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The relations of metabolic syndrome to anxiety
and depression symptoms in children and adults

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Abstract

Metabolic syndrome is a cluster of five factors (elevated systolic blood pressure, elevated blood glucose, elevated triglycerides, large waist circumference, and decreased HDL) that are related to a greater chance of heart disease, stroke, and diabetes. There is evidence that metabolic syndrome is correlated with depression, but the directionality and mechanism is unclear. There is also dispute in the literature as to whether there is a correlation with anxiety and metabolic syndrome. In this study, levels of depression and anxiety determined from questionnaires and interviews (Adult Self Report, Child Behavior Checklist, Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime, and the Composite International Diagnostic Interview) were compared with the five factors of metabolic syndrome in 100 three-person families. In children and adolescents, elevated triglycerides were predictive of elevated depressive behavior above the age of 12.68 ($p < .05$), with the significance increasing with age. In adults, anxious behavior was predictive of overall metabolic risk, HDL, and waist circumference when controlling for age, sex, and SES ($p < .01$, $p < .05$, and $p < .05$ respectively). Additionally, a lower SES, older age, greater anxious behavior, and being male were all predictive of greater overall metabolic risk. Results implicate an age-moderated difference in how metabolic factors affect depression in children, possibly having a mechanism coinciding or affected by puberty. In adults, the directionality seems to reverse, with the anxious behavior having an effect on the metabolic syndrome factor, possibly related to stress and inflammation. Further research is needed to study these mechanisms and elucidate the connections between the disorders.

Table of Contents

Introduction

Metabolic Syndrome

Metabolic syndrome is one of the most formidable challenges in contemporary public health (Kaur, 2014). The disease, defined as a group of risk factors, including visceral obesity, unhealthy blood fat content, high blood pressure, and high blood sugar, is associated with three of the six leading causes of death worldwide: cardiovascular disease, stroke, and diabetes (WHO World Health Report, 2017). Individuals with metabolic syndrome are five times more likely to develop type 2 diabetes, two times more likely to develop cardiovascular disease within 5 to 10 years of being diagnosed with metabolic syndrome, and 2 to 4 times more likely to suffer a stroke (Alberti et al., 2009; Alberti, Zimmet, Shaw, & Group, 2005). In the United States, between 2003 to 2012, the overall prevalence of the syndrome was 33%, with significantly higher rates among females (35.6%) than males (30.3%; (Aguilar, Bhuket, Torres, Liu, & Wong, 2015). Additionally, age is a risk factor for metabolic syndrome, with a prevalence of 50% in adults over the age of 60 (Aguilar et al., 2015).

More specifically, the National Institutes of Health National Heart, Lung, and Blood Institute (2016) defines metabolic syndrome as the presence of a cluster of at least three of the following factors in an adult over age 21:

1.) Large waist circumference (>40 inches for men; >35 inches for women)

2.) High triglycerides (>150 mg/dL)

3.) Low high-density lipoprotein (HDL), commonly referred to as "good cholesterol" (<40 mg/dL for men; <50 mg/dL for women)

4.) High fasting glucose (>100 mg/dL)

5.) Elevated blood pressure (>130/85 mm HG)

A BMI classified as "obese" (i.e. over 30 kg/m^2), though not an explicit component of the NIH's definition of metabolic syndrome, is also considered a risk factor for metabolic problems (Kaur, 2014). Each of these factors is related to metabolic risk, for related, but slightly different, reasons.

Larger waist circumference is indicative of increased stores of adipose tissue in the abdomen, often resulting from low physical activity and the consumption of calorie-dense foods (Kaur, 2014). Adipose tissue stores triglycerides, and contains a variety of cell types, such as adipocytes and immune cells (Halberg, Wernstedt-Asterholm, & Scherer, 2008; Schmidt et al., 2015). An adipocyte is an endocrine cell, known to secrete substances including cortisol, a stress hormone, and pro-inflammatory factors, such as cytokines (Lau, Dhillon, Yan, Szmitko, & Verma, 2005). When these substances are secreted, they can cause an adverse metabolic effect, as the adipose tissue's local inflammation proliferates into systemic chronic low-grade inflammation (Trayhurn & Wood, 2004). Inflammation often precedes the development of diabetes, and is predictive of future weight gain (Engstrom et al., 2003; Rodriguez-Hernandez, Simental-Mendia, Rodriguez-Ramirez, & Reyes-Romero, 2013; Thorand et al., 2003).

