THESIS

UNDERSTANDING THE LINK BETWEEN PARENTAL AND ADOLESCENT

DEPRESSIVE SYMPTOMS IN FAMILIES AT-RISK FOR TYPE 2 DIABETES

Submitted by

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ABSTRACT

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DEPRESSIVE SYMPTOMS IN FAMILIES AT-RISK FOR TYPE 2 DIABETES

Depression and type 2 diabetes (T2D) are serious chronic diseases that show familial aggregation. However, the connection between parent and child depression and T2D risk within families at risk for T2D is poorly understood. The primary objective of the current study was to examine associations among maternal depressive symptoms, adolescent depressive symptoms, and adolescent metabolic characteristics in at-risk families. The second objective was to examine to what extent adolescent coping techniques served as a mediator of the relationship between parental and adolescent depressive symptoms. To address these objectives, I conducted a secondary, cross-sectional data analysis of the baseline phase of a T2D prevention trial with adolescents. Participants were 119 girls (age 14±2y; 62% non-Hispanic Black) and a biological parent. All girls were at risk for T2D by being overweight or obese (BMI $\geq$ 85th percentile) and having a first- or second-degree relative with diabetes. By study design, girls also had at least mild-to-moderate depressive symptoms as determined with the Center for Epidemiologic Studies-Depression Scale (CES-D, total score $\geq$ 16). Adolescents reported a continuous measure of depressive symptoms on the Children's Depression Inventory, and parents described their own depressive/anxiety symptoms on the Adult Self-Report. Adolescent coping skills were measured by adolescents' report on the Responses to Stress Questionnaire-Social Stress Version. Metabolic risk factor measures included fasting glucose, fasting insulin, insulin sensitivity determined with oral glucose tolerance tests, and body composition by dual-energy x-ray

absorptiometry. Parental depressive/anxiety and adolescent depressive symptoms were positively correlated ($p < .05$), and this relationship remained even when accounting for race, age, puberty, body fat, lean mass, height, and presence of maternal diabetes ($p = .01$). Parental depression/anxiety symptoms were significantly related to adolescent BMI metrics, adjusting for similar covariates (all $p < .05$), but parental depression/anxiety did not relate to other insulin or glucose indices after accounting for body composition. Adolescent coping strategies of disengagement coping, involuntary engagement coping, and involuntary disengagement coping were all predictive of greater adolescent depressive symptoms in adjusted analyses (all $p < .05$). However, parental depression/anxiety and coping had independent main effects on adolescent depressive symptoms, and there was no evidence that coping mediated the relationship between parental depressive/anxiety symptoms and adolescent depressive symptoms (all $p \geq .34$). In conclusion, among adolescent girls at-risk for T2D with some depressive symptoms, higher levels of parental depressive/anxiety symptoms were related to relatively higher levels of adolescent depressive symptoms and higher adolescent BMI. Frequency of negative coping skills also predicted relatively greater depressive symptoms among adolescent girls at-risk for T2D. The positive relationship of parental depression/anxiety and adolescent adverse coping skills to depressive symptoms in teens at-risk for T2D may have applied implications for preventative efforts targeting depression and T2D in these youth. However, longitudinal data are required to help elucidate the directional nature of these associations.

TABLE OF CONTENTS

CHAPTER 1: INTRODUCTION

Type 2 diabetes is a serious chronic illness that causes severe health complications including cardiovascular disease and stroke, amputations, blindness, and kidney failure (Centers for Disease Control and Prevention, 2011). Although type 2 diabetes historically was a disease of middle and later adulthood, there has been a very alarming rise in the incidence of type 2 diabetes among adolescents and young adults, particularly in racial/ethnic minorities (Dabelea et al., 2014; Pettitt et al., 2014). Insulin resistance, also referred to as poor insulin sensitivity, is the major physiological precursor to type 2 diabetes (Cali et al., 2009; U.S. Department of Health and Human Services, 2008). Being overweight or obese and having a family history of diabetes significantly increase a child's or adolescent's risk of insulin resistance and ultimately, developing type 2 diabetes (Morrison et al., 2010; Weiss et al., 2004). Yet, psychosocial factors also may play a role in worsening insulin resistance and diabetes risk. In particular, depressive symptoms have been related to insulin resistance and type 2 diabetes in both adolescents (Shomaker et al., 2010) and adults (Golden et al., 2004; Golden et al., 2008; Pan et al., 2008), after accounting for obesity. Despite the familial risk of both depression and diabetes, very little work has sought to understand patterns of co-occurrence in depressive symptoms and metabolic risk factors for type 2 diabetes among families at-risk for type 2 diabetes. Thus, the primary aim of the current project was to evaluate the relationship between parental depressive symptoms and adolescent depressive symptoms in families at high risk for type 2 diabetes. We also sought to evaluate the relationship between parental depressive symptoms and adolescent metabolic risk factors for type 2 diabetes. Previous research and family/ecological systems theoretical accounts point to adolescent coping skills as a potential mechanism explaining the connection between

1

parental depressive symptoms and adolescent depressive symptoms. Yet, despite the potentially

important clinical applications of identifying mechanisms that explain this link, it is unclear if

this framework applies to families at-risk for type 2 diabetes. Thus, the secondary aim was to

investigate to what extent adolescent coping skills explain the association between parental and

adolescent depressive symptoms among families at-risk for type 2 diabetes.

CHAPTER 2: CONNECTING DIABETES AND DEPRESSION

What is Type 2 Diabetes?

Type 2 diabetes is a metabolic condition in which the body does not respond to its own production of insulin, known as insulin resistance (American Diabetes Association, 2013). Insulin is a hormone released by the pancreas in response to food intake, and it promotes the uptake and utilization of glucose (blood sugar) by almost all of the body's cells. Insulin resistance—also referred to as poor insulin sensitivity—occurs when the body creates insulin but does not use it effectively or efficiently, and as such, glucose accumulates in the blood (U.S. Department of Health and Human Services, 2008). As insulin resistance worsens, the body's glucose levels continue to rise without being metabolized for energy in the rest of the body, ultimately leading to type 2 diabetes (Cali et al., 2009). Diabetes causes very serious health complications such as heart disease, stroke, hypertension, kidney or nervous system disease, amputations, and depression (Centers for Disease Control and Prevention, 2011). Death as a consequence of type 2 diabetes is the seventh leading cause of death in the United States (Centers for Disease Control and Prevention, 2011). In the United States, approximately 40% of adults aged 20 years or older are estimated to develop type 2 diabetes in their lifetime (Gregg, Zhuo, Cheng, Albright, Narayan, & Thompson, 2014). Historically, type 2 diabetes has been known as 'adult-onset diabetes' (American Diabetes Association, 2013). However, in recent years, type 2 diabetes has been on the rise in adolescents across all racial/ethnic groups, with the highest prevalence in American Indian/Alaskan native, non-Hispanic Black, and Hispanic minority adolescent populations (Pettitt et al., 2014). From 2001 to 2009, there was a 35% increase in the prevalence of type 2 diabetes in adolescents (Dabelea et al., 2014). Type 2

diabetes in adolescents is associated with an increased risk for end of life renal disease and a three-fold increase in death during middle age as compared to non-diabetic peers (Pavkov et al., 2006). Furthermore, those with adolescent-onset type 2 diabetes have a greater risk of mortality during middle age than those with adult-onset type 2 diabetes, indicating both an increase in adverse effects from earlier onset of diabetes and a crucial need to understand risk factors for type 2 diabetes that can inform prevention efforts.

Etiology of Type 2 Diabetes

Familial risk. It is well established that type 2 diabetes runs in families. Utilizing both the National Growth and Health Study and the Princeton Follow-Up Study, adolescents who had a family history of type 2 diabetes were estimated to be up to six times more likely than youth with no diabetes family members to develop type 2 diabetes (Morrison et al., 2010). The more family members with diabetes, the greater the risk. For example, in the Framingham Heart Study and the Framingham Offspring Study, compared to individuals with no parental history of type 2 diabetes, individuals with one diagnosed biological parent had a 3.5-fold increased chance and those with two diagnosed biological parents had a 6-fold increased chance for type 2 diabetes in their lifetime (Meigs, Cupples, & Wilson, 2000). Family history also appears to be related to the age of onset of type 2 diabetes. In a cross-sectional study of 651 patients diagnosed with type 2 diabetes, those with a family history of the disease had higher body weight and an earlier age of diagnosis than those without a family history (Jeong et al., 2010). Studies have consistently demonstrated an inverse correlation between strength of family history of type 2 diabetes and age of onset of type 2 diabetes across diverse racial/ethnic groups (Molyneaux, Constantino, & Yue, 2004).

Genetic, environmental, and epigenetic factors likely interact in complex ways to explain how diabetes runs in families (Temelkova-Kurktschiev & Stefanov, 2012). Recent investigations have estimated that the heritability of type 2 diabetes ranges from 45-85% (Schwenk, Vogel & Shurmann, 2013). There is also emerging evidence that maternal characteristics while the child is still in utero, such as increased maternal glucose levels (Hillier et al., 2007), gestational diabetes (maternal diabetes during pregnancy) (Dabelea et al., 2008), maternal body mass index (BMI; weight in kg/height in m^2), and excessive gestational weight gain (Kaar, Crume, Brinton, Bischoff, McDuffie, & Dabelea, 2014), as well as fetal or developmental over-nutrition in utero (Dabelea & Harrod, 2013), increase future risk for obesity and type 2 diabetes in the offspring.

Obesity. Obesity is a significant risk factor for type 2 diabetes through its effects on increasing insulin resistance and individuals' risk for "metabolic syndrome," a cluster of metabolic problems that is a precursor of type 2 diabetes (Weiss et al., 2004). In youth, obesity is defined as having a BMI at or greater than the 95th percentile for age and sex (Ogden & Flegal, 2010). The contemporary emergence of type 2 diabetes incidence in youth has paralleled the dramatic rise in obesity in childhood and adolescence over the past half century (Rosenbloom, 2002). In a study that compared obese, overweight, and non-overweight children and adolescents, overweight and obese youth had higher levels of fasting insulin, insulin resistance, fasting glucose, impaired glucose tolerance, and the presence of metabolic syndrome compared to non-overweight youth (Weiss et al., 2004). Longitudinal data also indicate that obesity, as measured by both BMI and waist circumference, in childhood and adolescence is an independent risk factor for early-onset type 2 diabetes, even after accounting for diabetes family history (Franks et al, 2007). Multivariate analyses of two large longitudinal studies comprising almost

2,000 children and adolescents indicated that both obesity as well as fasting insulin, independent of each other, were two of the most salient risk factors that predicted type 2 diabetes onset either 9 or as much as 26 years later (Morrison, Glueck, Horn, & Wang, 2010).

Psychological factors. Psychological factors such as depressive symptoms also may be a risk factor for worsening insulin resistance and type 2 diabetes, even beyond traditional risk factors including family history of type 2 diabetes and obesity. Evidence for depression as a risk factor for insulin resistance and type 2 diabetes comes from studies of adults and youth.

Adults. A cross-sectional relationship exists between depressive symptoms and insulin resistance in adults. For instance, within young adult women and men, individuals with elevated depressive symptoms had higher insulin resistance than those without depressive symptoms, even after accounting for potentially confounding factors such as demographics and/or lifestyle habits (Pearson et al., 2010). It has also been identified that within young men, there was a positive association between elevated depressive symptoms and insulin resistance, after accounting for covariates such as BMI, socio-economic status, physical activity, and fasting serum level of cholesterol (Timonen et al., 2006). Likewise, in middle-aged and older adult men and women, insulin resistance was significantly higher in those who had elevated depressive symptoms, as determined by a threshold score, as compared to those with no or low symptoms, even after accounting for potential confounders including physical activity level, education level, BMI and use of alcohol and tobacco (Pan et al., 2008). Said differently, adults with elevated depressive symptoms had a 1.5 times greater likelihood of having insulin resistance in the top quartile than those without such depressive symptoms (Pan et al., 2008).

