

**FAMILY EATING BEHAVIOURS AND CHILD BODY MASS INDEX**

**FAMILY EATING BEHAVIOURS AND CHILD BODY MASS INDEX: CHANGES  
AFTER 12 MONTHS ENROLMENT IN A PEDIATRIC WEIGHT MANAGEMENT  
PROGRAM**

By JOSÉE IVARS, BSc.

A Thesis Submitted to the School of Graduate Studies in Partial Fulfilment of the Requirements  
for the Degree of Master of Science

McMaster University © Copyright by Josée Ivars, July 2020

MASTER OF SCIENCE (Department Medical Science, 2020)

McMaster University, Hamilton, Ontario

TITLE: Family eating behaviours and child body mass index: Changes after 12 months  
enrolment in a pediatric weight management program

AUTHOR: Josée Ivars, BSc. (Bishop's University)

SUPERVISOR: Dr. Katherine Morrison

COMMITTEE: Dr. Katherine Morrison, Dr. Hertzell Gerstein, Dr. Gita Wahi

NUMBER OF PAGES: i-viii, 54

## **ABSTRACT**

**Background:** Overweight and obesity affect approximately one-third of Canadian children and these children are at an increased risk of developing life-long obesity-related health consequences. Treatment efforts in the pediatric setting focus on behavioural modification – including modification of dietary intake. Eating behaviours encompass a modifiable aspect of dietary intake and could be a useful approach for weight reduction in the context of pediatric weight management (PWM) programs.

**Objectives:** 1) To describe changes in family, child and parent eating behaviours over a 12-month period after enrolment in a PWM program. 2) To examine if changes in family eating behaviours over the first 6 months in a PWM program can predict child BMI z-score at 12 months, even when controlling for other factors related to changes in BMI z-score.

**Methods:** Children ages 2-17 with a BMI >85<sup>th</sup> percentile were included in this analysis. Data was collected at baseline, 6 month and 12-month timepoints as part of the CANadian Pediatric Weight management Registry. BMI z-score was derived from height (cm) and weight (kg). Eating behaviours (eating related to hunger (ERH) and eating style (ES)) were measured in children, parents and families using the Family Eating and Activity Habits Questionnaire. Child and parental scores were summed to generate a family score. Decrease in scores over time indicated improvements in eating behaviours.

**Results:** 807 children were included in the analysis (48.3% male, 51.7% female, mean age 12.08, mean baseline BMI z-score 3.55). Family ERH scores decreased significantly from baseline to 6 months by 1.63% (-0.16 points, SD=0.09,  $p<0.05$ ) but by 12 months scores were the same as at baseline. Child ERH scores decreased significantly by 2.06% at 6 months (-0.14 points, SD=0.02,  $p<0.05$ ), but at 12 months scores were the same as at baseline. Parent ERH scores did not change significantly. Family ES scores decreased from baseline to 6 months by 1.88% (-1.99 points, SD=0.21,  $p<0.05$ ) and stayed approximately the same at 12 months. Child ES scores were similar, decreasing 2.86% (-1.68 points, SD=0.03,  $p<0.05$ ) from baseline to 12 months. Parents ES score decreased significantly by 6 months, but after 12 months, scores were the same as at baseline. Change in either family eating behaviours from baseline to 6 months did not predict child BMI z-score at 12 months when adjusting for age, sex and BMI z-score at baseline. In subsequent exploratory analysis, increases in child eating behaviour scores at 6

months was a significant predictor of increases in child BMI z-score at 12 months ( $p=0.02$ ) when adjusting for age, sex and BMI z-score at baseline.

**Conclusion:** In the context of PWM programs, eating style and eating for hunger improved over the first 6 months after enrolment. Improvements in child eating behaviour after 6 months could predict improvements in child BMI z-score after 12 months.

## **ACKNOWLEDGEMENTS**

I have so many individuals to thank as I come to the end of my MSc. I would first like to extend my deepest appreciation to my supervisor, Dr. Katherine Morrison. Dr. M has helped me become a great scientific writer, critical thinker, problem solver and academic. Thank you for believing in me, reminding me of my potential when I didn't always see it, and making sure I was always able to put my best work forward.

My committee members, Dr. Wahi and Dr. Gerstein, have played a tremendous roll in the development of my project from start to finish, especially helping me to define my research direction and methodology. Thank you for always, providing helpful insight, feedback and guidance. Thank you for encouraging me along the way, even when it felt like I wasn't getting anywhere, or going backwards for that matter!

Dr. Patrick McPhee joined our study team during the second half of my master's, and I am so thankful for his exceptional mentorship, professionalism and expertise, especially with statistics, methodology and editing. Thank you for not making me feel like there were silly questions and always being available for a pep-talk when I needed one (usually weekly!).

I would like to thank the CANPWR Study Team, past and present, especially Vivian Vaughan Williams and Kelly Bradbury, for their help and guidance as I learned about the CANPWR Study. Thank you for teaching me the ropes and always giving me reassurance.

The Morrison Research Team has been an amazing support along the way. Special thanks to Elizabeth Gunn, Dr. Basma Ahmed and Efraim Yousuf. It was a delight to be able to share this journey with my fellow MSc. colleagues Chiara Homann and Nina Varah. We shared many highs and lows along the way, and I am glad that I had two other lovely people that always knew and understood exactly what I was going through.

Finally, I need to extend my deepest thanks and appreciation to my wonderful family and friends. To my mom and dad, Nancy and René Ivars, thank you for putting up with my endless desire to be a "professional student", encouraging me to follow my dreams, and always giving me unconditional love, support, and kindness. To Brandon, my partner, thank you for always listening, loving and encouraging me, especially when times were the most stressful. To my two fur babies, Peppy and Princess, thank you for bringing so much joy, purpose and light to my life 🐾. Finally, thank you to my best friend of over 10 years and writing wizard, Emma Clue, for her dedication to helping me edit this entire thesis.

## **TABLE OF CONTENTS**

1. INTRODUCTION AND LITERATURE REVIEW	
1.1. Summary of project.....	1
1.2. Childhood obesity.....	1
1.2.1. Worldwide prevalence.....	2
1.2.2. Canadian prevalence.....	2
1.3. Health-related consequences of obesity.....	2
1.3.1. Physical health consequences.....	2
1.3.2. Psychosocial consequences.....	3
1.4. Classification of obesity in children .....	4
1.5. Etiology of childhood obesity.....	5
1.5.1. Population level contributory factors .....	5
1.5.1.1. Non-modifiable risk factors.....	5
1.5.1.2. Modifiable behaviours.....	6
1.5.1.2.1. Diet-related factors.....	7
1.5.1.2.2. Eating behaviours.....	8
1.5.1.2.3. Eating behaviours and obesity in children .....	9
1.5.1.2.4. Measuring eating behaviours.....	11
1.5.1.2.5. Other modifiable behaviours.....	12
1.6. Treatment of childhood obesity.....	13
1.6.1. Treatment overview.....	13
1.6.2. Factors that influence treatment outcomes .....	14
1.7. Eating behaviour modification in the context of pediatric weight management.....	15
1.8. Summary.....	17
2. PROJECT RATIONALE, OBJECTIVES, AND HYPOTHESES.....	17
2.1. Rationale.....	17
2.2. Project objectives.....	17
2.2.1. Objective 1.....	17
2.2.2. Objective 2.....	18
2.3. Hypotheses.....	18
3. METHODOLOGY .....	18

3.1. Participants and Study Design.....	18
3.2. Outcome measures .....	19
3.2.1. Outcomes for objective 1 .....	19
3.2.2. Outcomes for objective 2.....	19
3.3. Description of outcome measures .....	19
3.3.1. Changes in eating behaviours using The FEAHQ.....	19
3.3.2. Child BMI and BMI z-score.....	22
3.4. Covariates.....	23
3.5. Statistical analysis.....	24
3.5.1. Objective 1 (paired samples t-test) .....	26
3.5.2. Objective 2 (hierarchical multivariate regression) .....	26
4. RESULTS.....	27
4.1. Participant characteristics.....	27
4.2. Eating behaviours.....	31
4.2.1. Eating related to hunger.....	31
4.2.2. Eating style.....	31
4.3. Changes in eating behaviours as a predictor of child BMI z-score.....	34
5. DISCUSSION.....	39
5.1. Changes in eating behaviours throughout pediatric weight management.....	39
5.2. Changes in eating behaviours in relation to child BMI z-score.....	40
5.3. Strengths and limitations.....	42
5.4. Future directions for research.....	43
6. CONCLUSION.....	43
7. APPENDICES.....	44
Appendix I: The FEAHQ in the CANPWR Study.....	44
Appendix II: Child eating behaviour scores as predictors of family eating behaviour scores.....	46
8. REFERENCES.....	48



## **LIST OF FIGURES AND TABLES**

**Table 1:** Summary of methodology used to validate the FEAHQ

**Table 2:** Statistical analysis plan

**Table 3:** Participant characteristics at baseline, 6 months, and 12 months

**Table 4:** Participant characteristics at baseline between included and excluded groups

**Table 5:** Mean eating behaviour scores at baseline, 6 months and 12 months and percent change in score between timepoints

**Figure 1:** Percent changes in family, child and parent eating behaviours from baseline to 6 months and baseline to 12 months.

**Table 6:** Relationship between covariates of interest and child BMI z-score at 12 months, corrected for child BMI z-score at baseline

**Table 7:** Hierarchical regression model predicting child BMI z-score from BMI z-score at baseline, age, sex, and changes in family eating behaviours from baseline to 6 months

**Table 8:** Relationship between covariates of interest and child BMI at 12 months, corrected for child BMI at baseline

**Table 9:** Hierarchical regression model predicting child BMI at 12 months from age, sex, BMI at baseline, change in screen time hours and family eating behaviours at 6 months

**Table 10:** Relationship between covariates of interest and child BMI z-score at 12 months, corrected for child BMI at baseline with child eating behaviours as independent variables

**Table 11:** Hierarchical regression model predicting child BMI z-score from BMI z-score at baseline, age, sex, and changes in child eating behaviours from baseline to 6 months

## **LIST OF ABBREVIATIONS**

CANPWR: Canadian Pediatric Weight Management Registry

BMI: body mass index

CDC: Centre for Disease Control

SES: Socio-economic status

WHO: World Health Organization

IOTF: International Obesity Task Force

SSB: Sugar-sweetened beverage

FEAHQ: Family Eating and Activity Habits Questionnaire

PWM: Pediatric weight management

CAAT: Children's Appetite Awareness Training

FCET: Food Cue Exposure Training

ERH: Eating related to hunger

ES: Eating style

NSS: Not statistically significant ( $p > 0.05$ )

## 1. INTRODUCTION AND LITERATURE REVIEW

### 1.1. Summary of Project

Overweight and obesity affect approximately one-third of Canadian children and these children are at an increased risk of developing life-long obesity-related health consequences such as type 2 diabetes, cardiovascular disease and mental health issues. Typically, treatment efforts in the pediatric setting focus on behavioural modification – including modification of dietary intake, physical activity, sleep and sedentary time. Eating behaviours (how, when, where and with whom one eats) encompass a modifiable aspect of dietary intake for both children, and their caregivers. Eating behaviour modification could be a useful approach for weight reduction in the context of pediatric weight management programs but remains understudied. In this thesis project, eating behaviours of a large group of children and youth with obesity, measured using a validated questionnaire, are described using data from the multi-site CANadian Pediatric Weight Management Registry (CANPWR). The changes in eating behaviours over a 12-month period after enrolment in a pediatric weight management program are described. The influence of change in eating behaviours over the first 6 months of enrolment in a pediatric weight management program on body size at 12 months will be evaluated. These findings will inform clinicians if eating behaviours change during current weight management programs and the effectiveness of eating behaviour modification on weight-related outcomes when treating children with overweight or obesity.

The introductory section will discuss obesity in children, the health consequences of this disease, and individual and family characteristics that predispose children to the development of obesity. The treatment of obesity in children will be briefly discussed and the factors known to influence health outcomes during treatment will be described. This will allow for in-context description of the role that eating behaviour modification could potentially play in weight management programs.

### 1.2. Childhood obesity

Obesity is a pathophysiological condition that can result from an imbalance between energy intake and energy expenditure<sup>1</sup>, and is characterized by excess body fat<sup>2</sup>. Obesity in children and adolescents is a serious public health concern as it leads to both short-term health issues and has long-term implications into adulthood<sup>3</sup>.

### 1.2.1. Worldwide prevalence

Over the past 3 decades, child body mass index (BMI) trends for both males and females have increased steeply. More recently, in some developed countries, including Canada, BMI trends in children 5-19 years old have begun to plateau, although the prevalence of overweight and obesity remains greater than 30%<sup>4</sup>. The global age-adjusted prevalence of obesity has risen from 0.7% to 5.6% in girls and from 0.9% to 7.8% in boys from 1975 to 2016, respectively<sup>5</sup>. The rate of severe obesity in some countries such as the United States, where it is defined as a BMI greater than 20% of the 95th percentile on Center for Disease Control (CDC) growth charts, is still increasing. This is particularly seen in children of a lower socio-economic status (SES) and in minority groups<sup>6</sup>. In children with severe obesity, the risk of obesity-related health consequences is exacerbated, and obesity is more likely to persist into adulthood<sup>7</sup>.

### 1.2.2. Canadian prevalence

According to WHO cut-points, in 1979, 23.3% of Canadian children ages 2-17 were considered overweight or obese<sup>4</sup>. This estimate has now risen to over 30% as of 2016<sup>8</sup>. Despite this rise, in recent years, the prevalence of overweight and obesity in Canadian children has begun to plateau<sup>9</sup>. As of 2015, the prevalence of obesity alone is 10.4% in girls and 15.5% in boys ages 19 years of age or younger<sup>10</sup>. The prevalence of childhood overweight and obesity also varies by province or region. For example, the Northwest Territories, northern Manitoba and parts of Labrador are estimated to have an obesity prevalence of between 45.4% and 65.4%<sup>8</sup>(WHO). Thus, the prevalence of obesity in Canadian children remains high and varies by geographical region.

## 1.3. Health-related consequences of obesity

Obesity is a complex disease involving multiple organ systems, often resulting in multiple co-morbid conditions. These conditions, which include both physical and mental health issues, can exert both short-term and long-term effects on the individual.

### 1.3.1. Physical health consequences

There is a rich body of high-quality evidence describing the relationship of obesity in childhood with cardiovascular risk factors. The study of cardiovascular risk in children with

obesity is particularly relevant if this risk persists into adulthood. The prevalence of cardiovascular risk factors such as high cholesterol, triglycerides and blood pressure in these children is high. In one study, 58% of 5-10 year olds with obesity had at least 1 cardiovascular risk factor<sup>11</sup>. Clustering of risk factors has also been observed. For example, compared to children of normal weight, overweight children were 9.7 times more likely to have 2 cardiovascular risk factors and 43.5 times more likely to have 3 risk factors<sup>11</sup>. Chronic inflammation, non-alcoholic fatty liver disease, sleep apnea and other disorders are other health consequences associated with obesity in childhood<sup>3</sup>. Furthermore, persistence of obesity into adulthood is a likely outcome for children with obesity: 70% of children and 80% of adolescents with obesity will continue to have obesity as adults<sup>12</sup>. Persistence into adulthood is predicted by parental weight status, duration and severity of obesity, and presence of obesity at adolescence<sup>13</sup>. Longitudinal studies have demonstrated a link between obesity in childhood and cardiovascular disease risk in adulthood<sup>14</sup>, likely associated with thickening of the arteries and deposition of atherosclerotic plaques in early years<sup>15</sup>. In a study published in 1999, obesity in adolescence predicted a 1.5 times increased risk of premature mortality in adulthood<sup>16</sup>. Almost 20 years later, in 2016, these results were replicated in large cohort studies<sup>17</sup>.

### 1.3.2. Psychosocial consequences

The relationship between psychosocial difficulties and obesity might be considered bi-directional. That is, obesity may result in poor mental well-being and poor mental well-being may increase the risk of obesity. High quality evidence suggests that pediatric obesity is related to an increased risk of mental health issues, especially in teenaged girls<sup>13</sup>. The prevalence of depression in a population of 244 youth ages 8-17 entering weight management treatment at a Canadian treatment centre was 36.4%<sup>18</sup>. In the same study, body fat percentage was deemed a significant predictor of depression and health-related quality of life<sup>18</sup>. Longitudinal evidence suggests that children with overweight or obesity report more psychosocial distress than children of a normal weight, an observation that was stable over a period of two years<sup>19</sup>. Teasing and bullying are more commonly experienced by children with obesity<sup>3</sup>. The persistence of obesity into adulthood can predict social isolation and economic challenges such as lower socio-economic status<sup>20</sup>. The psychosocial effects of childhood obesity are important to consider as they can be involved in both short-term and long-term outcomes.

