

**The Weight of Parenting:  
A Cross-Sectional Analysis of Family Stress and Body Composition  
Among Parents in the Guelph Family Health Study**

**by**

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## ABSTRACT

### **THE WEIGHT OF PARENTING: A CROSS-SECTIONAL ANALYSIS OF FAMILY STRESS AND BODY COMPOSITION AMONG PARENTS IN THE GUELPH FAMILY HEALTH STUDY**

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This thesis is an investigation of the associations between family-level stress and adiposity in a sample of parents (64 mothers and 61 fathers) participating in the Guelph Family Health Study. The demands of parenthood may increase stress levels, placing parents at risk of stress-associated adiposity. We used linear regression models with the generalized estimating equation approach to investigate the associations between family-level stress (general stress, parent-specific stress, and household chaos) and body mass index, waist circumference (WC), waist-to-height ratio (WHtR), and percent fat mass, and associations of the potential mediators cortisol, dietary fat intake, sleep, and physical activity. Overall, parents reported moderately high levels of stress, but fathers' stress levels were not significantly associated with adiposity. Among mothers, parenting-specific stress was positively associated with WC and WHtR and household chaos was positively associated with all measures of adiposity. No mediators were identified in this study.

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## List of Abbreviations

BMI – body mass index

CHAOS – Confusion, Hubbub, And Order Scale

CI – confidence interval

FM – fat mass

GFHS – Guelph Family Health Study

HPA – hypothalamic-pituitary-adrenal

IPAQ – International Physical Activity Questionnaire

MET – metabolic equivalent

PA – physical activity

PSI – Parenting Stress Index

PSQI – Pittsburgh Sleep Quality Index

SD – standard deviation

SE – standard error

WC – waist circumference

WHO – World Health Organization

WHtR – waist-to-height ratio

## 1.0 Introduction

Many Canadians lead busy lives, and it is common to experience difficulties with balancing stressful demands. Nearly 25% of Canadian adults describe daily life as “quite a bit” or “extremely stressful” (Statistics Canada, 2017). Parents of young children may be especially at risk for chronic stress because they must cope with the demands of parenthood in addition to the general demands of daily life. Together, these stressors may combine to increase the risk of obesity, which 5.1 million Canadians currently face (Statistics Canada, 2014). Weight status and adipose tissue have been foci of past research, with evidence pointing towards excess abdominal adiposity in individuals with chronic stress (Björntorp, 2001). While the link between chronic stress and adiposity has been established, less is known about the association between parenting-specific stress and weight status in parents of young children in Canada. A better understanding of how stress may uniquely affect Canadian parents is important for identifying areas where support is needed and the ways in which those supports could help prevent chronic disease development. This includes cardiovascular disease, Type 2 diabetes, obesity, chronic inflammation and immune system malfunctioning, all of which have been independently linked with chronic stress.

The present study aims to fill these gaps in the literature by exploring two research questions. First, to what extent does family stress (a combination of general stress, parenting-specific stress, and home environment stress) contribute to adiposity in parents of the Guelph Family Health Study, as measured by body mass index (BMI), fat mass percentage (%FM), waist circumference (WC), and waist-to-height ratio (WHtR)? And secondly, are these relationships mediated physiologically by cortisol and

behaviourally by dietary fat intake, sleep efficiency, and physical activity (PA)? Findings from the present study will help to illuminate the complex and multifactorial issues surrounding overweight and obesity in Canada by investigating the role of parents' stress on various measures of weight and body composition. These results can be used to inform family-based supportive strategies.

## **2.0 Review of the Literature**

### **2.1 Chronic Stress in the Canadian Population**

The World Health Organization (WHO) states “there is no health without mental health” (2005). Mental health is defined as the state in which an individual can fully realize their own capabilities, cope with normal stressors of life, work productively, and positively contribute to their community (WHO 2005). Good mental health status is an integral component of one’s cognition, emotional and social interactions, and ability to enjoy life (WHO 2005). It is important to note that this definition of health extends beyond simply the absence of disease or ailment to include all factors that contribute to a state of complete physical, mental, and social well-being (WHO 2005). Chronic stress exposure can detract from mental health by repeatedly causing the body to make adaptive adjustments to maintain homeostasis. Over time, this can drain the body’s ability to repair its psychological networks, contribute to “wear and tear” on the body, and cause maladaptation and adverse health outcomes (Seib et al., 2014). The impact of chronic stress on health is being increasingly acknowledged as a public health issue (Seib et al., 2014).

Chronic stress is a relatively common condition, and many Canadians find themselves in stressful situations every day. Statistics Canada’s 2014 report showed

that 23.5% of Canadians (6.7 million) aged 15 or older described their average day as “quite a bit” or “extremely stressful”. The 35-54 year age bracket reported the highest levels of daily stress, with up to 30% of respondents describing most days as “quite a bit” or “extremely stressful” (Statistics Canada, 2017b). There is a gender difference in perceived life stress reporting; men are consistently less likely to report stress than their female counterparts (Statistics Canada, 2017a). The mechanisms behind these gender differences remain poorly understood, but some evidence suggests that women are more susceptible to stress from a greater diversity of life sources such as family relationship strain and perceived life constraints (Block, He, Zaslavsky, Ding, & Ayanian, 2009).

The stress response is coordinated by the hypothalamic-pituitary-adrenal (HPA) axis and includes collaborative behavioural and physiological processes. The stress response serves to maintain the body’s internal balance in the face of demands upon it (Torres, Turner, & Nowson, 2010). These demands made upon the body are known as stressors, and they can take many forms including physical (e.g., injury), physiological (e.g., extreme cold), or psychological factors (e.g., emotional distress) that threaten homeostasis (Foss & Dyrstad, 2011; Torres et al., 2010). Acute stressors trigger responses lasting from seconds to hours, while the effects of chronic stressors can last for days or months (Torres et al., 2010). Stress influences behavioural, endocrine, metabolic, and immune system functioning which can push the body outside of its normal resting state to protect itself (Chen & Qian, 2012), which is in turn connected to appetite, ingestion, energy storage, and energy mobilization (Adam & Epel, 2007). The disruption of the body’s energy budget that accompanies chronic stress is associated

with elevated circulating inflammatory markers, increased risk of diabetes and some cancers, suppressed immune functioning, impaired weight management, cardiovascular health, cognition, and poorer quality of life (DeCaro & Worthman, 2011; Goldman-Mellor, Brydon, & Steptoe, 2010).

## **2.2 Stress and Obesity**

Obesity has gained attention as a global public health problem. In 2014, an estimated 1.9 billion adults worldwide were overweight, over 600 million of whom were obese (Sampasa-Kanyinga & Chaput, 2017). Canadians contribute 5.1 million adults (over 20% of the adult population) to this statistic (Statistics Canada, 2014). As a global epidemic that negatively affects health and well-being, it is imperative that emphasis is placed on uncovering the many factors that may increase the risk of obesity including the ways in which stress may manifest itself as a contributor (Sampasa-Kanyinga & Chaput, 2017).

Block and colleagues (2009) followed a nationally-representative American cohort (n = 1355) for 9 years and found psychosocial stress to be significantly positively associated with weight gain in those with a higher baseline BMI. The association between stress and weight gain has been more consistently shown in women versus men, which may be due to the higher levels of stress experienced by women or differences in how women cope with stressors, or due to physiologic differences in responses among men and women (Block et al., 2009). In a nationally-representative sample of Canadian adults (n = 13,926), Sampasa-Kanyinga and Chaput (2017) found that work and life stresses were associated with excess weight. A study by Chen and Qian (2012) examined 112,716 Canadian subjects' (aged 18 and above) responses to a

survey about their daily stress levels. Nearly 20% of respondents reported being “quite a bit stressed” and an additional 3.7% reported being “extremely stressed” in their daily lives. Only 17% of respondents reported no daily stress. While the extremely stressed women had greater obesity than any other group, any level of stress was associated with greater risk of obesity.

The relationship between stress and weight is both acute and chronic in nature. A 15-year longitudinal study of 3,617 people hypothesized that maintaining high levels of stress or increased stress levels from childhood to adulthood would contribute to faster weight gain over time (Liu & Umberson, 2015). The results showed that women with higher childhood stress gained weight faster during the 15-year study observation period than women with low childhood stress. Among males, neither childhood nor adulthood stress was found to be associated with weight gain trajectories. Liu and Umberson (2015) conclude that stress “reverberates throughout the life course to foster cumulative disadvantage in body mass” in women. Chronic stress exposure may perpetuate the metabolic adjustments to stress and maintain these adjustments at a level that promotes weight gain (Isasi et al., 2015).

Psychological distress increases the risk of Type 2 diabetes, cardiovascular disease, and all-cause mortality; it is thought that increased adiposity may facilitate this reciprocal relationship (Murabito, Massaro, Clifford, Hoffmann, & Fox, 2013). Epidemiological studies show that obesity works synergistically with mental health to worsen severe health consequences (Mumford, Liu, Hair, & Yu, 2013). Block and colleagues (2009) found that American women with normal weight and depression have twice the cortisol and intraabdominal fat than their non-depressed counterparts.

Overweight and obesity contribute to the risk of preventable health conditions such as cardiovascular disease, Type 2 diabetes, hypertension, musculoskeletal diseases, and cancer (Ul-Haq, Mackay, Fenwick, & Pell, 2014). All-cause mortality is increased in obesity (Ul-Haq et al., 2014). Diet and PA have been the primary foci of obesity prevention and treatment research; however, much variance is left unexplained. Continued improvements in the preventative health care field depend on identifying and better understanding factors that contribute to poor health (Seib et al., 2014). This opens the door for chronic stress's role in partly explaining the pathogenesis of overweight and obesity.

### **2.3 Family Stress**

While a substantial proportion of the Canadian population experiences daily stress, the experiences of parents of young children remain largely unknown. This population may be at special risk for stress-related health consequences due to the unique challenges of parenthood. The many demands of parenthood may act as additional sources of psychosocial stress. Parenting stress encompasses the distress resulting from the demands of being a parent (Hayes & Watson, 2013). These include work-family conflict, parent-child engagement, fatigue, financial, and other strains (Berryhill & Durtschi, 2017).