Longitudinal data in adults also point to a connection between depressive symptoms and insulin resistance or type 2 diabetes. Women with elevated depressive symptoms were 66%

more likely to develop type 2 diabetes compared to those with low or no depressive symptoms, even when accounting for women's age, race, medication use, and education (Everson-Rose et al., 2004). Factors such as physical activity and waist circumference appeared to partially account for the association between depressive symptoms and diabetes risk. Similar prospective effects of elevated depressive symptoms have been documented in both women as well as men in middle adulthood (Golden et al., 2004; Golden et al., 2008) and in older adulthood (Carnethon et al., 2007), after accounting for BMI or adiposity. In a meta-analysis of 23 longitudinal studies, elevated depressive symptoms were a significant risk factor for onset of type 2 diabetes among adults (Rotella & Mannucci, 2013). These effects have also been documented regarding full-syndrome major depressive disorder in both men and women in middle adulthood (e.g., Eaton, Armenian, Gallo, Pratt, & Ford, 1996).

Despite this strong evidence indicating that depressive symptoms act as a risk factor for onset of type 2 diabetes in adults, it is important to note that not all studies have observed this effect (Saydah, Brancati, Golden, Fradkin, & Harris, 2003). Furthermore, some work suggests a bi-directional relationship such that depression promotes type 2 diabetes, and the burden of having type 2 diabetes as a chronic illness, in turn, may worsen depressed mood (Golden et al., 2008; Mezuk, Eaton, Albrecht, & Golden, 2008).

Adolescents. Much less work has investigated the relationship of depressive symptoms to insulin resistance or type 2 diabetes in youth. Yet, available data support a similar connection between depressive symptoms and insulin resistance. Across adolescents of all weight categories, depressive symptoms have been positively associated with metabolic risk factors for type 2 diabetes (Hannon, Rofey, Lee, and Arslanian, 2013; Jaser et al., 2009; Shomaker et al., 2010). In generally healthy adolescents, depressive symptoms were positively correlated with

fasting insulin and insulin resistance, even after accounting for individual's body composition of fat mass and fat-free mass (Shomaker et al., 2010). Comparatively, in adolescents who were considered at-risk for type 2 diabetes based upon both having a BMI at the 85th percentile or higher and having a biological parent with type 2 diabetes, there was a positive association between depressive symptoms and fasting insulin (Jaser et al., 2009). Moreover, among obese adolescents, Hannon et al. (2013) found that higher levels of depressive symptoms were associated with greater metabolic risk factors for type 2 diabetes, including higher glucose levels, lower pancreatic insulin secretion relative to insulin sensitivity as measured by a "disposition index," and higher incidence of pre-diabetes, as determined by either impaired fasting glucose or impaired glucose tolerance.

Much less longitudinal research has been conducted on the relationship between depressive symptoms and insulin resistance in youth. In one study, Shomaker et al. (2011) found that initial levels of depressive symptoms predicted worsening fasting insulin, insulin resistance, and fasting glucose, even when accounting for BMI and BMI change, over an approximate 5-year period in youth at-risk for adult obesity due to either their own or their parent's weight. The inverse relationship was not observed; initial levels of insulin resistance were not predictive of later depressive symptoms (Shomaker et al., 2011). In a similar vein, other longitudinal studies have found support for negative emotionality temperaments such as anger and aggression as risk factors for increased metabolic precursors to type 2 diabetes, including fasting insulin levels (Ravaja & Keltikangas-Jarvinen, 1995).

Depression epidemiology within families. The connection between depression and type 2 diabetes risk is particularly meaningful because, like type 2 diabetes, depression is a major

public health concern. I next consider what is known about the epidemiology of depression in adults and adolescents.

Adults. Approximately 9% of adults report current elevated depressive symptoms, referring to symptoms of major depressive disorder that may or may not meet criteria for a full-syndrome diagnosis as defined by the Diagnostic and Statistical Manual for Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013; Centers for Disease Control and Prevention, 2010; Strine et al., 2008). Specifically, women report more elevated depressive symptoms than men. Approximately 10% of women and 8% of men in community samples report current elevated depressive symptoms; 4% of women report current elevated depressive symptoms that meet criteria for major depressive disorder compared to 2.7 % of men (Centers for Disease Control and Prevention, 2010). Lifetime prevalence rates of major depressive disorder are even more gender differentiated. Approximately 12% of women will experience major depressive disorder in their lifetime versus about 6% of men (Ford, & Erlinger, 2004). Elevated depressive symptoms that do not necessarily meet full-syndrome criteria are even more prevalent. In the U.S. adult population, about 20% of women and 11% of men endorse elevated depressive symptoms at some point in their lifetime, as measured by a score at or greater than 10 on the widely-used Patient Health Questionnaire (Strine et al., 2008).

Depression in type 2 diabetes is of particular concern. A meta-analysis of prevalence rates of depression in adults with type 2 diabetes across the world found an overall occurrence of current elevated depressive symptoms, including major, minor, and subsyndromal depression, to be about 18% (Ali, Stone, Peters, Davies & Khunti, 2006). As in the general population, women with type 2 diabetes have nearly double the rates of current elevated depressive symptoms than men (approximately 24% versus 13%) (Ali et al., 2006). Taken together, these data indicate that

adults with type 2 diabetes experience more depression than adults in the general population, and that women with type 2 diabetes are particularly affected.

Depression is not only prevalent in people with type 2 diabetes but also appears to significantly increase diabetes-related poor health outcomes. Comorbid depression and diabetes is associated with lower metabolic control and poorer quality of life (Lustman & Clouse, 2005). Evidence shows that depression in type 2 diabetes is associated with a greater risk of developing a host of very serious negative outcomes, such as increased risk for myocardial infarction, otherwise known as heart attack (Scherrer et al., 2011), compared to diabetes uncomplicated by depression. Major depressive disorder and type 2 diabetes each increased the risk of myocardial infarction by approximately 30% individually, whereas having both major depressive disorder and type 2 diabetes increased the risk for myocardial infarction by 82%, when compared to adults with neither depression nor diabetes.

Adolescents. Approximately 25% of adolescents in the general population experience elevated depressive symptoms at some point during adolescence, with approximately 18% reaching the threshold for major depressive disorder (Lewinsohn Shankman, Gau, & Klein, 2004). Of those with elevated depressive symptoms, 58% are female and of those with major depressive disorder, 70% are female, indicating that depression is more common in adolescent females than males.

Fewer studies have been conducted on depression in adolescents with type 2 diabetes because the disease is still relatively rare in pediatric populations. One study compared depressive symptoms in adolescents with type 2 diabetes, type 1 diabetes (referring to an autoimmune disease in which the pancreas can no longer produce insulin), or a variety of other types of chronic illness such as celiac disease or asthma (Lawrence et al., 2006). The results

indicated that having type 2 diabetes in both girls and boys was associated with a greater likelihood of having mild or moderate-to-severe depressive symptoms, as compared to boys with type 1 diabetes or girls with other non-diabetes chronic illnesses (Lawrence et al., 2006). Mirroring community trends, among adolescents with type 2 diabetes specifically, girls were more likely to have elevated depressive symptoms than boys. Among all youth with type 2 or type 1 diabetes, negative correlates that were associated with having depressive symptoms included poorer glycemic control as measured by HbA1c and more emergency room visits (Lawrence et al., 2006).

     Connections between parent and adolescent depression. In community samples, depression has been estimated to be moderately familial, with a systematic meta-analysis of the literature finding that youth with first-degree relatives with clinical depression have up to a four-fold greater odds of developing clinical depression compared to youth without a family history of depression (Rice, Harold, & Thapar, 2002). In twin studies, heritability for clinical depression ranges very broadly from 15-80%, with lower estimates on adolescent self-report measures and higher estimates using parent-report measures (Rice et al., 2002).

     In community-wide longitudinal samples, parental major depressive disorder has been associated with a greater odds of offspring developing major depressive disorder, compared to children without depressed parents (Nomura, Wichramaratne, Warner, Mufson, & Weissman, 2002). Similarly, a series of longitudinal studies support parental internalizing symptoms, referring to depressed mood, as well as anxiety or withdrawal, as a prospective risk factor for increases in depressive symptoms among offspring. For example, maternal internalizing symptoms were positively associated with adolescents' depressive symptoms measured at age 15,

and were also predictive of continued greater depressive symptoms five years later (Agerup et al., 2014).

While considerable evidence supports a relationship between parental and adolescent depression in the general population, very little research has investigated the correspondence of parent and adolescent depressive symptoms among families at-risk for diabetes. Only one study investigated parental and adolescent depressive symptoms among 61 adolescents with either type 1 or type 2 diabetes. In this sample, elevated parental depressive symptoms were positively associated with depressive symptoms in adolescents who have diabetes (Eckshtain, Ellis, Kolmodin, & Naar-King, 2010). Teens who have developed frank diabetes as a major chronic illness can be distinguished from adolescents who are at-risk for type 2 diabetes, but who have not yet developed this serious medical syndrome. To what degree parental and adolescent depressive symptoms relate in families of at-risk adolescents has not been explicitly tested. Determining the relationship of parental depressive symptoms to adolescent depressive symptoms in families at-risk for diabetes is imperative for informing potentially novel family-based avenues for prevention strategies for type 2 diabetes in high-risk youth.

Parental depression and adolescent metabolic risk factors. While it has been established that both depressive symptoms and metabolic risk factors for type 2 diabetes show familial aggregation (Morrison et al., 2010; Rice et al., 2002), very few studies have examined the relationship between parental depressive symptoms and adolescent metabolic risk factors for type 2 diabetes. In adolescents with frank type 2 diabetes, parental depressive symptoms were related to adolescents' depressive symptoms cross-sectionally, but were not related to adolescents' metabolic characteristics or their response to diabetes treatment (Weinstock et al., 2015). Conversely, in youth with type 1 diabetes, a number of investigations show that parental

depression is associated with youth's poorer treatment adherence and glycemic control (Mackey, Struemph, Powell, Chen, Streisand, & Holmes, 2014). The relationship of parental depressive symptoms to metabolic risk factors may or may not be similar among adolescents at-risk for a chronic disease (i.e., type 2 diabetes), who are otherwise generally healthy. Understanding if there is a connection, however, would have important implications for diabetes prevention efforts. There is some small evidence that a link exists connecting parental depressive symptoms and adolescent metabolic characteristics adolescents at risk for depression. Specifically, in a cross-sectional study of adolescents that are generally healthy, those who were at risk for depression due to a parent with depression had increased systolic blood pressure and worse insulin sensitivity compared to a control group of adolescents with no family history of depression (Mannie, Williams, Diesch, Steptoe, Leeson & Cowen, 2013). Additionally, these results remained significant after controlling for adolescents' weight, exercise, smoking, or alcohol consumption. These preliminary data speak to the possibility that parental depressive symptoms could be related to a worse metabolic profile among adolescents at-risk for type 2 diabetes, a hypothesis that warrants further testing.

Coping as a Potential Mechanism Explaining the Link between Parental and Adolescent Depression

It is well accepted that the association between parental and adolescent depression is likely due to a combination of genetic and environmental factors (Rice, Lewis, Harold & Thapar, 2013). What is less well understood are the potentially modifiable mechanisms that explain the connection between parental and adolescent depression. One widely held view is that adolescents' learned coping skills for handling stress may be a key mechanism explaining the proposed association between parental depression and adolescent depression. From a family

systems theoretical perspective, depression and diabetes risk may be connected within families in part due to the qualities of adolescent coping mechanisms for handling stress. Specifically, inter-individual differences in coping skills to manage stress have been proposed to account for the association between parental depression and adolescent depression (Compas et al., 2010; Dunbar et al., 2013; Langrock, Compas, Keller, Merchant, & Copeland, 2002).

Defining coping. Coping has been defined as "cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person" (Lazarus & Folkman, 1984, p. 141). Although there are a number of frameworks for defining variations in coping skills, one approach that has been used extensively is to consider three main voluntary or volitional subsets of coping: primary control coping, secondary control coping, and disengagement coping (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). Primary control coping, such as problem solving, involves altering or changing the situation to cope with a stressor (Connor-Smith et al., 2000). Secondary control coping, such as acceptance, involves trying to adapt to the situation or stressor (Connor-Smith et al., 2000). Disengagement coping, such as avoidance, is distinguished by cognitive and/or behavioral strategies that involve avoiding the stressor or reaction to the stressor (Connor-Smith et al., 2000). This same framework also proposes that coping may additionally involve involuntary or automatic reactions to stress, known as involuntary engagement, such as rumination or physiological arousal that is experienced by the individual as automatic and non-volitional, and involuntary disengagement, such as emotional numbing (Connor-Smith et al., 2000).

