diagnosed). Consistently, the newly-diagnosed group had significantly lower z-BMI scores as compared to the pump transitioning group, most likely due to weight loss from the disease onset. In the combined group, there was a main effect for zBMI score in that lower zBMI scores accounted for unique variance in bulimic symptoms. Interestingly, when groups were examined separately, there was a similar effect of z-BMI score for the newly diagnosed group but no effects for z-BMI in the pump transitioning group.

Further, a significant two-way interaction revealed that a combination of high body image dissatisfaction and high depressive symptoms accounted for the most bulimic symptoms. There were no significant effects for dietary restraint. Previous studies have shown that youth with T1D and co-occurring DEB generally report lower dietary restraint than individuals with DEB and without T1D (e.g., Powers et al., 2013). It has been theorized that one reason for this is due to the focus on regularity of meal times as
prescribed by the T1D treatment regimen. However, given that the combined sample also included youth who are newly diagnosed and not living with the treatment regimen, it was expected that there would be a significant effect for dietary restraint. One plausible explanation for this factor not accounting for bulimic symptoms could be that dietary restraint was assessed using a single item. When the Dual Pathway Model was examined in the newly diagnosed group only, similar factors emerged as significant predictors of bulimic symptoms: a combination of higher body image dissatisfaction as well as lower z-BMI scores.

It was expected that treatment and disease related factors would be associated with DEB in the pump-transitioning group even when accounting for traditional eating disorder risk factors delineated by the Dual Pathway Model. Indeed, these additional factors proposed by the Modified Dual Pathway Model were found to be associated with bulimic symptoms in a subset of youth who have had experience living with the disease and treatment related factors (i.e., pump group). In the pump group, when controlling for traditional eating disorder risk factors (dietary restraint, body image dissatisfaction, and weight), depression and uncontrollable hunger related to insulin treatment and fluctuations in blood glucose levels accounted for significant unique variance in bulimic symptoms. This is a salient finding because it underscores the importance of accounting for disruptions to hunger and satiety due to T1D treatment and disease processes when assessing eating pathology in youth with T1D. It is also notable that there were no significant effects for body image dissatisfaction, dietary restraint, or weight. This finding is consistent with previous studies which suggest that youth with T1D and co-occurring DEB present with less weight concerns and dietary restraint as compared to
aged-matched peers with DEB (e.g., Powers, Richter, Ackard, Gerken, Meier, & Criego, 2012). This also suggests that for youth living with the disease and treatment regimen, experiences of hunger dysregulation related to hormonal deficits (e.g., amylin), over-insulinization, and blood glucose fluctuations combined with depressive symptoms were most predictive of bulimic symptoms, above and beyond dietary restraint, body image dissatisfaction, and weight status. Traditional models of eating pathology do not account for hunger and satiety disruptions and, therefore, may attribute more psychopathological causes to higher endorsement of DEB (Young-Hyman & Davis, 2010).

As there have been inconsistencies in findings related to sex differences in risk for DEB in youth with T1D, (Meltzer, Johnson, Prine, Banks, Desrosiers, & Silverstein, 2001; Neumark-Sztainer et al., 2002) analyses of potential sex differences were explored. There were some sex differences in this sample, including adolescent girls having higher A1c in the combined and newly diagnosed groups, and higher body image dissatisfaction in the pump transitioning group. Interestingly, there were significant group differences between pump vs. newly diagnosed on z-BMI, but no group differences on body image dissatisfaction. One reason for this may be that there were significant sex differences for body image dissatisfaction, with adolescent girls being higher on this factor. Thus, sex differences on body image dissatisfaction may have decreased the overall effects of body image dissatisfaction in relationship to DEB in this combined sex sample. Of note, there were no sex differences in bulimic symptoms, depression, dietary restraint, or dysregulated hunger in either group suggesting similar levels of symptoms across sexes. This is consistent with previous findings, suggesting that adolescent boys with T1D have twice as many body image concerns and are more likely to vomit for weight control.
purposes than their age-matched male peers without a chronic illness (Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995). Therefore, it is not surprising since adolescent boys seem to be at higher risk for DEB than their age matched peers that they would have comparable levels of DEB symptoms as compared to adolescent girls with T1D. In the current study, there were no significant sex differences with regards to DEB, and sex was not a significant predictor of DEB in regression models, suggesting that for these youth with T1D, adolescent girls and boys are similarly affected by DEB.