Normal developmental milestones of children may also act as sources of stress for parents. DeCaro and Worthman (2011) investigated biomarkers of parental stress following a child's entry to kindergarten in a study with 29 families (51 parents). Linear regression showed that greater disturbance of family routines and difficulty adapting were significantly and positively associated parents' salivary cortisol levels after

controlling for baseline. This study shows that disruptions in the home environment, even if the cause is a normal and positive one, can affect parental stress at a hormonal level which, in turn, can contribute to weight gain. Research has shown that maternal parenting stress remains relatively constant over time, meaning that highly stressed mothers of young children become highly stressed mothers of adolescents, and so on (Berryhill & Durtschi, 2017). Strategies to manage or lower this stress to protect parent health are important.

Family stress can foster unhealthy lifestyle behaviours that can contribute to obesity. MacRae and colleagues (2017) discovered a significant positive association between chaotic home environments and dietary fat consumption in parents of young children which can contribute to excess caloric intake and weight gain. Busy families frequently consume more convenience-based, less nutritious foods (Patrick & Nicklas, 2005). Additionally, busy families may be less likely to share meals together, which is known to be associated with lower intake of healthier foods in both parents and children (Patrick & Nicklas, 2005). Difficulties with managing parenting stress may impair a parent's ability to provide care for their child. Walton and colleagues (2013) found that greater levels of parenting stress were associated with children's unrestricted television viewing and decreased engagement in active play, two factors that may contribute to childhood obesity. Reducing parenting stress may help to restore family functioning and allow parents to engage in more positive parenting practices (Hayes & Watson, 2013). Understanding how stress among young parents may influence their obesity risk may inform strategies to help parents to develop the skills needed to overcome future challenges is paramount to protecting family health.

## **2.4 Physiological Impact of Chronic Stress**

### **2.4.1 Cortisol is the Primary Biomarker of Stress**

Despite the diversity of forms that stressors may take, there is a common chemical mediator produced that facilitates the stress response throughout the body. This mediator is cortisol, a glucocorticoid hormone synthesized by the adrenal gland. Cortisol initiates a chain of metabolic reactions that prepare the body for action, including increased lipolysis, gluconeogenesis, and glycogenolysis to mobilize energy for muscles, increased heart rate, heightened senses and alertness, and stimulation of the immune system (Konishi & O'Connor, 2016). As a result of its centrality in the metabolic aspects of the stress response, cortisol is considered the primary biomarker of the physiological burden of stress (DeCaro & Worthman, 2011). Levels of psychosocial stress, such as work- or relationship-related stress, are mirrored by cortisol levels (DeCaro & Worthman, 2011). Cortisol is a powerful influencer of metabolism that is intended to be reserved for acute responses to stress. Recognizing that chronic stress contributes to increased risk of preventable chronic disease is an important step towards protecting the health of stressed individuals.

### **2.4.2 Chronic Stress and Increased Risk of Adiposity**

As a hormone, cortisol exerts whole-body effects. This ability to effectively influence multi-tissue systems is an essential component of the acute stress response for survival purposes, however, chronic stress can easily disadvantage the whole body by the same principles. The multifactorial impacts of cortisol are understudied in terms of chronic health implications. Existing evidence supports the notion that cortisol is the central mediator of the chronic stress and health relationship, which warrants further

investigation into how cortisol may be associated with adiposity. Excess adiposity is a symptom of hypercortisolemic disorders, such as Cushing's Syndrome; conversely, hypocortisolemic disorders, such as Addison's Disease, are associated with weight loss and appetite suppression (Foss & Dyrstad, 2011). This demonstrates the centrality of cortisol in the maintenance of body weight.

While cortisol exerts whole-body effects, its power to influence adipocyte functioning is critical to understanding how chronic stress contributes to preventable disease and negative health outcomes. Cortisol alters adipocyte regulation, hormonal pathways, and receptor-level signaling. One key feature of adipose tissue is the high density and sensitivity of glucocorticoid receptors in visceral adipose tissue, which may help to explain how cortisol facilitates such a large impact (Black, 2006; Holmes, Ekkekakis, & Eisenmann, 2009). Higher HPA activity or hypercortisolemic states cause adipocyte proliferation and growth (Foss & Dyrstad, 2011) while correcting the balance of cortisol reverses these symptoms (Glasow & Bornstein, 2000). Glucocorticoid exposure is powerful enough to alter the phenotypic characteristics of adipose connective tissue cells to appear more like adipocytes (Glasow & Bornstein, 2000). In addition to adipose tissue having a greater density of glucocorticoid receptors, stress exposure is again magnified for adipose tissue by the enzyme 11 $\beta$ -hydroxysteroid dehydrogenase type 1 (11 $\beta$ -HSD1) which converts cortisone into the active molecule cortisol (Black, 2006). 11 $\beta$ -HSD1 is overly expressed in obese adipose tissue, and mRNA levels have a positive association with waist circumference and insulin resistance (Hirata et al., 2012). Obese adipose tissue has elevated 11-HSD1 activity, which combines with lower liver 11-HSD1 activity to promote visceral hypercortisolemia (Holmes et al., 2009). Visceral

hypercortisolemia is achieved by the greater density of glucocorticoid receptors and upregulation of cortisol activation by 11-HSD1 to further compromise adipocyte health and encourage adiposity.

Lipid metabolism is another route by which cortisol influences whole-body energy storage. Acute stress triggers whole-body lipolysis to mobilize energy (Adam & Epel, 2007). Hypercortisolemic environments promote increased lipoprotein lipase (LPL) and hormone-sensitive lipase (HSL) activity (Holmes et al., 2009). These enzymes convert triglycerides to free fatty acids in circulation and within cells, respectively (Holmes et al., 2009). Insulin pairs with cortisol to promote LPL activity, which favours lipid storage in adipocytes to contribute to visceral adiposity (Black, 2006; Holmes et al., 2009). HSL is chiefly responsible for atherosclerotic plaque development (Holmes et al., 2009). For individuals with a dysregulated stress response, glucocorticoids may promote esterification of blood vessel smooth muscle to worsen lipoprotein attack by macrophages and the resultant cholesterol accumulation and foam cell/fatty streak formation (Holmes et al., 2009). Together with insulin, cortisol promotes lipid accumulation by directly stimulating lipoprotein lipase or indirectly inhibiting the lipolytic influence of growth hormones (Adam & Epel, 2007).

Chronic stress, while exerting effects throughout the body, uniquely disadvantages adipose tissue health. This is achieved through cortisol's innate propensity to act on adipocytes, adipocytes' responsiveness to cortisol, and the lipogenic adipocyte environment, among other mechanisms. These stress-induced disruptions can contribute to weight gain, excess adiposity, and increase the risk for

obesity. Understanding the impact of stress on cortisol levels and obesity risk is important to elucidate the long-term impacts of chronic stress.

## **2.5 Behavioural Manifestations of Stress Which Increase Risk of Obesity**

### **2.5.1 Dietary Patterns are Influenced by Stress**

Past research has clearly identified the many links between diet and health. Stress can influence dietary patterns in several key ways to adversely affect health, including altering dietary preferences, altering amount consumed, and stimulating eating in the absence of hunger (Chen & Qian, 2012). Acute stress tends to increase caloric intake in 35-70% of the population, (Adam & Epel, 2007; Chen & Qian, 2012; Ulrich-Lai, 2016) and chronic stress is associated with overeating (Ulrich-Lai, 2016). Regardless of the amount of food consumed, chronic life stress tends to be associated with an increased preference for sugary and fatty foods (Chen & Qian, 2012; Zellner et al., 2006), consumption of which is made easier in the fat- and sugar-rich Western dietary landscape (Adam & Epel, 2007). This is especially the case for women (Epel, Lapidus, & McEwen, 2001; Zellner et al., 2006). Highly palatable foods, including those high in sugars and fats, have a calming effect on the HPA axis and trigger the opioid, dopamine, and endocannabinoid limbic systems (thus creating a “high” or rush of pleasure) which may attenuate the HPA axis in times of stress to provide a surrogate calmness (Adam & Epel, 2007; Groesz et al., 2012). Corticosterone administration can increase sugar cravings, hyperphagia, and weight gain (Epel et al., 2001), while adrenalectomy and glucocorticoid receptor antagonists can prevent or reverse obesity (Epel et al., 2001). Minor daily stress repetition may cause chronic activation of the stress system and prime the body to overeat to try to restore energy balance (Adam &

Epel, 2007). Groesz and colleagues (2011) determined that greater stress was positively associated with a stronger drive to eat and the consumption of more highly palatable, non-nutritive foods. Perceived stress was negatively associated with healthy eating, including vegetable and whole grain consumption (Groesz et al., 2012). In addition to vegetable and whole grains, fruit consumption is negatively associated with stress (Zellner et al., 2006). The combination of increased snacking and the tendency to choose snacks high in sugar or fat can increase daily caloric intake and promote weight gain (Zellner et al., 2006).

Emotional eating occurs when a stressed individual cannot distinguish between hunger and anxiety which can contribute to overconsumption (Conner, Fitter, & Fletcher, 1999). Lemmens and colleagues (2010) found that viscerally overweight subjects exposed to stress had higher energy intake in the absence of hunger, while their normal weight counterparts were consistent between stress and rest conditions. Stress eating differences by gender can be seen in individuals as young as 15 years of age (Block et al., 2009). In an analysis of high school students aged 15-19, stressful days were significantly associated with increased caloric intake by girls, though boys did increase fat consumption (Block et al., 2009). Together, this supports the notion that stress may influence macronutrient preference, and that the strength of that influence differs by gender. While there has been substantial interest in the association between stress and dietary patterns, the biological mechanisms driving these processes are not well understood (Appelhans, Pagoto, Peters, & Spring, 2010). Large studies do not tend to focus on chronic stress exposure and eating behaviours as much as they do acute stress (Groesz et al., 2012). Stress has the power to influence food preferences,

amount consumed, and eating in the absence of hunger. These relationships are complex, and much remains unknown. More research is needed to better clarify these relationships.