Parental depression and adolescent coping. In community samples, parental depression has been associated with adolescent coping skills. Langrock et al. (2002) found a

negative cross-sectional association between intensity of parental depressive symptoms and 7-17-year-olds' use of primary coping. Also, among youth with depressed parents, there was an inverse relationship between parental withdrawal and adolescents' primary or secondary control coping, and a positive relationship between parental withdrawal and adolescents' reports of disengaged coping, involuntary engagement and disengagement. There have been no investigations into the associations between parental depressive symptoms and coping skills in adolescents either with type 2 diabetes or among youth at high-risk for this disorder. Understanding the relationship between parental depressive symptoms and different dimensions of adolescent coping skills in this specific population would allow for more informed, nuanced, and targeted intervention or prevention efforts to address depression in this high-risk group and to potentially decrease diabetes risk in these adolescents.

Coping skills and adolescent depression. Coping skills have also been associated with adolescent depressive symptoms in a number of cross-sectional studies. For example, in adolescents at-risk for depression due to having at least one parent with a history of major depressive disorder, adolescents' primary and secondary control coping were significantly, inversely associated with adolescent depressive symptoms (Dunbar et al., 2013). Similarly, there was a positive correlation between adolescents' depressive symptoms and disengaged coping (Dunbar et al., 2013) and involuntary engagement (Langrock et al., 2002). Fear et al. (2009) found that within adolescents with at least one parent with a history of depression, adolescents' primary and secondary control coping were inversely correlated with depressive symptoms, whereas Langrock et al. (2002) found that only secondary coping skills were associated with fewer adolescent depressive symptoms. Even after controlling for gender, age, parental marital status, parental depressive symptoms, and conflict between parents, youths' secondary coping

skills continued to be inversely correlated with depressive symptoms, in that less usage of secondary coping predicted more depressive symptoms (Fear et al., 2009).

Few longitudinal investigations have been conducted to examine adolescent coping and depressive symptoms. In a relatively short-term, 8-week longitudinal study, adolescents' rumination over controllable events and their rumination over social events predicted increases in later depressive symptoms (Nicolai, Laney, & Mezulis, 2013). Additionally, negative cognitive appraisals, referring to negative thoughts about oneself or the causes or consequences of an action, prospectively predicted increases in adolescents' depressive symptoms (Nicolai et al., 2013). A similar study found that the combination of rumination and negative cognitive appraisals predicted depressive symptoms in adolescents over a six-month period (Black & Pössel, 2013).

Among individuals with type 2 diabetes, available evidence suggests that the use of different coping skills is important. Qualitative interviews with adolescents with type 2 diabetes have shown that coping skills are important factors in effective self-management of their diagnosis (Auslander, Sterzing, Zayas, & White, 2010; Mulvaney et al., 2008). In particular, coping skills or strategies were acknowledged as being a way to help in managing their disease (Auslander et al., 2010; Mulvaney et al., 2008). Similar patterns have been observed in adults with type 2 diabetes. Zhang et al., (2009) found that in Chinese adults with type 2 diabetes, coping that was characterized as "negative" had a significant positive association with depressive symptoms, whereas avoidant and active coping styles were inversely related to depressive symptoms. Understanding the extent to which coping skills relate to adolescent depressive symptoms in those teens at risk for type 2 diabetes may be important for informing intervention efforts to prevent the onset of type 2 diabetes or major depressive disorder.

Coping skills as a mediator of parental and adolescent depressive symptoms.

Langrock et al. (2002) examined coping skills as a possible mediator of the relationship between family stress associated with parental depression and adolescent depressive symptoms in a study of children and adolescents with at least one parent with depression. Secondary coping skills, specifically, served as a significant mediator. Family stressors were related to adolescents' less frequent use of secondary control coping skills, which in turn, were related to greater adolescent depressive symptoms. Interestingly, no other coping strategies were mediators. The need exists to determine what voluntary or involuntary coping mechanisms may play a role in the relationship between parental and adolescent depressive symptoms in adolescents at-risk for type 2 diabetes.

Connecting Adolescent and Adult Studies of Depression and Diabetes Risk

Despite the evidence supporting a relationship between depressive symptoms and insulin resistance or type 2 diabetes in adults and adolescents separately, virtually no work has investigated the relationship between depression and diabetes risk within families. Understanding patterns of correspondence among depression factors and diabetes risk factors in at-risk families is important for a number of reasons. Both depression and diabetes run in families, which likely reflects both shared genetic as well as environmental risk factors (Downey & Coyne, 1990). There is some suggestion that degree of diabetes family history not only may affect offspring's diabetes risk, but also may have implications for depression risk. For example, in a small cross-sectional study of children and adolescents with some family history of type 2 diabetes, Irving et al. (2008) observed a positive association between extent of diabetes family history and adolescent depressive symptoms, such that youth who had the greatest number of family members with type 2 diabetes also reported the highest levels of depressive symptoms.

Parental or familial depression – which might be expected to have relevance for youth's depressive symptoms (Agerup, Lydersen, Wallander & Sund, 2014; Downey & Coyne, 1990) – was not examined.

Theoretically, a family or ecological systems theoretical framework may be useful for understanding the potential connectedness among depression and diabetes risk at the parental and child levels. Each member of the family, or system, is posited to be interconnected with one another (White & Klein, 2008). Similarly, each part of the system is theorized to work in a bidirectional manner with each other and the environment (White & Klein, 2008; Bronfenbrenner, 1994). As such, both the parent-system and broader extended family-system are thought to influence the adolescent-subsystem. These interactions among the systems are influenced both by the environment in which they are happening and the genetic components involved in development (Bronfenbrenner, 1994). As such, rather than viewing parents as the primary causal agents in the development of depressive symptoms in adolescents, a family systems framework posits that the likely bi-directional interaction among parents and their adolescents may contribute to commonalities in depressive symptoms in both parent and adolescent. Independent of any etiological assumptions, a family systems theoretical approach assumes that parental depressive symptoms and adolescent symptoms are related, and that this association might serve as a maintenance factor in the compromised health of the family system. Clarifying the relationship between parental depressive symptoms and adolescent depressive symptoms within families specifically at risk for diabetes could, thus, have important implications for intervention and prevention efforts for the mental and physical health of these at-risk families.

The Current Study

  Summary of past literature. Strong evidence supports a possible role for depressive symptoms in worsening insulin resistance and the onset of type 2 diabetes (Pan et al., 2008; Pearson et al., 2010; Rotella & Mannucci, 2013; Shomaker et al., 2010; Timonen et al., 2006). This research has been conducted separately in adults (Pan et al., 2008; Pearson et al., 2010; Rotella & Mannucci, 2013; Timonen et al., 2006) and adolescents (Shomaker et al., 2010), with very little attention to potential patterns of correspondence between parent and adolescent depressive symptoms in families at-risk for diabetes. Likewise, factors that may help explain a possible connection in depressive symptoms within the family system among adolescents at-risk for type 2 diabetes have not been identified. Understanding parental-adolescent correspondence of depressive symptoms among these high-risk families is important because it has the potential to illuminate new intervention efforts targeting prevention of type 2 diabetes in youth.

  Consistent with studies of community samples (Nomura et al., 2002; Rice et al., 2002), preliminary data support a connection between depressive symptoms in parents and depressive symptoms in adolescents who have already developed type 2 diabetes or who have type 1 diabetes (Eckshtain et al., 2010). Evidence also exists to support a relationship between parental depression and adolescent coping skills in the general population (Dunbar et al., 2013; Langrock et al., 2002) and, in turn, between adolescent coping skills and adolescent depressive symptoms (Dunbar et al., 2013; Fear et al., 2009; Langrock et al., 2002). Coping skills are believed to act as a mediator in the relationship between parent and adolescent depressive symptoms (Compas et al., 2010; Langrock et al., 2002). Yet, to what extent coping skills – and particularly forms of voluntary or involuntary coping – might underlie the connection between parental and adolescent depressive symptoms in families at-risk for type 2 diabetes is not known. Due to familial

implications and the potential bidirectional nature of the relationship between depressive symptoms and type 2 diabetes (Golden et al., 2008; Mezuk, Eaton, Albrecht, & Golden, 2008), understanding the connection between parental and adolescent depressive symptoms among adolescents at high risk for diabetes has the potential to enhance our more nuanced understanding of risk factors for type 2 diabetes. This notion, combined with understanding the extent to which coping skills may explain this connection, may be helpful for informing more targeted, effective prevention efforts.

Objectives. In the current study, I evaluated the associations among parental depressive symptoms, adolescent depressive symptoms, and adolescent coping skills in a secondary analysis of adolescent girls at high risk for type 2 diabetes, but who had not yet developed the disease. Adolescents were at-risk for type 2 diabetes by virtue of their BMI (overweight or obese ≥85th BMI percentile for age and sex) and a close family history of type 2 diabetes (or pre-diabetes or gestational diabetes) in first- and/or second-degree relatives. Adolescents also endorsed at least mild to moderate depressive symptoms, but were not clinically depressed. The current project had the following specific objectives:

Objective #1: To determine among adolescent girls at-risk for type 2 diabetes to what extent parental depressive symptoms were related to (a) adolescent depressive symptoms and (b) metabolic risk factors for type 2 diabetes including fasting insulin, insulin sensitivity, and fasting glucose.

Hypothesis #1: I hypothesize that among adolescent girls at-risk for type 2 diabetes, parental depressive symptoms will be (a) positively associated with adolescent depressive symptoms and (b) related to metabolic factors such as higher fasting insulin and glucose and poorer insulin sensitivity.

Objective #2: To evaluate to what extent five different types of adolescent coping skills acted as a mediator of the link between parental depressive symptoms and adolescent depressive symptoms in adolescent girls at-risk for type 2 diabetes.

Hypothesis #2: I predict that the relationship between parent and adolescent depressive symptoms will be mediated by adolescent coping skills, in that adolescents' coping skills will partially explain the relationship between parent and adolescent depressive symptoms.

CHAPTER 3: METHODS

Participants

Participants were 12-17 year old girls who participated in the baseline phase of a diabetes prevention trial (ClinicalTrials.gov: NCT01425905). The current project utilized baseline data only, prior to girls' entry into the intervention phase of the trial. Inclusion criteria were: (i) 'at-risk' for type 2 diabetes determined by having a BMI at or above the 85th percentile for age, and having at least one first or second degree family member with type 2 diabetes, gestational diabetes (diabetes during pregnancy), or pre-diabetes (impaired fasting glucose of ≥100 mg/dL or impaired glucose tolerance of 2-hour glucose following an oral glucose load ≥140 mg/dL); (ii) elevated mild to moderate depressive symptoms defined as a total score of 16 or greater on the Center for Epidemiologic Studies-Depression scale (CES-D); (iii) good general health; and (iv) English-speaking. Exclusion criteria were: (i) current major depressive episode or any current psychiatric symptoms that would require treatment; (ii) major medical problem such as type 2 diabetes (fasting glucose level > 126 mg/dL or 2-hour glucose after an oral glucose administration > 200 mg/dL); (iii) medication use such as anti-depressants or stimulants that could affect insulin resistance or mood; (iv) current involvement in a structured weight loss or psychotherapy treatment program; and (v) pregnancy.

Participants were recruited through direct mailings to families within a 50-mile radius of Bethesda, Maryland, radio and social media advertisements, flyers distributed in local public schools, and letters to local school nurses and physicians. Recruitment materials invited adolescent girls who had a family history of type 2 diabetes to participate in a study testing

whether a brief, 6-week group program would lower adolescents' risk for developing type 2 diabetes.

Procedure

All study procedures were approved by the Institutional Review Board of the National Institute of Child Health and Human Development. Participants were seen for two baseline screening appointments at a pediatric outpatient clinic at the Mark O. Hatfield Clinical Research Center of the National Institutes of Health in Bethesda, Maryland. The first appointment took place during after-school hours. Following consent and assent, adolescents and parents completed questionnaires, and then a physical exam was conducted with the adolescent by a nurse practitioner or endocrinologist. Adolescents' family history of type 2 diabetes, pre-diabetes, and gestational diabetes was evaluated during a medical history conducted by a nurse practitioner or endocrinologist with the parent/guardian. A psychiatric interview conducted by a psychologist or trained psychology graduate student was administered to the adolescent to rule out a current psychiatric disorder that would warrant study exclusion and a referral. Adolescent volunteers who were eligible after the initial screening took part in a second baseline assessment at the NIH Clinical Research Center. Following an overnight fast, adolescents' height and fasting weight were measured to compute BMI, a 2-hour oral glucose tolerance test was administered, and adolescents completed additional questionnaires. Only individuals who were deemed to be eligible after the entire screening phase were included in the current study analyses.