#### 1.4. Classification of obesity in children

To fully understand the literature and compare studies, it is important to consider how obesity is classified. In particular, it is important to note that there is no universally accepted method for classifying childhood obesity, although all current methods rely on the use of body mass index (BMI), defined as weight in kilograms divided by height in meters, squared ( $\text{kg}/\text{m}^2$ ). As BMI changes with increasing age in children, the use of single BMI cut points for overweight and obesity cannot be utilized. Rather, reference to a population standard is generally utilized, typically considering the number of standard deviations above or below the mean (BMI z-score).

The difference in classification methods generally stems from use of a different standard comparator population. The World Health Organization (WHO) growth standard is considered by some to be the “gold standard” of normal growth. Given the rise in public health concern over childhood obesity, coupled with the release of WHO child growth standards for children under 5 years of age in 2006, there was a need to develop a single growth reference for children and adolescents<sup>21</sup>. The growth standard for children 0-5 years was developed from a prospective study to reflect how children should grow under optimal conditions, from multiple countries (prescriptive approach)<sup>4</sup>. In contrast, the WHO compiled data from three sources to create a growth reference for older children: the National Health Examination Survey (NHES), Cycle II (1963-1965) and III (1966-1970) and the National Health and Nutrition Examination Survey (NHANES), Cycle I (1971-1974). The NHES cycle II was a sample of 7,119 American children ages 6-11 years, and the NHES cycle III was a sample of 6,768 American children ages 12-17 years. The NHANES was a sample of approximately 23,000 Americans ages 1-74 years<sup>21</sup>. The NHES sampled the population in an attempt to best-represent all children in the United States<sup>22</sup>. The NHANES over-sampled groups thought to be at high risk of malnutrition at preset rates<sup>23</sup>. Using the same statistical methods for constructing the new growth curves for children under 5, the new growth references for children 5-19 were constructed<sup>24</sup>. Based on these curves, children who have a BMI between one and two standard deviations above the mean for age and sex are considered overweight (BMI z-score between 1 and 2) whereas children with a BMI more than two standard deviations above the mean are considered obese (BMI z-score greater than or equal to 2)<sup>25</sup>.

CDC growth curves are based on the US national survey data collected between 1963 and 1994. According to the CDC, overweight is defined as a BMI equal to or greater than the 85<sup>th</sup>

percentile and below the 95<sup>th</sup> percentile. Obesity is defined as BMI equal to or greater than the 95<sup>th</sup> percentile. The CDC method of classification is the most commonly used indicator in the United States<sup>26</sup>. According to the CDC, there are 3 classes of obesity. Class I obesity is defined by age and sex-specific BMI  $\geq$  the 95<sup>th</sup> percentile (class I) and severe obesity is defined as<sup>27</sup>; class II obesity (age/sex specific BMI  $\geq$  120% of the 95<sup>th</sup> percentile) and class III obesity (age/sex specific BMI  $\geq$  140% of the 95<sup>th</sup> percentile)<sup>28</sup>.

The International Obesity Task Force (IOTF) guidelines have taken a slightly different approach. The IOTF developed sex-specific BMI curves using observations from an international sample of children and plotted the BMI centile curves that passed through the adult cut points of 25 and 30 for overweight and obesity respectively. The respective age and sex specific BMI was then used to classify overweight or obesity<sup>29</sup>. In Canada, use of the WHO growth standard for defining obesity is recommended and will be utilized in this thesis.

### 1.5. Etiology of childhood obesity

Obesity is a disorder with complex, multifactorial causality. The heritability of obesity-related phenotypes is cited between 6% and 85% in varying populations<sup>30</sup>. Still, in population-based studies, it is evident that there are a series of non-modifiable and modifiable factors that can contribute to the development and perpetuation of overweight and obesity in children.

#### 1.5.1. Population-level contributory factors

##### 1.5.1.1. Non-modifiable risk factors

Age, sex, socio-economic status, and race and ethnicity are non-modifiable risk factors that can influence weight status in children. In Canada, for both boys and girls, the prevalence of obesity increases with increased age (10.8% of 3-6 year olds, 11.8% of 7-12 year olds and 15% of 13-19 year olds)<sup>10</sup>. Additionally, there are well-defined gender differences in the presentation of childhood obesity<sup>31</sup>. The prevalence of obesity in Canadian children is higher in males across all age groups: on average, the prevalence is 5.1% higher in males than females (males=15.5%, females=10.4%)<sup>10</sup>. The prevalence of obesity in males tends to increase as they get older, while in females, adolescents have the highest prevalence, pre-adolescents have the lowest, and girls ages 3-6 fall in the middle<sup>10</sup>.

Race and ethnicity are also significant contributors to the development of overweight and obesity. In the US, the prevalence of obesity in Hispanic children ages 2-19 is 21.9%. The prevalence in non-Hispanic black children is 19.5%, while obesity in Non-Hispanic white children is significantly lower at 14.7%<sup>32</sup>. The prevalence of class I and II obesity follow a similar trend based on race. Class III obesity remains lower in White children compared to other ethnicities, although the differences are not as drastic as for the other weight classes<sup>6</sup>.

Finally, there is a known relationship between socio-economic status and the prevalence of childhood obesity. Typically, obesity prevalence is highest in the lowest socio-economic groups of high-income countries<sup>2</sup>. Canadian children of families with income less than the poverty level are more likely to be obese compared to those of higher socioeconomic status (19.4% versus 15.2%, respectively)<sup>33</sup>. The number of dependents in a home is also important. Canadian families with the lowest level of income adequacy (income per number of dependents) had a 23.9% prevalence of overweight and 16.9% prevalence of obesity (for both sexes of children) compared to a 16.8% prevalence of overweight and a 9.3% prevalence of obesity in families with the highest levels of income adequacy. Parental education level is closely associated with socioeconomic status, thus it is unsurprising that there are associations between parental education level and childhood obesity as well. The CDC analyzed data from the National Health and Examination Survey from 2011-2014. They confirmed that education level was negatively associated with the risk of obesity. That is, children with a head of household with a high school education or less had a 21.6% chance of having obesity compared to 18.3% for children living in families where the head of household had some college education and 9.6% for a complete college education. The gap was even greater in families that identified as Black non-Hispanic and Hispanic families<sup>34</sup>.

In summary, children that are older, male, of a racial or ethnic minority and of a lower socio-economic background in higher-income countries are predisposed to a higher risk of developing obesity in the child and adolescent years.

#### 1.5.1.2. Modifiable behaviours

A number of modifiable behaviours are also related to the development of childhood obesity. In population studies that include both lean and overweight/obese children, diet-related



variables, screen time, physical activity and sleep, parental weight status, and child mental health status are all related to obesity prevalence.

#### 1.5.1.2.1 Diet-related factors

Diet-related variables such as sugar-sweetened beverage (SSB) consumption, fruit and vegetable intake and frequency of breakfast consumption are related to obesity and overweight in children.

SSB consumption is associated with increased cardiometabolic risk in adolescents, especially in girls, regardless of their weight status. One study examined children at age 14 and again at age 17. Girls who increased SSB consumption over the three-year period to >1.3 servings per day had 3.2 times increased overall cardiometabolic risk and a 4.8 times greater likelihood of overweight and obesity<sup>35</sup>. Reviews of the literature confirm this link between SSB intake and risk of obesity<sup>36,37,38</sup>. In a review of 17 longitudinal studies including over 56,000 children (mean age 9 years), 94% of the studies reported a positive relationship between SSB intake and measures of body mass such as BMI<sup>36</sup>. A 2015 evaluation of systematic literature reviews had similar findings<sup>37</sup>. This review assessed 13 reviews, which included a total of 30 longitudinal studies, 34 cross-sectional studies and 12 intervention studies. In 70% of the studies cited, a direct association between SSB consumption and obesity and overweight (cross-sectional) and weight gain (longitudinal and interventional) in children and adolescents was suggested. A recent review (2020) including 20 cross-sectional and longitudinal studies determined that consuming more than 4 SSBs per week lead to a 24% greater risk of childhood obesity. Based on evidence from these studies, there is an association between SSB consumption and measures of body mass, weight gain, and cardio-metabolic risk factors related to obesity.

In the US, children and adolescents do not meet current recommended intakes of fruit and vegetables<sup>39</sup>. Because they are made up of mostly water and fiber, increased fruit and vegetable consumption are thought to play an important role in the consumption of a nutrient-dense, well-balanced diet. However, evidence for the association between increased intake of fruit and vegetables and the risk of obesity is largely inconclusive. A 2020 review of the literature concluded that in 14 cross-sectional and longitudinal studies, increased fruit and vegetable intake could not reduce the risk of obesity in children<sup>38</sup>. Other reviews have highlighted that increasing

fruit and vegetable consumption in the experimental setting can lead to decreased measures of adiposity in adults but not children<sup>40</sup>.

Children that consume breakfast everyday are more likely to consume healthier foods throughout the day, achieve a greater quality of life, and have increased cognitive performance<sup>41</sup>. Historically, the prevalence of breakfast consumption amongst children has declined over time. In 1965, 85-90% of American children consumed breakfast. 25 years later, the proportion of children that consumed breakfast decreased by 15-20%<sup>42</sup>. According to a systematic review of the literature including 26 studies (cross-sectional and longitudinal), daily breakfast consumption could reduce the risk of obesity by 34%<sup>38</sup>. In longitudinal studies, increased frequency of breakfast consumption was related to the greatest improvements in BMI and waist circumference over time<sup>43</sup> and breakfast skippers are at an increased risk of developing central adiposity (approximately 2.2 times greater odds)<sup>44</sup>. Additionally, in a 2 year longitudinal study including 271 children ages 6-9, breakfast consumption was significantly, negatively related to changes in BMI z-score<sup>45</sup>. Daily breakfast consumption is an important predictor of weight status in children and has a protective effect against obesity and overweight.

Despite strong evidence in some cases, studies evaluating the relationship between certain dietary variables and measures of body mass in children have limitations. Mainly, these studies collect data using differing methods of self-reporting, typically in the form of food frequency questionnaires, dietary recall or food diaries. Because of this, it is difficult to generalize these results to the population.

#### 1.5.1.2.2 Eating behaviours

Dietary intake is influenced and largely determined by the eating behaviours that accompany the consumption of food<sup>46</sup>. Eating behaviours are a complex, intersection of biological, social and environmental factors that influence how, when and what an individual may eat<sup>47</sup>. Eating behaviours, at least in part, can be considered modifiable, and may be a potential target in weight management strategies<sup>48</sup>.

According to Davison and Birch and their application of Ecological Systems Theory to childhood overweight<sup>49</sup>, dietary intake is considered a risk factor for increased child weight status. Eating behaviours can be described as specific behaviours that accompany the consumption of food such as: how and when one might eat (e.g. eating speed, eating

environment/location), how one may respond to cues to eat or to not eat (hunger and satiety) or other stimuli in their environment (e.g. smell of cooking food, seeing others eating, seeing food)<sup>47</sup>.

Eating-related behaviours for children occur within the context of family and community niches<sup>49</sup>. This can be further influenced by socio-economic status, ethnicity, parental work hours, and accessibility of fast food within the neighbourhood. It is uncertain which parental or family factors most influence child and parental eating patterns, though environmental factors are likely to play a role. For example, child feeding practices, types of food available at home, nutritional knowledge, parental dietary intake, and parental food preferences are all family-level moderating variables related to eating behaviours and child weight status<sup>49</sup>.

Although certain eating behaviours may evolve as a result of underlying biological mechanisms, for the purpose of this thesis, eating behaviours will be considered modifiable since the relative impact of biology versus environment is not well understood, especially in children with obesity. The role and the effectiveness of eating behaviour modification in the context of pediatric weight management programs has been previously studied; however, the majority of these studies take place in a cross-sectional setting and therefore the ability to draw causal associations is limited.

#### 1.5.1.2.3 Eating behaviours and obesity in children

It is known that children with obesity differ in their appetitive traits when compared to their normal-weight counterparts. Mainly, children with obesity have a lower responsiveness to internal cues of fullness<sup>50,51</sup>, eat at a faster rate<sup>52,47</sup>, and are more likely to eat in response to external stimuli rather than internal cues of hunger<sup>53</sup>. Eating behaviours are known to track throughout the childhood years. Higher measures of body fat in the pre-school years predict more “food-approaching” behaviours at age 10, suggesting that higher fat mass at a younger age may upregulate certain drivers of appetite<sup>54</sup>. There are many structural components and factors associated with eating behaviours in children that are described in detail elsewhere<sup>52</sup>. This thesis project highlighted two components of eating behaviours: eating in response to hunger and eating style, described below.

Eating in response to hunger reflects the functioning of underlying mechanisms such as food cue responsiveness (response to cues of hunger) and satiety responsiveness (response to

cues of fullness). Abnormalities in these mechanisms can cause an individual to eat when they may not be hungry, therefore, unnecessarily consuming excess energy<sup>55</sup>. For example, heightened food cue responsiveness may lead to eating in response to external stimuli rather than in response to internal hunger sensations and satiety responsiveness, and, when considered alongside eating speed, can predict BMI Z-score and waist circumference z-score<sup>56</sup>. Why children eat in the absence of hunger is not well-understood although it appears to be influenced by dysfunction of the satiety response, disinhibited eating and/or emotional stressors<sup>56</sup>. Individuals that are responsive to satiety cues will adjust their energy intake of any additional food following the consumption of a prior meal based on the pre-loading paradigm. For example, children with obesity between the ages of 8-12 years old showed no downregulation of energy intake of a “test meal” directly following a “pre-load meal” compared with a non-pre-load meal control condition. Children with a normal BMI, on the other hand, downregulate subsequent energy intake<sup>53</sup>. Studies have shown that eating in the absence of hunger is a behaviour that tracks throughout the childhood years, in children with and without obesity, if not addressed<sup>55</sup>. This fact, coupled with other susceptibilities to obesity, could result in excess weight acquisition in children. Interventions that are focused on decreasing the response to external cues to eat, especially when not hungry, may be successful at reducing energy intake overall and addressing risk of weight gain<sup>55</sup>.

Eating style is a component of dietary intake which describes the environment and habits related to food consumption<sup>46</sup>. An “obesogenic environment” pertains to a setting that promotes unhealthy food consumption habits. Family-related characteristics of eating style such as family meals, parental control, influences and attitudes<sup>57</sup> are significant determinants of overweight and obesity in children. Frequent fast food intake, sedentary lifestyles that intersect the consumption of energy-dense, nutrient-poor foods, especially high levels of electronic device usage during meal time, and children that eat meals without a parent are all characteristics of an obesogenic eating environment that may relate to poor eating styles<sup>46</sup>.

Eating styles that include frequent electronic device usage and television viewing during mealtime are associated with poor diet quality in children<sup>58,59</sup> and an increased odds of being overweight<sup>60</sup>. Further, the protective effects of eating family meals seem to be negated when families consume meals together in front of the TV<sup>59</sup>.

Previous studies have evaluated components of family eating behaviours and how they are related to obesity and overweight in children<sup>61</sup>. Family eating styles that involve negative feeding strategies such as rigid meal settings, using food as a reward for eating or good behaviour, as well as parental persuasion for eating, were all positively associated with child BMI. That is, these factors were more prevalent in families with a child with obesity or overweight. Likely, this is because children are eating in response to external stimuli (parental influence) rather than internal cues of hunger. Authoritative parenting styles are characterized by higher levels of control, yet high levels of warmth. Authoritative parents offer a moderate degree of control that might facilitate the development of children's ability to self-regulate appetite<sup>62</sup>. The absence of family meals is a strong predictor of childhood overweight and obesity. Children that eat neither breakfast nor dinner with their families are at approximately a 4-fold risk of being overweight or obese. Eating at least dinner as a family decreases this risk to 1.2-fold<sup>63</sup>. Eating family meals is not only related to child weight status but also to the development of his or her eating patterns, nutrient intake and prevention of the development of disturbed eating practices<sup>63,46</sup>.

Eating related to hunger and eating style are two components of eating behaviours that have been associated with weight status in children. Eating related to hunger gives insight to the health of underlying mechanisms like the satiety response. This behaviour has a biological basis but can be modified with certain behavioural interventions, described in section 1.7. Eating style describes the environment and context related to food consumption. Obesogenic components of eating style such as device usage during mealtime and absence of family meals are also related to development of obesity in population studies.