HDL ("good cholesterol") functions by carrying low-density lipoproteins ("bad cholesterol") to the liver, which then removes them. High levels of low-density lipoproteins are related to artery hardening, which results in impeded blood flow. Therefore, low levels of low-density lipoprotein have negative metabolic effects, whereas high levels HDL promote good metabolic health (Mayo Clinic, 2015; American Heart Association, 2017).

Triglycerides are a measure of the amount of lipids in the blood. They store unused calories and provide the body with energy when there is low blood glucose between meals. High levels of triglycerides contribute to artery wall thickening and hardening, known as atherosclerosis, and are linked to the increased risks of stroke and cardiovascular disease (American Heart Association, 2017; Mayo Clinic, 2015).

Fasting glucose levels are a measure of the amount of glucose in the blood after fasting for at least eight hours. Glucose, a simple sugar, is derived from food and is used to generate energy for the body's cells. After eating, glucose enters the bloodstream via the intestines, and results in a blood sugar "spike". After fasting, if levels are high it indicates that the body is not utilizing insulin properly for transporting glucose into cells. This suggests the presence of diabetes or a prediabetic state, and over time can also lead to problems such as heart attacks and strokes (American Diabetes Association, 2017; Joslin Diabetes Center, 2017).

Systolic blood pressure refers to the pressure in the arteries during cardiac muscle contraction (ventricular systole). A high systolic blood pressure may be caused by an unhealthy lifestyle, medications, and stress-induced vasoconstriction (artery narrowing) among other reasons. High systolic blood pressure is a known risk factor of cardiovascular disease and stroke (Reece, Wasserman, Campbell, & Yunagisawa, 2011).

Finally, a high BMI is indicative of large quantities of visceral body fat, and can be regarded as a predictor of metabolic syndrome independent of waist circumference. In clinical practice and research, BMI can serve as a simple indicator of body fat and metabolic risk (Janssen, Heymsfield, Allison, Kotler, & Ross, 2002).

Psychological Connections

In addition to negative physiological outcomes, several studies have indicated a link between metabolic syndrome and psychiatric disorders. In particular, these associations have been well studied in relation to schizophrenia and bipolar disorder. Metabolic syndrome has a comorbidity rate as high as 60% among individuals with schizophrenia (Meyer, Koro, & L'Italien, 2005). The literature suggests many possible reasons, including side effects of medication usage, lifestyle, and a common pathogenic pathway involving sphingolipids. Amongst their other functions sphingolipids are involved in inflammation. Inflammation as a result of obesity can lead to glucose intolerance. It can also reduce white matter and myelination in the brain, leading to abnormal brain circuitry and psychotic symptoms resulting in schizophrenia (Castillo et al., 2016).

Bipolar disorder is also associated with a higher risk of metabolic syndrome than in the general population (McIntyre et al., 2010). Even when controlling for medication and health habits, individuals with bipolar disorder have been shown to be at greater cardiovascular risk. This has lead researchers to believe that dysregulated inflammatory pathways may underlie these disorders (Leboyer et al., 2012) This pathway is possibly mediated by cytokines, which are part of the immune system's inflammatory response, and are elevated during episodes of mania and depression (Leboyer et al., 2012). In a study examining metabolic syndrome, schizophrenia, and bipolar disorder, metabolic syndrome was more likely to occur in individuals with schizophrenia and bipolar disorder than controls, even when controlling for exercise, diet, and smoking (Bly et al., 2014). These findings support the multi-systemic nature of bipolar disorder, as it is indicates that

its effects are related to more than one body system. In response to this research on

bipolar disorder and schizophrenia and multi-systemic theories, increased emphasis has

been placed on conceptualizing and studying psychiatric disorders as disorders of not

only the mind, but also the body (Leboyer et al, 2012). However, to date, the evidence

supporting this theory in relation to other psychiatric disorders, namely anxiety and

depression, remains mixed.

With regard to anxiety disorders, several studies have identified metabolic

associations. For example, a study looking at Vietnam war veterans found that a

diagnosis of generalized anxiety disorder was positively associated with metabolic

syndrome, whereas depression was not (Carroll et al., 2009). Similarly, another study by

Luppino and colleagues (2011) found a strong association between most metabolic

syndrome components and anxiety-specific affects, but not depression-specific affects.