### **2.5.2 Physical Activity May be Influenced by Stress**

PA can be conceptualized as the increase of energy expenditure above resting levels due to skeletal muscle activity (Stults-Kolehmainen & Sinha, 2014). Broadly, this includes both deliberate exercise and physical activities done during normal daily life. There is strong evidence supporting the health benefits of PA, including reduction of obesity (Stults-Kolehmainen & Sinha, 2014) by increasing energy expenditure. This can help attenuate the effects of chronic stress on weight gain. Additionally, it is widely accepted that PA is an effective treatment for stress and can elevate mood, partly due to endorphin release, and these benefits are independent of fitness level (Salmon, 2001). Interestingly, Stults-Kolehmainen & Sinha (2014) found that PA can improve tolerance of future stressors and lessen the severity of perceived stress. However, the reverse relationship of how PA is affected by mental health is under-researched (Stults-Kolehmainen & Sinha, 2014). Stults-Kolehmainen & Sinha (2014) concluded from their systematic review of the literature that evidence points towards stress impairing efforts to engage in PA. Their evidence suggests that the tasks associated with being physically active outweigh the perceived benefits, or that those tasks are “overwhelming”, “inconvenient”, or otherwise intrinsically stressful. Despite evidentiary support for PA’s many benefits, it can be viewed as a burdensome chore which deters people from engaging in PA especially in stressful times where it would be most beneficial (Langlie, 1977; Stults-Kolehmainen & Sinha, 2014). Stress may compound

adverse health effects by impairing desire to engage in PA, thereby limiting the ability of PA to mitigate stress symptoms, and cumulatively contribute to adipose tissue development.

### **2.5.3 Stress Can Influence Sleep**

Together with adequate PA, sleep is known to contribute to good quality of life, healthy weight maintenance, and chronic disease prevention (Sampasa-Kanyinga & Chaput, 2017). Disrupted sleep patterns are an important correlate of stress with substantial health consequences including higher BMI, greater perceived stress levels, disordered neuroendocrine appetite control, and greater caloric intake (Dweck, Jenkins, & Nolan, 2014; Sampasa-Kanyinga & Chaput, 2017). Additionally, preference for fats and sugars increases after sleep restriction (less than 7 hours per night) (Dweck et al., 2014). Chronic sleep deprivation can increase cortisol, leptin, and ghrelin to promote adiposity (Gunderson et al., 2008), as well as proinflammatory cytokines (Milrad et al., 2016; Santos, Tufik, & Mello, 2007).

Stress is associated with altered sleep patterns as both a cause and consequence, the result of which is an increased risk of overweight or obesity. Stress is viewed as the primary cause of chronic insomnia (Kerstedt, 2006). Cortisol production has a positive association with reported sleep disturbance (Lopresti, Hood, & Drummond, 2013). A cross-sectional analysis has found a 25% higher incidence of sleep disruptions in those with high job stress (Kalimo, Tenkanen, & Ha, 2000). However, sleep duration and health is a delicate balance; epidemiological studies have shown men and women to be at greater risk of obesity and chronic disease with both restricted and extended sleep durations (Gunderson et al., 2008). Chronic sleep

restriction is a characteristic of many modern lifestyles and may pose long-term health consequences. At 6 year follow-up, those who slept less than 6 hours per day had greater BMI and fat mass than short sleepers who increased sleep to 7-8 hours per day (n = 43) (Chaput, Despres, Bouchard, & Tremblay, 2012). Uncovering how sleep and stress combine to impact adiposity is an important step forward for health promotion.

## **2.6 Chronic Stress Poses Bio-Behavioural Risks on Parents' Adiposity**

Many behavioural factors that contribute to health may be compromised in parents to increase adiposity and chronic disease risk. Sleep is an important contributor to the stress-health relationship, and parents of young children may be at special risk for sleep-related health consequences because the transition to parenthood is strongly associated with sleep deprivation. Gunderson and colleagues (2007) found that mothers who experienced sleep deprivation (< 5 hours per day) at 6 months postpartum were 2.3 times more likely to retain 5 kg or more at 1 year postpartum (n = 940). Limited research has investigated maternal sleeping patterns beyond 1 year postpartum (Gunderson et al., 2008) however Taveras and colleagues (2011) confirm in a sample of 586 new mothers that  $\leq 5$  hours sleep per day during the first year postpartum is associated with greater overall and central maternal adiposity at 3 years postpartum. PA patterns may also be disrupted by the transition to parenthood. Hull and colleagues (2011) found that parents participated in an average 2.5 ( $\pm 5.0$ ) hours per week less PA than their childless counterparts. The factors associated with parenthood may act in concert with the inhibitions identified by Stults-Kolehmainen & Sinha (2014) to lessen engagement in PA, such as viewing PA as burdensome or a chore. Likewise, dietary patterns may shift as parents adjust to the demands of their child's diet. More research

is needed to explore this relationship further into parenthood, and to identify how these behavioural factors contribute to parental health.

Research has found associations between chaotic home environments, excess weight gain, and poor health behaviours, including poor sleep and emotional eating (Dumas et al., 2005; Lumeng et al., 2014). However, the effect of home chaos on parental health is largely unknown (MacRae, Darlington, Haines, & Ma, 2017). Using pilot data from the Guelph Family Health Study, MacRae and colleagues found home chaos to be significantly positively associated with parents' dietary fat intake and highlighted the importance of these novel findings for parents' modeling of high-fat eating behaviours to their children. Supporting parents through stress management strategies is an important contributor to family health. Higher levels of parenting stress are associated with poorer parenting practices and family functioning (DeCaro & Worthman, 2011; Walton, 2013). Walton and colleagues (2013) found that parenting stress was significantly associated with unhealthy family behaviours including failure to meet recommended PA levels and excess television viewing. This suggests that parental stress extends beyond the individual level to impact the healthy practices of the family unit. It is important to better understand the stress patterns in Canadian parents so that strategies can be designed and implemented to support good health behaviours in parents as well as to prevent the modeling of poor health behaviours from parents to their children (Walton, Filion, Darlington, Morrongojello, & Haines, 2015). What remains unknown is how these environmental stressors interact with the physiological and behavioural mechanisms of chronic stress on adiposity risk among Canadian parents with young children.

### **3.0 Rationale, Objectives, and Hypotheses**

#### **3.1 Rationale**

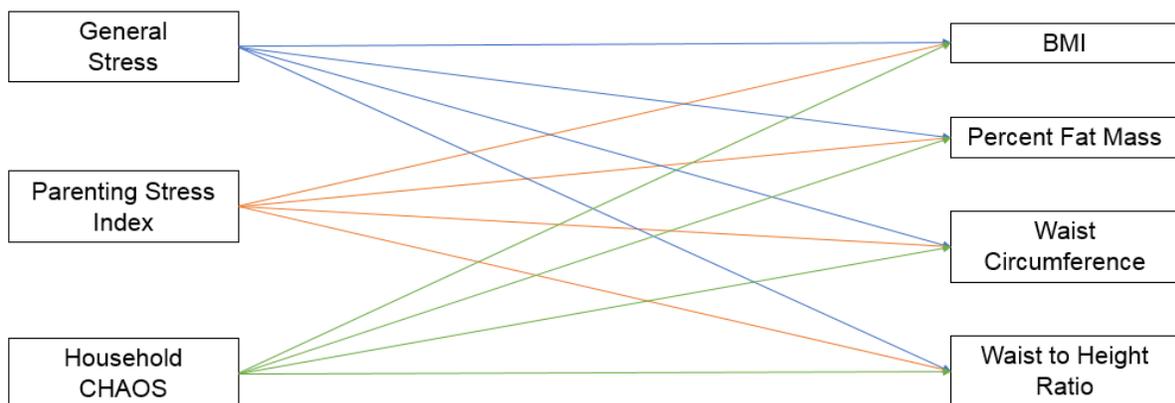
The relationship between stress and obesity risk is largely unknown in the general population (Chen & Qian, 2012), and few studies have examined this association in the Canadian population. Research is also needed to elucidate the behavioural and physiological mechanisms by which stress may lead to obesity risk. To best encompass all elements of “stress”, both physiological measures (such as cortisol) and psychosocial measures (such as perceptions of distress) should be included in the operationalization of stress in future research (Chen & Qian, 2012; Holmes et al., 2009), which is what the proposed research aims to do. For obesity treatment and prevention, a focus on the conventional energy imbalances of diet and exercise is a narrow perspective that leaves much variance unaccounted for and highlights the need to examine other influences including chronic stress (Holmes et al., 2009). Identifying solutions and preventative strategies is dependent on a deeper understanding of the factors that contribute to obesity.

Previous research within the Guelph Family Health Study (GFHS), a family-based cohort study, found a significant positive association between household stress and dietary fat intake (MacRae et al., 2017). This proposed research builds upon this work by investigating two additional measures of parents’ stress (Parent-Perceived Stress and the Parenting Stress Index) and explores the impact of stress on parents’ body composition outcomes. The inclusion of cortisol and dietary fat intake as mediators is supported by MacRae et al.’s investigation (2017), and the mediation model investigation is further strengthened by examining sleep quality and PA as potential

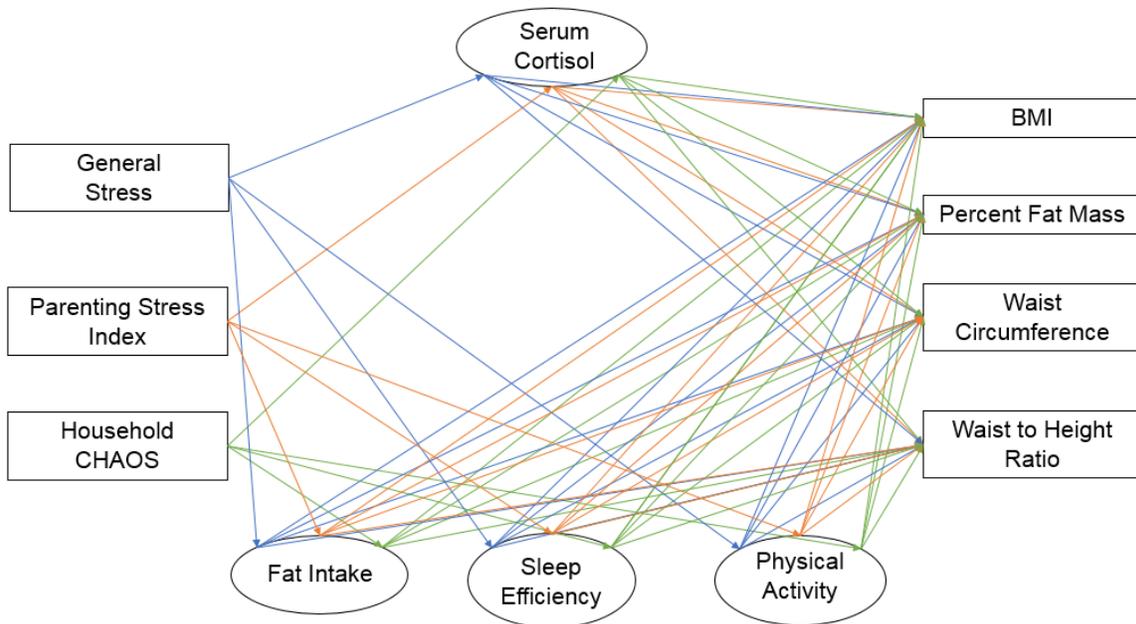
mediators. Additionally, this study benefits from increased sample size due to the recruitment of new waves of participants into the GFHS since MacRae et al.'s investigation (2017). Findings from this proposed study will help to illuminate the complex and multifactorial issues surrounding overweight and obesity in Canada by investigating the role of parents' stress on various measures of weight and body composition. These results can be used to inform family-based supportive health strategies.