#### 1.5.1.2.4 Measuring eating behaviours

Measurement tools have been developed in an attempt to quantify eating behaviours in children<sup>64,65</sup>. Measurement tools typically take the form of a self-reported or parent-reported questionnaire and are based on specific behaviours such as satiety responsiveness, food-cue responsiveness, eating speed and eating environment. In 1998, Moria Golan and colleagues developed the Family Eating and Activity Habits Questionnaire (FEAHQ)<sup>66</sup>. This questionnaire was designed for use in pediatric weight management in order to evaluate changes in the obesogenic nature of the home environment over time. This questionnaire measures eating

behaviours on 4 sub-scales including eating related to hunger and eating style. Eating related to hunger is intended to capture how well the child is able to eat in response to their own internal cues of hunger, irrespective of environmental stimuli. Eating style is related to the feeding environment, pace of food consumption, presence of a parent during mealtime, tendency to eat under emotional distress, and the location where meals are taken. The FEAHQ has been previously validated in children with obesity<sup>66</sup>. More detail on the validation process is included in the methods section. A copy of the FEAHQ used in this study can be found in Appendix I.

Eating behaviours such as eating related to hunger and characteristics of eating style are frequently related to measures of body mass in population studies that involve both children of a normal weight and children with obesity or overweight. There is some evidence that these behaviours can be changed, and their modification may lead to improvements in weight status – the focus of this study.

#### 1.5.1.2.5 Other modifiable behaviours

As noted above, there are multiple other factors associated with obesity development in childhood and these will be briefly described here.

Screen time has been identified as one of the most important factors associated with obesity and overweight. In a study of 930 children, age 7 – 11 years, for every additional three hours of computer or television viewing, the odds of obesity increased by 50%<sup>67</sup>. This relationship was not observed in adolescents ages 12-17. Further, TV viewing time is positively associated with adiposity in females and increased total cholesterol in males ages 7-11, in a large cohort study<sup>68</sup>. While this may in part be related to reduced energy expenditure, in a Canadian study of children age 8-10 years, screen time was also associated with less favourable, more obesogenic dietary choices. More than two hours of screen time per day (the current recommended maximum) was related to higher energy intake, and consumption of less fibre and fewer fruits and vegetables<sup>69</sup>. Estimates were even higher in those with overweight or obesity. In the adolescent population, screen time is positively associated with increased metabolic risk in young adulthood, including increased BMI and adiposity, and unfavourable levels of triglycerides in a longitudinal study<sup>70</sup>.

Increased levels of habitual physical activity, in studies in which activity was measured with devices (objective measures), are associated with a lower prevalence of obesity<sup>71</sup>. A 2016

review evaluating 162 studies (longitudinal and interventional) of 5-17-year-old children cited physical, cognitive and social benefits of objectively measured physical activity. Low intensity physical activity was associated with decreased cardio-metabolic risk<sup>72</sup>. Still, the associations are not completely understood, especially in populations of solely children with obesity, in the context of weight management interventions.

Sleep is an essential aspect to growth and development in the pediatric population and disturbances in sleep can lead to detriments in the physical and mental health of children and adolescents<sup>73</sup>. When compared with data from 20 years ago, children and adolescents are spending significantly less time engaging in high quality sleep<sup>74</sup>. There is clear evidence to suggest an inverse relationship between sleep, more specifically sleep duration, and overweight or obesity in the pediatric population. Insufficient sleep is associated with poorer diet quality and increased child BMI z-score, especially in mid-childhood (7-11 years old)<sup>75</sup>. Sleep duration of less than 9 hours for children 10 or older, 10 hours for children 5 – 10 years old and 11 hours for children less than 5 years old, is coupled with a 58% increased risk of being overweight or obese. This same study determined that for every hour of additional sleep, the risk decreases by approximately 9% <sup>76</sup>.

Overall, a number of modifiable behaviours are proposed to play a causal role in the development of overweight and obesity in children. In addition to dietary intake and eating behaviours, other factors also influence the development of obesity including screen time, physical activity and sleep.

## 1.6 Treatment of childhood obesity

### 1.6.1 Treatment overview

It is clear that overweight and obesity pose a significant health threat to Canadian children, and therefore, identifying effective treatment options is critical. Obesity treatment in the pediatric population generally has focussed on behavioural change and lifestyle modifications<sup>77</sup>. According to Canadian guidelines, children should engage in 60 minutes a day of moderate to vigorous physical activity, sleep 8-11 hours (depending on their specific age) and not exceed 2 hours of daily recreational screen time<sup>78</sup>. Thus, treatment is focused around diet modification, increasing physical activity, decreasing sedentary behaviour, improving sleep hygiene and addressing mental health barriers. However, for those with severe obesity, bariatric

surgery is more effective than lifestyle modification<sup>79</sup> and can lead to weight reductions of up to 27%<sup>80</sup>. Bariatric surgery can also address other obesity-related comorbidities such as type 2 diabetes, hypertension and dyslipidemia<sup>80</sup>, but is only recommended in post-pubescent adolescents with severe obesity or related co-morbidities who have not had success with lifestyle modifications alone<sup>81</sup>. Nonetheless, the recommendation stands that children and adolescents with overweight or obesity of any degree, should be involved in multidisciplinary treatment programs<sup>81</sup>.

Currently, 89% of Canadian pediatric weight management programs offer some type of behavioural/lifestyle counselling as part of their treatment program<sup>82</sup>. A 2015 systematic review and meta-analysis, including 31 randomized trials (behavioural and pharmacological interventions over 6 months), provided low to moderate-quality evidence that behavioural intervention-type treatments are associated with moderate declines in BMI and BMI z-scores<sup>83</sup>. Low to moderate-quality evidence suggested that combined behavioural-pharmacological interventions only demonstrated a small effect on declines in BMI and BMI z-score<sup>83</sup>. As most programs in Canada are only 6 months-2 years in duration, and attrition rates can be high, it is difficult to study the long-term outcomes of weight management intervention in childhood and adolescence. It has been reported however, that in randomized trials of at least 6 months in length, overall there were significant benefits in favour of treatment (versus non-treatment controls) for reductions in BMI or BMI z-score, blood pressure and quality of life<sup>83</sup>. Clinical practice guidelines recommend that a comprehensive weight management intervention should include structured behavioural modifications that are family-centered, age-appropriate and culturally sensitive<sup>81</sup>. Multi-disciplinary weight management programs (endocrinology, psychology, nutrition, exercise counselling, etc.) can be tailored to fit the individual needs of the families involved and hopefully, address any barriers to treatment<sup>84</sup>. Behavioural interventions may include routine evaluations of diet and physical activity levels from a multidisciplinary team of practitioners like dietitians, exercise physiologists, social workers, physicians, nurse practitioners and nurses. Clinical practice guidelines also recommend that care-providers support healthy eating habits, including the promotion of structured mealtimes and the elimination of anything that could cause interference of cues of hunger or satiety (i.e. boredom, stress, excess screen time)<sup>81</sup>.



### 1.6.2 Factors that influence treatment outcomes

There are some aspects of weight management interventions that are known to predict a greater degree of improvement in weight-related outcomes. Children that begin treatment with a lesser degree of obesity have a greater chance of weight reduction during treatment<sup>85</sup>. Individuals that already have previous experience being physically active and are currently partaking in an exercise program upon entry into a weight management program are also likely to have more successful treatment (i.e. weight reduction or weight stabilization)<sup>86</sup>. Typically, children that are less than 8 years old when treatment is first initiated are more likely to have greater decreases in BMI. In one study, this was still observed 5 years after treatment ended<sup>86</sup>. For children greater than 13 years of age however, the opposite was true<sup>86</sup>. Parental changes and motivation<sup>87</sup>, family readiness to change<sup>88</sup>, and program attendance<sup>89</sup> are other factors that may influence changes. For example, 84 children (mean age 11 years) with obesity and their parents received a combination of group and individual weight management interventions over a time period of 18 weeks. Parents that were more confident about program success and were more motivated to make healthy changes at baseline predicted better program adherence and earlier evidence of program success in their children<sup>90</sup>. Despite this evidence, much is still unknown about treatment success and favourable outcomes in pediatric weight management programs. The specific determinants of change in body mass and other obesity-related health implications still needs further research<sup>91</sup>.

### 1.7 Eating behaviour modification in the context of pediatric weight management

As described previously, in the context of pediatric weight management, behavioural change represents the foundation for weight stabilization or weight reductions. One aspect of behavioural change involves dietary intake, which in part, is influenced by eating behaviours.

There is evidence for a genetic component in the development of appetitive traits. In twin studies, it was found that the heritability of satiety and hunger traits could be up to 75%<sup>92</sup>. Despite this evidence, eating behaviours can still be modified though behavioural intervention.

Eating in response to hunger is a behaviour that can be modified by deconstructing the psychological associations between food exposures and feelings of hunger, although this may be difficult given its strong biological basis. Children's Appetite Awareness Training (CAAT), and Food Cue Exposure Training (FCET) are two methods that have previously been explored in

children (8-12-year olds)<sup>48</sup>. CAAT involves increasing the child's sensitivity and awareness to internal cues of hunger and fullness. FCET involves experiential exercises where children are exposed to foods that would normally cause cravings and learn methods to cope when not physically hungry. When used in 8-week randomized interventions, FCET was most successful in reducing eating in the absence of hunger, even 6 months post-treatment period<sup>48</sup>. Eating in response to hunger can also be improved by promoting child self-regulation for feelings of fullness through moderate, controlled portion sizes and organization of the feeding environment (shopping for, choosing and cooking various foods)<sup>46</sup>. Additionally, in 60 children and their families (ages 6-11) in a 12 month weight management program, improvements in eating related to hunger were moderately correlated with reductions in weight in children ( $r=0.59$ ,  $p<0.05$ )<sup>66</sup>.

Eating style may be modified by making specific changes to the eating environment at home. Sharing family meals, turning off electronics during meal time, ensuring no meals are skipped, reducing snacking, and socialization with family during eating times are all examples<sup>46</sup>. These changes can be best executed through parental role modeling, stimulus control (setting children up for success by removing unhealthy food from sight and reach) and limit-setting (limiting behaviours such as the consumption of unhealthy foods to establish healthy eating patterns)<sup>93</sup>. It is suggested that clinical cohorts of children with obesity have more obesogenic eating behaviours, compared to their peers in the community, especially their ability to respond to cues of fullness<sup>94</sup>. Previous studies have evaluated how eating behaviour modification may be related to weight status through pediatric weight management programs, although evidence is limited. One study, using 30 parents as the "exclusive agents of change" for their child with obesity were counselled on eating behaviour modification strategies such as food stimulus control. Over the course of 1 year, children with parents in this experimental group experienced greater reductions in weight as opposed to children of families in the "control condition" where children were at the center of the intervention, and parents were not involved<sup>87</sup>. This approach represented a family-oriented treatment strategy, addressing many aspects of a healthy lifestyle. In 60 children and their families (ages 6-11) in a 12 month weight management program, improvements in eating style were highly correlated with weight reductions in children ( $r=0.73$ ,  $p<0.01$ )<sup>66</sup>.

Eating behaviour modification, in the context of family-centered, pediatric weight management is a potential avenue for assisting families with treatment success but has been relatively understudied to date.

## 1.8 Summary

Obesity and overweight in children are common and are associated with multiple adverse health consequences. Given this, development of effective treatment interventions is critical. Yet, efforts to date have shown moderate effects at best, and typically only over the short term. Treatment efforts include a multidisciplinary approach with a focus on behavioural modification. Eating behaviours are a driver of dietary intake and are associated with weight related outcomes in cross-sectional and longitudinal studies – though the evidence is limited to date. The role of eating behaviour modification in the context of a pediatric weight management programs is not well characterized but may present a possible intervention strategy or avenue for weight management in children.

## 2. PROJECT RATIONALE, OBJECTIVES AND HYPOTHESES

### 2.1. Rationale

Eating behaviours in children and families have been previously studied but are not well defined in the context of pediatric weight management (PWM), particularly in a longitudinal setting. Based on previous research, eating behaviour modification may be an important strategy for weight management in children, but the influence of current weight management programs is unknown. Thus, it is important to demonstrate how eating behaviours change over time in the context of weight management efforts and whether these changes in eating behaviours are associated with changes in weight-related outcomes.

### 2.2. Project Objectives

#### 2.2.1. Objective 1

The first objective of this project was to study if changes in the eating behaviours, eating related to hunger and characteristics of eating style occur in the family unit during the first 12 months of PWM treatment. Changes in the parent and child scores were secondary components of this objective. The following research questions were assessed:

1. a) Do eating related to hunger and/or eating style in families change from time of enrolment in a pediatric weight management program (baseline) to 6 months, baseline to 12 months and from 6 months to 12 months?
1. b) Do eating related to hunger and/or eating style change in children alone, and in parents alone, as described in Objective 1a?

### 2.2.2. Objective 2

The second objective was to determine if changes in family eating related to hunger and/or eating style over the first 6 months in a PWM program predict child BMI-related measures at 12 months, independent of baseline BMI and other factors known to influence BMI. The following research questions were assessed:

2. a) Do changes in family eating related to hunger and/or eating style 6 months after entry to a pediatric weight management program predict **child BMI z-score at 12 months** (primary outcome), independent of baseline BMI z-score and other factors known to influence changes in BMI z-score such as age and sex?
2. b) Do changes in family eating related to hunger and/or eating style 6 months after entry to a pediatric weight management program predict **child BMI at 12 months** (secondary outcome), independent of baseline BMI and other factors known to influence changes in BMI such as age and sex?

## 2.3 Hypotheses

It was hypothesized that families (1A) (children and parents together) and children and parents alone (1B) would demonstrate significant improvements in eating related to hunger and/or eating style 6 and 12 months after enrolling in a pediatric weight management program. It was further hypothesized that changes in family eating related to hunger and/or eating style after the first 6 months would predict changes in child BMI z-score (2A) and child BMI (2B) even when controlling for other factors related to changes in body mass.

## 3. METHODOLOGY

### 3.1 Participants and Study Design

Participants for this project were enrolled in the CANadian Pediatric Weight Management Registry (CANPWR) study. The CANPWR study is an ongoing national, multi-centre observational study. CANPWR included children and adolescents aged 2-17 years old that had a BMI greater than the 85<sup>th</sup> percentile and were enrolled in 1 of 10 weight management programs across Canada. All weight management programs involved operated in affiliation with a pediatric hospital. Participants in the CANPWR study were assessed at baseline, 6 months, 1-year, 2-year and 3-year timepoints. Research personnel collected data on health outcomes and biological, sociodemographic and behavioural characteristics by means of standardized measurements and questionnaires. Information was collected and stored in a database, housed at the Population Health Research Institute using a research focussed software (iDataFax). The main objectives and other details about the CANPWR study are described elsewhere<sup>91</sup>. This thesis project analyzed CANPWR Study data from the baseline, 6 month and 12-month time points.

### 3.2 Outcome Measures

#### 3.2.1 Outcomes for objective 1

- a) The primary outcome for objective 1 was the change in the Family Eating and Activity Habits Questionnaire sub-scale scores (eating related to hunger and eating style) for the family from baseline to 6 months, 6 months to 12 months and baseline to 12 months.
- b) The secondary outcome for objective 1 was similar to the primary outcome but instead, considered changes in the scores for the child alone and parents alone across timepoints.

#### 3.2.2 Outcomes for objective 2

- a) The primary outcome for objective 2 was the child BMI z-score at 12 months, independent of the baseline BMI z-score.
- b) The secondary outcome for objective 2 was the BMI at 12 months, independent of the baseline BMI.

### 3.3 Description of exposures and outcome measures

#### 3.3.1 Changes in eating behaviours using the FEAHQ

The FEAHQ was developed in 1994 by Golan et al<sup>66</sup> to identify factors associated with obesity and monitor changes in the family eating environment that might be related to improvements in child weight status<sup>66</sup>. The FEAHQ evaluates the eating and activity habits of each parent and the child; together these comprise the family score<sup>95</sup>. A higher score on the questionnaire, is indicative of a more obesogenic home environment. The FEAHQ is comprised of four sub-scales: 1. Activity and leisure time; 2. Food stimulus control; 3. Eating related to hunger; and 4. Eating style. The subscales have been evaluated independently for their relationship to health outcomes in the context of pediatric weight management<sup>66</sup>. Of these four subscales, two were collected in CANPWR: eating related to hunger and eating style. The food stimulus control sub-scale was not approved by study psychologists for use in the CANPWR study. The activity and leisure subscale was also not collected as other, validated measures of activity were collected by the CANPWR study and, as this subscale score was not related to outcomes in pediatric weight management in previous studies<sup>66</sup>.