By contrast, however, other recent studies have found that the prevalence of

metabolic syndrome was greater only in individuals with depression, but not anxiety

(Butnoriene, Bunevicius, Norkus, & Bunevicius, 2014). Another study found that as

levels of depression increased, the number of components of metabolic syndrome

exhibited also increased, even when controlling for obesity, smoking status,

socioeconomic status, age, lifestyle, and comorbidity with anxiety. This relationship was

not observed with anxiety symptoms (Skilton, Moulin, Terra, & Bonnet, 2007).

Other studies have found no association between depression or anxiety and

metabolic problems. For example, a study in Finland found that after sex, alcohol

consumption, smoking, marital status, education level, and physical activity were

controlled for, metabolic syndrome was not associated with anxiety or depression in

young adults (Herva et al., 2006). Affirming these findings, a large longitudinal study conducted in Norway found no association between anxiety and depression and future metabolic syndrome (Hildrum, Mykletun, Midthjell, Ismail, & Dahl, 2009).

In summary, the literature on the relations between anxiety, depression and metabolic problems remains unclear. To date, the majority of the research has focused on depressive disorders and additional research is needed to understand the metabolic effects of both anxiety alone and comorbid with depression.

Significance

More research is needed to investigate the interactions between metabolic syndrome, anxiety disorders, and depressive disorders in order to address the inconsistencies in the literature and determine if a common mechanism may connect these disorders. This is particularly important to understand, as anxiety and depressive disorders are very common. In the United States, there is an 18.1% prevalence rate of past year anxiety disorders in adults (Kessler, Berglund, et al., 2005; Kessler, Chiu, Demler, Merikangas, & Walters, 2005) and a 6.7%, prevalence rate of past year major depressive episodes (National Survey on Drug Use and Health, 2016). Given this high prevalence, it is important to research to have scientific consensus regarding whether these disorders may be a risk factor for metabolic syndrome, or its components. This will enable further research into specific mechanisms and associations, which can help improve treatments, diagnoses, and screening. As stated by Chapman and colleagues, "Research examining the association between depressive disorders and chronic disease suggests that timely diagnosis and treatment of psychiatric disorders could greatly affect

the impact of chronic disease. The presence of mental illness may be an important

contributor to the etiology of chronic disease. Thus, the promotion of mental health

would likely result in reducing a considerable proportion of the burden of chronic

disease." (Chapman, Perry, & Strine, 2005). There is sufficient evidence to believe that

this rationale could be extended to anxiety and depressive disorders.

Furthermore, understanding the relations between these disorders may also help

control the cost of healthcare. In the United States, 75% of healthcare finances are spent

on chronic diseases. The Centers for Disease Control and Prevention (CDC), reported

that $313.8 billion dollars were spent on cardiovascular disease and stroke in 2009, as

well as $116 billion spent on diabetes in 2007 (2009). In contrast with the costs of

depression and anxiety, which average $210.5 billion and $42 billion per year,

respectively. Therefore, if a connection between anxiety and depression can be

empirically established, it is possible that early identification and treatment of depression

and anxiety could prevent the development of more costly and chronic metabolic

conditions. This has the potential to reduce healthcare cost by billions of dollars

(Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015; Greenberg et al., 1999).

Finally, a novel element of the current study is that it examines metabolic

symptoms in relation to anxiety and depression in both adults and children. While

previous studies have looked at obesity and anxiety and depression in children, to our

knowledge, the specific associations between elevated metabolic syndrome components

and anxiety and depression symptoms have not been examined (Anderson, Cohen,

Naumova, Jacques, & Must, 2007; Weiss et al., 2004). It is important to understand the

development of disorders from an early age, as it can lead to earlier screening, identification and intervention.

Hypotheses

Based on the existing literature, it was hypothesized that all of the individual components of metabolic syndrome would be positively correlated with the presence of depressive symptoms. As the literature indicates that depression is correlated with metabolic syndrome more often than anxiety, it was also hypothesized that the presence of anxiety symptoms would not be significantly correlated with metabolic syndrome components. Finally, it was hypothesized that the relations between anxiety, depression, and metabolic syndrome components would be consistent for adults and children. As there is a lack of literature looking at children, adults, and metabolic syndrome, there was no reason to hypothesize that there would be a difference between children and adults.

Methods

Participants

This study constitutes a secondary analysis of data obtained from a family study on emotion regulation conducted in the University of Vermont Child Emotion Regulation Laboratory under the direction of Dr. Robert Althoff (Committee on Human Research in Medical Sciences #13-275). Eighty-three family units, each consisting of one proband accompanied by either two biological parents, or one biological parent and one full biological sibling, were recruited from both a pediatric psychiatry clinic and the community. In addition to the presence of biological family members, recruitment criteria

for the proband included either the presence of emotion dysregulation problems, or the ability to be a clean control or psychiatric control. Subjects were chosen for this secondary analysis based on the availability of questionnaire, blood draw, and morphometric data.