It is postulated that youth with T1D are at a higher risk for a multitude of psychological disorders including depression and eating pathology (e.g., Mannucci et al., 2005, Kanner et al., 2003). However, in the current study, when group comparisons were examined, the pump group did not have significantly different scores on depressive symptoms, body image dissatisfaction, bulimic symptoms, binge eating, purging, or dietary restraint as compared to newly diagnosed youth. In other words, adolescents with treatment experience did not endorse more clinical symptoms when compared to newly diagnosed youth. It is important to note those newly diagnosed may represent a unique subset of youth who report slightly higher acute distress due to recently being diagnosed with a chronic illness. However, few individuals across this combined sample scored in the clinical range for depressive symptoms (12%), bulimic symptoms (15%), or body image dissatisfaction (3%). These rates are similar to prevalence of these disorders in the non-diabetic adolescent population (Merikangas, He, Burstein, Swanson, Avenevoli et al., 2010). Relatedly, a recent study compared youth with T1D and co-occurring DEB with age-matched peers with DEB and without T1D. They concluded that their sample of youth with T1D and DEB reported less psychopathology (less depression, anxiety,
dietary restraint, food preoccupation, weight concerns, and drive for thinness) overall as compared to the group with DEB and without T1D (Powers, Richter, Ackard, Gerken, Meier, & Criego, 2012). In the current study, youth living with T1D did not report more psychopathology when compared to newly diagnosed youth who have not been living with the illness. Thus, youth with T1D living with the treatment regimen do not necessarily present with more psychopathology than age-matched peers who have just been diagnosed.

Despite a lack of group differences in level of bulimic symptoms in the current study, there were different predictors of bulimic symptoms based on group. For youth who were newly diagnosed with T1D and naïve to treatment regimen, a combination of high body image dissatisfaction and high depressive symptoms were related to the most bulimic symptoms. For youth living with T1D and treatment regimen, a combination of high depressive symptoms and uncontrollable hunger related to insulin and fluctuations in blood glucose was related to the most bulimic symptoms. This finding is consistent with previous studies which have found that depression is a significant risk factor for DEB in youth with T1D (e.g., Colton, Olmsted, Daneman, & Rodin, 2013), and extends those findings by suggesting that for youth with high depressive symptoms, high levels of hunger dysregulation due to disease and treatment factors potentiates the highest risk for DEB.

**Clinical Implications**

The implications of these findings suggest that disruptions to hunger and satiety are important in conceptualizing risk for DEB in youth with T1D. Therefore, treatment of DEB in this population should implement a multidisciplinary approach to
appropriately address both psychological and physiological factors associated with DEB. It is suggested that once DEB is detected in youth with T1D, early psychological treatment is indicated, particularly for youth with concomitant poor glycemic control (Gagnon, Aime, Belanger, & Markowitz, 2012) given the potential deleterious effects of DEB on A1c (Goebel-Fabbri et al., 2008). Previous studies have found support for the use of modified CBT to treat DEB in youth with T1D within the context of the treatment regimen (Peveler & Fairburn, 1992). Although these treatments may previously have been at odds with one another, recent shifts in diabetes treatment affords more flexibility within the dietary regimen (ADA, 2012). This flexibility is conducive to eating disorder treatments such as CBT, where rigid food rules are discouraged, and more nonrestrictive attitudes towards eating are promoted (Agras & Apple, 2004). Thus, it seems that CBT for bulimia could be modified and implemented without interfering with the T1D treatment regimen. In addition to incorporating regular meals and snacks (which should be adapted to insulin regimen; Goebel-Fabbri, 2009), cognitions regarding weight and shape, as well as fears of hypoglycemia, can be targeted through this treatment modality. CBT could also be implemented in an inpatient setting, particularly for adolescents with very poor glycemic control, as inpatient treatment for bulimia nervosa has shown to be more effective than outpatient treatment in reducing binge eating, insulin omission, and other compensatory behaviors (Takii et al., 2003). However, previous treatments for DEB, including psychoeducational interventions and CBT, have not addressed factors associated with insulin treatment and blood glucose (Alloway et al., 2001; Peveler & Fairburn, 1992; Takii et al., 2003).
Given that disruptions to hunger and satiety proprioception are associated with disordered eating, it is important to address the effects of insulin treatment and glycemia on hunger and satiety within the context of treatment for DEB. Previous treatments for DEB in youth with T1D have not targeted these disease and treatment related factors. In a 6-week brief psychoeducation intervention, the material presented in the intervention sessions related to issues such as set point theory, and emphasized a nondieting approach to eating (Olmsted et al., 2002). Despite also incorporating psychoeducation regarding the dangers of insulin misuse and encouraging T1D treatment providers to promote a flexible eating plan to patients, education regarding the relationships between hunger, insulin use, and glycemia were absent. Further, despite finding decreases in dietary restraint and body image dissatisfaction as a result of the intervention, there were no significant effects on insulin misuse for weight management, or changes to glycemic control (Olmsted et al., 2002). Providing education regarding the effects of insulin and blood glucose levels on subsequent control over hunger could be a helpful addition to current treatment interventions, and potentially affect insulin misuse and glycemic control. The multidisciplinary DEB treatment team approach could incorporate diabetes educators, as well as nurse practitioners and endocrinologists to provide education on these disease and treatment-related factors, and assist the patients in making modifications to their insulin regimen in an effort to reduce dysregulated hunger associated with insulin use and blood glucose.