Two studies have examined the reliability and validity of the subscales of FEAHQ, considering each sub-scale individually. Information on the reliability and validity of the two sub-scales used in this study (eating related to hunger and eating style) can be found in Table 1. First, the questionnaire was administered to 40 mothers (20 had a child with obesity, 20 had a child of a normal weight). From this, the questionnaire was found to be internally consistent, that is, individual responses to each item were consistent with other like-items and questions actually measured what they were intended to measure. Cronbach's alpha, a measure of internal consistency and scale reliability, was computed for each individual sub-scale; the mean  $r$  was 0.83 overall, and 0.86 and 0.88 for eating related to hunger and eating style sub-scales, respectively. Test-retest reliability was measured when the group of 40 mothers were asked to re-take the questionnaire two weeks later. Pearson correlation coefficients revealed a strong, positive correlation between testing scores ( $r=0.85$ ,  $p<0.01$ ), indicating a high test-retest reliability. Lastly, an independent t-test between scores of children with obesity and scores of normal weight children was used to determine concurrent validity. The scores were significantly different for all sub-scales, and the total family scores were higher for families with a child with obesity, indicating a more obesogenic home environment.

The second study ensured that the questionnaire could be completed by either parent (to minimize bias across participant scores) and predict outcomes of weight management treatment. Parents of 60 children with obesity (mean age=9.2 years, SD=1.0) completed the questionnaire in the context of a 12-month randomized trial. Thirty participants were randomized to the experimental (family centered- parents as the exclusive agents of change) intervention and 30 were randomized to the conventional (child-centered- child as the exclusive agents of change) intervention (control). Parents in the experimental intervention received 14 sessions on behavioural modification, including information to build relevant parenting skills in relation to eating behaviours. Children in the conventional intervention received 30 group sessions to learn skills on diet monitoring, calorie intake and increasing exercise. Both parents completed the questionnaire before and after the intervention and the results were compared. After 12 months, there were significant improvements in eating behaviours (i.e. eating while standing or occupied by other activities, eating following stress and eating between meals) in the experimental group. The average reduction in weight was greater in the experimental group compared to the conventional group (14.6% reduction versus 8.4%)<sup>66</sup>. Pearson correlation coefficient indicated that reports of the mothers and fathers were significantly correlated for all subscales (Table 1). Children in the experimental intervention lost more weight and FEAHQ questionnaire scores were lower over the course of the intervention, compared to the child-focused intervention. Weight loss in the child was correlated with changes in the family score for each of the two subscales Eating related to hunger and Eating Style (see Table 1). Behavioural changes (i.e. improvements in eating behaviours) explained 27% of the variance observed in weight reduction<sup>66</sup>. The results from these studies confirmed that information on eating behaviours, as measured by this questionnaire, may predict weight-related changes in children enrolled in family-centered weight management interventions.

The scoring system used in the FEAHQ is simple yet is specific enough to accurately capture the behaviours of parents and child as well as the home environment as a whole<sup>66</sup>. Likert-type questions are answered by one parent on behalf of the other spouse and the child and then are totaled at the end to generate a family score. Some questions pertain to only the child, some to only the mother and father, and some to the mother, father and child. To derive the family score, parental and child scores are summed. Child and parent scores may also be considered individually as demonstrated in prior studies<sup>97</sup>. The total score is considered as an

index: the higher the total score, the less appropriate the eating patterns and the more obesogenic the home environment might be. Theoretical possible scores for eating related to hunger varied from 1 – 7 for children, 0 – 3 for parents and 1-10 for families. Theoretical possible scores for eating style varied from 1 – 59 for children, 1 – 47 for parents and 2-106 for families. The questions in each subscale can be viewed in Appendix I.

For the purposes of this project, data collected at baseline, 6 month and 1-year timepoints was used. This questionnaire was completed by the parents, in a room separate from their child. The changes in eating related to hunger and eating style were the outcomes for objective 1 and the exposures of interest for objective 2.

**Table 1:** Summary of methodology used to validate the FEAHQ

Measure	Method of testing	Intent of testing	Results
Content validity	Expert panel	Do the questions ask what is intended to be captured? Are the questions phrased and presented clearly?	Experts determined the content was valid
Internal consistency	Cronbach’s alpha	Are responses to individual items consistent with other items that measure the same thing?	Mean $r=0.83$ (total) $r=0.86$ and $0.88$ for eating related to hunger and eating style sub-scales, respectively ( $P<0.05$ )
Test-retest reliability	Pearson correlation coefficients	Can the results be replicated at a later occasion?	$r=0.85$ ( $P<0.01$ )
Concurrent validity	Independent t-test	Can the questionnaire discriminate between obese and normal weight children?	Mean difference= 2 ( $p<0.05$ ), 9 ( $p<0.01$ ) for eating related to hunger, and eating style, respectively.
Reliability: similarities in mother’s vs. father’s report	Pearson correlation coefficient, MANCOVA	Are there differences in whether the mother or the father completes the questionnaire?	$r=0.94$ , $0.83$ for eating related to hunger and eating style sub-scales, respectively ( $P<0.05$ )
Predictive validity	Pearson correlation coefficient, ANCOVA	Can the questionnaire predict responses to treatment?	$r=0.59$ , $0.73$ ( $P<0.05$ , $<0.01$ ), for eating related to hunger and eating style sub-scales, respectively ( $P<0.05$ )

### 3.3.2 Child BMI and BMI z-score

Child height in centimeters (to the nearest 0.1cm) as well as body mass in kilograms (to the nearest 0.1kg) were measured at the baseline, 6 month and 12-month time points. From these two measures, child BMI ( $\text{kg}/\text{m}^2$ ) was calculated and a standardized BMI z-score was also determined utilizing the World Health Organization growth standard (WHO, 2007), consistent with the CANPWR protocol<sup>91</sup>. As BMI changes with age, a BMI z-score is utilized which relates the child’s BMI to a growth standard, that is specific for age and sex. BMI z-score is frequently



used in the pediatric population to allow for accurate comparison of males and females of different ages.

The use of BMI z-score in children with obesity has limitations. Due to the nature of the curve, changes in BMI z-score are not as detectable in children with severe obesity<sup>98</sup>. Therefore, for a child with severe obesity, a given change in BMI would result in a smaller change in BMI z-score<sup>99</sup>. A proposed alternative in this case may be to evaluate changes in absolute BMI over time<sup>100,101</sup>. Both these methods will be evaluated, with changes in absolute BMI as a secondary outcome for objective 2.

### 3.4 Covariates

To address Objective 2, it was important to consider potential covariates. Covariates were included in the analysis because of their known associations to the dependent variables in question, based on previous literature. These variables are controlled for in order to be able to clearly examine the independent relationship between eating behaviours and child BMI-related measures.

*Age:* Age was collected by self-report and/or chart review conducted by research personnel. Age is known to be related to successful treatment outcomes in pediatric weight management. Typically, children that are less than 8 years old when treatment is first initiated are more likely to have greater decreases in BMI<sup>86</sup>. Child age (in years) was used as a continuous variable in regression analysis.

*Sex:* Sex was collected by self-report. There are differences in the presentation of pediatric obesity based on sex, mainly because of differences in hormones between males and females<sup>31</sup>. Because of these differences, it was important to control for sex in regression models. Sex was collected and analyzed as a dichotomous, categorical variable where 1=female and 2=male.

*Socio-economic status:* Obesity prevalence is highest in the lowest socio-economic groups of high-income countries<sup>2</sup>. Canadian children of families with income less than the poverty level are more likely to be obese compared to those of higher socioeconomic status<sup>33</sup>. Children from low-socioeconomic status groups have increased difficulty attending weight management programs and often have poorer obesity treatment outcomes<sup>102</sup>. Indicators of socio-economic status (total annual household income and highest parental education levels) were

collected by parent self-report. Parental education levels were categorized into no high school, some high school, high school diploma, university/college or post-graduate. Total annual household income was categorized into <\$49,999, \$50,000-\$79,000, \$80,000-\$99,000, or >\$100,000.

*Maternal BMI:* Obesity-related phenotypes are observed to have a heritability between 6% and 85%<sup>30</sup> and because of this, other studies have considered maternal BMI to be an important co-variate<sup>103,104,105</sup>. Maternal BMI (kg/m<sup>2</sup>) was used as a continuous variable in regression models. Maternal BMI was derived from self-reported height and weight.

*Child BMI z-score at baseline:* Utilizing child BMI z-score at baseline as a covariate allows for evaluation of change in BMI z-score at 12 months. Children that begin treatment with a lesser degree of obesity have a greater chance of weight reduction during treatment<sup>85</sup>. For this reason, in regression analysis, child BMI or BMI z-score at baseline was adjusted for. Child BMI (kg/m<sup>2</sup>) and BMI z-score was derived using standardized height and weight measurements by trained study personnel.

*Lifestyle behaviours:* There is evidence that increased screen time, decreased physical activity and poor sleep duration are associated with obesity and overweight and children<sup>68,106,107</sup>. Changes in child lifestyle behaviours from baseline to 6 months were controlled for to remain synonymous with evaluating changes in eating behaviours in a 6-month timeframe: screen time (hours/day), sleep (hours/day), physical activity (organized hours/week, unorganized hours/week). These variables were all collected by self-report, consistent with the CANPWR protocol<sup>91</sup>.

### 3.5 Statistical analysis

Statistical analysis was completed using SPSS Statistical Software (version 26.0). The statistical analysis plan is outlined in Table 2. Some of the variables used in the analyses were derived. Derived variables included BMI, BMI z score and the eating behaviour sub-scale scores.

*Child BMI & BMI z-score:* Child BMI was derived by dividing weight in kilograms by height in centimeters, squared. BMI z-score was derived by comparing BMI to age and sex-specific curves (WHO, 2007).

*Eating behaviour score:* Answer selections were first converted to their numerical score equivalent according to Golan's scoring criteria<sup>66</sup>. These scores were summed according to the question and which family member it corresponded with. The child score was calculated for both sub-scales; eating related to hunger and eating style. Parental scores were averaged to generate 1 parental average score for each sub-scale. This method assumes that children have an equal contribution from each parent in the development of their eating behaviour characteristics, if the child comes from a 2-parent household. If answers were only recorded for 1 parent, the score for those answers was used. This was done in light of the fact that some families are single-parent families and the methodology for addressing single-parent families when using this questionnaire was unclear. This method assumed that children from single-parent households will be influenced solely by this parent's behaviours. The family score was calculated by summing the parental average score (or single parent score) and the child score.

Only participants with complete family eating behaviour scores at baseline and 6 months, as well as a child BMI z-score at baseline and 12 months were included in analysis. Further, participants must have had complete paired data between timepoints to be analysed in objective 1. Participant demographics were completed for the entire sample at each time point (baseline, 6 months and 12 months). To confirm that there were no significant differences between those with complete FEAHQ information and those with incomplete FEAHQ information, an independent sample t-test (for continuous variables) and a Pearson's Chi-Square test (for categorical variables) was completed at baseline for the following variables: age, SES, BMI z-score, maternal BMI, physical activity levels, screen time, and sleep.

**Table 2: Statistical analysis plan**

<b>Objective</b>	<b>Outcome measure</b> (C)= Continuous (D)=Discrete	<b>Hypothesis</b>	<b>Method of analysis</b>
1. (A) To determine if family eating behaviours change from time of enrolment into a PWM program to six months and 12 months, as well as from 6 months to 12 months.	<b>Primary:</b> family scores for eating in response to hunger and eating style sub-scales (measured by FEAHQ) (C)	(1A) Family eating behaviours will decrease (i.e. improve) after enrollment in a PWM program	Paired samples t-test
1. (B) To determine if child and parent eating behaviours change from time of enrolment into a PWM program to six months and 12 months, as well as from 6 months to 12 months.	<b>Secondary:</b> Child and parents scores for eating in response to hunger and eating style sub-scales (measured by FEAHQ) (C)	(1B) Child and parent eating behaviours will decrease (i.e. improve) after enrollment in a PWM program	
2. (A) Determine if changes in family eating behaviours over the first 6 months in PWM program can predict child BMI z-score at 12 months, independent of baseline BMI and other factors known to influence BMI.	<b>Primary:</b> Child BMI z-score (WHO) (C)	(2A) Eating in response to hunger and eating style over 6 months will predict child BMI z-score at 12 months	Hierarchical multiple regression models
2. (B) Determine if changes in child and parent eating behaviours over the first 6 months in PWM program can predict child BMI z-score at 12 months, independent of baseline BMI and other factors known to influence BMI.	<b>Secondary:</b> Child BMI (C)	(2B) Eating in response to hunger and eating style over 6 months will predict child BMI at 12 months	

### 3.5.1 Objective 1

The first objective was to evaluate if eating related to hunger and eating style scores changed significantly between baseline and 6 months, baseline and 12 months and 6 months and 12 months, primarily for the families and secondarily for children and parents.

Differences in scores were tested for normality (baseline to 6 months, 6 months to 12 months and baseline to 12 months) using a protocol outlined by Hae-Young, 2013<sup>108</sup>. Decreases in score (or a negative mean difference) was indicative of improvements in that eating behaviour. As the differences were normally distributed, a parametric paired-sample t-test was used to compare mean differences for each individual (child, parent and family) for three time points (baseline to 6 months, 6 months to 12 months, and baseline to 12 months). A significance level

of 0.05 was used. Means and standard deviations were calculated for each sub-scale, for family, child and parent. Percent change in score was calculated by dividing mean scores by total possible theoretical scores, multiplying by 100, and subtracting baseline score from follow-up score. Levene's test was used to test equality of variance across all variables ( $p > 0.05$ ).

### 3.5.2 Objective 2

The primary aim of objective 2 was to determine if change in each of the family eating behaviour scales was related to child BMI z-score at 12 months, independent of other factors known to influence changes in BMI. To first assess univariate relationships between BMI z-score at 12 months and other variables of interest, partial Pearson's correlations were performed, accounting for child BMI z-score at baseline. A more conservative p-value (0.10) was used to determine which variables from the Pearson's correlations were best suited to go into the hierarchical multivariate regression model. A hierarchical model was used in order to control for variables known to influence changes in BMI in pre-determined "steps". Other variables like changes in eating behaviours were subsequently added to evaluate if these variables had the ability to add a significant level of prediction to child BMI z-score at 12 months. Age and sex were included in the final models regardless of their significance as they are strongly associated with changes in BMI z-score. Changes in family eating behaviours were also included in the final models regardless of significance in univariate models, since they are central to the research question.

Tests for the following were performed to address the assumptions for multivariate regression: linearity, normality, homoscedasticity, equality of variance and identification of outliers. A hierarchical multiple regression model was constructed to assess objective 2. As we were interested in understanding the influence of change in eating behaviours on BMI z-score at 12 months, independent of baseline BMI z-score, model 1 included BMI z-score at baseline as the predictor variable. This allows for the evaluation of change in BMI z-score over time. This protocol was repeated for the secondary objective of objective 2, instead with child BMI in place of family eating behaviours.

## 4. RESULTS

### 4.1 Participant characteristics

Table 3 reports overall participant characteristics, including the total number of observations (N) available for that variable at baseline, 6 months, and 12 months. The average age of participants (n=1297) at baseline was 12.08 (SD=3.47) years and included approximately equal numbers of males and females. Of all the families enrolled in the study, 72% of parents had a college or university-level education. Approximately 31% of the sample came from a family that had an annual household income of more than \$100,000 and approximately 27% came from a family that had an annual household income of \$49,000 or less at baseline. The remainder of families were distributed between the two intermediate income categories. This distribution trend of household income was similar at 6 months and 12 months.

The majority of children were not meeting the recommended 7 hours a week (60 minutes a day) of moderate to vigorous physical activity<sup>78</sup>. On average, participants were engaging in 3.77 (SD 3.05) hours a week of a combination of organized and unorganized physical activity at baseline. However, children were meeting the recommendation for sleep across all timepoints of the study (mean=8.71 (SD 1.50) hours of sleep per night at baseline). Screen time hours per day at baseline were almost double the recommended threshold, mean = 3.95(SD 2.0). Averages for these lifestyle behaviours were similar at follow-up time points (6 months and 12 months).

At baseline, 56.2% of mothers were considered to have obesity based on BMI. At 6 months and 12 months there were considerable decreases in the number of observations, however the relative proportions of mothers in each weight category remained similar. Child BMI z-score at baseline was on average, 3.55(SD 1.29), which is considered severe obesity<sup>109</sup>.