As seen in Table 1, the mean socioeconomic status of families in this sample was 65.43 on the Hollingshead scale, which corresponds to middle class status. There were 140 total children, of which 65.7% of the children in the sample were male (0=female, 1=male) and the mean age was 11.36 years. All children were between the ages of 7 to 17. Among the children there were 56 sibling pairs in the sample. There were 117 adults, with 30.8% of the sample being male (0=female, 1=male), and a mean age of 43.51 years.

The original purpose of this study was to examine the relations between emotion dysregulation and metabolic problems. Therefore, the mean level of emotional-behavioral problems was higher in the children than in the general population. Based on the Child Behavior Checklist, the average score for total emotional-behavioral problems was at the 69th percentile. More specifically, the mean level of withdrawn and anxious symptoms were at the 71st and 69th percentile, respectively, for children in this sample. Adults had a slightly above average total problems score in the 54th percentile. They had an average withdrawn score in the 63rd percentile and an average anxious-depressed score in the 67th percentile. Written informed consent and assent was obtained from all subjects prior to participation in the study. No exclusions were made based on sex, race, or ethnic background.

Measures

Child Behavior Checklist

The Child Behavior Checklist (CBCL) is a parent-report questionnaire that assesses children's emotional, behavioral and social problems during the past 6 months. The questionnaire consists of 113 problem items that are rated on a 3-point scale of 0 ("Not true"), 1 ("Somewhat or Sometimes True"), or 2 ("Very True or Often True"), which have been factor-analytically reduced to 8 syndrome scales that are consistent across age, informant, and culture (Achenbach & Rescorla, 2001). The psychometrics of these scales have been well characterized with test-retest reliabilities ranging from 0.74 to 0.95 and Cronbach alphas ranging from 0.79 to 0.97. For the purposes of this study, raw scores on the Anxious-Depressed and Withdrawn-Depressed syndrome scales were used. Because most parents in the sample were mothers, whenever available, the maternal report was used for consistency.

Adult Self Report

The Adult Self Report (ASR) is a self-report questionnaire analogous to the CBCL, that assesses emotional, behavioral, social, and substance use problems during the past 6 months. Similar to the CBCL, adults reported on problem behaviors on a 3-point scale for 123 problem items. These items have been factor-analytically reduced to 8 syndrome scales that are consistent across age, informant, and culture. The psychometrics for these scales have been well characterized with rest-retest reliabilities ranging from 0.87 to 0.91 and Cronbach alphas ranging from 0.83 to 0.88. For this study, raw scores on the Anxious-Depressed and Withdrawn-Depressed syndrome scales were used.

Blood Draw and Morphometric Data

In accordance with the recommendations of the National Cholesterol Education Program (NCEP), height, weight, and waist circumference (measured halfway between the iliac crest and the lowest rib), blood pressure and resting heart rate data were collected from all participants. Body Mass Index (BMI) was calculated from height and weight.

Additionally, a blood sample was taken to obtain plasma glucose, triglycerides, HDL, and insulin measurements. Participants were required to fast for at least eight hours prior to the blood draw. After the blood draw, participants were given $5.00 vouchers to purchase breakfast at the University of Vermont Medical Center Dining Facilities. If adults were treated for hypertension, diabetes, or hyperlipidemia, the type and dosage of medication was recorded. In order to standardize the scales for each morphometric and metabolic factor (waist circumference, systolic blood pressure, fasting glucose, high-density lipoproteins, and triglycerides), the factors were first z-transformed. High-density lipoprotein scores were transformed so that higher scores would reflect lower levels, and therefore, a greater metabolic risk. Then, the factors that comprise metabolic syndrome were averaged to compute overall metabolic risk. Due to a smaller sample size for data from the blood draw ($n_{total} = 178$, $n_{kids} = 89$, $n_{adults} = 89$) the Z scores were not summed in order to avoid underestimating metabolic risk for people with missing data. The metabolic risk score was calculated separately for the full sample, for adults only, and for children only.

Statistics

Pearson's correlation and multiple hierarchical regression analyses were

conducted using IBM SPSS Statistics version 24. Then, the PROCESS macro 2.16 was

used to conduct follow-up simple slope analyses to examine the nature of interaction

effects (Hayes, 2016).