### 3.2 Research Objectives

The present study aims to fill gaps in the literature by asking two questions. First, to what extent does family stress (measured independently as parent-perceived stress, parenting-specific stress, and home environment chaos) contribute to adiposity as measured by BMI, WC, WHtR, and %FM in Canadian parents (Figure 1)? And secondly, are these relationships mediated by physiological factors (i.e., cortisol) and behavioural factors (i.e., dietary fat intake, sleep quality, and PA) (Figure 2)?



**Figure 1** Linear regression of body composition outcomes on family-level stress.



**Figure 2** Linear regression of body composition outcomes on family-level stress as potentially mediated by serum cortisol, dietary fat intake, sleep efficiency, and physical activity.

### 3.3 Hypotheses

We hypothesize that each of the three family-level stress measures will be positively associated with each of BMI, WC, WHtR, and %FM. We also hypothesize that both cortisol and behavioural mechanisms (dietary fat intake, sleep efficiency, and PA) will mediate this relationship; specifically, greater stress is predicted to be associated with markers of adiposity, as explained by greater cortisol and dietary fat intake as well as lower sleep efficiency and PA.

## 4.0 Materials and Methods

### 4.1 Study Participants

The Guelph Family Health Study is a longitudinal cluster randomized trial focusing on childhood obesity prevention. As such, all participants must reside in

Wellington County, Ontario, Canada, with at least one child aged 18 months to 5 years. Using baseline data from participants of the Guelph Family Health Study (GFHS) pilot cohorts, 152 parent participants from 86 families were recruited for this analysis. Ten participants were excluded from analyses due to missing data. Additionally, 16 female participants were excluded due to inability to accurately assess body composition while pregnant or breastfeeding. One male participant was excluded due to a religious exemption from body composition assessment. This yielded a final analytic sample of 125 parent participants from 72 families. Data were collected between December 2014 and August 2016 as part of baseline assessment. The GFHS consent form is available in Appendix A.

## **4.2 Measures**

### **4.2.1 Stress Measures**

Participants completed three stress surveys either online or on paper. General stress was evaluated using the question “Using a scale from 1 to 10, where 1 means ‘no stress’ and 10 means ‘an extreme amount of stress,’ how much stress would you say you have experienced in the last year?” from the Public Health Management Corporation Community Health Data Base’s Southeastern Pennsylvania Household Health Survey (2015) (Appendix B). Parenting-specific stress was evaluated using the 12-question Parental Distress subscale of the Parenting Stress Index (PSI) developed by Abidin et al. (1990). On a 5-point Likert scale from 1 (strongly disagree) to 5 (strongly agree), participants responded to statements such as “I often have the feeling that I cannot handle things very well”, “I feel trapped by my responsibilities as a parent”, and “Having a child has caused more problems than I expected in my relationship with my

spouse (or male/female friend)”; the full survey used can be found in Appendix C. A total score out of 60 was obtained by summing the responses with higher scores indicating greater parental distress. Standardized Cronbach’s alpha for this survey in our sample was 0.76. Home environment chaos was evaluated using the Confusion, Hubbub, And Order Scale (CHAOS) developed by Matheny et al. (1995) (Appendix D). This scale quantifies chaos as the frustration, disorganization, and noisiness of a home. On a 4-point Likert scale from 1 (very much like your own home) to 4 (not at all like your own home), one parent from each family responded to 15 statements such as “We almost always seemed to be rushed” or “It’s a real zoo in our home”. A total score out of 60 was obtained by summing the responses, where higher scores indicate greater home chaos. Standardized Cronbach’s alpha for this survey in our sample was 0.89.

#### **4.2.2 Adiposity Measures**

Adiposity was evaluated using the anthropometric measures BMI, WC, WHtR, as well as %FM using BOD POD™. Height was measured using a wall-mounted stadiometer with participants barefoot or in sock feet. Height was measured at the apex of the inhaling breath to the nearest 0.1 cm. Two measurements were taken; if measures differed by more than 0.5 cm, a third measurement was taken. The final data point was an average of the two closest measures. The full protocol can be found in Appendix E. Weight was measured in kg using the BOD POD scale, whose high reliability means that only one measurement was needed (Appendix F). WC was measured at the ileac crest to the nearest 0.1 cm using a Gulick II measuring tape (Country Technology Inc., Gay Mills, WI). In accordance with Statistics Canada and NHANES recommendations, the measurement was taken at the top of the iliac crest

over bare skin or over a thin clothing layer during the pause after expiration and before inhalation. Two measurements were taken; if measures differed by more than 0.5 cm, a third measurement was taken. The final data point was the average of the two closest measurements. The full protocol is described in Appendix G.

BMI was calculated by dividing weight (kg) by height (m<sup>2</sup>). BMI categories are defined by the World Health Organization (1995) as underweight (< 18.5 kg/m<sup>2</sup>), normal weight (18.5-24.9 kg/m<sup>2</sup>), pre-obese (25.0-29.9 kg/m<sup>2</sup>), obesity class I (30.0-34.9 kg/m<sup>2</sup>), obesity class II (35.0-39.9 kg/m<sup>2</sup>), and obesity class III (> 40 kg/m<sup>2</sup>).

Participants' WHtR was calculated by dividing WC (cm) by height (cm). WHtR is complementary to WC because it is adjusted for the individual's height and improves the accuracy in predicting disease risk (Ashwell, Gunn, & Gibson, 2012; Browning, Hsieh, & Ashwell, 2010; Man Ying Lee, Huxley, Wildman, & Woodward, 2008).

Percent fat mass was assessed in 124 participants using BOD POD, an air displacement plethysmograph. The BOD POD measured body volume, calculated body density, and used the Siri equation (Siri, 1961) to estimate fat mass and fat-free mass (kg and % body weight). Participants wore tight-fitting clothing such as a bathing suit and a bathing cap to minimize air trapping and removed jewelry and glasses (all that which is not 'self'). Participants were instructed to sit quietly, limit movement, and breathe normally while in the test chamber. Thoracic gas volume was measured (n=92) or calculated (n=32) to give body volume. By calculating body volume, body fat percentage can be approximated. The entire BOD POD test takes approximately 15 minutes per participant. By calculating body volume, %FM can be approximated. Our reliability testing of %FM measurement showed an intra-individual day-to-day variation

of 2.1% and intra-operator variation of 2.6, which align with reference values of 1.7-4.5% and 2.7%, respectively (Fields, Goran, & McCrory, 2002). The instrument was calibrated twice in the morning of each data collection day; once with the test chamber empty, and once with a 49.980 L calibration cylinder in the chamber. The full protocol is described in Appendix H.

#### **4.2.3 Potential Mediators**

For this analysis, the potential mediators of cortisol, dietary fat intake, PA, and sleep efficiency were considered. Blood cortisol samples were collected in the morning by LifeLabs Medical Laboratory Services in Guelph and analyzed by LifeLabs using the Siemens Advia Centaur cortisol assay, a competitive immunoassay with chemiluminescent detection. Fasting was not a requirement of this test. Collection of cortisol samples in the morning provides a stronger indication of basal diurnal rhythms than other times of day (DeCaro & Worthman, 2011).

Parents' dietary fat intake was quantified using the Block fat screener (Block, Gillespie, Rosenbaum, & Jenson, 2000) (Appendix I). The screener listed 16 high-fat food items and the participant responded with their consumption frequency from 1 (once per month or less) to 5 (5 or more times per week). A total score was calculated by summing the responses with a score of 23 or higher indicating high fat consumption (Block et al., 2000). Due to a change in protocol subsequent to Phase I of this study, dietary fat intake data are only available for the 52 parents in the Phase I cohort.

PA data were collected by parent self-report using the International Physical Activity Questionnaire Short Form (IPAQ), a validated tool developed to assess health-related PA (Booth, 2000) (Appendix J). Participants recorded the duration and intensity

of their PA for an average week. The total PA for the average week was calculated by multiplying the active minutes per week by the IPAQ coefficient for that type of activity, called a MET score, where METs are multiples of the resting metabolic rate if one MET-minute is equal to kilocalories of expenditure for a 60kg person. Walking, moderate PA, and vigorous PA have MET coefficients of 3.3, 4.0, and 8.0, respectively. As per IPAQ protocol, if a range of values was given the mean value was used as the data point (i.e., a response of 10-20 minutes was converted to 15 minutes).

Sleep efficiency (a ratio of the time spent sleeping relative to the time spent in bed) data were collected using a subset of questions from the Pittsburgh Sleep Quality Index (PSQI), a validated self-report survey (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) (Appendix K). As for PA, for sleep responses with a range of values, the mean value was used as the data point (i.e., a response of 10-20 minutes was converted to 15 minutes).

### **4.3 Analytic Technique**

Statistical analyses were performed using SAS University Edition Version 3.6 (SAS Institute Inc. 2015). Linear regression coefficient estimates ( $\hat{\beta}$ ) and 95% confidence intervals (CI) were calculated using generalized estimating equations (GEEs). The GEE approach was selected to account for the correlation between cohabitating participants (Liang & Zeger, 1986). Post hoc, models were stratified by gender based on preliminary evidence of differences by parent gender. Independently, each body composition measure was regressed onto each of the stress measures. Models were adjusted for age, family size (total number of people living in the home),

and annual household income. A  $p$ -value  $\leq 0.05$  was considered statistically significant for all tests.