**Table 3:** Participant characteristics at baseline, 6 months and 12 months

	Baseline			6 months			12 months		
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
Age (years)	1297	12.08	3.47	1291	12.61	3.47	1274	13.18	3.49
	<i>N</i>	%		<i>N</i>	%		<i>N</i>	%	
Sex	1297			1077			944		
Male	627	48.3		520	51.9		461	49.0	
Female	670	51.7		557	48.1		483	51.0	
	<i>N</i>	%		<i>N</i>	%		<i>N</i>	%	
Parental education*	1153			807			744		
No high school	6	0.5		4	0.5		2	0.3	
Some high school	25	2.2		15	1.9		14	1.9	
High school	124	10.8		82	10.2		76	10.2	
College/University	830	72.0		582	72.1		546	73.4	
Post-graduate	168	14.6		124	15.4		106	14.2	
Total income**	1207			847			780		
<\$49999	356	27.4		257	28.1		233	27.6	
\$50000-79999	255	19.7		191	20.9		166	19.7	
\$80000-99999	187	14.4		140	15.0		117	13.9	
>\$100000	409	31.5		259	28.3		264	31.3	
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
Lifestyle behaviours***									
PA- total (h/week)	1199	3.77	3.05	872	4.00	2.98	823	3.89	3.12
PA-org. (h/wk.)	1202	1.41	1.93	882	1.50	1.96	832	1.34	1.91
PA-unorg. (h/wk.)	1204	2.36	2.12	876	2.50	2.21	829	2.56	2.24
Sleep (h/day)	1285	8.71	1.50	890	8.75	2.83	840	8.62	1.46
Screen time (h/day)	1250	3.95	2.00	896	3.98	1.99	840	4.12	2.03
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
Maternal BMI (kg/m <sup>2</sup> )	1097	32.6	8.6	653	32.1	8.42	613	32.3	8.3
	<i>N</i>	%		<i>N</i>	%		<i>N</i>	%	
Maternal BMI****	1069			634			596		
Underweight	10	0.9		9	1.4		9	1.5	
Normal weight	195	18.2		117	18.5		111	18.6	
Overweight	263	24.6		156	24.6		138	23.2	
Obese	601	56.2		352	55.6		338	56.7	
Obese: class I	247	23.1		154	24.3		142	23.8	
Obese: class II	139	13.0		93	14.7		85	14.3	
Obese: class III	215	20.1		105	16.6		111	18.6	
	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>N</i>	<i>Mean</i>	<i>SD</i>
Child BMI z-score	1288	3.55	1.29	1076	3.49	1.32	904	3.60	1.61

\*highest parental education level

\*\*total annual household income from all sources (CAD)

\*\*\*moderate-vigorous physical activity. Asked to participants as "activity that makes you out of breath, increases your heart rate or makes you sweaty."

\*\*\*\*BMI classified as: <18.5kg/m<sup>2</sup>=underweight, 18.5-24.9 kg/m<sup>2</sup>=normal weight, 25-29.9 kg/m<sup>2</sup>=overweight, 30-34.9 kg/m<sup>2</sup>=class I obesity, 35-39.9 kg/m<sup>2</sup>=class II obesity, >40 kg/m<sup>2</sup>=class III obesity<sup>110</sup>

For inclusion into the analysis of this study, study participants had to have a family score for both eating behaviour scales at baseline and 6 months and a child BMI z-score at baseline and 12 months. In an effort to assess sampling bias, a comparison of those individuals included and not included in the analysis is shown in Table 4. Out of the total 1297 participants, 807 participants were included based on complete data at baseline. The majority of participants that were excluded had missing family eating behaviour scores at 6 months. There were no identified differences in characteristics between the included participants versus the excluded participants at baseline except the maternal BMI. Mean maternal BMI was 1.09 kg/m<sup>2</sup> (95%CI, 0.4 to 2.1; p=0.041) higher on average in those who met inclusion for this analysis compared to those who did not.

**Table 4:** Participant characteristics at baseline between included and excluded groups

Variable	Included		Excluded		Independent samples t-test			
	N	Mean	N	Mean	Mean diff.	95 % CI		Sig.
Age (months)	807	143.83	490	146.84	-3.02	-7.70	1.66	0.21
Lifestyle behaviours								
*PA-org. (h/week)	801	1.40	401	1.43	-0.03	-0.26	0.20	0.80
*PA- unorg.(h/week)	803	2.40	401	2.29	0.11	-0.15	0.36	0.40
Screen time (h/day)	804	4.03	446	3.80	0.23	0.00	0.46	0.05
Sleep (h/day)	804	8.73	481	8.68	0.05	-0.12	0.22	0.57
Maternal BMI	680	32.98	417	31.89	1.09	0.04	2.14	0.04
BMI z-score	807	3.59	484	3.48	0.11	-0.04	0.25	0.15
Variable	Included		Excluded		Pearson Chi-Square			
	N	%	N	%	Value			sig.
Sex	807		490		2.26			0.13
Males	430	53.3	240	49.0				
Females	377	46.7	250	51.0				
Parental Edu.**	690		422		12.78			0.05
No high school	4	0.6	2	0.5				
Some high school	14	2.0	11	2.6				
High school	81	11.7	43	10.20				
College/University	515	74.6	315	74.6				
Post-graduate	76	11.0	51	12.1				
Total income***	757		450		8.08			0.15
<\$49999	211	27.9	145	32.2				
\$50000-79999	170	22.5	85	18.9				
\$80000-99999	119	15.7	68	15.1				
>\$100000	257	33.9	152	33.8				

\*moderate-vigorous physical activity, \*\*highest parental education level, \*\*\*total annual household income from all sources (CAD)



## 4.2 Eating behaviours

### 4.2.1 Eating related to hunger

*Family:* At baseline, the average family eating related to hunger score was 4.09 (SD 2.02) out of 10 (Table 5). By 6 months, family eating related to hunger scores decreased 1.63% to 3.93 (SD 1.93) out of 10. Overall, scores decreased significantly from baseline to 6 months ( $p<0.01$ ) and then did not change significantly from 6 months to 12 months. By 12 months, the average score was 3.95 (SD 2.09). Scores from baseline to 12 months did not change significantly. Percent changes in scores over time are visually represented in Figure 1.

*Child:* Over the course of the first 6 months average child eating related to hunger scores, decreased, but by 12 months, scores were not statistically different from baseline. At baseline the average eating related to hunger score was 3.25 (SD 1.68) out of a possible 7 points. This score decreased by 2.1% ( $p<0.01$ ) to 3.11 (SD 1.66) after 6 months. Because of a slight, non-significant increase in score by 12 months, there was no statistically significant difference in score from baseline to 12 months. Child age at baseline was negatively associated with child eating related to hunger score at baseline, 6 months and 12 months ( $r=-0.09$ ,  $p=0.003$ ;  $r=-0.14$ ,  $p<0.01$ ;  $r=-0.13$ ,  $p<0.01$ , respectively). That is, younger children had higher eating related to hunger scores than older children, on average. Child sex was not a significant predictor of eating related to hunger scores at any time point.

*Parent:* Parental eating related to hunger scores were not significantly different between timepoints. At baseline, parents had an average score of 0.83 (0.78) out of a possible 3 points. This score decreased by 0.7% at 6 months to 0.81 (SD 0.75) (NSS). By 12 months, this score decreased an additional 0.6% (-1.0% from baseline) to 0.80 (SD 0.77) (NSS).

### 4.2.2 Eating style

*Family:* Families had an average eating style score of 41.29 (SD 11.40) out of a possible 106 points at baseline. By 6 months this score decreased significantly by 1.9% to 39.40 (SD 11.19) ( $p<0.01$ ). From 6 months to 12 months no significant changes occurred. From baseline to 12 months there was a significant decrease in score (2.1%,  $p<0.01$ ) to 39.12 (SD 11.41).

*Child:* Average child scores at baseline were 25.29 (SD 7.72) out of a possible 59 points. The average score decreased by 2.65% at 6 months (23.73 (SD 7.56)) ( $p<0.01$ ). There was no

statistically significant change in scores from 6 months to 12 months. Child scores decreased significantly from baseline to 12 months by 2.86% to 23.61 (SD 7.75) ( $p < 0.01$ ). Age and child eating style scores were moderately and positively correlated at baseline, 6 months and 12 months ( $r = 0.39$ ,  $p < 0.01$ ;  $r = 0.40$ ,  $p < 0.01$ ;  $r = 0.44$ ,  $p < 0.01$ , respectively). Child sex was not a significant predictor of eating style at any timepoint.

*Parent:* Parent eating style scores were on average, 16.02 (SD 5.51) out of a possible 47 points at baseline. By 6 months, parent eating style scores significantly decreased by 0.9% to 15.58 (SD 5.49). No significant changes occurred from 6 months to 12 months. From baseline to 12 months, no significant changes in score were observed.

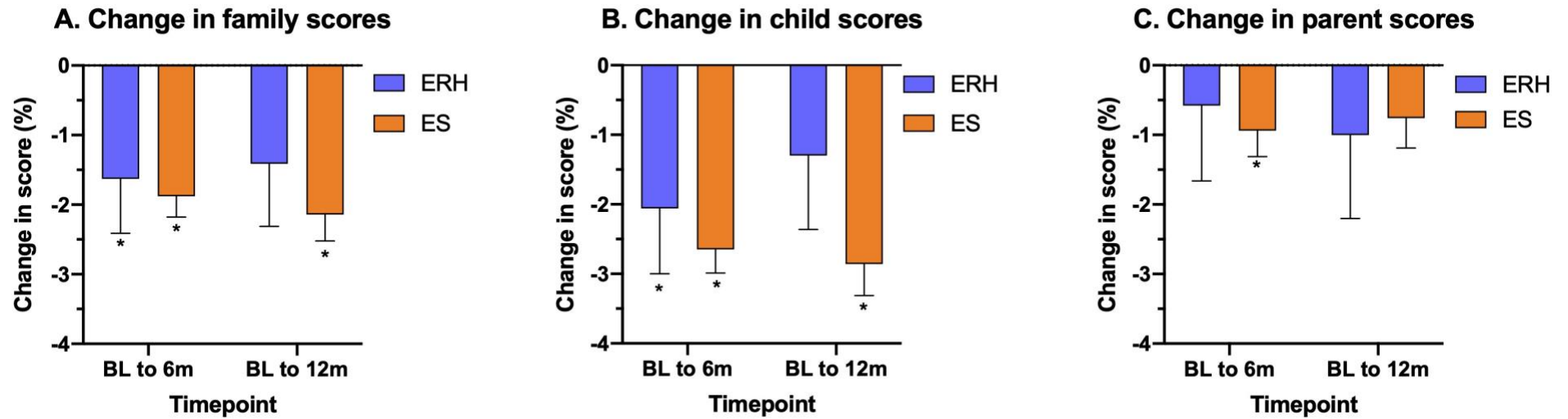
**Table 5:** Mean eating behaviour scores at baseline, 6 months and 12 months and percent change in score between timepoints

<b>Eating related to hunger</b>												
	<b>Baseline</b>			<b>6 months</b>			<b>12 months</b>			<b>Change (%)</b>		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	BL-6m	BL-12m	6m-12m
Family	781	4.09	2.02	755	3.93	1.93	618	3.95	2.09	<b>-1.63*</b>	-1.41	0.22
Child	785	3.25	1.68	758	3.11	1.66	625	3.16	1.76	<b>-2.06*</b>	-1.30	0.76
Parent	801	0.83	0.78	804	0.82	0.74	638	0.80	0.77	-0.58	-1.00	-0.41

<b>Eating style</b>												
	<b>Baseline</b>			<b>6 months</b>			<b>12 months</b>			<b>Change (%)</b>		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	BL-6m	BL-12m	6m-12m
Family	719	41.39	11.40	711	39.40	11.19	561	39.12	11.41	<b>-1.88*</b>	<b>-2.14*</b>	-0.26
Child	737	25.29	7.72	729	23.73	7.56	574	23.61	7.75	<b>-2.65*</b>	<b>-2.86*</b>	-0.21
Parent	751	16.02	5.51	746	15.58	5.49	598	15.66	5.45	<b>-0.94*</b>	-0.76	0.18

\*indicates  $p < 0.05$



**Figure 1:** Percent changes in family, child and parent eating related to hunger (ERH) and eating style (ES) scores from baseline, 6 months and 12 months.\* indicated a significant change ( $p < 0.05$ ) between timepoints. A. describes changes in family eating behaviours scores over time. B. describes changes in child eating behaviours scores over time. C. describes changes in parent eating behaviour scores over time.

#### 4.3 Changes in eating behaviours as a predictor of child BMI z-score

The primary objective was to determine if changes in family eating behaviours after 6 months could predict changes in child BMI z-score at 12 months, independent of other factors known to be related to changes in child BMI z-score.

In table 6, the univariate analysis is presented of the relationship of potential covariates with BMI z-score, independent of baseline BMI z score. In this analysis, only age and sex were significantly related to BMI z-score at 12 months when controlling for BMI z-score at baseline ( $r=0.18$ ,  $p<0.01$ ).

Table 7 includes the hierarchical multivariate analysis. There was no independent influence of either the change in the family eating for hunger or the family eating style scores from baseline to 6 months on BMI z-score at 12 months. Age and sex were independently related to BMI z-score at 12 months, independent of baseline BMI z-score. This suggests that older children and males had less change in BMI z-score from baseline to 12 months. The addition of age and sex into the model explained an additional 2.7% of the variability in BMI z-score at 12 months (adjusted  $R^2=0.55$ ). The full model of BMI z-score at baseline, age, sex, and the change in family eating behaviours (model 3) was statistically significant and explained 55.1% of the variability in BMI z score at 1 year ( $R^2=0.56$ ,  $F(5, 485)=121.35$ ,  $p<0.005$ , adjusted  $R^2=0.55$ ).

**Table 6:** Relationship between covariates of interest and child BMI z-score at 12 months, corrected for child BMI z-score at baseline

Independent Variable	Partial Pearson Correlation	
	<i>r</i>	<i>p</i>
Age	0.18	<0.01*
Sex	0.10	<0.01*
SES (total annual income)	-0.05	0.18
Maternal BMI at baseline	0.05	0.19
Change in screen time at 6m	0.06	0.13
Change in sleep at 6m	-0.01	0.74
Change in organized PA at 6m	-0.05	0.17
Change in unorganized PA at 6m	0.03	0.41
Change in family hunger at 6m	-0.02	0.58
Change in family style at 6m	0.07	0.11

**Table 7:** Hierarchical regression model predicting child BMI z-score at 12 months from BMI z-score at baseline, age, sex, and changes in family eating behaviours from baseline to 6 months

Variable	Model 1 BMI z-score at BL			Model 2 BMI z-score at BL+ age+sex			Model 3 BMI z-score at BL+ age+sex+ Δfamily eating behaviours		
	B	β	p	B	β	p	B	β	p
Constant	0.50		<0.01*	-0.96		<0.01*	-0.96		<0.01*
BMI z-score at BL	0.86	0.73	<0.01*	0.92	0.78	<0.01*	0.93	0.79	<0.01*
Age				0.01	0.16	<0.01*	0.01	0.16	<0.01*
Sex				0.20	0.06	0.04*	0.20	0.06	0.04*
Δfamily eating style							0.01	0.06	0.06
Δfamily hunger							0.00	0.00	0.95
R <sub>2</sub>	0.53			0.55			0.56		
Adj. R <sub>2</sub>	0.52			0.55			0.55		
F	541.06			200.50			121.35		
Δ adj. R <sub>2</sub>	0.52			0.03			0.00		
ΔR <sub>2</sub>	0.53			0.03			0.00		
ΔF	541.06			14.87			1.73		
			<0.01*			<0.01*			0.18

\*indicates p<0.05

A similar analysis was conducted to examine predictors of change in BMI from baseline to 12 months. Univariate regression analyses (Table 8) were repeated with each predictor variable of interest and BMI at 12 months as the dependent variable while adjusting for BMI at baseline. From these univariate analyses, sex and change in screen time at 6 months (p=0.034 and p=0.078, respectively), met the criteria for inclusion (p<0.10) in the multivariate model.

The multivariate analysis is presented in Table 9. Only the baseline BMI was related to BMI at 12 months. None of the covariates were related to BMI at 12 months and change in family eating for hunger and eating style scores were also not related. The final model was statistically significant with BMI at baseline, age, sex, and change in screen time and family eating behaviours at 6 months included in the model (F(6, 480)=335.82, p<0.01, adjusted R<sub>2</sub>=0.81).