Measures

Parental depressive/anxiety symptoms. Parents' perceptions of their own depressive/anxiety symptoms over the past six months were assessed with the depressed/anxious scale of the reliable, validated and normed Adult Self-Report (ASR) (Achenbach & Rescorla,

2003). Normed T-scores were used for descriptive purposes, and the total raw score was used in primary analyses.

Adolescent depressive symptoms. Adolescents' degree of depressive symptoms in the past two weeks were assessed with the Children's Depression Inventory (CDI) (Kovacs & Beck, 1977), a 27 item self-report measure of depressive symptoms in the past two weeks. The CDI is widely used and has very good reliability and validity (Smucker, Craighead, Craighead, & Green, 1986). The CDI total score, ranging from 0-57, is derived from the sum of all items.

Adolescent coping skills. The Responses to Stress Questionnaire (RSQ)-Social Stress Version is a reliable and well-validated measure of adolescent coping in response to social stressors (Connor-Smith et al., 2000). The social stress version captures coping in response to social stressors common to teenagers such as peer conflict, peer pressure, and peer rejection (Connor-Smith et al., 2000). This 57-item questionnaire is divided into 5 factor-analytically derived subscales including: (i) primary control engagement coping (comprised of problem solving, emotional regulation and expression), (ii) secondary control engagement coping (comprised of positive thinking, cognitive restructuring, and acceptance), (iii) disengagement coping (comprised of avoidance, denial, wishful thinking, and distraction), (iv) involuntary engagement (comprised of rumination, intrusive thoughts, physiological and emotional arousal, and involuntary action), and (v) involuntary disengagement (comprised of emotional numbing, cognitive interference, inaction, and escape). Scales were derived by calculating the average of all respective items. For the purposes of the current investigation and consistent with past literature (Compas et al., 2001; Connor- Smith et al., 2000), each of the five scales were examined separately.

Adolescent pubertal and anthropometric characteristics. Pubertal status, which has been related both to depressive symptoms and insulin sensitivity (Hannon, Janosky, & Arslanian, 2006; Negriff & Susman, 2011), was determined by breast Tanner staging conducted by a nurse practitioner or endocrinologist: [Tanner Stage 1: pre-adolescent; Tanner Stage 2-4: pubertal; Tanner Stage 5: post-pubertal] (Marshall & Tanner, 1969). For the current study, pubertal status was converted to a dichotomy defined as pre/current pubertal (Tanner Stages 1-4) or post-pubertal (Tanner Stage 5). Height was measured in triplicate by stadiometer and fasting weight by calibrated digital scale. These values were used to compute BMI (kg/m^2), BMI standard score (BMI z-score) and BMI percentile for age and sex according to the Centers for Disease Control and Prevention (CDC) standards (Kuczmarski et al., 2000). Dual-energy x-ray absorptiometry (DXA) scan was used to determine percentage body fat and lean mass (kg). An external standard simulating fat and muscle was scanned for calibration.

Adolescent metabolic profile. A standard oral glucose tolerance test (OGTT) was administered to measure insulin and glucose metabolic indices. Fasting blood sampled prior to the administration of the OGTT was used to measure fasting insulin levels and fasting glucose levels. We also evaluated the presence or absence of hyperinsulinemia (fasting insulin >20 O U/mL), and the presence or absence of impaired fasting glucose (fasting glucose 100-125 mg/dL), given these clinical cut-points are often used in practice. An OGTT is less invasive, less costly, and less labor intensive than clamp-derived measures of insulin sensitivity, but it yields more valid estimates of insulin sensitivity than calculations derived from fasting measures alone (Eldredge, & Agras, 1997). Insulin sensitivity levels were estimated with the whole body insulin sensitivity index, estimated with insulin and glucose sampled at baseline and 30, 60, 90 and 120 minutes after glucose administration (Matsuda & DeFronzo, 1999). This metric has been

validated for use in overweight and obese youth (Yeckel et al., 2004). Also, presence or absence of impaired glucose tolerance was determined as a fasting glucose of 140-199 mg/dL 120 minutes after glucose administration.

Demographic information. Parents reported their daughters' date of birth, which was used to calculate age. Parents reported racial/ethnic background.

Analysis Plan

Analyses proceeded from basic to more advanced. Data were cleaned and adjusted for outliers. Specifically, outliers were corrected to fall within 1.5 times the interquartile range above the 75th percentile or below the 25th percentile (e.g., to the whiskers in Tukey's boxplot) (Tukey, 1977). Descriptive information was generated to describe the study sample. Preliminary analyses utilized Pearson correlations to describe the associations among the key variables of parental depressive/anxiety symptoms (ASR), adolescent coping skills (RSQ primary, secondary, voluntary disengagement, involuntary disengagement, and involuntary engagement), adolescent depressive symptoms (CDI), and adolescents' metabolic characteristics (continuous fasting insulin, fasting glucose, and insulin sensitivity), as well as their relationship with possible covariates including age and body measurements (percentage body fat, lean mass, and BMI z-score). Independent samples *t*-tests were used to describe differences in adolescent coping skills, metabolic characteristics, adolescent depressive symptoms, and parent depressive/anxiety symptoms by race (non-Hispanic Black vs. other), maternal history of diabetes (presence vs. absence), weight status (overweight vs. obese), and puberty status (Tanner Stages 1-4 vs. Tanner Stage 5). Independent samples *t*-tests also were used to describe the relationship between continuous measures of parental and adolescent depressive symptoms, adolescent coping skills, and adolescent metabolic characteristics with the categorical measures

of adolescent hyperinsulinemia, impaired fasting glucose, and impaired glucose tolerance. Chi-square tests were employed to describe the bivariate associations among dichotomous variables, including adolescent impaired fasting glucose, impaired glucose tolerance, hyperinsulinemia, weight status, race/ethnicity, and puberty.

To address the primary objectives, a series of linear multiple regressions were evaluated. Adolescent depressive symptoms was the dependent variable. In the first step, the pre-determined covariates of race, age, puberty, percent fat, lean mass, height, and presence of maternal diabetes were entered. We controlled for these factors because they have been related to depression or insulin resistance and because we wanted to determine the relationship of parental depression and adolescent coping to adolescent depressive symptoms independent of these potential confounds. In the second step, parental depression/anxiety was entered to determine the relationship of parental depressive/anxiety symptoms to adolescent depressive symptoms, adjusting for the covariates. To determine the relationship of parental depression/anxiety symptoms with adolescent metabolic characteristics, the continuous metabolic outcomes of fasting insulin, fasting glucose, insulin sensitivity, BMI, BMI z-score, lean mass, and percent fat were regressed on race, age, puberty, presence of maternal diabetes, and parental depressive/anxiety symptoms. In addition, body composition, referring to lean mass, height, and percent fat, was controlled for in the analyses predicting fasting insulin, fasting glucose, and insulin sensitivity, since these measures are highly related to body composition and we wished to determine the relationship of parental depressive/anxiety to metabolic risk for T2D independent of body composition. Binary logistic regressions were used to evaluate the relationship of parental depressive/anxiety symptoms to the odds of having insulin or glucose levels which met clinical cut-points for hyperinsulinemia, impaired fasting glucose, and impaired glucose

27

tolerance. In these analyses, I adjusted for race, age, puberty, percent fat, lean mass, height, and presence of maternal diabetes.

To test coping skills as a possible mediator of parental and adolescent depressive symptoms, we used a product of coefficients approach (MacKinnon, Lockwood, & Hoffman, 2002). We obtained the product of two estimates: α (the association of parental depression with adolescent coping) and β (the association of adolescent coping with adolescent depression) and compared the product to a standard distribution to test for significance of the indirect effect. The estimates for β – one for each of the five coping skills – were obtained by regressing adolescent depressive symptoms on the covariates (race, age, puberty, percent fat, lean mass, height, and presence of maternal diabetes), parental depressive/anxiety symptoms, and each of the coping skills (considered separately). The estimates for α were obtained by regressing each adolescent coping skill subscale on race, age, puberty, percent fat, lean mass, height, and presence of maternal diabetes and parental depressive/anxiety symptoms. Using a sample size of 119 adolescents, post-hoc power analyses indicated adequate power to detect moderate (.83) and large (.99) effects based upon regression modeling containing 9 predictor variables but insufficient power to detect small effects (.13).

CHAPTER 4: RESULTS

Descriptive information and preliminary analyses

One hundred and nineteen adolescent girls, mean (SD) age of 14.5 (1.6) years, comprised the study sample. Descriptive information is provided in Table 1. A very small amount (1.3%) of data points were missing. Most adolescent girls were non-Hispanic Black (62.2 %). Non-Hispanic Black adolescents had higher lean mass ($M = 49.21$ kg, $SD = 7.81$) compared to adolescents of other racial/ethnic backgrounds ($M = 45.59$ kg, $SD = 8.54$); $t(116) = 2.35$, $p < .05$, and they reported more frequent disengagement coping ($M = 1.36$, $SD = .57$ vs. $M = 1.15$, $SD = .50$); $t(113) = 2.05$, $p < .05$. They were also more likely to be post-pubertal than adolescents of other racial/ethnic backgrounds, X^2 (1, N = 119) = 5.72, $p = .02$. There were no other racial/ethnic differences in key variables.

Seventy-three percent of participants were obese (at or above the 95th percentile for BMI), with the remaining adolescents being overweight. Those with obesity were more likely to have higher fasting insulin ($M = 24.41$, $SD = 23.68$ vs. $M = 12.13$, $SD = 21.08$); $t(117) = -2.58$, $p < .05$, and lower levels of insulin sensitivity ($M = 2.20$, $SD = 1.31$) compared to those who were overweight ($M = 3.48$, $SD = 1.48$); $t(114) = 4.47$, $p < .001$. Participants who were obese were more likely to have hyperinsulinemia compared to participants who were overweight, X^2 (1, N = 119) = 16.96, $p < .001$.

While all participants, by study design, had at least one first- or second-degree relative with type 2 diabetes, prediabetes, or gestational diabetes, approximately 20% ($n = 23$) had a mother with diabetes. Participants who had a mother with diabetes had lower levels of depressive symptoms than those with other diabetic relatives ($M = 10.70$, $SD = 5.74$ vs. $M =$

Table 1. Descriptive information about study participants (*N* = 119)

	Mean (SD)
Age, years	14.45 (1.61)
Socioeconomic status [d]	2.73 (0.91)
Tanner breast stage	4.54 (0.82)
BMI, kg/m^2	32.96 (6.60)
BMI z-score	1.97 (0.47)
BMI percentile for age	95.89 (3.30)
Fat mass, % [a]	41.67 (5.77)
Lean mass, kg [a]	47.86 (8.24)
Parental depressed/anxious, T score [b]	52.43 (3.52)
Parental depressed/anxious, raw score [b]	5.16 (4.29)
Teen depressive symptoms	24.91 (7.33)
Primary coping [c]	1.57 (0.54)
Secondary coping [c]	1.60 (0.49)
Disengagement coping [c]	1.28 (0.55)
Involuntary engagement coping [c]	1.00 (0.60)
Involuntary disengagement coping [c]	0.84 (0.52)
Fasting glucose, mg/dL	89.05 (6.77)
Fasting insulin, OU/mL	21.11 (23.56)
Insulin sensitivity, WBISI [b]	2.58 (1.57)

	Frequency (%)
Race/ethnicity	
Black	74 (62.2)
White	19 (16.0)
Hispanic	13 (10.9)
Multiple races	9 (7.6)
Asian	4 (3.4)
Obesity (BMI ≥95th percentile)	87 (73.1)
Impaired fasting glucose (100-125 mg/dL)	7 (5.9)
Impaired glucose tolerance (140-199 mg/dL)	8 (6.7)
Hyperinsulinemia (>20 OU/mL)	63 (52.9)
Maternal T2D, pre- or gestational diabetes (presence)[a]	23 (19.3)

BMI- Body Mass Index; WBISI- Whole Body Insulin Sensitivity Index; T2D- Type 2 Diabetes
[a] = 1 missing value
[b] = 3 missing values
[c] = 4 missing values
[d] = 13 missing values

14.09, $SD = 5.87$); $t(116) = 2.50$, $p < .05$, and they more frequently utilized primary coping ($M = 1.84$, $SD = .48$ vs. $M = 1.49$, $SD = .52$); $t(112) = -2.90$, $p < .01$.