Mediation was investigated by first regressing the potential mediators onto the stresses, and then regressing the body composition measures on the potential mediators. Both of these regressions must show significant associations between the potential mediating variable, predictor, and outcome for mediation to occur (Mackinnon, Lockwood, Hoffman, West, & Sheets, 2002).

#### **4.4 Sample**

This analysis involved parent participants in both the Pilot Phase 1 ( $n = 83$ ) and Pilot Phase 2 ( $n = 69$ ) cohorts, yielding a total 152 individuals from 86 families. Due to inability to accurately measure body composition using the BOD POD during pregnancy or while breastfeeding, 16 female participants were excluded from analyses. Additionally, one male participant was excluded due to a religious exemption from body composition assessment. Health assessment data were missing for 10 individuals, and one individual did not complete any stress surveys. Finally, 8 mothers and 8 fathers were excluded because their body composition or stress data points were considered outliers because they exceeded the third quartile by an excess of three times the interquartile range. With these considerations in mind, the final analytic sample size consists of 61 fathers and 64 mothers from 72 families.

The CHAOS survey was completed only by parent one, i.e. the first parent to sign up for the GFHS. In dual-caregiver households, they share the same CHAOS score as it represents a shared environment. In cases where parent one did not complete the CHAOS survey ( $n = 3$ ), both caregivers are excluded from that analysis. The mediation

models have several cases of participant exclusion due to incomplete data. Additionally, the Block Rapid Fat Screener was not used in the Phase 2 cohort due to a change in the study protocol. Availability of all measures is described in Appendix A.

## **5.0 Manuscript: Family-Level Stress is Associated with Adiposity in Mothers, But Not in Fathers, of Preschoolers**

### **5.1 Abstract**

**Keywords:** parenting stress, home environment, chaos, obesity, children

Background: Stress may act both biologically and behaviourally to increase adiposity.

The demands associated with parenthood may increase stress levels, placing parents at increased risk of stress-associated adiposity.

Objectives: This study aimed to investigate the associations between family-level stress (general stress, parent-specific stress, and household chaos) and adiposity, and whether these relationships were mediated by cortisol, dietary fat intake, sleep, or physical activity (PA) among parents of preschoolers.

Methods: The sample consisted of 64 mothers and 61 fathers from 72 families. Linear regression using generalized estimating equations (GEEs) was used to investigate associations between stress measures and body mass index, waist circumference (WC), waist-to-height ratio (WHtR), and percent fat mass. GEEs were also used to investigate potential mediators.

Results: Parents reported moderately high levels of stress on all three measures of family-level stress. Parenting-specific stress was positively associated with WC and WHtR among mothers. Household chaos was positively associated with all measures of adiposity among mothers. Neither cortisol, dietary fat intake, sleep efficiency, nor PA mediated the mothers' stress-adiposity relationships. Fathers' stress levels were not significantly associated with any of the adiposity measures explored.

Conclusions: Our results suggest that family-level stress is associated with adiposity in mothers, but not in fathers. Future research is needed to elucidate mechanisms by which higher stress leads to excess adiposity and how these mechanisms may differ by parent gender.

## **5.2 Introduction**

Nearly 25% of Canadians (6.7 million) aged 15 and older experience chronic stress (Chen & Qian, 2012; Statistics Canada, 2017). Stress is the body's non-specific response to any factor that may threaten or overwhelm the body's ability to maintain homeostasis (Sampasa-Kanyinga & Chaput, 2017). Chronic stress is associated with increased risk of obesity, Type 2 diabetes, cardiovascular disease, immune compromise, cancer, and other disorders (DeCaro & Worthman, 2011; Goldman-Mellor et al., 2010). As such, chronic stress is increasingly recognized as an important public health issue (Seib et al., 2014).

Chronic stress is hypothesized to influence adiposity through the disruption of the biological stress system and through the promotion of unhealthful eating, activity, and sleep behaviours. The hormone cortisol is considered the primary biomarker of the stress response; chronic stress, as indicated by elevated cortisol levels, can increase the propensity for adiposity (Per Bjorntorp & Rosmond, 2000). Cortisol disproportionately targets adipose tissue to promote excess adiposity, especially within visceral adipose tissue (Bjorntorp & Rosmond, 2000) which in turn increases the risk of chronic disease. In addition to hormonal mechanisms by which stress may increase adiposity, several behavioural mechanisms have also been explored. Chronic stress has been shown to increase snacking, particularly of high sugar and fat foods, as well

as reduce fruit, vegetable, and whole grain consumption (Zellner et al., 2006). Sleep patterns are also commonly disrupted by stress (Kerstedt, 2006). Paradoxically, physical activity (PA) has been shown to be inversely associated with stress (Stults-Kolehmainen & Sinha, 2014) despite the anxiolytic benefits of PA (Salmon, 2001).

While a substantial proportion of the Canadian population experiences daily stress, the experiences of parents of young children remain largely unknown. This population may be at special risk for high levels of stress due to the many competing demands on their time (Berryhill & Durtschi, 2017) as well as the potential stress adjusting to their role as a parent (Hayes & Watson, 2013). This high level of stress among parents may adversely impact their own health, but research also suggests that high stress may also impair a parent's ability to provide care for their child. Walton and colleagues (2013) found that greater levels of parenting stress were associated with children's unrestricted television viewing and decreased engagement in active play, two factors that may contribute to childhood obesity. Thus, reducing stress among parents may allow parents to engage in more positive parenting practices. Understanding how stress among young parents may influence their obesity risk can inform strategies to help parents develop the skills needed to successfully manage current and future stressors and is paramount to protecting family health.

Thus, the objective of this study was to investigate the associations between three measures of family-level stress (general stress, parenting-specific stress, and household chaos), and adiposity among a sample of Canadian parents of young children. It was hypothesized that family-level stress would be positively associated with adiposity. This study also examined whether these associations were mediated by

cortisol, dietary fat intake, sleep, or PA; it was hypothesized that increased cortisol and dietary fat intake as well as lower PA and sleep efficiency in the face of stress would help to explain the mechanism of any stress-adiposity association.

## **5.3 Materials and Methods**

### **5.3.1 Study Participants**

This study used baseline data collected between December 2014 and August 2016 among parents participating in the Guelph Family Health Study (GFHS) phase 1 and 2 pilot studies. The GFHS is a family-based cohort study designed to identify early life risk factors for obesity and chronic disease and to investigate family-based approaches for health promotion. Families were eligible to participate in the pilot studies if they had at least one child aged 18-months to 5 years, resided in Wellington County, Ontario, Canada, and had a parent who could respond to questionnaires in English. Forty-four participants were excluded due to missing data ( $n = 11$ ), outlying data points ( $n = 16$ ), pregnancy/breastfeeding ( $n = 16$ ), or religious exemption from body composition assessment ( $n = 1$ ). This yielded a final analytic sample of 125 parent participants (64 mothers, 61 fathers) from 72 families. The study was approved by the University of Guelph Research Ethics Board (REB14AP008).

### **5.3.2 Measures**

#### *Stress Measures*

We assessed three different types of stress via online or paper surveys. *General stress* was evaluated in both parents using the question “Using a scale from 1 to 10, where 1 means ‘no stress’ and 10 means ‘an extreme amount of stress,’ how much

stress would you say you have experienced in the last year?” (Philadelphia Health Management Corporation, 2015). *Parent-specific stress* was evaluated in both parents using the 12-item Parental Distress subscale of the Parenting Stress Index (PSI) (Abidin, 1990); the standardized Cronbach’s alpha was 0.76 for this survey. On a 5-point Likert scale from 1 (strongly disagree) to 5 (strongly agree), participants responded to statements such as “I often have the feeling that I cannot handle things very well”, “I feel trapped by my responsibilities as a parent”, and “Having a child has caused more problems than I expected in my relationship with my spouse (or male/female friend)”. A total score out of 60 was obtained by summing the responses with higher scores indicating greater parental distress. *Home environment chaos* was evaluated only in parent one (defined as the first parent to sign up for the study, of whom 80.6% were mothers) using the 15-item Confusion, Hubbub, And Order Scale (CHAOS) (Matheny, Washs, Ludwig, & Phillips, 1995), for which the standardized Cronbach’s alpha was 0.89. This scale quantifies chaos as the frustration, disorganization, and noisiness of a home. On a 4-point Likert scale from 1 (very much like your own home) to 4 (not at all like your own home), one parent from each family responded to statements such as “There is very little commotion in our home” and “We can usually find things when we need them”. A total score out of 60 was obtained by summing the responses, where higher scores indicate greater home chaos.

### *Adiposity Measures*

Adiposity was evaluated using the anthropometric measures waist circumference (WC), body mass index (BMI), and waist to height ratio (WHtR) as well as body composition assessment using air displacement plethysmography (BOD POD™,

Cosmed Inc., Concord CA). Height was measured using a wall-mounted stadiometer with participants barefoot or in sock feet. Height was measured at the apex of the inhaling breath to the nearest 0.1 cm. Two measurements were taken; if measures differed by more than 0.5 cm, a third measurement was taken. The final data point was the average of the two closest measures. Weight was measured in kg using the BOD POD digital scale, whose high reliability means that only one measurement was needed. WC was measured at the iliac crest to the nearest 0.1 cm using a Gulick II measuring tape (Country Technology Inc., Gay Mills, WI). In accordance with Statistics Canada and NHANES recommendations, the measurement was taken at the top of the iliac crest over bare skin or over thin clothing during the pause after expiration and before inhalation. Two measurements were taken; if measures differed by more than 0.5 cm, a third measurement was taken. The final data point was the average of the two closest measurements.

BMI was calculated by dividing weight (kg) by height ( $m^2$ ). BMI categories are defined by the World Health Organization (WHO; 1995) as underweight ( $< 18.5 \text{ kg}/m^2$ ), normal weight ( $18.5\text{-}24.9 \text{ kg}/m^2$ ), pre-obese ( $25.0\text{-}29.9 \text{ kg}/m^2$ ), obesity class I ( $30.0\text{-}34.9 \text{ kg}/m^2$ ), obesity class II ( $35.0\text{-}39.9 \text{ kg}/m^2$ ), and obesity class III ( $\geq 40 \text{ kg}/m^2$ ). WHtR was calculated by dividing waist circumference (cm) by height (cm). WHtR is complementary to WC because it is adjusted for the individual's height and improves the accuracy in predicting disease risk (Ashwell et al., 2012; Browning et al., 2010; Man Ying Lee et al., 2008).