Results

Tables 1 and 2 present the descriptive statistics and Pearson's correlations

between all variables of interest. Using the results of the Pearson's correlations, analyses

were initially run on the full sample, and then broken down into adults and children.

Full Sample

A hierarchical multiple regression analysis was conducted predicting the overall

metabolic risk for the full sample. At step one, SES, sex, and age were entered as

covariates. At step two, anxious-depressed symptoms were added to the model. The

overall model was significant and accounted for 35% of the variability in the overall

metabolic risk score (R^2 .35, F [4, 192] = 25.81, p <.001) with the addition of anxious-

depressed symptoms uniquely contributing 1.4% of the variance (ΔR^2 = .01, ΔF [1, 192]

= 4.20, p <.05). More specifically, age (b = .02, t[192] = 8.32, p <.001), and anxious-

depressed symptoms (b = .02, t[192] = 2.05, p < .05) had significant positive effects on

overall metabolic risk. SES (b = -.01, t[192] = -3.38, p = .001) had a significant negative

effect on overall metabolic risk, and sex (b = .52, t[192] = 5.61, p <.001) had an effect

such that males had a higher metabolic risk than females. The same procedure was

repeated using withdrawn-depressed symptoms at step two to predict overall metabolic

risk. The model was significant and predicted 33% of the variance in withdrawn-

depressed symptoms (R^2 =.33, F [4, 192] = 25.20, p <.001). However, the addition of

withdrawn-depressed symptoms at step two was not significant.

Adults

　　　　We repeated the hierarchical multiple regression analysis to investigate anxious-

depressed symptoms on overall metabolic risk in adults only. Sex, age, and SES were

used in step one of the analysis to predict overall metabolic risk in adults. In step two,

anxious-depressed symptoms were added. The overall model was significant, accounting

for 38% of the variability in metabolic risk (R^2 = .38, F [4, 92] = 13.92, p <.001). The

addition of anxious-depressed symptoms uniquely contributed 4.8% of the variance (ΔR^2

=.05, ΔF [1, 92] = 7.07, p <.01). Age (b = .03, t[92] = 2.84, p < .01) and anxious-

depressed symptoms (b = .03, t[92] = 2.66, p < .01) had positive significant effects on

metabolic risk, whereas SES (b = -.007, t[92] = -2.36, p < .05) had a significant negative

effect on metabolic risk. Sex (b = .71, t[92] = 5.39, p <.001) also had an effect such that

adult males had a higher metabolic risk than adult females.

　　　　To further investigate the relations between anxious-depressed symptoms and

metabolic problems, we conducted hierarchical multiple regressions predicting each of

the individual metabolic components that were significantly correlated with anxious-

depressed symptoms in Table 2. In the first regression with HDL as the dependent

variable, age, sex, and SES were entered at step one. In step two, anxious-depressed

symptoms were added. The overall model was significant, accounting for 21% of the

variance in HDL (R^2 = .21, F [4, 74] = 4.97, p <.001), with anxious-depressed symptoms

uniquely accounting for 5.4% of the variance (ΔR^2 =.05, ΔF [4, 74] = 5.07, p <.05).

Anxious-depressed symptoms (b = .04, t[74] = 2.25, p <.05) had a significant positive

effect on HDL. Sex (b = .82 t[74] = 3.72, p <.001) also had an effect such that being male

was associated with having lower levels of HDL.

This model was repeated predicting waist circumference. The overall model was

significant, predicting 32% of the variance in waist circumference (R^2 =.32, F [4, 91] =

10.79, p <.001) with anxious-depressed symptoms uniquely predicting 4.4% of the

variance in waist circumference (ΔR^2 =.04, ΔF [1, 91] = 5.86, p <.05). SES (b = -.01

t[91] = -2.90, p < .01) had a significant negative effect, anxious-depressed symptoms (b =

.03 t[91] = 2.42, p <.05) had a significant positive effect, and sex (b = .75 t[91] = 4.39,

p <.001) was significant such that to being male was associated with waist circumference.

Finally, the third iteration of this model predicting BMI was statistically significant,

predicting 17.2% of the variance (R^2 =.17, F [4, 90] = 4.68, p <.01). However, the

addition of anxious-depressed symptoms did not uniquely contribute to the significance.

Further analyses examining the possibility of interactions effects of between age, sex, and

SES with anxious-depressed symptoms on HDL, and waist circumference did not yield

significant findings.