Adolescent girls with hyperinsulinemia (fasting insulin >20 μU/mL) utilized involuntary engagement coping more often than girls without hyperinsulinemia ($M = 1.16$, $SD = .63$ vs. $M = .83$, $SD = .51$); $t(113) = -3.09$, $p < .01$. They also reported more frequent involuntary disengagement coping ($M = .95$, $SD = .51$) than girls without hyperinsulinemia ($M = .72$, $SD = .51$); $t(113) = -2.44$, $p < .05$. Girls with and without IFG or IGT did not differ in parental depressive symptoms, adolescent coping, or adolescent depressive symptoms.

Table 2 displays the bivariate correlations among adolescent age, body composition, parental depressive/anxiety symptoms, adolescent coping skills, adolescent depressive symptoms, and adolescent insulin and glucose metabolism. There was a positive correlation between parental depressive/anxiety symptoms and child depressive symptoms. Parental depressive/anxiety symptoms also were inversely related to adolescents' use of primary control coping, but were unrelated to any of the other dimensions of coping. Adolescent depressive symptoms were positively correlated with teens' use of disengagement, involuntary engagement, and involuntary disengagement coping, such that higher levels of depressive symptoms were related to more frequent use of these coping strategies.

Adolescent depressive symptoms were positively correlated with lean body mass, but were nonsignificantly associated with other body or metabolic characteristics. Parental depressive symptoms were positively correlated with adolescent girls' BMI z-score, such that parents with relatively higher levels of depressive symptoms had daughters with greater BMI z-scores. Adolescent coping was related to a few different body measurements and metabolic indicators. Primary coping was inversely associated with adolescents' percent body

31

Table 2. Correlations among key variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age, yrs	---												
2. BMI z-score	-.08	---											
3. Body fat, %	.05	.78**	---										
4. Lean mass, kg	.26**	.67**	.30**	---									
5. Parental dep/anx	-.06	.19*	.16	.13	---								
6. Teen dep symptoms	.13	.11	.01	.19*	.25**	---							
7. Primary coping	.01	.11	-.20*	-.03	-.23*	-.15	---						
8. Secondary coping	.13	-.09	-.05	-.05	-.15	-.04	.39**	---					
9. Diseng coping	.05	-.03	-.10	.02	.04	.34**	.20*	.36**	---				
10. Invol engage coping	-.17	.26**	.12	.21*	.13	.34**	.31**	.05	.58**	---			
11. Invol diseng coping	-.12	.19*	.07	.15	.12	.29**	.22*	.22*	.68**	.80**	---		
12. Fasting glucose, mg/dL	-.06	.33**	.33**	.26**	.08	.09	.18	.13	.08	.26**	.15	---	
13. Fasting insulin, μU/mL	-.26**	.40**	.25**	.33**	.07	.12	.04	-.06	.00	.20**	.15	.22*	---
14. Insulin sensitivity	.12	-.55**	-.50**	-.39**	-.19	-.02	-.03	.10	-.01	-.23*	-.12	-.45**	-.81**

***$p \leq .001$. **$p \leq .01$. *$p \leq .05$. †$p \leq .10$.

fat, such that teens with relatively greater body fat used primary coping skills less often.

Involuntary engagement coping was positively correlated with BMI z- score, fasting insulin and

glucose, and inversely related to insulin sensitivity, indicating that adolescents more frequently

using involuntary engagement to cope were heavier and at higher metabolic risk for type 2

diabetes.

Primary analyses

Table 3 displays the series of regression analyses predicting adolescent depressive

symptoms. Covariates (model 1) accounted for 13% of the variance in adolescent depressive

symptoms. The only significant predictors of adolescent depressive symptoms in this model

were lean mass and maternal diabetes. Heavier teens had more depressive symptoms, whereas

adolescents with diabetic mothers had less depressive symptoms. Adding parental

depressive/anxiety symptoms to the equation (model 2) explained an additional, significant 5%

of variance. Adolescent girls with parents who had relatively higher levels of depressive

symptoms had more depressive symptoms themselves.

Table 3. Multiple linear regression predicting adolescent girls' depressive symptoms from
parental depressive/anxiety symptoms

	Adolescent Depressive Symptoms					
	Model 1			Model 2		
Predictors	B	SE	β	B	SE	β
Race (black)	-.19	1.22	-.02	-.29	1.19	-.02
Age (y)	.38	.43	.10	.43	.42	.11
Puberty (pubertal)	-.86	1.51	-.07	-.61	1.47	-.05
Body fat (%)	-.04	.11	-.04	-.07	.10	-.07
Lean mass (kg)	.22	.10	.30*	.19	.09	.26*
Height (cm)	-.12	.10	-.14	-.12	.10	-.14
Mother diabetes (presence)	-4.03	1.44	-.26**	-3.60	1.41	-.24*
Parental depression/anxiety symptoms				.34	.13	.24**
Model R^2		.13*			.18**	
Δ R^2		.13*			.05**	

***$p \leq .001$. **$p \leq .01$. *$p \leq .05$. †$p \leq .10$.

After accounting for potential covariates (race, age, puberty, and presence of maternal diabetes), parental depression/anxiety symptoms were a significant predictor of adolescent BMI z-score and BMI, accounting for 3.8% of the variance in adolescent BMI z-score (p = .04), and 4% of the variance in adolescent BMI (p = .03). Parental depression/anxiety symptoms were positively related to adolescent girls being of heavier BMIs. Parental depression/ anxiety was not a significant predictor of any other metabolic risk factors for type 2 diabetes.

In logistic regression analysis, lean mass (OR: 1.16, p = .004), age (OR: .62, p = .02), and percent body fat (OR: 1.20, p = .001) were related to a greater odds of having hyperinsulinema. Lean mass was related to a greater odds of having impaired fasting glucose (OR: 1.19, p = .03), and percent body fat was related to a greater odds of having impaired glucose tolerance (OR: 1.18, p = .03). However, parental depressive/anxiety symptoms were not related to the odds of having hyperinsulinemia, impaired fasting glucose, or impaired glucose tolerance when accounting for race, age, puberty, percent fat, lean mass, height, and presence of maternal diabetes.

Table 4 displays the series of regression models evaluating adolescent coping as a predictor of adolescent depressive symptoms, accounting for all covariates as well as for parental depressive symptoms. Primary coping and secondary coping were unrelated to adolescent depressive symptoms. In contrast, disengagement coping, involuntary engagement coping, and involuntary disengagement coping were all predictive of greater adolescent depressive symptoms. Disengagement coping explained an additional, significant 9% of the variance in adolescent depressive symptoms, accounting for all covariates as well as parental depressive symptoms. Parental depression/anxiety, as well as lean mass and maternal diabetes, remained significant when disengagement coping was included in the model. Involuntary engagement coping

34

Table 4. Series of multiple linear regressions predicting adolescent girls' depressive symptoms from voluntary and involuntary coping

	Adolescent Depressive Symptoms														
	Voluntary Coping Models									Involuntary Coping Models					
	Model 1			Model 2			Model 3			Model 4			Model 5		
Predictors	B	SE	β	B	SE	β	B	SE	β	B	SE	β	B	SE	β
Race (black)	-.13	1.20	-.01	-.30	1.20	-.02	.47	1.15	.04	-.25	1.14	-.02	-.09	1.17	-.01
Age (y)	.43	.42	.11	.42	.42	.11	.36	.40	.10	.71	.41	.19†	.57	.41	.15
Puberty (pubertal)	-.65	1.48	-.05	-.62	1.48	-.05	-.25	1.40	-.02	-.33	1.41	-.03	-.42	1.44	-.03
Body fat (%)	-.09	.11	-.09	-.07	.10	-.07	-.04	.10	-.04	-.09	.10	-.09	-.08	.10	-.07
Lean mass (kg)	.20	.09	.27*	.20	.09	.26*	.19	.09	.25*	.12	.09	.17	.15	.09	.21
Height (cm)	-.13	.10	-.15	-.12	.10	-.14	-.11	.10	-.12	-.09	.10	-.10	-.09	.10	-.11
Mother diabetes (presence)	-3.22	1.46	-.21*	-3.61	1.42	-.24*	-3.43	1.34	-.22*	-3.83	1.35	-.25**	-3.57	1.38	-.23*
Parental depression/anxiety	.31	.13	.23*	.34	.13	.24*	.32	.12	.23*	.30	.12	.22*	.31	.13	.22*
Primary coping	-1.10	1.11	-.10	--	--	--	--	--	--	--	--	--	--	--	--
Secondary coping				.11	1.13	.01	--	--	--	--	--	--	--	--	--
Disengagement							3.46	.97	.31***	--	--	--	--	--	--
Involuntary engagement										3.22	.97	.31***	--	--	--
Involuntary disengagement													2.75	1.12	.23*
Model R^2	.19**			.18*			.27***			.26***			.23***		
Δ R^2	.01			.00			.09***			.08***			.05*		

***$p \leq .001$. **$p \leq .01$. *$p \leq .05$. †$p \leq .10$.

accounted for 8% of the variance in adolescent depressive symptoms. Parental

depression/anxiety, as well as maternal diabetes, remained significantly predictive of adolescent

depressive symptoms. Lastly, involuntary disengagement coping accounted for 5% of the

variance in adolescent depressive symptoms, and again, parental depressive/anxiety symptoms

and maternal diabetes remained significant. Adolescents that utilized more disengagement

coping, involuntary engagement coping, or involuntary disengagement coping had greater levels

of depressive symptoms. To explore the unique contribution of the different coping strategies,

we ran a single regression model that included all covariates, parental depression/anxiety, and

the three coping skills together (disengagement, involuntary engagement, and involuntary

disengagement) that were significant in the separate regressions at the $p < .05$ level. In this

model, only disengagement coping remained significant ($p = .03$). However, involuntary

disengagement approached significance ($p = .068$).

Table 5 displays the results determining to what extent parental depressive/anxiety

symptoms had an indirect effect on adolescent depression through each of the five adolescent

coping strategies. All five of the proposed mediation models were nonsignificant, indicating that

adolescent coping did not serve as a significant mediator of parental depressive/anxiety

symptoms and adolescent depressive symptoms.

Table 5. Indirect effects of parental depression/anxiety symptoms on adolescent girls' depressive symptoms through adolescents' coping strategies

Indirect path	α (SE)	β (SE)	Sobel test statistic (SE)	p
Primary coping	-.02 (.01)	-1.10 (1.11)	.89 (.02)	.37
Secondary coping	-.01 (.01)	.11 (1.13)	-.10 (.01)	.92
Disengagement coping	.01 (.01)	3.46 (.97)	.96 (.04)	.34
Involuntary engagement coping	.01 (.01)	3.22 (.97)	.96 (.03)	.34
Involuntary disengagement coping	.01 (.01)	2.75 (1.12)	.93 (.03)	.35

CHAPTER 5: DISCUSSION

This study examined the relationship of parental depressive/anxiety symptoms to depressive symptoms and metabolic risk factors for T2D in adolescent girls determined to be at high risk for T2D and the role that coping played within the association between parental and adolescent depressive symptoms. Specifically, the primary objective was to examine to what extent parental depressive/anxiety symptoms were related to adolescent depressive symptoms and metabolic risk factors for T2D. The secondary objective was to examine to what extent adolescent coping skills mediated the relationship between parental depressive symptoms and adolescent depressive symptoms.

Key Study Findings

Link between parental and adolescent depressive symptoms. Consistent with hypotheses and with past literature examining the relationship between parental and adolescent depression (Agerup et al., 2014; Nomura et al., 2002; Rice et al., 2002), the current results indicated that there was a positive and significant correlation between parental depressive/anxiety symptoms and depressive symptoms in adolescent girls at-risk for T2D. Furthermore, even after accounting for potential confounding and significant variables such as adolescent lean mass and maternal presence of diabetes, parental depressive symptoms remained a significant predictor of higher levels of adolescent depressive symptoms. Due to the cross-sectional nature of the study, it is not possible to identify whether parental depressive/anxiety symptoms cause more depressive symptoms in adolescent girls at-risk for T2D, or if the relationship is reversed. For example, on one hand, it is possible that parental depressive/anxiety symptoms affect adolescent depressive symptoms through mechanisms such as modeling of

depressed affect or intervening factors including adverse effects on parenting (Hayden, Olino, Mackrell, Jordan, Desjardins, & Katsiroumbas, 2013), increased parent-teen conflict (Cummings, Koss, & Davies, 2015), or impaired emotional support or unavailability (Easterbrooks, Bureau, & Lyons-Ruth, 2012). However, on the other hand, it is possible that adolescent depressive symptoms affect parental depressive symptoms. For instance, if an adolescent is suffering from depressive symptoms, the parent may feel unable to help his or her child, and in turn, experience depressive symptoms or anxiety about the adolescent's depressive symptoms. Yet a third scenario is also possible, that a third variable, such as family stress or shared genes, affects both the parent and adolescent. From a family systems approach, it is likely that the relationship between parent and adolescent depressive symptoms is bi-directional in nature (White & Klein, 2008; Bronfenbrenner, 1994), indicating that these three possible explanations are not mutually exclusive.