**Table 8:** Relationship between covariates of interest and child BMI at 12 months, corrected for child BMI at baseline

Independent Variable	Partial Pearson Correlation	
	<i>r</i>	p
Age	0.04	0.28
Sex	0.07	0.03*
SES (total annual income)	-0.03	0.33
Maternal BMI at baseline	0.06	0.11
Change in screen time at 6m	0.06	0.08*
Change in sleep at 6m	-0.01	0.84
Change in organized PA at 6m	-0.06	0.11
Change in unorganized PA at 6m	0.05	0.22
Change in family hunger at 6m	0.06	0.20
Change in family style at 6m	-0.04	0.38

\*p<0.10

**Table 9:** Hierarchal regression model predicting child BMI at 12 months from age, sex, BMI at baseline, change in screen time hours and family eating behaviours at 6 months

Variable	Model 1 BMI z-score at BL			Model 2 BMI z-score at BL+ age+sex			Model 3 BMI z-score at BL+ age+sex+ Δfamily eating behaviours+ Δscreen time		
	B	β	p	B	β	p	B	β	p
Constant	0.98		0.20	-0.10		0.92	-0.06		0.95
BMI at baseline	1.01	0.90	<0.01*	0.98	0.88	<0.01*	0.99	0.88	<0.01*
Age				0.01	0.04	0.13	0.01	0.03	0.19
Sex				0.50	0.03	0.15	0.47	0.03	0.17
Δfamily eating style							0.03	0.03	0.19
Δfamily hunger							-0.03	-0.01	0.74
Δscreen time							0.17	0.04	0.08
R <sub>2</sub>	0.80			0.81			0.81		
Adj. R <sub>2</sub>	0.80			.0.80			0.81		
F	1985.34			666.87			335.82		
Δadj R <sub>2</sub>	0.001			0.001			0.001		
ΔR <sub>2</sub>	0.80			0.002			0.002		
ΔF	1985.34			1.74			0.10		

\*indicates p<0.05

Not surprisingly, given that child score contributed up to 70% (7/10) of family score proportionally, child score was the best predictor of family eating related to hunger score at baseline and 6 months. In multivariate regression models (Appendix II), child eating related to hunger scores at baseline explained 86% of the variance that was observed in family score (adj  $R^2=0.86$ ,  $p<0.01$ ). The remaining 14% of the variance was predicted by parental score at baseline (adj  $R^2=0.14$ ,  $p<0.01$ ). At 6 months, child eating related to hunger scores had a similar ability to predict family scores (adj  $R^2=0.67$ ,  $p<0.01$ ). The remaining 33% could be predicted by family score at baseline (19%, adj  $R^2=0.19$ ,  $p<0.01$ ) and parental score at 6 months (14%, adj  $R^2=0.14$ ,  $p<0.01$ ). Likewise, Child eating style score contributed up to 56% (59/106) of the family score proportionally. Child score was the best predictor of family eating style score at baseline and 6 months. In regression analysis (Appendix II), child eating style score at baseline could predict 83% (adj.  $R^2=0.83$ ,  $p<0.01$ ) of the variance in family eating style score at baseline. The additional 17% (adj.  $R^2=0.17$ ,  $p<0.01$ ) of variance was predicted by parent eating style score at baseline. Family eating style at baseline was significantly associated with family eating style at 6 months (adj.  $R^2=0.55$ ,  $p<0.01$ ). Child eating style scores at 6 months could predict and additional 28% (adj.  $R^2=0.28$ ,  $p<0.01$ ) of the variance in family eating style scores at 6 months. The remaining 17% of variance was explained by parent eating style score at 6 months (adj.  $R^2=0.17$ ,  $p<0.01$ ).

Given that child score was a strong predictor of family score and that there was a significant changes in child eating for hunger and eating style scores and not in the respective parent scores from baseline to 6 months, it was considered whether or not a change in child score alone might be related to change in the child's BMI z-score. Thus, an exploratory analysis was conducted post hoc in which we examined the change in child eating behaviour scores at 6 months on BMI z-score at 12 months. The univariate and multivariate regression results are presented in Tables 10 and 11. In univariate analysis, change in child hunger score was unrelated to BMI z score at 12 months. Change in child eating style score from baseline to 6 months was related ( $p=0.03$ ). In the multivariate model, change in child eating style was a significant predictor of BMI z-score at 12 months ( $p=0.02$ ). For every 1-point increase in child eating style score, the BMI z-score increased by 0.21 units. Adding change in child eating behaviours at 6

months to the model explained an additional 0.4% ( $p < 0.01$ ) of the variance observed in BMI z-score at 12 months than model 2 (age, sex, BMI z-score at baseline).

**Table 10:** Relationship between covariates of interest and child BMI z-score at 12 months, corrected for child BMI at baseline with child eating behaviours as independent variables

Independent Variable	Partial Pearson Correlation	
	<i>r</i>	p
Age	0.18	<0.01*
Sex	0.10	<0.01*
SES (total annual income)	-0.05	0.18
Maternal BMI at baseline	0.05	0.19
Change in screen time at 6m	0.06	0.13
Change in sleep at 6m	-0.01	0.74
Change in org. PA at 6m	-0.05	0.17
Change in unorg. PA at 6m	0.03	0.41
Change in child hunger at 6m	0.01	0.93
Change in child style at 6m	0.09	0.03*

\* $p < 0.10$

**Table 11:** Hierarchical regression model predicting child BMI z-score at 12 months from age, sex, BMI at baseline, and change in child eating behaviours at 6 months

Variable	Model 1 BMI z-score at BL			Model 2 BMI z-score at BL+ age+sex			Model 3 BMI z-score at BL+ age+sex+ $\Delta$ child eating behaviours		
	B	$\beta$	p	B	$\beta$	p	B	$\beta$	p
Constant	0.55		0.00	-0.96		0.00	-0.96		0.00
BMI z-score at baseline	0.84	0.73	<0.01*	0.91	0.79	<0.01*	0.91	0.79	<0.01*
Age				0.01	0.17	<0.01*	0.01	0.17	<0.01*
Sex				0.22	0.07	0.02	0.22	0.07	0.02*
$\Delta$ child eating style							0.21	0.07	0.02*
$\Delta$ child hunger							0.02	0.03	0.36
R <sub>2</sub>	0.53			0.56			0.56		
Adj. R <sub>2</sub>	0.53			0.56			0.56		
F	592.2			<0.01*			135.19		
$\Delta$ adj. R <sub>2</sub>	0.53			0.03			0.00		
$\Delta$ R <sub>2</sub>	0.53			0.03			0.01		
$\Delta$ F	592.2			<0.01*			17.4		
							<0.01*		
							3.22		
							0.04*		



## 5. DISCUSSION

This study has afforded an opportunity to examine two eating behaviours (eating for hunger and eating style) in a large cohort of children with obesity enrolled in pediatric weight management programs. Further, these eating behaviours were examined in the child, their parents and together in the family scores. Consistent with the primary hypothesis, we identified significant improvements in family eating behaviours over a 6-month period. Family eating related to hunger scores decreased significantly from baseline to 6 months, but these improvements were not sustained at 12 months. Family eating style scores decreased significantly from baseline to 6 months and still remained significantly lower than baseline at 12 months. The decline in family scores was largely related to the decline in child scores as parental scores did not change over time. Contrary to our hypothesis however, neither changes in family eating style or changes in family hunger were related to changes in the child BMI z-score at 12 months, independent of BMI z-score at baseline, age and sex. Based on a post-hoc analysis, carried out when we observed changes in the child score only, it was noted that changes in child eating style after 6 months was a significant predictor of child BMI z-score at 12 months, independent of BMI z-score at baseline, age, sex, and other lifestyle behaviours.

### 5.1 Changes in eating behaviours throughout pediatric weight management

As noted, improvements in child eating for hunger and child eating style were identified 6 months after entering a weight management program. The change in eating style was maintained at 12 months. In contrast, the parental eating style score improved less than the child score and this change was not maintained at 12 months. There were no changes in the parents eating for hunger score.

The majority of pediatric weight management programs in Canada emphasize family-centered, behavioural changes<sup>82</sup>. Our findings may suggest that families engaged in family-centered weight management treatment may still have the child at the centre of efforts to implement behavioural change. Parents are known to be effective drivers of change in pediatric weight management interventions<sup>87</sup>, and therefore, ensuring that family-centered interventions stress the involvement of parents is crucial. Additionally, parents are models of their child's dietary intake and eating behaviours<sup>57</sup> and therefore, if the parent is not changing their eating behaviours, this may limit the potential of the child maintaining any improvements.

In this study it was observed that the majority of significant improvements in eating behaviours for children, parents and families occurred in the initial 6 months, and from 6 months to 12 months no significant changes occurred. Families may strongly adhere to behavioural change strategies involving eating behaviour modification for the initial 6 months of a weight management intervention, and then face declines over time. This could be related to program attrition. Consistent with previous literature, attrition is common in pediatric weight management programs<sup>111</sup> and is related to stress and time commitments<sup>112</sup> as well as lack of child weight-loss<sup>89</sup>. The sustainability of behavioural changes that occur during weight management interventions may be related to a multitude of factors. Children may not have the psychological support from their parents, or parents may not possess the necessary parenting tools to be able to maintain these behaviours<sup>113</sup>. Coupled with attrition, motivations to engage in behavioural change are likely highest during the initial stages of treatment; a theory that is supported by these results.

Improvements in scores were more drastic for the eating style sub-scale than the eating related to hunger sub-scale. Previous literature suggests that eating related to hunger may have a biological basis given its involvement with appetitive hormones and reward pathways in the brain<sup>114</sup> and satiation and satiety can be measured by the presence of certain biomarkers<sup>115</sup>. Appetitive traits such as eating related to hunger may also be strongly heritable<sup>92</sup>. This could support the idea that eating related to hunger may not be as easily modified, especially in short-term, in weight management interventions that are not necessarily targeting eating behaviour modification. Characteristics of eating style might be more easily modified in the short-term as they pertain to lifestyle behaviours that could be more easily addressed (e.g. who does the child eat with, what room do they eat in, under what circumstances do they eat, etc.). Not consuming family meals has previously been related to obesity and overweight in children<sup>63</sup>, the odds of being overweight is lower in children that do not eat in the presence of electronic devices<sup>60</sup> and eating under emotional distress is common in children with overweight or obesity<sup>116</sup>.

## 5.2 Changes in eating behaviours in relation to child BMI z-score

Changes in the family eating behaviour scores after 6 months were not related to changes in BMI z-score at 12 months. In the post-hoc analysis, the change in the child eating style score had a modest influence on the BMI z-score. It also suggests that the child scores contributed

more to the changes in family score than the parent scores did. This is likely due to a combination of factors: 1) overall, child score contributed a greater proportion of possible points to the family score (7/10 and 59/106 for eating related to hunger and eating style, respectively) and 2) because children demonstrated a greater capacity to change their eating behaviours compared to their parents, especially during the first 6 months. Thus, by using changes in child eating behaviours in the model instead, a stronger association between eating behaviour and child BMI z-score was observed.

In contrast to previous studies, changes in eating behaviours in our study were relatively minor contributors to the change in BMI z-score. The FEAHQ foundational study by Golan et al., was a 12-month intervention study conducted in a selective group of 60 children with obesity (ages 6-11) in a weight management program, with no history of psychiatric treatment, both parents living at home and parental agreement to not miss any follow-up visits. At baseline, children with obesity had an average eating related to hunger score of 6/7 and an eating style score of 32/59, compared to 3/7 and 25/59 observed in this study, respectively. Family and parent scores were not explicitly reported so a comparison could not be made. In the Golan study, changes in child eating behaviours explained 27% of the variance observed in weight reductions<sup>66</sup>. In this study, changes in child eating behaviours predicted 0.4% of the variance in BMI z-score. The results presented by Golan are likely different than the results presented in this thesis because in this multi-step analysis, other factors known to influence changes in BMI over time were controlled for, which allowed for the true associations of changes in eating behaviours to be effectively isolated. Secondly, the conditions of the Golan study and this thesis project greatly differ in that the CANPWR cohort is comprised of “real-world” participants who may come from one-parent households, have diagnosed mental illnesses or have imperfect follow-up; all factors that may impede an individual’s ability to undergo meaningful behavioural change. Family-centred behavioural modifications seen in pediatric weight management programs in Canada typically involve multi-disciplinary care, and therefore, eating behaviour modification may not necessarily be the focus of the interventions. This could explain why changes in eating behaviours may only predict a small (yet significant) percentage of changes in BMI z-score in this sample. Additionally, changes in eating behaviours likely need to be combined with improvements in dietary quality (a variable that was not considered in this analysis) to induce the highest degree of change in BMI z-score since dietary intake can be a predictor of BMI in

children<sup>35,37,36,38,45</sup>. Even so, previous literature suggests that improvements in BMI-related measures over a period of approximately 1 year are likely to be modest in most children attending weight management programs in Canada<sup>111</sup>. Synonymous with this evidence, it is to be expected that there will only be minimal changes in BMI z-score observed in this cohort.

### 5.3 Strength and limitations

This study was successful in analysing results from a validated questionnaire that has not frequently been used previously, especially in the longitudinal setting. Research in this field is rich in cross-sectional evidence, or in samples that include both children with obesity and children of a normal weight<sup>97,47,55,117</sup>. The CANPWR cohort is a “real world” sample that includes children from different backgrounds and varying levels of affluence attending weight management treatment in Canada.

The results of this study, although informative, do not come without limitations. Response bias can occur in self-administered questionnaires<sup>118</sup>, however the FEAHQ was designed to be a “self-monitoring questionnaire” to minimize the degree to which someone responds based on how they believe the interviewer wants them to<sup>66</sup>. The scoring system of the questionnaire comes with limitations as well. The eating related to hunger sub-scale is based on less total points, so the opportunity to detect significant behavioural change over time is minimized, especially for parents. Co-variables used in this analysis (SES, maternal BMI, PA, sleep and ST) were also collected by self-report which comes with limitations. Response bias could lead participants to answer questions in a way that they believe is the most socially acceptable<sup>118</sup>. Although previously validated, the FEAHQ could lead to some degree of measurement error and may not effectively capture information about the intended behaviours. Physical activity and measures of sedentary behaviours such as sleep and screen time are best captured by objective measures like accelerometry and self-report has shown only moderate reliability<sup>119</sup>. As it was not feasible to collect device-measured physical activity, the relationship of changes in physical activity to BMI z-score may not be accurate. Lastly, there are limits inherent to using BMI z-score to evaluate weight change in growing children. Although it is a good metric because it is based on a numerical scale and easily derived, it is not the only method of detecting program success. It should also be noted that BMI is a measure that does not differentiate between lean mass and fat mass. Therefore, children may have increases in lean

mass and decreases in fat mass from weight management intervention that may not necessarily be reflected in changes in BMI.

#### 5.4 Future directions for research

There are many possible avenues to consider when extending the outcomes of this research. Future research could focus on the development of other reliable and consistent measures of eating behaviours and the obesogenic nature of the home environment. Interventional studies specifically targeting eating behaviour modification might be necessary to better characterize the relationship with changes in BMI z-score. Lastly, more research is needed to learn more about other factors that may relate to the ability of families, children and parents to modify eating behaviours, and how this may predict outcomes of weight management interventions.

## 6. CONCLUSION

Eating behaviours are shown to improve in families attending multi-disciplinary weight management programs in Canada, especially within the first 6 months of a program. Changes in eating style were sustainable, while those in eating for hunger were not. This may be related to an underlying biological basis of eating related to hunger and an underlying behavioural basis of characteristics of eating style. The improvement in family scores is largely driven by improvements in the children's scores. Improvements in child eating style in the first 6 months of a weight management program predict changes in their BMI z-score after 12 months. Changes in family eating behaviours did not have this same predictive capability. Further research is needed to better understand how eating behaviour modification may predict outcomes of weight management.