Additionally, to investigate the possibility that metabolic problems may predict

anxious-depressed symptom, additional hierarchical multiple regression analyses were

conducted with anxious-depressed symptoms as the dependent variable. In step one, age,

sex, and SES were entered. In step two, HDL was entered. In step three, waist

circumference was added followed by BMI in step four. No significant effects were

found, indicating that, in adults, anxious-depressed symptoms are more predictive of metabolic problems than vice versa. The analysis was repeated with withdrawn-depressed symptoms as the dependent variable, and there were also no significant findings.

Children

Sex, age, and socioeconomic status were used in step one of a hierarchical multiple regression analysis to predict overall metabolic risk in children and adolescents. In step two, anxious-depressed symptoms were added. The overall model was significant, and predicted 26.3% of the variability in metabolic risk ($R^2 = .26$, $F [4, 95] = 8.46$, $p < .001$). However, the addition of anxious-depressed symptoms was not significant.

Next, we performed additional hierarchical multiple regressions predicting each of the metabolic risk factors that were significantly correlated with anxious-depressed and withdrawn symptoms. First, using withdrawn symptoms as the dependent variable, age, SES, and sex were entered at step one of the regression. At step two, triglycerides were added, followed by waist circumference at step three, and BMI at step four. The overall model predicted 21% of the variance in withdrawn-depressed symptoms ($R^2 = .21$, $F [6, 64] = 2.84$, $p < .05$). Though the overall model was significant, none of the individual predictors had significant independent effects on withdrawn-depressed symptoms, suggesting a possible interaction effect. The analysis was repeated, predicting anxious-depressed symptoms, and was not significant.

To examine possible interactive effects on withdrawn-depressed symptoms, we computed cross product terms of "age x triglycerides", and added it as a fifth step to the model. The addition of the interaction term significantly improved the overall model,

which now predicted 25% of the variance in withdrawn-depressed symptoms ($R^{2=}$ .25, F [7, 63] = 3.05, p <.01). The interaction of age x triglycerides uniquely predicted 4.3% of the variance, which was a marginally significant increase (ΔR^2 =.04, ΔF [1, 63] = 3.59, p = .063). To further explore this interaction, we used PROCESS to conduct follow-up simple slope analyses at values of +/-1 standard deviation for age and triglycerides. For younger children (-1 standard deviation), there was no significant effect of triglycerides on withdrawn-depressed symptoms. However, for older children (+1 standard deviation), there was a significant positive effect of triglycerides on withdrawn symptoms (b = .47, t[63] = 2.47, p < .05; see Figure 1). Additionally, the Johnson-Neyman technique was used to identify regions of significance, indicating a statistically significant effect of triglycerides on withdrawn scores for children over the age of 12.68 years.

There were no significant interactions of between other variables in the model. Finally, meeting full criteria for metabolic syndrome did not significantly predict anxious-depressed or withdrawn symptoms in children

Discussion

The purpose of this study was to evaluate if there was a connection between metabolic syndrome and anxiety and depression. The findings of the study partially support the hypothesis, as anxiety was related to metabolic syndrome components in adults and depression was related to metabolic syndrome components in children. Although in the full sample, 1.4% of the overall metabolic risk was uniquely predicted by anxious-depressed symptoms, further analyses indicated that the relations of anxious-depressed symptoms and metabolic risk in adults drove this effect. Anxious-depressed

symptoms predicted of 4.8% of the variance in overall metabolic risk in adults, and had

an effect such that higher levels of anxious-depressed symptoms were associated with

greater metabolic risk. Additionally, being older, male and of lower SES background

were risk factors for higher overall metabolic risk, lower levels of good cholesterol and a

large waist circumference. This finding is consistent with previous research insofar as

low SES and age have been previously identified as risk factors for metabolic syndrome

(Hildrum, Mykletun, Hole, Midthjell, & Dahl, 2007; Matthews, Raikkonen, Gallo, &

Kuller, 2008). However, inconsistent with the literature, most research has found that

women tend to have a greater risk of metabolic syndrome than men (Beigh & Jain, 2012;

Tonstad, Sandvik, Larsen, & Thelle, 2007). These inconsistencies may be due to the large

proportion of female adults in this sample of which may have reduced this studies ability

to detect effects of anxiety or depression on metabolic risk for males. Additionally, most

participants in this study resided in Vermont, a state that is healthier than other areas in

the United States (United Health Foundation America's Health Rankings, 2016).