Link between parental depressive/anxiety symptoms and adolescent metabolic risk factors for type 2 diabetes. Parental depressive symptoms were positively related to some, but not all, adolescent metabolic risk factors for type 2 diabetes. Specifically, parental depressive/anxiety symptoms were positively associated with adolescent girls' BMI and BMI z-score, accounting for race, age, puberty, and presence of maternal diabetes. These findings are consistent with recent research in young children finding that parental depressive symptoms were associated with children's BMI through the first six years of the child's life (Morrissey & Dagher, 2014). Interestingly, in a recent, prior study of parental depression and BMI in adolescents with type 2 diabetes, there was no significant relationship between the two variables at the baseline time point (Weinstock et al., 2015). Therefore, the current findings extend the sparse literature

in this area and encourage further cross-sectional and longitudinal examinations of the connection between parental depression and child BMI during adolescence.

In contrast, parental depressive/anxiety symptoms were not related to any insulin or glucose indices or to percent body fat or lean mass, as indicated in previous research with adolescents at risk for depression (Mannie et al., 2013). This pattern of results suggests that any relationship of parental depressive/anxiety symptoms to insulin or glucose may operate indirectly through the relationship of parental depression/anxiety to adolescent BMI. One possible explanation that warrants testing is to what extent adolescents internalize their parents' depressive symptoms and that these behaviors manifest in ways that may increase adolescent BMI, such as overeating. For instance, adolescent BMI might be impacted by parental depressive symptoms through a lack of encouraging positive eating habits or failing to make healthy food choices available for the adolescent, leading to excessive weight. The positive observed relationship between parental depressive/anxiety symptoms and adolescent BMI indicates that efforts to reduce parental depression could potentially have implications for reducing adolescent BMI. Yet, this hypothesis warrants explicit testing. Alternatively, is it also possible that this relationship occurs in the opposite direction. Parents may feel responsible for their child's overweight, which may manifest in increased depressive or anxiety symptoms. It would be interesting to evaluate to what extent efforts to reduce adolescent BMI could have positive implications for reducing parental depressive/anxiety symptoms.

Coping as a mediator of parental and adolescent depressive symptoms. Inconsistent with my hypotheses, coping did not act as a significant mediator of the relationship between parental depressive/anxiety symptoms and adolescent depressive symptoms. Although disengagement coping, involuntary engagement coping, and involuntary disengagement coping

were all significant predictors of adolescent depressive symptoms, parental depressive/anxiety symptoms did not have an indirect effect of adolescent depressive symptoms through any adolescent coping measure. Instead, both parental depressive/anxiety symptoms and adolescent coping were uniquely, independently related to teen girls' depressive symptoms.

Inconsistent with prior research (Dunbar et al., 2013; Fear et al., 2009; Langrock et al., 2002), primary and secondary coping were unrelated to adolescent depressive symptoms. It is possible that this lack of association could be due to the specific nature of the population of adolescents at risk for T2D. For example, adolescents with mothers with T2D had less depressive symptoms than those without mothers with diabetes. This result, along with the lack of association between adolescent depressive symptoms and primary or secondary coping, indicates that there may be protective factors within these families that are not fully elucidated and merits further investigation. Within families with mothers with T2D, it is likely that adolescents may have different experiences than those without mothers with diabetes, and that these experiences shape how the adolescent interprets or responds to their parent's behaviors. For example, Jaser, Champion, Dharamsi, Riesing, and Compas (2011) found that among children living with mothers who suffered from clinical depression, having higher levels of positive mood or affect predicted less levels of depressive symptoms in the children. That is, those that experienced more positive moods or emotions had lower levels of depressive symptoms. Therefore, adolescent mood or affect may serve as a significant protective factor within the family and familial stress, in a way that is unique from how coping may mediate the relationship.

However, consistent with prior research (Black & Pössel, 2013; Dunbar et al., 2013; Langrock et al., 2002; Nicolai et al., 2013), disengagement coping, involuntary engagement

coping, and involuntary disengagement coping were all positively related to adolescent depressive symptoms. Disengagement coping and involuntary engagement coping have been related in past research to less effective regulation of negative affect and higher levels of depressive symptoms (Silk, Steinberg, & Morris, 2003). Stress and low self-esteem have also been identified as significant predictors of disengagement coping and depressive symptoms (Martyn-Nemeth, Penckofer, Gulanick, Velsor-Friedrich, & Bryant, 2008). Due to the relationship between obesity and T2D risk, it is possible that adolescents at risk for T2D may have lower self-esteem than their non-overweight peers. Furthermore, it is possible that within adolescents at risk for T2D, additional factors such as stress and emotion regulation may have a negative impact on the type of coping that youth utilize. On the one hand, it is possible that these factors make it more difficult to utilize more effective coping techniques, and in turn, increase depressive symptoms. For example, Calvete, Camara, Estevez, & Villardón (2011) identified that within females, disengagement coping specifically increased the impact that social stressors had on depressive symptoms, whereas secondary coping minimized these effects. On the other hand, it is possible that increased depression could cause increased utilization of these coping skills. For example, as adolescent depressive symptoms increase, it may become more and more difficult to utilize positive coping techniques, and in turn, coping such as disengagement, involuntary engagement, and involuntary disengagement, may become "negative ruts" to dealing with depressed affect.

It is possible that other family stressors impact the types of coping that adolescents utilize. Brown, Oudekerk, Szwedo, & Allen (2013) identified that inter-parent aggression while offspring were adolescents predicted greater usage of disengagement coping during young adulthood, indicating that conflict within the family system may also act negatively on

adolescent coping use. Additionally, it was identified that friendship competence, or social support, acted as a buffer on dependence on disengagement coping (Brown et al., 2013). Therefore, it is necessary to determine in adolescents at-risk for T2D whether providing youth with psychoeducation about coping styles- including in particular decreasing voluntary and involuntary disengagement and involuntary engagement- and could help adolescents to utilize more effective coping techniques to reduce depressive symptoms.

It is also possible that more maladaptive forms of coping are related to a specific form of depression that is not typically characterized, such as atypical depressive symptoms. Atypical depression includes behaviors such as over or under sleeping, and increased or decreased eating (Lee, Ng, & Tsang, 2009). It is possible that these symptoms of depression may increase the likelihood of utilizing techniques such as voluntary or involuntary disengagement, or that the opposite relationship exists that allows disengagement techniques to manifest into atypical depressive symptoms. Atypical depressive symptoms are more characteristic in females rather than in males (Lee, et al., 2009) and those with atypical features have been associated with an earlier age of onset of depressive symptoms (Matza, Revicki, Davidson, & Stewart, 2003). In this particular sample, the nature of being an adolescent female makes participants more likely to express atypical depressive symptoms compared to other groups at risk for type 2 diabetes.

Although there is limited prior research regarding coping as a mediator of parental and adolescent depressive symptoms, with only Langrock et al. (2002) finding secondary coping as a mediator, the current study was inconsistent with this finding. No coping mechanisms were significant mediators in the current study. Possible explanations of this finding include the limitation of utilizing only one time point for data collection, and also the potential for other factors to partially explain the relationship to a greater degree than adolescent coping skills.

There may be other covariates such as adolescent sleep or academic rigor that may be influencing the relationship and cannot be determined in this study. Additionally, there may be a third factor, such as stress or socioeconomic variables, which influences both parent depression/anxiety symptoms and adolescent depression symptoms. Future studies should address other important aspects of an adolescent's life that may play a role within both the parent's life and the adolescent's daily life. It is also likely that this study did not identify all types of stressors in an adolescent's life. Specifically, the current study utilized a measure of coping that looked only at social stress. It did not look at family stress, stress about health, or even academic stressors that adolescents at-risk for T2D may have to cope with on a routine basis. Future studies should identify other types of stressors most pertinent to adolescents at-risk for T2D and how youth cope with them. Finally, it is possible that coping may act as a moderator in this relationship, rather than a mediator, and this was not something that was evaluated.

Study Strengths and Limitations

The current study had a variety of different strengths and limitations. Addressing the study's strengths and limitations are important to understand how to best integrate the results with prior research and how to more effectively direct future research.

Strengths. This particular study had a relatively large sample size, which in post-hoc analyses was adequate to detect moderate and large size effects. Additionally, there was very little missing data. The study also included measures that were consistent in both reliability and validity, speaking to the integrity of the findings. The sample also was racially/ethnically diverse, with a majority of non-Hispanic Black youth, a racial group that is disproportionately affected by T2D (Pettitt et al., 2014).

Limitations. There were some limitations of the current study. In particular, we were only able to utilize one time point. As such, it was not possible to infer causation or directionality. Additionally, mediation is most appropriately tested with a longitudinal study design rather than a cross-sectional study design. While a majority of respondents were the participant's mothers, a few fathers participated, and we were not able to explicitly determine whether a mother or father filled out the depressive/anxiety questionnaire. This is important in shaping our conclusions because the majority of previous research has studied maternal or paternal depressive symptoms independently. Because we were not able to determine which parent responded, it limits our ability to compare to past research and make inferences about specific intervention or prevention efforts. Finally, this study only used girls with elevated depressive symptoms that were at risk for T2D. Therefore, it is not possible to make general statements or recommendations for all adolescents based on the results of the study.

Conclusions and Future Directions

In this study, parental depressive/anxiety symptoms were positively related to adolescent depressive symptoms and adolescent BMI. Disengagement coping, involuntary engagement coping, and involuntary disengagement coping were all significant predictors of adolescent depressive symptoms. However, none of the coping techniques served as a mediator between parental depressive/anxiety symptoms and adolescent depressive symptoms.

More research is needed to evaluate coping as a mediator of parental and adolescent depressive symptoms over time as compared to a single, cross-sectional time point. Future studies should also aim characterize the protective factors that exist within families at risk for T2D, which help to mitigate teens' risk of T2D and depression. Such knowledge will help guide future intervention and prevention efforts to lessen the burden of personal and familial T2D on

adolescents. Additionally, examining whether there are factors other than coping mechanisms that better explain the relationship between parental and adolescent depressive symptoms is necessary to best understand the complicated relationship between parental and adolescent depressive symptoms in families at risk for T2D.

REFERENCES

Achenbach, T.M., & Rescorla, L.A. (2003). *Manual for the ASEBA adult forms and profiles*.
Burlington, VT: University of Vermont, Research Center for Children, Youth, and
Families.

Ali, S., Stone, M.A., Peters, J.L., Davies, M.J., & Khunti, K. (2006). The prevalence of co-
morbid depression in adults with type 2 diabetes: A systematic review and meta-analysis.
Diabetic Medicine, 23, 1165-1173. doi: 10.1111/j.1464-5491.2006.01943.x

American Diabetes Association. (2013). Diagnosis and classification of diabetes mellitus.
Diabetes Care, 36(Suppl. 1), S67–S74. doi: 10.2337/dc13-S067.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders*
(5th ed.). Arlington, VA: American Psychiatric Publishing.

Agerup, T., Lydersen, S., Wallander, J., & Sund, A.M. (2014). Longitudinal course of
diagnosed depression from ages 15 to 20 in a community sample: Patterns and parental
risk factors. *Child Psychiatry and Human Development.* In print. doi: 10.1007/s10578-
014-0444-8

Auslander, W.F., Sterzing, P.R., Zayas, L.E., & White, N.H. (2010). Psychosocial resources and
barriers to self- management in African American adolescents with type 2 diabetes: A
qualitative analysis. *The Diabetes Educator, 36,* 613-622. doi:
10.1177/0145721710369706

Bener, A., Darwish, S., Al-Hamaq, A.O.A., Yousafzai, M.T., & Nasralla, E.A. (2014). The
potential impact of family history of metabolic syndrome and risk of type 2 diabetes

mellitus: In a highly endogamous population. *Indian Journal of Endocrinology and Metabolism, 18*, 202-209. doi: 10.4103/2230-8210.129112

Black, S.W., & Pössel, P. (2013). The combined effects of self-referent information processing and ruminative responses on adolescent depression. *Journal of Youth and Adolescence, 42,* 1145-1154. doi: 10.1007/s10964-012-9827-y

Bronfenbrenner, U. (1994). Ecological models of human development. In *International Encyclopedia of Education, Vol. 3, 2nd. Ed.* Oxford: Elsevier. Reprinted in: Gauvain, M., & Cole, M. (Eds.), *Readings on the development of children, 2nd Ed.* (1993, pp. 37-43). NY: Freeman.