7. APPENDICES

Appendix I: The FEAHQ in the CANPWR Study. Questions 37-40: ERH sub-scale. Questions 41-45: ES sub-scale

**CANPWR FOLLOW-UP Questionnaire- Self Administered** Page 11

6 month    12 month  
 24 month    36 month

CANPWR #184   Plate #050

Participant ID:             Initials      
Centre #   Participant#   F   M   L

Guardian Unavailable  
 Completed over the telephone

**36. How frequently does your child buy his/her own sweets?**  
 Never    Almost Never    Sometimes    Frequently    Always

**37. When your child asks to eat, does he/she claim to be hungry?**  
 No    Yes

**38. Usually when the child eats:**  
 He/she asks for it  
 The food was offered by Father/Mother

**39. If it is meal time and your child is not hungry, how would you respond? (please select one)**

<input type="checkbox"/> You suggest that the child will eat later	<input type="checkbox"/> You convince the child to eat with the rest of the family
<input type="checkbox"/> You suggest that the child sit at the table with the rest of the family but would not eat	<input type="checkbox"/> This is an irrelevant question, the child is always hungry
<input type="checkbox"/> You suggest that the child sit at the table with the rest of the family but would eat less	

**40. When it is meal time and you are not hungry, what would you do?**

<b>Mother:</b>	<b>Father:</b>
<input type="checkbox"/> Not eat	<input type="checkbox"/> Not eat
<input type="checkbox"/> Eat less	<input type="checkbox"/> Eat less
<input type="checkbox"/> Eat the same	<input type="checkbox"/> Eat the same
<input type="checkbox"/> It never happens	<input type="checkbox"/> It never happens
<input type="checkbox"/> Unknown	<input type="checkbox"/> Unknown

CANPWR Main Version 3.0 - 2019Mar4

**CANPWR FOLLOW-UP Questionnaire- Self Administered** Page 12

6 month    12 month  
 24 month    36 month

CANPWR #184   Plate #051

Participant ID:             Initials      
Centre #   Participant#   F   M   L

**41. How frequently do the following behaviours occur for each family member?**

	<input type="checkbox"/> N/A	<b>Mother:</b>	<input type="checkbox"/> N/A	<b>Father:</b>	<input type="checkbox"/> N/A	<b>Child:</b>
		never	almost never	sometimes	frequently	Always
Eat while standing	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat straight from the pot/baking pan/bowl/frying pan	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat while watching television, reading, working	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat when bored	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat when angry or in other negative mood states	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat in disorderly way during the afternoon	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Eat late in the evening or at night	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

**42. How often do you eat in the following rooms?**

		<b>Mother:</b>	<b>Father:</b>	<b>Child:</b>
		N/A	never	almost never
		never	sometimes	frequently
		almost never	Always	Always
Living Room/TV Room	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Bedroom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Office	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

**43. Compared to other people of the same age, how would you rate the eating pace of:**

<b>Mother:</b>	<b>Father:</b>	<b>Child:</b>
<input type="checkbox"/> Slow	<input type="checkbox"/> Slow	<input type="checkbox"/> Slow
<input type="checkbox"/> Average	<input type="checkbox"/> Average	<input type="checkbox"/> Average
<input type="checkbox"/> Fast	<input type="checkbox"/> Fast	<input type="checkbox"/> Fast
<input type="checkbox"/> Unknown	<input type="checkbox"/> Unknown	

CANPWR Main Version 3.0 - 2019Mar4



Appendix II: Child eating behaviour scores as predictors of family eating behaviour scores

**Supplementary table 1:** Child and parental scores as predictors of family eating related to hunger scores at baseline

Variable	Model 1			Model 2		
	B	$\beta$	p	B	$\beta$	p
Constant	0.50		0.00	<<<		0.00
Child hunger: BL	1.10	0.93	0.00	1.00	0.84	0.00
Parent hunger: BL				1.00	0.38	0.00
R <sub>2</sub>	0.86			1.00		
Adj. R <sub>2</sub>	0.86			1.00		
$\Delta R_2$	0.86			0.14		
$\Delta$ adj. R <sub>2</sub>	0.86			0.14		
F	6938		0.00	-	-	
$\Delta F$	6938		0.00	-	-	

**Supplementary table 2:** Child and parental scores as predictors of family eating style scores at baseline

Variable	Model 1			Model 2		
	B	$\beta$	p	B	$\beta$	p
Constant	6.90		0.00	<<<		0.00
Child eating style: BL	1.36	0.91	0.00	1.00	0.67	0.00
Parent eating style BL				1.00	0.48	0.00
R <sub>2</sub>	0.83			1.00		
Adj. R <sub>2</sub>	0.83			1.00		
$\Delta R_2$	0.83			0.17		
$\Delta$ adj. R <sub>2</sub>	0.83			0.17		
F	5058		0.00	-	-	
$\Delta F$	5058		0.00	-	-	



**Supplementary table 3:** Child and parental scores as predictors of family eating related to hunger scores at 6 months

Variable	Model 1			Model 2			Model 3		
	B	$\beta$	p	B	$\beta$	p	B	$\beta$	p
Constant	2.24		0.00	0.44		0.00	<<<<		0.00
Family hunger: BL	0.41	0.44	0.00	0.05	0.06	0.00	<<<<	0.00	0.00
Child hunger: 6m				1.05	0.90	0.00	1.00	0.86	0.00
Parent hunger: 6m							1.00	0.39	0.00
R <sub>2</sub>	0.19			0.86			1.00		
Adj. R <sub>2</sub>	0.19			0.86			1.00		
$\Delta R_2$	0.19			0.67			0.14		
$\Delta$ adj. R <sub>2</sub>	0.19			0.67			0.14		
F	170.0			2170.53			>>>>		
$\Delta F$	170.0			3381.70			>>>>		

**Supplementary table 4:** Child and parental scores as predictors of family eating style scores at 6 months

Variable	Model 1			Model 2			Model 3		
	B	$\beta$	p	B	$\beta$	p	B	$\beta$	P
Constant	9.09		0.00	4.70		0.00	<<<<		1.00
Family eating style: BL	0.74	0.74	0.00	0.19	0.19	0.00	<<<<	0.00	1.00
Child eating style: 6m				1.13	0.76	0.00	1.00	0.67	0.00
Parent eating style 6m							1.00	0.49	0.00
R <sub>2</sub>	0.55			0.83			1.00		
Adj. R <sub>2</sub>	0.55			0.83			1.00		
$\Delta R_2$	0.55			0.28			0.17		
$\Delta$ adj. R <sub>2</sub>	0.55			0.28			0.17		
F	788.3			1573.9			>>>>		
	7			8					
$\Delta F$	788.3			1065.0			>>>>		
	7			5					

## 8. REFERENCES

1. Eckel R. Obesity: a disease or a physiologic adaptation for survival? *Obes Mech Clin Manag.* 2003;3-30.
2. Güngör NK. Overweight and obesity in children and adolescents. *J Clin Res Pediatr Endocrinol.* 2014;6(3):129-143. doi:10.4274/Jcrpe.1471
3. Reilly JJ. Descriptive epidemiology and health consequences of childhood obesity. *Best Pract Res Clin Endocrinol Metab.* 2005;19(3 SPEC. ISS.):327-341. doi:10.1016/j.beem.2005.04.002
4. Shields M, Tremblay MS. Canadian childhood obesity estimates based on WHO, IOTF and CDC cut-points. *Int J Pediatr Obes.* 2010;5(3):265-273. doi:10.3109/17477160903268282
5. Bentham J, Di Cesare M, Bilano V, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet.* 2017;390(10113):2627-2642. doi:10.1016/S0140-6736(17)32129-3
6. Cockrell Skinner A, Perrin EM, Skelton JA. Prevalence of Obesity and Severe Obesity in US Children. 2016. doi:10.1002/oby.21497
7. Bass R, Eneli I. Severe childhood obesity: an under-recognised and growing health problem. *Postgrad Med J.* 2015;91(1081):639-645. doi:10.1136/postgradmedj-2014-133033
8. Rao DP, Kropac E, Do MT, Roberts KC, Jayaraman GC. Childhood overweight and obesity trends in Canada. *Heal Promot chronic Dis Prev Canada Res policy Pract.* 2016;36(9):194-198. doi:10.24095/hpcdp.36.9.03
9. Olds T, Maher C, Zumin S, et al. Evidence that the prevalence of childhood overweight is plateauing: Data from nine countries. *Int J Pediatr Obes.* 2011;6(5-6):342-360. doi:10.3109/17477166.2011.605895
10. Carroll MD. Prevalence of Obesity Among Children and Adolescents in the United States and Canada. *CDC.gov.* 2015;(211). [https://stacks.cdc.gov/view/cdc/33062/cdc\\_33062\\_DS1.pdf](https://stacks.cdc.gov/view/cdc/33062/cdc_33062_DS1.pdf).
11. Freedman DS, Dietz WH, Srinivasan SR, et al. The Relation of Overweight to Cardiovascular Risk Factors Among Children and Adolescents : The Bogalusa Heart Study. *Am J Epidemiol.* 1999;103(2):181-204. doi:10.1542/peds.103.6.1175
12. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting Obesity in Young Adulthood from Childhood and Parental Obesity. *N Engl J Med.* 1997;337(13):869-873. doi:10.1056/NEJM199709253371301
13. Reilly JJ, Methven E, McDowell ZC, et al. Health consequences of obesity. *Arch Dis Child.* 2003;88(9):748-752. <http://www.ncbi.nlm.nih.gov/pubmed/12937090>. Accessed March 4, 2019.
14. G T, G Y, H L, et al. Body-Mass Index in 2.3 Million Adolescents and Cardiovascular Death in Adulthood. *N Engl J Med.* 2016;374(25). doi:10.1056/NEJMOA1503840
15. Lamotte C, Iliescu C, Libersa C, Gottrand F. Increased intima-media thickness of the carotid artery in childhood: a systematic review of observational studies. *Eur J Pediatr.* 2011;170(6):719-729. doi:10.1007/s00431-010-1328-y
16. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes.* 1999;23:S2. <http://dx.doi.org/10.1038/sj.ijo.0800852>.
17. Aune D, Sen A, Prasad M, et al. BMI and all cause mortality: systematic review and non-

- linear dose-response meta-analysis of 230 cohort studies with 3.74 million deaths among 30.3 million participants. *BMJ*. 2016;353:i2156. doi:10.1136/bmj.i2156
18. Morrison KM, Shin S, Tarnopolsky M, Taylor VH. Association of depression & health related quality of life with body composition in children and youth with obesity. *J Affect Disord*. 2015;172:18-23. doi:10.1016/J.JAD.2014.09.014
  19. Gibson LY, Allen KL, Davis E, Blair E, Zubrick SR, Byrne SM. The psychosocial burden of childhood overweight and obesity: evidence for persisting difficulties in boys and girls. *Eur J Pediatr*. 2017;176(7):925-933. doi:10.1007/s00431-017-2931-y
  20. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and Economic Consequences of Overweight in Adolescence and Young Adulthood. *N Engl J Med*. 1993;329(14):1008-1012. doi:10.1056/NEJM199309303291406
  21. De Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Organ*. 2007;85(9):660-667. doi:10.2471/BLT.07.043497
  22. NHES III (1966-1970). <https://wwwn.cdc.gov/nchs/nhanes/nhes3/Default.aspx>. Accessed September 16, 2020.
  23. NHANES I (1971-1974). <https://wwwn.cdc.gov/nchs/nhanes/nhanes1/Default.aspx>. Accessed September 16, 2020.
  24. Borghi E, de Onis M, Garza C, et al. Construction of the World Health Organization child growth standards: selection of methods for attained growth curves. *Stat Med*. 2006;25(2):247-265. doi:10.1002/sim.2227
  25. WHO | What is overweight and obesity? *WHO*. 2014. [https://www.who.int/dietphysicalactivity/childhood\\_what/en/](https://www.who.int/dietphysicalactivity/childhood_what/en/). Accessed March 4, 2019.
  26. Defining Childhood Obesity | Overweight & Obesity | CDC. <https://www.cdc.gov/obesity/childhood/defining.html>. Accessed March 4, 2019.
  27. Daniels SR, Kelly AS. Pediatric Severe Obesity: Time To Establish Serious Treatments for a Serious Disease. *Child Obes*. 2014;10(4):283. doi:10.1089/CHI.2014.1041
  28. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat 11*. 2002;(246):1-190.
  29. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7(4):284-294. doi:10.1111/j.2047-6310.2012.00064.x
  30. Yang W, Kelly T, He J. Genetic Epidemiology of Obesity. *Epidemiol Rev*. 2007;29(1):49-61. doi:10.1093/epirev/mxm004
  31. Wisniewski AB, Chernausk SD. Gender in childhood obesity: Family environment, hormones, and genes. *Gend Med*. 2009;6:76-85. doi:10.1016/J.GENM.2008.12.001
  32. Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of obesity among adults and youth: United States, 2011-2014. *Signif Heal Stat Sel Reports from Fed Agencies*. 2016;(219):91-101.
  33. Phipps SA, Burton PS, Osberg LS, Lethbridge LN. Poverty and the extent of child obesity in Canada, Norway and the United States. *Obes Rev*. 2006;7(1):5-12. doi:10.1111/j.1467-789X.2006.00217.x
  34. Ogden CL, Carroll MD, Fakhouri TH, et al. Prevalence of Obesity Among Youths by Household Income and Education Level of Head of Household — United States 2011–2014. *MMWR Morb Mortal Wkly Rep*. 2018;67(6):186-189. doi:10.15585/mmwr.mm6706a3

35. Ambrosini GL, Oddy WH, Huang RC, Mori TA, Beilin LJ, Jebb SA. Prospective associations between sugar-sweetened beverage intakes and cardiometabolic risk factors in adolescents. *Am J Clin Nutr.* 2013;98(2):327-334. doi:10.3945/ajcn.112.051383
36. Luger M, Lafontan M, Bes-Rastrollo M, Winzer E, Yumuk V, Farpour-Lambert N. Sugar-Sweetened Beverages and Weight Gain in Children and Adults: A Systematic Review from 2013 to 2015 and a Comparison with Previous Studies. *Obes Facts.* 2017;10(6):674-693. doi:10.1159/000484566
37. Keller A, Bucher Della Torre S. Sugar-sweetened beverages and obesity among children and adolescents: A review of systematic literature. reviews. *Child Obes.* 2015;11(4):338-346. doi:10.1089/chi.2014.0117
38. Poorolajal J, Sahraei F, Mohamdadi Y, Doosti-Irani A, Moradi L. Behavioral factors influencing childhood obesity: a systematic review and meta-analysis. *Obes Res Clin Pract.* 2020;14(2):109-118. doi:10.1016/j.orcp.2020.03.002
39. Banfield EC, Liu Y, Davis JS, Chang S, Frazier-Wood AC. Poor adherence to U.S. dietary guidelines for children and adolescents in the NHANES population. *J Acad Nutr Diet.* 2016;116(1):21. doi:10.1016/J.JAND.2015.08.010
40. Ledoux TA, Hingle MD, Baranowski T. Relationship of fruit and vegetable intake with adiposity: a systematic review. *Obes Rev.* 2011;12(5):e143-e150. doi:10.1111/j.1467-789X.2010.00786.x
41. Lundqvist M, Vogel NE, Levin L-Å. Effects of eating breakfast on children and adolescents: A systematic review of potentially relevant outcomes in economic evaluations. *Food Nutr Res.* 2019;63. doi:10.29219/FNR.V63.1618
42. Siega-Riz AM, Popkin BM, Carson T. Trends in breakfast consumption for children in the United States from 1965-1991. *Am J Clin Nutr.* 1998;67(4):748S-756S. doi:10.1093/ajcn/67.4.748S
43. Smith KJ, Gall SL, McNaughton SA, Blizzard L, Dwyer T, Venn AJ. Skipping breakfast: longitudinal associations with cardiometabolic risk factors in the Childhood Determinants of Adult Health Study. *Am J Clin Nutr.* 2010;92(6):1316-1325. doi:10.3945/ajcn.2010.30101
44. Wennberg M, Gustafsson PE, Wennberg P, Hammarström A. Poor breakfast habits in adolescence predict the metabolic syndrome in adulthood. *Public Health Nutr.* 2015;18(1):122-129. doi:10.1017/S1368980013003509
45. Carlson JA, Crespo NC, Sallis JF, Patterson RE, Elder JP. Dietary-related and physical activity-related predictors of obesity in children: a 2-year prospective study. *Child Obes.* 2012;8(2):110-115. doi:https://dx.doi.org/10.1089/chi.2011.0071
46. Scaglioni S, De Cosmi V, Ciappolino V, Parazzini F, Brambilla P, Agostoni C. Factors influencing children's eating behaviours. *Nutrients.* 2018;10(6):1-17. doi:10.3390/nu10060706
47. Freitas A, Albuquerque G, Silva C, Oliveira A. Appetite-Related Eating Behaviours: An Overview of Assessment Methods, Determinants and Effects on Children's Weight. *Ann Nutr Metab.* 2018;73(1):19-29. doi:10.1159/000489824
48. Boutelle KN, Peterson CB, Rydell SA, Zucker NL, Cafri G, Harnack L. Two Novel Treatments to Reduce Overeating in Overweight Children: A Randomized Controlled Trial. *J Consult Clin Psychol.* 2011;79(6):759. doi:10.1037/A0025713
49. KK D, LL B. Childhood overweight: a contextual model and recommendations for future research. *Obes Rev.* 2012;29(6):997-1003. doi:10.1016/j.biotechadv.2011.08.021.Secretd