Body composition was assessed in 124 participants using a BOD POD, an air displacement plethysmograph. The BOD POD measures body volume, calculates body

density, and uses the Siri equation (Siri, 1961) to convert body density to fat mass. Raw body volume was measured with participants wearing tight-fitting clothing such as a bathing suit and a bathing cap to minimize air trapping, with jewelry and glasses (all that which is not 'self') removed. Participants were instructed to sit quietly, limit movement, and breathe normally while in the test chamber. Thoracic gas volume was either measured (n=92) using a single-use tube connected to the rear of the test chamber, or for those participants unable to complete thoracic gas volume testing, calculated (n=32). The entire BOD POD test takes approximately 15 minutes per participant. By calculating body volume, body fat percentage (%FM) can be approximated. Our reliability testing of %FM measurement showed an intra-individual day-to-day variation of 2.1% and intra-operator variation of 2.6, which align with reference values of 1.7-4.5% and 2.7%, respectively (Fields et al., 2002). The instrument was calibrated twice in the morning of each data collection day; once with the test chamber empty, and once with a 49.980 L calibration cylinder in the chamber.

### *Potential Mediators*

For this analysis, the potential mediators of cortisol, dietary fat intake, PA, and sleep efficiency were considered. Blood cortisol samples were collected in the morning by LifeLabs Medical Laboratory Services in Guelph and analyzed by LifeLabs using the Siemens Advia Centaur cortisol assay, a competitive immunoassay with chemiluminescent detection. Fasting was not a requirement of this test. Collection of cortisol samples in the morning provides a stronger indication of basal diurnal rhythms than other times of day (DeCaro & Worthman, 2011).

Parents' dietary fat intake was quantified using the Block fat screener (Block, Gillespie, Rosenbaum, & Jenson, 2000). The screener listed 16 high-fat food items and the participant responded with their consumption frequency from 1 (once per month or less) to 5 (5 or more times per week). A total score was calculated by summing the responses with a score of 23 or higher indicating high fat consumption (Block et al., 2000). Due to a change in protocol after Phase I of this study, dietary fat intake data were only available for the 52 parents (23 mothers and 29 fathers) in the Phase I study.

PA data were collected by parent self-report using the International Physical Activity Questionnaire Short Form (IPAQ), a validated tool developed to assess health-related PA (Booth, 2000). Participants recorded the duration and intensity of their PA for an average week. The total PA for the average week was calculated by multiplying the active minutes per week by the IPAQ coefficient for that intensity of activity, called a MET score, where METs are multiples of the resting metabolic rate if one MET-minute is equal to kilocalories of expenditure for a 60kg person. Low intensity, moderate intensity, and vigorous intensity PA have MET coefficients of 3.3, 4.0, and 8.0, respectively.

Sleep efficiency (a ratio of the time spent sleeping relative to the time spent in bed) was assessed by dividing the average time spent sleeping per night by the difference between average bedtime and waketime. These questions are part of the Pittsburgh Sleep Quality Index, a validated self-report survey (Buysse et al., 1989).

### 5.3.3 Analytic Technique

Statistical analyses were performed using SAS University Edition Version 3.6 (SAS Institute Inc. 2015). Linear regression coefficient estimates ( $\hat{\beta}$ ) and 95% confidence intervals (CI) were calculated using generalized estimating equations (GEEs). The GEE approach was selected to account for the correlation between cohabitating participants (Liang & Zeger, 1986). Post hoc, models were stratified by gender based on preliminary evidence of differences in adiposity by parent gender. Independently, each body composition measure was regressed onto each of the stress measures. Models were adjusted for age, family size (total number of people living in the home), and annual household income. A p-value  $\leq 0.05$  was considered statistically significant. Mediation was investigated by first exploring the association between family-level stressors and the potential mediators, and then exploring the association between potential mediators and body composition measures.

## 5.4 Results

### 5.4.1 Descriptive Data

The average age of mothers and fathers was 35.7 and 36.8 years, respectively (Table 1). Most participants were white (84.0%), married (86.4%), highly educated (73.6% university education or more), and had relatively high annual household income (47.3% earn \$100,000+). Average family size (number of people living in the home) was 4.6. Using WHO BMI classification guidelines, 40.3% of the sample was normal weight, 31.5% was overweight/pre-obese, and 28.2% was obese (class I, II, or III).

Both mothers and fathers reported moderately high levels of stress on all three measures of family-level stress (Table 1). Parents in this sample were also moderately

overweight according to their BMI (mothers' mean 26.9, fathers' mean 28.9) compared to the WHO recommendations of < 25.0. Mothers' and fathers' mean WC also exceed recommendations of 80 and 94 cm, respectively (WHO, 2008). Mean WHtR for mothers and fathers was above 0.5, which indicates greater health risk (Ashwell et al., 2012; Browning et al., 2010). Mean %FM was 26.3% for fathers and 34.1% for mothers. While no specific guidelines exist for determining which level of body fat increases health risk, it is generally accepted that fat mass of 10-22% for males and 20-32% of body weight for females is within healthy ranges (Esmat, 2016; Kelly, Wilson, & Heymsfield, 2009).

Mean serum cortisol levels for both genders were within the clinically normal range of 170-720 nmol/L suggested by LifeLabs (Table 1). Sleep efficiency means indicate that 90% of fathers' and 88% of mothers' time spent in bed was spent asleep. While no sleep efficiency recommendations exist, scores closer to 100% objectively indicate better quality sleep (Jackowska, Dockray, Hendrickx, & Steptoe, 2011). Dietary fat intake scores, assessed using the Block Rapid Fat Screener, show means of 37.5 for fathers and 39.7 for mothers. This is above the Block Screener's "high fat" category of 23 points. Current PA guidelines recommend that adults should engage in moderate-intensity activity for 30 minutes per day on most if not all days of the week (Bergman, Grijibovski, Hagströmer, Bauman, & Sjöström, 2008). With respect to the IPAQ scoring system, this equates to 600 MET-minutes of moderate activity (5 days x 30 minutes x 4.0 MET-score) or 480 minutes of vigorous activity (3 days x 20 minutes x 8.0 MET-score) (Bergman et al., 2008). On average, both mothers and fathers in this sample achieved these recommendations from a combination of low, moderate, and vigorous intensity activity.

**Table 1** Background characteristics of the analytic sample

Characteristic	Fathers	Mothers	Combined Total
	Mean (SD)		
<b>Body Composition</b>			
BMI (kg/m <sup>2</sup> )	28.85 (5.95) N = 60	26.89 (6.20) N = 64	27.84 (6.14) N = 124
Normal weight (18.5-24.9)	19 (31.67%)	31 (48.44%)	50 (40.32%)
Overweight/pre-obese (25.0-29.9)	22 (36.67%)	17 (26.56%)	39 (31.45%)
Obese (≥30.0)	19 (16.67%)	16 (25.00%)	35 (28.23%)
Waist circumference (cm)	101.68 (15.86) N = 61	90.81 (12.64) N = 63	96.15 (15.27) N = 124
Waist to height ratio	0.57 (0.09) N = 61	0.55 (0.08) N = 63	0.56 (0.08) N = 124
Fat mass (%)	26.50 (9.54) N = 59	34.05 (9.01) N = 64	30.40 (9.98) N = 123
<b>Stress Scores</b>			
General stress	6.66 (1.92) N = 58	6.67 (2.00) N = 63	6.66 (1.95) N = 121
Parenting-specific stress	28.48 (7.61) N = 58	29.19 (8.80) N = 62	28.85 (8.22) N = 120
Home chaos, reported by parent 1	31.7 (7.7) N = 59	32.26 (8.27) N = 61	32.00 (7.99) N = 120
<b>Potential Mediators</b>			
Serum cortisol (nmol/L)	304.56 (102.62) N = 39	313.62 (91.08) N = 47	309.51 (96.01) N = 86
Sleep efficiency (%) <sup>*</sup>	90.19 (10.28) N = 54	87.95 (11.03) N = 61	89.00 (10.69) N = 115
Dietary fat intake	37.52 (5.80) N = 29	39.74 (6.40) N = 23	38.50 (6.12) N = 52
Physical activity (MET-minutes <sup>**</sup> )	2077.43 (1919.25) N = 50	1948.09 (1839.04) N = 61	2006.98 (1868.58) N = 112

<sup>\*</sup>Sleep efficiency is the proportion of the total sleep period spent asleep.

<sup>\*\*</sup>One MET-minute is equal to kilocalories of energy expenditure for an average 60 kg person.

### 5.4.2 Linear Regression of Adiposity on Stress

Associations between measures of stress and body composition for both mothers and fathers are presented in Table 2. Among mothers, significant positive associations were found between home chaos and all four markers of adiposity (BMI  $\hat{\beta} = 0.30$ , 95% CI = 0.11, 0.48; WC  $\hat{\beta} = 0.56$ , 95% CI = 0.22, 0.90; WHtR  $\hat{\beta} = 0.003$ , 95% CI = 0.001, 0.005; %FM  $\hat{\beta} = 0.40$ , 95% CI = 0.18, 0.62). PSI was also significantly positively associated with mothers' WC ( $\hat{\beta} = 0.61$ ,  $p = 0.008$ ) and WHtR ( $\hat{\beta} = 0.004$ ,  $p = 0.006$ ). No significant associations between stress and adiposity were observed in fathers.

**Table 2** Linear regression results of parents' body composition on stress using generalized estimating equations<sup>1, 2</sup>

Body Composition		General Stress $\hat{\beta}$ (95% CI)	Parenting Stress $\hat{\beta}$ (95% CI)	Household Chaos $\hat{\beta}$ (95% CI)
Mothers	BMI	0.42 (-0.27, 1.10) N = 60	0.22 (-0.02, 0.42) N = 59	<b>0.29 (0.11, 0.48)</b> N = 58
	WC	1.15 (-0.15, 2.45) N = 59	<b>0.61 (0.16, 1.07)</b> N = 58	<b>0.56 (0.22, 0.90)</b> N = 57
	WHtR	0.002 (-0.007, 0.011) N = 59	<b>0.004 (0.001, 0.007)</b> N = 58	<b>0.003 (0.001, 0.005)</b> N = 57
	%FM	0.11 (-1.01, 1.24) N = 60	0.26 (-0.05, 0.57) N = 59	<b>0.40 (0.18, 0.62)</b> N = 58
Fathers	BMI	0.45 (-0.38, 1.29) N = 56	-0.01 (-0.22, 0.24) N = 56	0.14 (-0.03, 0.30) N = 56
	WC	0.84 (-1.55, 3.22) N = 56	-0.08 (-0.54, 0.70) N = 56	0.20 (-0.24, 0.63) N = 57
	WHtR	0.006 (-0.007, 0.018) N = 56	0.001 (-0.002, 0.005) N = 56	0.002 (-0.001, 0.005) N = 57
	%FM	0.14 (-1.22, 1.50) N = 56	-0.08 (-0.27, 0.42) N = 56	0.10 (-0.22, 0.41) N = 56

<sup>1</sup> Models were adjusted for age, household income, and family size.