**Strengths, Limitations, and Future Directions**

The present study was novel in that it examined the putative role of T1D treatment and disease based factors in conjunction with traditional risk factors for DEB in
a sample of youth with T1D. The aim of this study was to test the Modified Dual Pathway Model of disordered eating in youth with T1D, which posits that established risk factors for eating pathology in the general population (e.g., body image dissatisfaction, dietary restraint, depression, weight status) in conjunction with treatment and disease-related factors specific to T1D (e.g., hunger and satiety dysregulation, A1c, length of illness) interact to potentiate risk for DEB in youth with T1D. The Modified Dual Pathway Model postulates that it is necessary to account for these disease and treatment related processes to avoid inaccurately attributing pathological causes for DEB in this population.

The current study is not without limitations. The analyses conducted in this study are cross-sectional and therefore, we cannot assume the causal or directional nature of these variables in reference to DEB. Further, given that we were analyzing data on two groups of youth (one with disease and treatment experience, and the newly diagnosed group), we could not utilized a diabetes-specific measure of eating pathology such as the DEPS-R (Markowitz et al., 2010) to assess diabetes-specific DEB across groups. A prospective examination of these two groups would allow for the use of such an assessment measure. Additionally, given that our disease and treatment-experienced group was transitioning to an insulin pump treatment and their regimen was changing, we were unable to control for within group differences regarding insulin administration and dosing. It is also notable that youth with T1D who are transitioning to an insulin pump may represent a distinct group. Previous studies have shown that insulin pump users are more likely to be female, white, older adolescents, and have a higher socioeconomic status (Paris, Imperatore, Klingensmith, Petitti, Rodriguez, et al., 2009). Finally, in this
study, our measurement of dietary restraint was limited to a single item, limiting the variability with which we could assess this behavior in our sample.

The present study demonstrated a relationship between disruptions in proprioception of hunger cues to disordered eating when controlling for other known risk factors for DEB in a sample of youth with established T1D. This finding is novel, and lends support to the Modified Dual Pathway Model. Future studies should examine the relationships between hunger and satiety dysregulation, and DEB, along with other putative risk factors consistent with the Modified Dual Pathway Model in a prospective design. Examination of the newly diagnosed and pump transitioning groups prospectively has the potential to isolate the role of treatment and disease related factors in conjunction with DEB, provide stronger support for the Modified Dual Pathway Model and inform more accurate conceptualization and treatment of DEB in youth with T1D.
CHAPTER 5

REFERENCES


Figure 1. Stice’s Original Dual Pathway Model
Figure 2. Modified Dual Pathway Model
Table 1