Brown, C.L., Oudekerk, B.A., Szwedo, D.E., & Allen, J.P. (2013). Inter-parent aggression as a precursor to disengagement coping in emerging adulthood: The buffering role of friendship competence. *Social Development, 22*(4): 683-700. doi: 10.1111/sode.12026

Cali, A.M.G., Man, C.D., Cobelli, C., Dziura, J., Seyal, A., Shaw, M., …Caprio, S. (2009). Primary defects in beta-cell function further exacerbated by worsening of insulin resistance mark the development of impaired glucose tolerance in obese adolescents. *Diabetes Care, 32*(3), 456-461. doi: 10.2337/dc08-1274

Calvete, E., Camara, M., Estevez, A., & Villardón, L. (2011). The role of coping with social stressors in the development of depressive symptoms: Gender differences. *Anxiety, Stress, & Coping, 24*(4), 387-406. doi: 10.1080/10615806.2010.515982

Carnethon, M.R., Biggs, M.L., Barzilay, J.I., Smith, N.L., Vaccarino, V., Bertoni, A.G., …Siscovick, D. (2007). Longitudinal association between depressive symptoms and incident type 2 diabetes mellitus in older adults: The cardiovascular health study. *Archives of Internal Medicine, 167,* 802-807.

Centers for Disease Control and Prevention. (2010). Current depression among adults—United

    States, 2006 and 2008. *Morbidity and Morality Weekly Report, 59,* 1229-1235.

Centers for Disease Control and Prevention. (2011). National diabetes fact sheet: national

    estimates and general information on diabetes and prediabetes in the United States, 2011.

    Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease

    Control and Prevention; 2011.

Compas, B.E., Champion, J.E., Forehand, R., Cole, D.A., Reeslund, K.L., Fear, J., …Roberts, L.

    (2010). Coping and parenting: Mediators of 12-month outcomes of a family group

    cognitive-behavioral preventative intervention with families of depressed parents.

    Journal of Consulting and Clinical Psychology, 78, 623-634. doi: 10.1037/a0020459

Connor-Smith, J.K., Compas, B.E., Wadsworth, M.E., Thomsen, A.H., & Saltzman, H. (2000).

    Responses to stress in adolescence: measurement of coping and involuntary stress

    responses. *Journal of Consulting and Clinical Psychology, 68*, 976-992. doi:

    10.1037/0022-006X.68.6.976

Cummings, E.M., Koss, K.J., & Davies, P.T. (2015). Prospective relations between family

    conflict and adolescent maladjustment: Security in the family system as a mediating

    process. *Journal of Abnormal Child Psychology, 43*(3), 503-515. doi: 10.1007/s10802-

    014-9926-1

Dabelea, D., & Harrod, C.S. (2013). Role of developmental overnutrition in pediatric obesity

    and type 2 diabetes. *Nutrition Reviews, 71,* (Suppl. 1), S62-S67. doi:

    10.1111/nure.12061

Dabelea, D., Mayer-Davis, E.J., Lamichhane, A.P., D'Agostino, R.B. Jr., Liese, A.D., Vehik,

    K.S., …Hamman, R.F. (2008). Association of intrauterine exposure to maternal diabetes

and obesity with type 2 diabetes in youth: The SEARCH Case-Control Study. *Diabetes Care, 31*, 1422–1426. doi: 10.2337/dc07-2417

Dabelea, D., Mayer-Davis, E.J., Saydah, S., Imperatore, G., Linder, B., Divers, J., …Hamman, R.F. (2014). Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. *Journal of the American Medical Association, 311*(17), 1778-1786. doi: 10.1001/jama.2014.3201

Downey, G., & Coyne, J.C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin, 108,* 50-76.

Dunbar, J.P., McKee, L., Rakow, A., Watson, K.H., Forehand, R., & Compas, B.E. (2013). Coping, negative cognitive style and depressive symptoms in children of depressed parents. *Cognitive Therapy and Research, 37,* 18-28. doi: 10.1007/s10608-012-9437-8

Easterbrooks, M.A., Bureau, J.F., & Lyons-Ruth, K. (2012). Developmental correlates and predictors of emotional availability in mother-child interaction: a longitudinal study from infancy to middle childhood. *Developmental Psychopathology, 24*(1), 65-78. doi: 10.1017/S0954579411000666

Eaton, W.W., Armenian, H., Gallo, J., Pratt, L., & Ford, D.E. (1996). Depression and risk for onset of type II diabetes. *Diabetes Care, 19,* 1097- 1102.

Eckshtain, D., Ellis, D.A., Kolmodin, K., & Naar-King, S. (2010). The effects of parental depression and parenting practices on depressive symptoms and metabolic control in urban youth with insulin dependent diabetes. *Journal of Pediatric Psychology, 35*(4), 426-435. doi:10.1093/jpepsy/jsp068

Eldredge, K.L., & Agras, W.S. (1997). The relationship between perceived evaluation of weight and treatment outcome among individuals with binge eating disorder. *International Journal of Eating Disorders, 22*, 43-49.

Everson-Rose, S.A., Meyer, P.M., Powell, L.H., Pandey, D., Torrens, J.I., Kravitz, H.M., … Matthews, K.A. (2004). Depressive symptoms, insulin resistance, and risk for diabetes in women at midlife. *Diabetes Care, 27,* 2856-2862.

Fear, J.M., Champion, J.E., Reeslund, K.L., Forehand, R., Colletti, C., Roberts, L., & Compas, B.E. (2009). Parental depression and interparental conflict: Children and adolescents' self-blame and coping responses. *Journal of Family Psychology, 23,* 762-766. doi: 10.1037/a0016381

Ford, D.E., & Erlinger, T.P. (2004). Depression and C-reactive protein in US adults: data from the Third National Health and Nutrition Examination Survey. *Archives of Internal Medicine, 164* (9), 1010-1014. doi: 10.1001/archinte.164.9.1010

Franks, P.W., Hanson, R.L., Knowler, W.C., Moffett, C., Enos, G., Infante, A.M., …Looker, H.C. (2007). Childhood predictors of young-onset type 2 diabetes. *Diabetes, 56,* 2964-2972. doi: 10.2337/db06-1639

Golden, S.H., Williams, J.E., Ford, D.E., Yeh, H., Sanford, C.P., Nieto, F.J., & Brancati, F.L. (2004). Depressive symptoms and the risk of type 2 diabetes: The atherosclerosis risk in communities study. *Diabetes Care, 27,* 429-435.

Golden, S.H., Lazo, M., Carnethon, M., Bertoni, A.G., Schreiner, P.J., Diez Roux, A.V., … Lyketsos, C. (2008). Examining a bidirectional association between depressive symptoms and diabetes. *Journal of the American Medical Association, 299,* 2751-2759. doi: 10.1001/jama.299.23.2751

Gregg, E.W., Zhuo, X., Cheng, Y.J., Albright, A.L., Narayan, K.M., & Thompson, T.J. (2014). Trends in lifetime risk and years of life lost due to diabetes in the USA, 1985-2011: A modeling study. *The Lancet Diabetes & Endocrinology,* In Press. doi: 10.1016/S2213-8587(14)70161-5

Hannon, T.S., Janosky, J., & Arslanian, S.A. (2006). Longitudinal study of physiological insulin resistance and metabolic changes of puberty. *Pediatric Research, 60,* 759-763. doi: 10.1203/01.pdr.0000246097.73031.27

Hannon, T.S., Rofey, D.L., Lee, S.J., & Arslanian, S.A. (2013). Depressive symptoms and metabolic markers of risk for type 2 diabetes in obese adolescents. *Pediatric Diabetes, 14,* 497-503. doi: 10.1111/pedi.12035

Hayden, E. P., Olino, T. M., Mackrell, S. V. M., Jordan, P. L., Desjardins, J., & Katsiroumbas, P. (2013). Cognitive vulnerability to depression during middle childhood: Stability and associations with maternal affective styles and parental depression. *Personality and Individual Differences*, *55*(8), 892–897. doi:10.1016/j.paid.2013.07.016

Hillier, T.A., Pedula, K.L., Schmidt, M.M., Mullen, J.A., Charles, M.A., & Pettitt, D.J. (2007). Child obesity and metabolic imprinting. *Diabetes Care, 30,* 2287-2292. doi: 10.2337/dc06-2361

Irving, R.R., Mills, J.L., Choo-Kang, E.G., Mclaughlin, W., Wright-Pascoe, R., Morrison, E.Y., & Brown, P. (2008). Diabetes and psychological co-morbidity in children with a family history of early-onset type 2 diabetes. *International Journal of Psychology, 46,* 937-942. doi: 10.1080/00207590802547308

Jaser, S.S., Holl, M.G., Jefferson, V., & Grey, M. (2009). Correlates of depressive symptoms in urban youth at risk for type 2 diabetes mellitus. *Journal of School Health, 79,* 286-292.

Jaser, S.S., Champion, J.E., Dharamsi, K.R., Riesing, M.M., & Compas, B.E. (2011). Coping and positive affect in adolescents of mothers with and without a history of depression. *Journal of Child and Family Studies, 20*(3), 353-360. doi:10.1007/s10826-010-9399-y

Jeong, S.U., Kang, D.G., Lee, D.H., Lee, K.W., Lim, D.M., Kim, B.J., … Koh, G. (2010). Clinical characteristics of type 2 diabetes patents according to family history of diabetes. *Korean Diabetes Journal, 34* (4), 222-228. doi: 10.4093/kdj.2010.34.4.222

Kaar, J.L., Crume, T., Brinton, J.T., Bischoff, K.J., McDuffie, R., & Dabelea, D. (2014). Maternal obesity, gestational weight gain, and offspring adiposity: The exploring perinatal outcomes among children study. *The Journal of Pediatrics, 165,* 509-515. doi: 10.1016/j.jpeds.2014.05.050

Kovacs, M., & Beck, A. T. An empirical-clinical approach toward a definition of childhood depression. In: Raskin JGSA, ed. *Depression in childhood: diagnosis, treatment, and conceptual models.* NY: Raven.

Kuczmarski, R.J., Ogden, C.L., Grummer-Strawn, L.M., Flegal, K.M., Guo, S.S., Wei, R., … Johnson, C.L. (2000). CDC growth charts: United States. *Advance Data,* (314), 1-27.

Langrock, A.M., Compas, B.E., Keller, G., Merchant, M.J., & Copeland, M.E. (2002). Coping with the stress of parental depression: Parents' reports of children's coping, emotional, and behavioral problems. *Journal of Clinical Child and Adolescent Psychology, 31,* 312-324. doi: 10.1207/S15374424JCCP3103_03

Lawrence, J.M., Standiford, D.A., Loots, B., Klingensmith, G.J., Williams, D.E., Ruggiero, A., …McKeown, R.E. (2006). Prevalence and correlates of depressed mood among youth with diabetes: The SEARCH for Diabetes in Youth Study. *Pediatrics, 117,* 1348-1358. doi: 10.1542/peds.2005-1398

Lazarus, R.S., & Folkman, S. (1984). *Psychological stress and the coping process.* New York, NY; Springer.

Lee, S., Ng, K.L., & Tsang, A. (2009). Prevalence and correlates of depression with atypical symptoms in Hong Kong. *Australian and New Zealand Journal of Psychiatry, 43,* 1147-1154. doi: 10.3109/00048670903279895

Lewinsohn, P.M., Shankman, S.A., Gau, J.M., & Klein, D.N. (2004). The prevalence and co-morbidity of subthreshold psychiatric conditions. *Psychological Medicine, 34,* 613-622. doi: 10.1017/S0033291703001466

Lustman, P.J., & Clouse, R.E. (2005). Depression in diabetic patients: The relationship between mood and glycemic control. *Journal of Diabetes and Its Complications, 19,* 113-122. doi: 10.1016/j.jdiacomp.2004.01.002

Mackey, E.R., Struemph, K., Powell, P.W., Chen, R., Streisand, R., & Holmes, C.S. (2014). Maternal depressive symptoms and disease care status in youth with type 1 diabetes. *Health Psychology, 33*(8), 783-791. doi: 10.1037/hea0000066

MacKinnon, D.P., Lockwood, C.M., & Hoffman, J.M. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7,* 83-104.