<sup>2</sup> Significant associations are shown in bold.

#### **5.4.3 Potential Mediation of Stress on Adiposity by Cortisol, Sleep Efficiency, Dietary Fat, and PA**

Prerequisite conditions for mediation analyses are that 1) a statistically significant association must exist between the predictor and outcome in the absence of the mediator, and 2) that a mediator is significantly associated with both the predictor and outcome variables (Mackinnon et al., 2002). In this analysis, the first condition was not met among fathers (Table 2). Among mothers, this first condition was met for two predictor variables; parent-specific stress and household chaos. However, while mothers' sleep efficiency was significantly associated with PSI ( $\hat{\beta} = 0.003$ , 95% CI = 0.0001, 0.006), it was not significantly associated with any of the body composition measures. Likewise, dietary fat intake was significantly associated with household chaos ( $\hat{\beta} = 0.59$ , 95% CI = 0.27, 0.91), but not with parenting-specific stress or any of the body composition measures. Serum cortisol was not significantly associated with any of the predictor or outcome measures among mothers. Thus, these results do not suggest any potential mediators.

#### **5.5 Discussion**

The objective of this study was to examine associations between various measures of family-level stress and adiposity among a sample of Canadian mothers and fathers of preschool-aged children. We hypothesized that family-based stress would be positively associated with adiposity in our sample. Our results reveal that this

was the case for mothers, but not fathers. This research suggests that parenting-specific and home environment stresses may be important determinants of maternal adiposity. Despite the gender-based difference in associations between stress and adiposity, both mothers and fathers reported relatively high levels of stress. The high level of stress reported by these parents underscores the importance of examining the influence of stress among parents of young children.

The differential influence of stress on adiposity by gender found in this study is supported by previous research which has found that associations between stress and weight status are more consistently demonstrated in women compared to men (Block et al., 2009; Overgaard, Gyntelberg, & Heitman, 2004). Chen and Qian (2012) investigated associations between stress and BMI in a sample of over 112,000 Canadian adults and found that highly stressed women had greater obesity compared to women with lower stress levels, whereas obesity prevalence among men was similar across all stress levels. Results from a prospective cohort of men and women suggest that women may respond to stress differently than men with more women reporting stress-related eating (Laitinen, Ek, & Sovio, 2002), which may help explain gender differences regarding the association between stress and adiposity. In the present study, household chaos was significantly associated with higher intake of dietary fat among women, which may suggest that the women in our study did respond to higher levels of stress through increased dietary intake. However, dietary fat intake was not associated with adiposity in our sample. While there has been research associating stress-driven eating, including foods high in fat, with weight outcomes in women (Laitinen et al., 2002), future prospective research that uses a more comprehensive dietary assessment than used in

the present study is needed to determine specific mechanisms by which stress leads to obesity risk among parents of young children.

Socialized gender norms and expectations may also explain why our results differ among mothers and fathers. Although fathers' involvement in active caregiving has been on the rise in recent history, in current Canadian society, mothers are still considered the primary caregivers to young children (Wall & Arnold, 2007). Thus, although mothers and fathers in our sample report similar levels of family-level stress, it is possible that mothers are more likely than fathers to perceive that it is their role or responsibility to address or reduce family-level stress, thus leading to more distress as a result of the perceived stress. Future research is needed to examine how perceptions of family stress may differ among mothers and fathers and how this perception may influence behaviour and health outcomes.

This study examined several putative bio-behavioural mediators of the association between stress and adiposity, none of which were found to be significant. Similar to findings from Jones et al. (2017) and Stone et al. (1998), PA remained consistent across stress levels in our sample. Sleep efficiency was positively associated with PSI, but not associated with adiposity. It is possible that mothers with greater parenting stress felt more exhausted, thus contributing to better sleep quality. The lack of association between perceived stress and cortisol levels is consistent with other work demonstrating that the physiological response does not always parallel the subjective stress response (Buske-Kirschbaum et al., 2003; Kudielka & Kirschbaum, 2005; Zimmer, Basler, Vedder, & Lautenbacher, 2003). However, our inability to detect a mediation effect may reflect the measures used to assess these mediators. Blood

cortisol measures are common, but may differ from other acute measures such as saliva (Hellhammer, Wu, & Kudielka, 2009; Konishi & O'Connor, 2016), and may not reflect the response to chronic stress in the way that hair cortisol measures would (Gidlow, Randall, Gillman, Silk, & Jones, 2016; Incollingo et al., 2015; Larsen, Fahrenkrug, Olsen, & Heitmann, 2016; Stalder et al., 2012). Our assessment of dietary intake was only measured in approximately half of our sample due to study design changes and, as mentioned previously, measured only one aspect of dietary intake; a more comprehensive dietary analysis is needed to clearly quantify diet-stress-adiposity associations. Likewise, the measure of PA and sleep used here is based on self-report and not objective measures of movement, such as wearable accelerometry devices. Additional research with more comprehensive assessments of potential mediators is needed to elucidate how higher stress may lead to increased adiposity among parents.

A further limitation of this study is the use of a single-item assessment of general stress. While significant stress-adiposity associations have been found in past research with a similar one-question survey (Sampasa-Kanyinga & Chaput, 2017), the use of a more comprehensive measure of general stress likely would have provided a more accurate reflection of general stress in this population. Level of household chaos was only reported by the first parent to sign up for the study (80.6% mothers). While it is possible that fathers may have perceived household chaos differently than mothers, the fact that fathers and mothers reported similar levels of general and parent-specific stress suggests that overall stress levels were similar between mothers and fathers. In addition, this sample contains a large proportion of white individuals of high socioeconomic status, which may reduce the generalizability of our findings.

Despite these limitations, this study extends previous findings reported in the literature. The inclusion of both mothers and fathers addresses a gap in the existing literature (Hayes & Watson, 2013). Another strength is the consideration of both biological and behavioural factors as mechanisms that may facilitate the stress-adiposity relationship. Additionally, the complementation of BMI with more comprehensive measures, such as WHtR and %FM from air displacement plethysmograph, is a strength of this study. While BMI is extensively used as a quick and easy proxy for adiposity, a shortcoming is that it indicates excess weight without specifying excess fat; had only proxy measures of adiposity been used here, significant associations would have been missed.

Overall, our study found that family stress may influence adiposity differently in mothers and fathers. Mothers' adiposity seems to be uniquely affected by parent-specific and home environment stresses. Despite reporting similar levels of stress as mothers, fathers' adiposity does not seem to be influenced by family stress. Future work should investigate the mechanisms by which stress leads to obesity including differences by gender. Additional work is also needed to better understand the effectiveness of stress-reduction strategies both for reducing parental distress and for adiposity reduction.

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## 6.0 Summary and Conclusions

The need for research uncovering the contributing factors to obesity has never been more prevalent than in today's climate; an estimated 20% of Canadians (5.1 million) are overweight or obese (Statistics Canada, 2014), which places these individuals at increased risk of many chronic illnesses and strains Canada's healthcare system (Public Health Agency of Canada, 2011). The role of stress in obesity has not been well-researched in the general population (Chen & Qian, 2012) and even less so in Canadians. The ways in which stress may facilitate obesity through biological and behavioural pathways has also received little research attention (Chen & Qian, 2012; Holmes et al., 2009). The demands associated with the role of caring for a young child may place additional strain on Canadian parents to increase their risk of obesity, which has not before been researched.

To address these gaps, the purpose of this study was to examine the relationship between family-level stress and adiposity in parents of preschool-aged children. This study investigated three measures of family stress: general stress, parenting-specific stress, and home environment chaos. This research extends previous findings by including fathers' perspectives (Hayes & Watson, 2013), as well as using multiple measures of stress and body composition. It also examined the potential mediation of the stress-adiposity association physiologically by the hormone cortisol and behaviourally by dietary fat intake, sleep efficiency, and PA in hopes of creating a comprehensive picture of the stress-adiposity relationship. Consistent with our hypotheses, maternal stress was positively associated with indices of adiposity; greater parenting-specific stress was associated with greater WC and WHtR, and greater home

chaos was associated with greater BMI, WC, WHtR, and %FM. However, despite reporting very similar levels of family stress and contrary to our hypotheses, none of the fathers' stress measures were associated with their body composition. Furthermore, the associations between maternal stress and adiposity were not mediated by our measures of cortisol, dietary fat intake, sleep efficiency, or PA.

It is evident that gender differences exist in the stress-adiposity relationship, however, some social factors may also be involved. While fathers' involvement in active caregiving for children has been on the rise in recent history and is considered important for children's healthy development and wellbeing (Ball, 2010; Yeung, Sandberg, Davis-Kean, & Hofferth, 2001), mothers are still considered the primary caregivers to young children in Canadian society (Wall & Arnold, 2007). In anticipation of the continued evolution of gender norms surrounding caregiving responsibilities and for the inclusion of non-nuclear families, it would perhaps be of importance to use self-report assessments of engagement and roles within the family rather than assumptions about domestic labour and childcare responsibilities. In future research, measuring degree and nature of involvement in parenting could help to ascertain which stress-adiposity differences exist because of gender-based differences and which are associated with specific elements of parenting.

While each of parenting-specific stress and home environment was associated with adiposity in mothers in our study, general stress failed to reveal any significant associations with adiposity in either mothers or fathers. Rather than indicating a true lack of association, it may be that this measure inadequately captures "general life stress" despite some significant stress-adiposity associations found in past research

with a similar one-question survey (Sampasa-Kanyinga & Chaput, 2017). Significant associations were found most consistently with home chaos, yet, the survey was completed only by Parent 1 (of whom, 80.6% were female). It was assumed that Parent 1 would be best able to provide an accurate picture of the home environment, but it is possible that perceptions of home environment stress would differ between parents in dual-parent homes.