50. Fisher JO, Birch LL. Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *Am J Clin Nutr.* 2002;76(1):226. doi:10.1093/AJCN/76.1.226
51. Jansen PW, Roza SJ, Jaddoe VW, et al. Children's eating behavior, feeding practices of parents and weight problems in early childhood: results from the population-based Generation R Study. *Int J Behav Nutr Phys Act.* 2012;9:130. doi:https://dx.doi.org/10.1186/1479-5868-9-130
52. Obregón AM, Pettinelli PP, Santos JL. Childhood obesity and eating behaviour. *J Pediatr Endocrinol Metab.* 2015;28(5-6):497-502. doi:10.1515/jpem-2014-0206
53. Jansen A, Theunissen N, Slechten K, et al. Overweight children overeat after exposure to food cues. *Eat Behav.* 2003;4(2):197-209. doi:10.1016/S1471-0153(03)00011-4
54. Derks IPM, Sijbrands EJG, Wake M, et al. Eating behavior and body composition across childhood: a prospective cohort study. *Int J Behav Nutr Phys Act.* 2018;15(1):96. doi:10.1186/s12966-018-0725-x
55. Fogel A, Mccrickerd K, Fries LR, et al. Eating in the absence of hunger: Stability over time and associations with eating behaviours and body composition in children. *Physiol Behav.* 2018;192:82-89. doi:10.1016/j.physbeh.2018.03.033
56. Carnell S, Wardle J. Appetite and adiposity in children: Evidence for a behavioral susceptibility theory of obesity. *Am J Clin Nutr.* 2008;88(1):22-29.
57. Savage J, Fisher J, Birch L. Parental Influences on Eating Behavior: Conception to Adolescence. *J Law, Med Ethics.* 2007;Spring(Childhood Obesity Symposium):22-34. doi:10.1111/j.1748-720X.2007.00111.x
58. Liang T, Kuhle S, Veugelers PJ. Nutrition and body weights of Canadian children watching television and eating while watching television. *Public Health Nutr.* 2009;12(12):2457-2463. doi:10.1017/S1368980009005564
59. Fulkerson JA, Loth K, Bruening M, Berge J, Eisenberg ME, Neumark-Sztainer D. Time 2 tlc 2nite: use of electronic media by adolescents during family meals and associations with demographic characteristics, family characteristics, and foods served. *J Acad Nutr Diet.* 2014;114(7):1053-1058. doi:10.1016/j.jand.2013.10.015
60. Vik FN, Bjørnarå HB, Overby NC, et al. Associations between eating meals, watching TV while eating meals and weight status among children, ages 10-12 years in eight European countries: the ENERGY cross-sectional study. *Int J Behav Nutr Phys Act.* 2013;10:58. doi:10.1186/1479-5868-10-58
61. Boswell N, Byrne R, Davies PSW. Family food environment factors associated with obesity outcomes in early childhood. *BMC Obes.* 2019;6:17. doi:10.1186/s40608-019-0241-9
62. Vollmer RL, Mobley AR. Parenting styles, feeding styles, and their influence on child obesogenic behaviors and body weight. A review. *Appetite.* 2013;71:232-241. doi:10.1016/j.appet.2013.08.015
63. Lee HJ, Lee SY, Park EC. Do family meals affect childhood overweight or obesity?: nationwide survey 2008-2012. *Pediatr Obes.* 2016;11(3):161-165. doi:10.1111/ijpo.12035
64. Wardle J, Guthrie CA, Sanderson S, Rapoport L. Development of the Children's Eating Behaviour Questionnaire. *J Child Psychol Psychiatry.* 2001;42(7):963-970. doi:10.1111/1469-7610.00792
65. Birch L., Fisher J., Grimm-Thomas K, Markey C., Sawyer R, Johnson S. Confirmatory factor analysis of the Child Feeding Questionnaire: a measure of parental attitudes, beliefs and practices about child feeding and obesity proneness. *Appetite.* 2001;36(3):201-210.

- doi:10.1006/appe.2001.0398
66. Golan M, Weizman A. Reliability and validity of the Family Eating and Activity Habits Questionnaire. *Eur J Clin Nutr.* 1998;52(10):771-777. doi:10.1038/sj.ejcn.1600647
  67. Siddarth D. Risk factors for obesity in children and adults. *J Investig Med.* 2013;61(6):1039-1042. doi:https://dx.doi.org/10.2310/JIM.0b013e31829c39d0
  68. Bergmann GG, Tassitano RM, Bergmann ML de A, Tenório MCM, Mota J. Screen time, physical activity and cardiovascular risk factors in adolescents. *Rev Bras Atividade Física Saúde.* 2018;23(August):1-12. doi:10.12820/rbafs.23e0008
  69. Shang L, Wang J, O'Loughlin J, et al. Screen time is associated with dietary intake in overweight Canadian children. *Prev Med reports.* 2015;2:265-269. doi:10.1016/j.pmedr.2015.04.003
  70. Grøntved A, Ried-Larsen M, Møller NC, et al. Youth screen-time behaviour is associated with cardiovascular risk in young adulthood: The European Youth Heart Study. *Eur J Prev Cardiol.* 2014;21(1):49-56. doi:10.1177/2047487312454760
  71. Jiménez-Pavón D, Kelly J, Reilly JJ. Associations between objectively measured habitual physical activity and adiposity in children and adolescents: Systematic review. *Int J Pediatr Obes.* 2010;5(1):3-18. doi:10.3109/17477160903067601
  72. Poitras VJ, Gray CE, Borghese MM, et al. Systematic review of the relationships between objectively measured physical activity and health indicators in school-aged children and youth. *Appl Physiol Nutr Metab.* 2016;41(6 (Suppl. 3)):S197-S239. doi:10.1139/apnm-2015-0663
  73. Gruber R, Carrey N, Weiss SK, et al. Position statement on pediatric sleep for psychiatrists. *J Can Acad Child Adolesc Psychiatry.* 2014;23(3):174-195. <http://www.ncbi.nlm.nih.gov/pubmed/25320611>. Accessed May 4, 2020.
  74. Keyes KM, Maslowsky J, Hamilton A, Schulenberg J. The Great Sleep Recession: Changes in Sleep Duration Among US Adolescents, 1991-2012. *Pediatrics.* 2015;135(3):460-468. doi:10.1542/peds.2014-2707
  75. Cespedes EM, Hu FB, Redline S, et al. Chronic Insufficient Sleep and Diet Quality: Contributors to Childhood Obesity. *Obesity (Silver Spring).* 2016;24(1):184. doi:10.1002/OBY.21196
  76. Chen X, Beydoun MA, Wang Y. Is Sleep Duration Associated With Childhood Obesity? A Systematic Review and Meta-analysis. *Obesity.* 2008;16(2):265-274. doi:10.1038/oby.2007.63
  77. Spear BA, Barlow SE, Ervin C, et al. Recommendations for Treatment of Child and Adolescent Overweight and Obesity. *Pediatrics.* 2007;110(1):210-214. doi:10.1542/peds.109.2.210
  78. ParticipACTION. Canadian 24-Hour Movement Guidelines for Children and Youth. 2016;(January 2015):1-2. <https://csepguidelines.ca/>.
  79. TH I, AP C, TM J, et al. Weight Loss and Health Status 3 Years After Bariatric Surgery in Adolescents. *N Engl J Med.* 2016;374(2). doi:10.1056/NEJMOA1506699
  80. Michalsky MP, Inge TH, Jenkins TM, et al. Cardiovascular Risk Factors After Adolescent Bariatric Surgery. *Pediatrics.* 2018;141(2). doi:10.1542/PEDS.2017-2485
  81. Styne DM, Arslanian SA, Connor EL, et al. Pediatric obesity-assessment, treatment, and prevention: An endocrine society clinical practice guideline. *J Clin Endocrinol Metab.* 2017;102(3):709-757. doi:10.1210/jc.2016-2573
  82. Ball GDC, Ambler KA, Chanoine JP. Pediatric weight management programs in Canada:

- Where, What and How? *Int J Pediatr Obes.* 2011;6(2-2):1-4.  
doi:10.3109/17477166.2010.512390
83. Peirson L, Fitzpatrick-Lewis D, Morrison K, Warren R, Usman Ali M, Raina P. Treatment of overweight and obesity in children and youth: a systematic review and meta-analysis. *C Open.* 2015;3(1):E35-E46. doi:10.9778/cmajo.20140047
  84. Avis JL, Bridger T, Buchholz A, et al. It's like rocket science...only more complex: Challenges and experiences related to managing pediatric obesity in Canada. *Expert Rev Endocrinol Metab.* 2014;9(3):223-229. doi:10.1586/17446651.2014.897605
  85. Kolsgaard MLP, Joner G, Brunborg C, Anderssen SA, Tonstad S, Andersen LF. Reduction in BMI z-score and improvement in cardiometabolic risk factors in obese children and adolescents. The Oslo Adiposity Intervention Study - a hospital/public health nurse combined treatment. *BMC Pediatr.* 2011;11(1):47. doi:10.1186/1471-2431-11-47
  86. Reinehr T, Brylak K, Alexy U, Kersting M, Andler W. Predictors to success in outpatient training in obese children and adolescents. *Int J Obes.* 2003;27(9):1087-1092. doi:10.1038/sj.ijo.0802368
  87. Golan M, Weizman A, Apter A, Fainaru M. Parents as the exclusive agents of change in the treatment of childhood obesity. *Am J Clin Nutr.* 1998;67(6):1130-1135. doi:10.1093/ajcn/67.6.1130
  88. Rhee KE, McEachern R, Jelalian E. Parent readiness to change differs for overweight child dietary and physical activity behaviors. *J Acad Nutr Diet.* 2014;114(10):1601-1610. doi:10.1016/j.jand.2014.04.029
  89. Skelton JA, Goff DC, Jr., Ip E, Beech BM. Attrition in a Multidisciplinary Pediatric Weight Management Clinic. *Child Obes.* 2011;7(3):185. doi:10.1089/CHI.2011.0010
  90. Gunnarsdottir T, Njardvik U, Olafsdottir AS, Craighead LW, Bjarnason R. The Role of Parental Motivation in Family-Based Treatment for Childhood Obesity. *Obesity.* 2011;19(8):1654-1662. doi:10.1038/oby.2011.59
  91. Morrison KM, Damanhoury S, Buchholz A, et al. The CANadian Pediatric Weight Management Registry (CANPWR): Study protocol. *BMC Pediatr.* 2014;14(1):1-8. doi:10.1186/1471-2431-14-161
  92. Carnell S, Haworth CMA, Plomin R, Wardle J. Genetic influence on appetite in children. *Int J Obes (Lond).* 2008;32(10):1468-1473. doi:10.1038/ijo.2008.127
  93. Wilfley DE, Kass AE, Kolko RP. Counseling and behavior change in pediatric obesity. *Pediatr Clin North Am.* 2011;58(6):1403-1424, x. doi:10.1016/j.pcl.2011.09.014
  94. Croker H, Cooke L, Wardle J. Appetitive behaviours of children attending obesity treatment. *Appetite.* 2011;57(2):525-529. doi:10.1016/J.APPET.2011.05.320
  95. Golan M. Fifteen years of the Family Eating and Activity Habits Questionnaire (FEAHQ): an update and review. *Pediatr Obes.* 2014;9(2):92-101. doi:10.1111/j.2047-6310.2013.00144.x
  96. Golan M, Fainaru M, Weizman A. Role of behaviour modification in the treatment of childhood obesity with the parents as the exclusive agents of change. *Int J Obes Relat Metab Disord.* 1998;22(12):1217-1224.  
<http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=med4&NEWS=N&AN=9877257>. Accessed July 3, 2019.
  97. Hajna S, LeBlanc PJ, Faught BE, et al. Associations between family eating behaviours and body composition measures in peri-adolescents: Results from a community-based study of school-aged children. *Can J Public Heal.* 2014;105(1):e15-e21.

- doi:10.17269/cjph.105.4150
98. Coppock JH, Ridolfi DR, Hayes JF, St Paul M, Wilfley DE. Current approaches to the management of pediatric overweight and obesity. *Curr Treat Options Cardiovasc Med.* 2014;16(11):343. doi:10.1007/s11936-014-0343-0
  99. Cole TJ, Faith MS, Pietrobelli A, Heo M. What is the best measure of adiposity change in growing children: BMI, BMI %, BMI z-score or BMI centile? *Eur J Clin Nutr.* 2005;59(3):419-425. doi:10.1038/sj.ejcn.1602090
  100. Daniels SR. The Use of BMI in the Clinical Setting. *Pediatrics.* 2009;124(1):35-41. doi:10.1542/peds.2008-3586F
  101. Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: A validation study. *J Pediatr.* 1998;132(2):204-210. doi:10.1016/S0022-3476(98)70433-0
  102. Demeule-Hayes M, Winters MW, Getzoff EA, Schwimmer BA, Rogers VS, Scheimann AO. Pediatric Weight Management Program Outcomes in a Largely Minority, Low Socioeconomic Status Population. *Clin Med Insights Pediatr.* 2016;10:CMPed.S38457. doi:10.4137/CMPed.S38457
  103. L.Y. G, S.M. B, E.A. D, E. B, P. J, S.R. Z. The role of family and maternal factors in childhood obesity. *Med J Aust.* 2007;186(11):591-595.  
<http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L46929642%0Ahttp://sfx.library.uu.nl/utrecht?sid=EMBASE&issn=0025729X&id=doi:&atitle=The+role+of+family+and+maternal+factors+in+childhood+obesity&stitle=Med.+J.+Aust.&title=Medical+J.>
  104. Rosenkranz RR, Bauer A, Dzewaltowski DA. Mother-daughter resemblance in BMI and obesity-related behaviors. *Int J Adolesc Med Health.* 2010;22(4):477-489.  
<http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=med7&NEWS=N&AN=21404879>.
  105. Chen J-L, Wall D, Kennedy C, Unnithan V, Yeh C-H. Predictors of increased body mass index in Chinese children. *Prog Cardiovasc Nurs.* 2007;22(3):138-144.  
<http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=med5&NEWS=N&AN=17786089>.
  106. Tremblay MS, Willms JD. Is the Canadian childhood obesity epidemic related to physical inactivity? *Int J Obes.* 2003;27(9):1100-1105. doi:10.1038/sj.ijo.0802376
  107. Felso R, Lohner S, Hollody K, et al. Relationship between sleep duration and childhood obesity: Systematic review including the potential underlying mechanisms. *Nutr Metab Cardiovasc Dis.* 2017;27(9):751-761.  
doi:<https://dx.doi.org/10.1016/j.numecd.2017.07.008>
  108. Kim H-Y. Statistical notes for clinical researchers: assessing normal distribution (2) using skewness and kurtosis. *Restor Dent Endod.* 2013;38(1):52.  
doi:10.5395/RDE.2013.38.1.52
  109. World Health Organization. WHO BMI Z-score cut-offs. WHO.  
[https://www.who.int/growthref/who2007\\_bmi\\_for\\_age/en/](https://www.who.int/growthref/who2007_bmi_for_age/en/). Published 2007. Accessed December 10, 2019.
  110. Defining Adult Overweight and Obesity | CDC.  
<https://www.cdc.gov/obesity/adult/defining.html>. Accessed April 28, 2020.
  111. Avis JL, Ambler KA, Jetha MM, Boateng H, Ball GD. Modest treatment effects and high program attrition: The impact of interdisciplinary, individualized care for managing



- paediatric obesity. *Paediatr Child Health*. 2013;18(10):e59. doi:10.1093/PCH/18.10.E59
112. Skelton JA, Martin S, Irby MB. Satisfaction and attrition in paediatric weight management. *Clin Obes*. 2016;6(2):143-153. doi:10.1111/cob.12138
  113. Reinehr T. Lifestyle intervention in childhood obesity: changes and challenges. *Nat Rev Endocrinol*. 2013;9(10):607-614. doi:10.1038/nrendo.2013.149
  114. Carnell S, Wardle J. Appetitive traits and child obesity: Measurement, origins and implications for intervention. *Proc Nutr Soc*. 2008;67(4):343-355. doi:10.1017/S0029665108008641
  115. De Graaf C, Blom WAM, Smeets PAM, Stafleu A, Hendriks HFJ. Biomarkers of satiation and satiety. *Am J Clin Nutr*. 2004;79(6):946-961.
  116. Govey MA, Lim CS, Clifford LM, Janicke DM. Disordered eating and health-related quality of life in overweight and obese children. *J Pediatr Psychol*. 2014;39(5):552-561. doi:10.1093/jpepsy/jsu012
  117. Loh DA, Moy FM, Zaharan NL, Mohamed Z. Eating behaviour among multi-ethnic adolescents in a middle-income country as measured by the self-reported Children's Eating Behaviour Questionnaire. *PLoS One*. 2013;8(12):e82885. doi:https://dx.doi.org/10.1371/journal.pone.0082885
  118. Furnham A. Response bias, social desirability and dissimulation. *Pers Individ Dif*. 1986;7(3):385-400. doi:10.1016/0191-8869(86)90014-0
  119. Foley L, Maddison R, Olds T, Ridley K. Self-report use-of-time tools for the assessment of physical activity and sedentary behaviour in young people: systematic review. *Obes Rev*. 2012;13(8):711-722. doi:10.1111/j.1467-789X.2012.00993.x