　　　In adults, only anxious-depressed symptoms predicted metabolic problems,

specifically waist circumference and high-density lipoproteins. This finding is contrary to

the hypotheses that depression, but not anxiety would be related to all the components of

metabolic syndrome. This finding provides further evidence that anxiety is related to

metabolic problems, and supports the assertion that more research should be done

investigating anxiety and metabolic outcomes.

　　　Inferences from the data imply anxiety may have a negative metabolic effect,

whereas depression may not. This may be explained by the fact that anxiety is a

psychological reaction to stress, even if the stressor is no longer present (Faravelli &

Pallanti, 1989; Finlay-Jones & Brown, 1981; Schneiderman, Ironson, & Siegel, 2005).

Additionally, people with high levels of stress may have altered eating habits, choosing to

eat higher-calorie foods and increase overall consumption (Zellner et al., 2006). High

levels of stress are related to emotional eating, weight gain around the abdomen, and

increased blood pressure, all factors that contribute directly or indirectly to metabolic

syndrome (A. Chao et al., 2016). Behavioral changes, such as increased physical activity,

improved eating habits, and stress reduction techniques, such as meditation or

mindfulness may serve to both alleviate anxiety and reduce its negative metabolic

consequence (Daubenmier et al., 2011; Salmon, 2001).

In particular, large waist circumference and low HDL levels were the metabolic

risk factors predicted by anxious-depressed symptoms. Increased adipose tissue around

the abdomen contributes to lower levels of HDL (Nguyen-Duy, Nichaman, Church, Blair,

& Ross, 2003). Increased visceral adipose tissue has been shown to be a consequence of a

stress-induced cortisol response (Moyer et al., 1994). Negative metabolic outcomes may

be caused by an increase in the stress hormone cortisol due to chronic stress in

individuals with anxiety (A. M. Chao, Jastreboff, White, Grilo, & Sinha, 2017). A

proposed mechanism is that increased interleukins (cytokines) stimulate the release of

cortisol (Urwyler, Schuetz, Ebrahimi, Donath, & Christ-Crain, 2017). In a recent study,

interleukin antagonism in obese participants was found to lead to a decrease in cortisol,

systolic blood pressure, and heart rate (Urwyler et al., 2017). Anxiety, through stress and

cortisol release, may be related to negative metabolic outcomes. This relates anxiety to

metabolic syndrome.

Unlike in adults, anxious-depressed symptoms did not predict metabolic outcomes in children. However, the interaction between age and triglycerides did have an effect on withdrawn-depressed symptoms, such that for children over the age of 12.68 years of age, high levels of triglycerides were associated with more severe withdrawn-depressed symptoms, whereas for children under the age 12.68, triglycerides had no effect. This is in contrast with the hypothesis that the relations between anxiety, depression and metabolic problems would be consistent across age. It is possible that triglyceride accrual around the age of puberty may increase the risk of depressive symptoms, possibly related to self-esteem and body dissatisfaction in adolescents, especially as steady weight gain and increased body fat is associated with the onset of puberty (Lehrer, 2015; Ozmen et al., 2007). This is further supported by a substantial body of research that has suggests that obesity, which is associated with high triglyceride levels, may contribute to the early onset of puberty (Ahmed, Ong, & Dunger, 2009). The early onset of puberty is also a risk factor for depression (Birmaher et al., 1996; Wang, Lin, Leung, & Schooling, 2016; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). It is possible that high triglycerides may lead to weight gain, premature puberty, and depressive symptoms during adolescence.

It is noteworthy, that in adults, internalizing psychiatric symptoms (e.g. anxious-depressed) were found to uniquely contribute to metabolic risk, whereas in children, the inverse relationship was observed. That is, in children, metabolic risk (specifically triglycerides) uniquely predicted internalizing symptoms (i.e. withdrawn-depressed). This may reflect an age-specific effect or separate mechanistic pathways. It is possible that similarly to schizophrenia and bipolar disorder, inflammatory processes may underlie

both the psychiatric and physiological symptoms in some individuals with anxiety and

depression. However, isolating those mechanisms was beyond the scope of the present

study. This study demonstrated a connection between symptoms of anxiety and

depression and metabolic problems, supporting the necessity for further research into the

cellular mechanisms of the disorders.

Limitations & Future Directions

This study had several limitations. First, because the design of the study was

cross-sectional, we could not make temporal inferences regarding the directionality of the

relations between anxiety and depression symptoms and metabolic problems.