Mannie, Z.N., Williams, C., Diesch, J., Steptoe, A., Leeson, P., & Cowen, P.J. (2013). Cardiovascular and metabolic risk profile in young people at familial risk of depression. *The British Journal of Psychiatry, 203,* 18-23. doi: 10.1192/bjp.bp.113.126987

Marshall, W.A., & Tanner, J.M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood, 44,* 291- 303.

Martyn-Nemeth, P., Penckofer, S., Gulanick, M., Velsor-Friedrich, B., & Bryant, F.B. (2009). The relationships among self-esteem, stress, coping, eating behavior, and depressive

mood in adolescents. *Research in Nursing and Health, 32,* 96-109. doi:
10.1002/nur.20304

Matsuda, M., & DeFronzo, R.A. (1999). Insulin sensitivity indices obtained from oral glucose
tolerance testing: comparison with the euglycemic insulin clamp. *Diabetes Care, 22*,
1462-1470.

Matza, L.S., Revicki, D.A., Davidson, J.R., & Stewart, J.W. (2003). Depression with atypical
features in the National Comorbidity Survey: Classification, description, and
consequences. *Archives of General Psychiatry, 60*(8) 817-826.
doi:10.1001/archpsyc.60.8.817

Meigs, J.B., Cupples, L.A., & Wilson, P.W. (2000). Parental transmission of type 2 diabetes: the
Framingham Offspring Study. *Diabetes*, *49*(12):2201-2207.

Mezuk, B., Eaton, W.W., Albrecht, S., & Golden, S.H. (2008). Depression and type 2 diabetes
over the lifespan. *Diabetes Care, 31,* 2383-2390. doi: 10.2337/dc08-0985

Molyneaux, L., Constantino, M., & Yue, D. (2004). Strong family history predicts a younger
age of onset for subjects diagnosed with type 2 diabetes. *Diabetes, Obesity, and
Metabolism, 6*, 187-194.

Morrison, J.A., Glueck, C.J., Horn, P.S., & Wang, P. (2010). Childhood predictors of adult type
2 diabetes at 9- and 26- year follow-ups. *Archives of Pediatrics & Adolescent Medicine,
164*, 53-60. doi: 10.1001/archpediatrics.2009.228

Morrissey, T.W., & Dagher, R.K. (2014). A longitudinal analysis of maternal depressive
symptoms and children's food consumption and weight outcomes. *Public Health
Nutrition, 17*(12), 2759-2768. doi: 10.1017/S1368980013003376

Mulvaney, S.A., Mudasiru, E., Schlundt, D.G., Baughman, C.L., Fleming, M., VanderWoude, A., …Rothman, R. (2008). Self-management in type 2 diabetes: The adolescent perspective. *The Diabetes Educator, 34,* 674-682. doi: 10.1177/0145721708320902

Negriff, S., & Susman, E.J. (2011). Pubertal timing, depression, and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on Adolescence, 21*(3), 717-746. doi: 10.1111/j.1532-7795.2010.00708.x

Nicolai, K.A., Laney, T., & Mezulis, A.H. (2013). Different stressors, different strategies, different outcomes: How domain-specific stress responses differentially predict depressive symptoms among adolescents. *Journal of Youth and Adolescence, 42,* 1183-1193. doi: 10.1007/s10964-012-9866-4

Noruma, Y., Wickramaratne, P.J., Warner, V., Mufson, L., & Weissman, M.M. (2002). Family discord, parental depression, and psychopathology in offspring: Ten-year follow up. *Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 402-409.

Ogden, C.L., & Flegal, K.M. (2010). Changes in terminology for childhood overweight and obesity. *National Health Statistics Reports, 25,* 1-5.

Pan, A., Ye, X., Franco, O.H., Li, H., Yu, Z., Zou, S., … Lin, X. (2008). Insulin resistance and depressive symptoms in middle-aged and elderly Chinese: Findings from the Nutrition and Health of Aging Population in China Study. *Journal of Affective Disorders, 109,* 75-82. doi: 10.1016/j.jad.2007.11.002

Pavkov, M.E., Bennett, P.H., Knowler, W.C., Krakoff, J., Sievers, M.L., & Nelson, R.G. (2006). Effect of youth-onset type 2 diabetes mellitus on incidence of end-stage renal disease and mortality in young and middle-aged Pima Indians. *Journal of the American Medical Association, 296*(4), 421-426.

Pearson, S., Schmidt, M., Patton, G., Dwyer, T., Blizzard, L., Otahal, P., & Venn, A. (2010).

> Depression and insulin resistance: Cross-sectional associations in young adults. *Diabetes Care, 33,* 1128-1133. doi: 10.2337/dc09-1940

Pettitt, D.J., Talton, J., Dabelea, D., Divers, J., Imperatore, G., Lawrence, J.M., … Standiford, D.A. (2014). Prevalence of diabetes in U.S. youth in 2009: The SEARCH for diabetes in youth study. *Diabetes Care, 37,* 402- 408. doi: 10.2337/dc13-1838

Ravaja, N., & Keltikangas-Jarvinen, L. (1995). Temperament and metabolic syndrome precursors in children: A three-year follow-up. *Preventative Medicine, 24,* 518-527. doi: 10.1006/pmed.1995.1082

Rice, F., Harold, G., & Thapar, A. (2002). The genetic aetiology of childhood depression: A review. *Journal of Child Psychology and Psychiatry, 43,* 65-79.

Rice, F., Lewis, G., Harold, G.T., & Thapar, A. (2013). Examining the role of passive gene-environment correlation in childhood depression using a novel genetically sensitive design. *Development and Psychopathology, 25,* 37-50. doi: 10.1017/S0954579412000880

Rosenbloom, A.L. (2002). Increasing incidence of type 2 diabetes in children and adolescents: treatment considerations. *Pediatric Drugs, 4*, 209-221.

Rotella, F., & Mannucci, E. (2013). Depression as a risk factor for diabetes: a meta-analysis of longitudinal studies. *Journal of Clinical Psychiatry, 74* (1), 31-37. doi: 10.4088/JCP.12r07922

Saydah, S.H., Brancati, F.L., Golden, S.H., Fradkin, J., & Harris, M.I. (2003). Depressive symptoms and the risk of type 2 diabetes mellitus in a US sample. *Diabetes/Metabolism Research and Reviews, 19,* 202-208. doi: 10.1002/dmrr.353

Scherrer, J.F., Garfield, L.D., Chrusciel, T., Hauptman, P.J., Carney, R.M., Freedland, K.E.,

 …Lustman, P.J. (2011). Increased risk of myocardial infarction in depressed patients

 with type 2 diabetes. *Diabetes Care, 34,* 1729-1734. doi: 10.2337/dc11-0031

Schwenk, R.W., Vogel, H., & Schurmann, A. (2013). Genetic and epigenetic control of

 metabolic health. *Molecular Metabolism, 2,* 337-347. doi:

 10.1016/j.molmet.2013.09.002

Séguin, M., Manion, I, Cloutier, P., McEnvoy, L., & Cappelli, M. (2003). Adolescent

 depression, family psychopathology and parent/child relations: A case control study. *The*

 Canadian Child and Adolescent Psychiatry Review, 12, (1), 1-9.

Shomaker, L.B., Tanofsky-Kraff, M., Young-Hyman, D., Han, J.C., Yanoff, L.B., Brady, S.M.,

 …Yanovski, J.A. (2010). Psychological symptoms and insulin sensitivity in adolescents.

 Pediatric Diabetes, 11, 417-423. doi: 10.1111/j.1399-5448.2009.00606.x

Shomaker, L.B., Tanofksy-Kraff, M., Stern, E.A., Miller, R., Zocca, J.M., Field, S.A.,

 …Yanovski, J.A. (2011). Longitudinal study of depressive symptoms and progression of

 insulin resistance in youth at risk for adult obesity. *Diabetes Care, 34,* 2458-2463. doi:

 10.2337/dc11-1131

Silk, J.S., Steinberg, L., & Morris, A.S. (2003). Adolescents' emotion regulation in daily life:

 Links to depressive symptoms and problem behavior. *Child Development, 74*(6), 1869-

 1880. doi: 10.1046/j.1467-8624.2003.00643.x

Smucker, M.R., Craighead, W.E., Craighead, L.W., & Green, B.J. (1986). Normative and

 reliability data for the Children's Depression Inventory. *Journal of Abnormal Child*

 Psychology, 14(1), 25-39.

Soonthornpun, S., Setasuban, W., Thamprasit, A., Chayanunnukul, W., Rattarasarn, C., & Geater,

A. (2003). Novel insulin sensitivity index derived from oral glucose tolerance test.
Journal of Clinical Endocrinology and Metabolism, 88, 1019-1023. doi:
10.1210/jc.2002-021127

Strine, T.W., Mokdad, A.H., Balluz, L.S., Gonzalez, O., Crider, R., Berry, J.T., & Kroenke, K.
(2008). Depression and anxiety in the United States: Findings from the 2006 Behavioral
Risk Factor Surveillance System. *Psychiatric Services, 59,* 1383-1390. doi:
10.1176/appi.ps.59.12.1383

Temelkova-Kurktschiev, T., & Stefanov, T.S. (2012). Lifestyle and genetics in obesity and type
2 diabetes. *Experimental and Clinical Endocrinology & Diabetes, 120,* 1-6. doi:
10.1055/s-0031-1285832

Timonen, M., Rajala, U., Jokelainen, J., Keinänen-Kiukaanniemi, S., Meyer-Rochow, V.B., &
Räsänen, P. (2006). Depressive symptoms and insulin resistance in young adult males:
Results from the Northern Finland 1966 birth cohort. *Molecular Psychiatry, 11,* 929-933.
doi: 10.1038/sj.mp.4001838

Tukey, J.W. (1977). *Exploratory data analysis.* Reading: Addison-Wesley.

U.S. Department of Health and Human Services, National Institutes of Heath, National Institute
of Diabetes and Digestive and Kidney Diseases. (2008). *Insulin resistance and
prediabetes* (NIH Publication No. 09-4893). Retrieved from
http://diabetes.niddk.nih.gov/dm/pubs/insulinresistance/insulinresistance_508.pdf

van Dam, N.T., & Earleywine, M. (2011). Validation of the Center for Epidemiologic Studies
Depression Scale- Revised (CESD-R): Pragmatic depression assessment in the general
population. *Psychiatry Research, 186*, 128-132. doi: 10.1016/j.psychres.2010.08.018

Weinstock, R.S., Trief, P.M., El ghormli, L., Goland, R., McKay, S., Milazewski, K., … Yasuda, P.M. (2015). Parental characteristics associated with outcomes in youth with type 2 diabetes: Results from the TODAY Clinical Trial. *Diabetes Care, 38,* 784-792. doi: 10.2337/dc14-2393

Weiss, R., Dziura, J., Burgert, T.S., Tamborlane, W.V., Taksali, S.E., Yeckel, C.W., … Caprio, S. (2004). Obesity and the metabolic syndrome in children and adolescents. *The New England Journal of Medicine, 350,* 2362-2374. doi: 10.1056/NEJMoa031049

White, J. M., & Klein, D. M. (2008). *Family theories* (3rd ed.). Thousand Oaks, CA: Sage.

Yeckel, C.W., Weiss, R., Dziura, J., Taksali, S.E., Dufour, S., Burgert, T.S., … Caprio, S. (2004). Validation of insulin sensitivity indices from oral glucose tolerance test parameters in obese children and adolescents*. Journal of Clinical Endocrinology and Metabolism, 89,* 1096-1101. doi: 10.1210/jc.2003-031503

Zhang, C.X., Tse, L.A., Yet, X.Q., Lint, F.Y., Chen, Y.M., & Chen, W.Q. (2009). Moderating effects of coping styles on anxiety and depressive symptoms caused by psychological stress in Chinese patients with type 2 diabetes. *Diabetic Medicine, 26,* 1282-1288. doi: 10.1111/j.1464-5491.2009.02840.x