*Descriptive Statistics*

<table>
<thead>
<tr>
<th>Variable</th>
<th>New</th>
<th>Pump</th>
<th>Combined</th>
</tr>
</thead>
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<tr>
<td>Age</td>
<td>12.74 (2.24)</td>
<td>12.86 (1.83)</td>
<td>12.79 (2.10)</td>
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<td>z-BMI*</td>
<td>-.14 (1.36)</td>
<td>.60 (.88)</td>
<td>.18 (1.23)</td>
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<tr>
<td>Length of Illness*†</td>
<td>4.14 (3.76)</td>
<td>1558.81 (1274.46)</td>
<td>666.03 (1130.94)</td>
</tr>
<tr>
<td>A1c*</td>
<td>11.4 (2.21)</td>
<td>8.3 (1.29)</td>
<td>9.93 (2.40)</td>
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<td>CDI</td>
<td>6.6 (6.6)</td>
<td>6.7 (4.9)</td>
<td>6.6 (5.9)</td>
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<tr>
<td>Image</td>
<td>5.12 (5.93)</td>
<td>6.40 (8.47)</td>
<td>5.66 (7.11)</td>
</tr>
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<td>Bulimia</td>
<td>2.7 (4.7)</td>
<td>1.5 (3.5)</td>
<td>2.2 (4.2)</td>
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<td>DTSS Total</td>
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<td>DTSS U</td>
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<td>9.88 (2.52)</td>
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</tr>
<tr>
<td>DTSS R</td>
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<td>9.12 (2.54)</td>
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<td>DTSS Hisugar</td>
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<td>3.33 (2.00)</td>
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</tr>
</tbody>
</table>

Note. All values are means (standard deviations).
*statistically significant differences (p < .01) between New and Pump. † length of illness in days. CDI=Children’s Depression Inventory; Image=Eating Disorder Inventory-III Body Image Dissatisfaction Subscale; DTSS U=DTSS Uncontrollable Hunger Subscale; DTSS R=DTSS Remain Hungry Subscale.
Table 2

*Correlations of Combined Group Variables (n=101)*

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<th>C2</th>
<th>C3</th>
<th>C4</th>
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<th>C6</th>
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<td>.12</td>
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<td>--</td>
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<td>.61**</td>
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*Note.* *p* < .05. **p** < .01.

Length=Length of illness measured in days; A1c=Hemoglobin A1c (index of glycemic control); Image=EDI Body Image Dissatisfaction Subscale.
Table 3

*Correlations of Newly Diagnosed Group Variables (n=58)*

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*Note.* *p* < .05, **p** < .01.

Length=Length of illness measured in days; A1c=Hemoglobin A1c (index of glycemic control); Image=EDI Body Image Dissatisfaction Subscale.
Table 4

Correlations of Pump Transitioning Group Variables (n=43)

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Note. * p<.05. **p<.01. Length=Length of illness measured in days; A1c=Hemoglobin A1c (index of glycemic control); Image=EDI Body Image Dissatisfaction Subscale; DTSS U= DTSS Uncontrollable Hunger Subscale; DTSS R= DTSS Remain Hungry Subscale.
Table 5

*Hierarchical Multiple Regression Analysis: Combined Group Predicting Bulimic Symptoms (n = 101)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>R</th>
<th>R²</th>
<th>β</th>
<th>t</th>
<th>p</th>
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</tbody>
</table>

*Note.* Only statistically significant steps are shown; Site= 0:GPI, 1:Emory, 2:Joslin; CDI=Children’s Depression Inventory; Image= Eating Disorder Inventory-III Body Image Dissatisfaction Subscale; ImageXCDI=Interaction of Eating Disorder Inventory-III Body Image Dissatisfaction Subscale and Children’s Depression Inventory.
Table 6

*Hierarchical Multiple Regression Analysis: Newly Diagnosed Group Predicting Bulimic Symptoms (n = 58)*

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*Note.* Only statistically significant steps are shown; CDI=Children’s Depression Inventory; Image= Eating Disorder Inventory-III Body Image Dissatisfaction Subscale.
Table 7

_Hierarchical Multiple Regression Analysis: Pump Transitioning Group Predicting Bulimic Symptoms (n = 43)._

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</table>

_Note._ Only statistically significant steps are shown; Site= 0:GPI, 1:Emory, 2:Joslin; DTSS U = DTSS Uncontrollable Hunger Subscale; CDI=Children’s Depression Inventory.