Additionally, lifestyle (e.g. exercise, diet), medication usage, and comorbidity were not

controlled for in this study. Because these factors may have been related to both

psychiatric and metabolic symptoms, it will be important to examine these effects in

future research.

A larger sample size and more even ratios of females to males would also make

the study more generalizable. Additionally, as the participants were part of a family

study, and thus shared genetics and environment, there may have been an inflated

likelihood of significant findings.

Future directions include adding diet, exercise, biological relatedness, and

medication use to the hierarchical regression model. This can help to elucidate the impact

each of these factors has on psychological wellbeing and metabolic syndrome

components. Future studies should investigate participants longitudinally, as this can help

to determine directionality. Lastly, it would be interesting to look at this data set in

relation to emotional eating and the HPA (hypothalamic-pituitary-adrenal) axis. This is

the idea that stress causes an increased desire for calorie-dense food. When comfort food

is eaten, it may alter signals in the brain, lowering the response to stress (Yau & Potenza,

2013). The HPA axis could be examined in part by adding blood cortisol levels, delay

discounting data, and diet to the model, as well as making the study longitudinal.

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Appendix

Sample N=257				Children N=140			Adults N=117		
	Sex	SES	Age	Sex	SES	Age	Sex	SES	Age
Mean	.50	65.98	26.00	.66	65.43	11.36	.31	66.65	43.51
Standard Deviation	.50	22.97	16.76	.48	23.99	2.81	.46	21.73	6.51
Minimum	0	0	7	0	0	7	0	0	28
Maximum	1	90	60	1	90	17	1	90	60
N	257	251	257	140	139	140	117	112	117
Missing (N)	0	6	0	0	1	0	0	5	0
% Male	50.2	-	-	65.7	-	-	30.8	-	-
% Female	49.8	-	-	34.3	-	-	69.2	-	-

Table 1. Descriptive Statistics

Pearson's Correlation
(N= 89-249)

Measure	Anxious Depressed Raw Score	Withdrawn Raw Score
All Subjects Average Z Score	.235**	.119
Adults Average Z Score	.245*	.108
Children Average Z Score	.132	.263**
Children & Adults Average Z Score	.169*	.191*
BMI	.296**	.140*
BMI Children Average Z Score	.195	.358**
BMI Adults Average Z Score	.228*	.134
Waist Circumference All Subjects Average Z Score	.286**	.146*
Waist Circumference Adults Average Z Score	.257*	.150
Waist Circumference Children Average Z Score	.132	.292**
HDL Adult Average Z Score	.215	.043
Triglycerides Children Average Z Score	.198	.286*
Metabolic syndrome Diagnosis	.077	.039

**$p<.01$, *$p <.05$ (2-tailed)
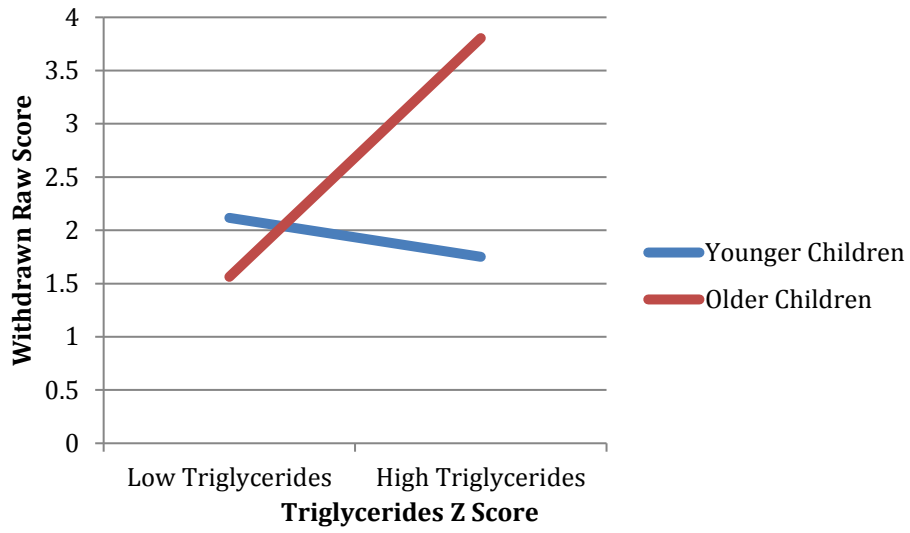*Table 2.* Pearson's Correlations

Figure 1. The moderating effect of age on triglycerides predicting depressive behavior