The present study builds on past work with the GFHS population by MacRae and colleagues (2017). MacRae et al. were the first to investigate the association between home chaos and parents' dietary fat intake; this analysis investigated if this association mediated the relationship between stress and adiposity. However, dietary fat intake was not associated with markers of adiposity, which precludes mediation analyses. Overall, both mothers and fathers reported relatively high levels of fat consumption. Consistent with MacRae et al.'s findings, mothers consumed greater dietary fat at higher levels of home chaos (2017) which is suggestive of dietary coping mechanisms (Fanelli Kuczumski et al., 2017; Frankenhaeuser, 1996; Zellner et al., 2006).

An unexpected finding was that maternal sleep quality appeared to increase with greater levels of stress. Past literature has found stress to impair sleep quality (Bastien, Vallieres, & Charles, 2004; Bernert, Merrill, Braithwaite, Van Orden, & Joiner, 2007; Jansson & Linton, 2006; Knudsen, Ducharme, & Roman, 2007; Sampasa-Kanyinga & Chaput, 2017), with some reports that nearly half of all women report lying awake at night due to stress (Frankenhaeuser, 1996). These contrasting findings suggest that feelings of parenting-specific stress contribute to faster falling asleep with fewer sleep interruptions, perhaps due to the mothers' feelings of exhaustion. The lack of

association with our adiposity measures perhaps indicates an issue with the measures used and suggests a need for objective rather than self-report measures.

It was hypothesized that stress would inhibit motivation to engage in PA, however, this was not found in this study. While stress has been shown to deter PA efforts (Stults-Kolehmainen & Sinha, 2014), other literature shows that those with good PA habits are more likely to engage in PA as a stress coping strategy (Frankenhaeuser, 1996). Perhaps the divergent responses blurred the association in this sample, which suggests that more research into the inhibiting and enabling factors of engaging in PA in stressed families is needed.

Non-significant associations between perceived stress and cortisol levels are consistent with other work demonstrating that the physiological response does not always parallel the subjective stress response (Buske-Kirschbaum et al., 2003; Kudielka & Kirschbaum, 2005; Zimmer et al., 2003). However, future research should explore how cortisol measures that assess chronic stress, such as hair cortisol, are associated with adiposity levels among parents.

None of the mediators explored in these analyses were shown to be significant. This is perhaps due to shortcomings in the measure, rather than a lack of association altogether. Of note is that all behavioural mediation pathways explored here were self-reported measures; a more comprehensive dietary measure would capture a broader spectrum of dietary habits, and accelerometer measures of PA and sleep patterns would provide real-time evaluation of lifestyle habits that are not prone to subjective bias. Small sample size may have limited the analysis of cortisol and dietary fat intake as these measures were only available for a subset of the analytic sample.

The CHAOS survey was used to evaluate confusion, disorganization, and lack of routine and order within the home. While there is support for elements of home chaos to be more representative of vibrancy than stress within the home dynamic (Dumas et al., 2005), home chaos should be considered a primary target for family stress reduction intervention due to its association with all markers of adiposity examined here. More focused measures of specific sources of family stress could be helpful in elucidating from where parental stress arises. For example, the Dinnertime Scale of the Family Routines Questionnaire developed by Fiese and colleagues (2016) could better ascertain where chaos is found in the home and where supports in maintaining routines would be useful. High levels of parenting stress are negatively associated with positive parenting practices, such as encouraging children's PA and monitoring screen time (Walton et al., 2015). By reducing these stress levels, family functioning may increase, and children's health may also be indirectly improved. Future research should focus on developing tools to assist parents in managing their stress to help families achieve higher goals across a broad spectrum of health factors.

In conclusion, the present study provides novel insight into family-level stress as a precipitating factor in maternal adiposity. While these associations were not found in fathers, their inclusion addresses a gap in the literature. Future research should address the mechanisms by which these associations occur, as our measures of dietary fat intake, sleep efficiency, PA, or cortisol were not able to explain this relationship. Future research should also focus on methods to reduce parents' feelings of stress because the levels reported here were greater than those of other populations.

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## 8.0 Appendices

### Appendix A - Guelph Family Health Study – Longitudinal Study Consent to Participate – Parent #1

The purpose of this form is to provide you with the information you need to make an informed decision for you and your family about participating in this research study. Participation in this study is voluntary.

#### Part 1: Understanding the Study

##### About the study

The Guelph Family Health Study is a unique research study that is following a large group of families with young children in Guelph over many years.

The study and its related costs are funded through the Better Planet Project at the University of Guelph.

##### Definitions

**Parents:** We define parents as the main caregivers of children. Parents can be biological, related or adoptive.

**Family:** For this study we define family as parents and their children who are 18 months to five years of age. Families can have one or many parents. A maximum of two parents from each family can register in this study.

##### What's required?

After you and your family complete your registration in this study, you will be asked to:

1. Complete online questionnaires Estimated time: 40 minutes per questionnaire

This study requires you to answer questions that will help us understand your family's health behaviours. Families with one parent who completes the online questionnaires will receive a \$50 grocery gift card and families with two parents who complete the online questionnaires will receive a \$75 grocery gift card as a thank you. For more information, visit what's required.

2. Meet with a member of our study team at your home Estimated time: 1 hour

The Study Coordinator will meet you at a convenient time to provide instructions on how to complete the rest of the study.

3. Track food and activity for a few days Estimated time: 15 minutes per day

At your home visit we will teach you how to keep a three-day record of the food eaten by your children. We will also ask your children to wear an activity monitor on their waist and/or their wrist for seven days. For more information, visit [what's required](#).

4. Come to the University of Guelph for a family health assessment Estimated time: 1.5 hours + travel time

Your family will be asked to visit the University of Guelph for a health assessment to:

Measure your height and weight using a scale and height board, similar to the ones at your doctor's office.

Measure waist using a tape measure.

Measure body fat and muscle.

**For adults**, this will be done using a machine called a BOD POD™. For more information, visit [How do you measure body fat and muscle?](#)

**For children**, this will be done using a machine called a Bioelectric Impedance Analyzer (BIA). BIA uses small patches that produce an electrical signal. For more information, visit [How do you measure body fat and muscle?](#)

Measure blood pressure using a cuff that wraps around your arm, similar to the one at your doctor's office.

Collect a saliva sample. For more information, visit [How do you collect saliva?](#)

You will receive a \$75 grocery gift card if one parent attends the health assessment or a \$100 grocery gift card if two parents attend the health assessment.

5. Provide a blood sample at a local laboratory Estimated time: 15 minutes per family member + travel time

You can choose for your family to provide blood samples at a local medical laboratory within three weeks of your health assessment. Giving a blood sample is optional and is not a requirement of the study. You and your family can still participate in the study even if you choose not to provide blood samples. You will receive a \$25 grocery gift card as a thank you for completing the laboratory visit. For more information, visit [giving blood](#).

6. Provide a fecal sample Estimated time: 5 minutes

You can choose for your family to provide fecal samples for the study. Giving a fecal sample is optional and is not a requirement of the study. You and your family can still participate in the study even if you choose not to provide fecal samples.

7. Participate in follow up assessments

Your family will be invited to complete a combination of questionnaires, home visits, food and activity tracking, health assessments, blood and fecal samples every six or 12 months for up to 20 years. Your follow up assessments will help us learn how human behaviours affect health over time.

### **What benefits are associated with this study?**

If you choose to participate in this study, you will be part of an important project that is helping us better understand the human behaviours that affect health. We will use this information to develop programs that reduce the risk for disease in families.

### **What risks are associated with this study?**

#### **Measuring body fat and muscle:**

**For parents:** You may experience claustrophobia while sitting in the BOD POD™. You can exit the BOD POD™ at any time. You may feel slightly embarrassed about wearing a bathing suit in the BOD POD™. The BOD POD™ is located in a private room. For more information, visit [How do you measure body fat and muscle?](#)

**For children:** There is a very small risk that your child's skin may be sensitive to the glue we use to apply the patches. Your child can ask us to remove the patches at any time, if they are uncomfortable.

**Giving blood:** You may experience the usual pain and bruising that people get when they give blood. You may also experience dizziness. Rarely, giving blood can cause an unusually small vein to collapse. The laboratory staff who will collect your blood have extensive training and experience taking blood in adults and young children.

**Fecal sample:** You may come in physical contact with the fecal sample, but this is a small risk. Full instructions for fecal donation collection, including an explicit statement suggesting hand-washing after collection, will be provided.

**Privacy:** The privacy of your information is very important to us. Any time you allow someone to access your information, there is a small risk to your privacy from human error or technical error. We have taken many precautions to ensure that the information and samples you provide us will remain safe and private, and that your identity will be protected to the extent required by law. For more information, visit [privacy](#).

### **Who is conducting this study?**

This study is being conducted by a team of researchers at the University of Guelph.

Lead Researchers

David Ma, Associate Professor, Human Health & Nutritional Sciences

Jess Haines, Assistant Professor, Family Relations & Applied Nutrition  
Research Team

Emma Allen Vercoe, Associate Professor, Molecular and Cellular Biology

Paula Brauer, Associate Professor, Family Relations & Applied Nutrition

Andrea Buchholz, Associate Professor, Family Relations & Applied Nutrition

Gerarda Darlington, Professor, Mathematics and Statistics

Alison Duncan, Professor, Human Health & Nutritional Sciences

David Mutch, Associate Professor, Human Health & Nutritional Sciences

Lawrence Spriet, Professor and Chair, Human Health & Nutritional Sciences

Study Coordinator

Angela Annis, Human Health & Nutritional Sciences and Family Relations & Applied  
Nutrition

### **Contact us**

You can contact the Study Coordinator for the Guelph Family Health Study by email at [coordinator@guelphfamilyhealthstudy.com](mailto:coordinator@guelphfamilyhealthstudy.com) or by telephone at 519-824-4120 ext. 56168.

This study has been approved by the University of Guelph Research Ethics Board. If you have questions about your rights as a research participant, please contact this group by email at [reb@uoguelph.ca](mailto:reb@uoguelph.ca) or by telephone at 519-824-4120 ext. 56606.

### **Part 2: Agreeing to the study**

Click on the box next to each statement that you agree with. When you are done, click 'Submit' at the bottom of the page.

By completing this consent form, I declare that:

I understand the Guelph Family Health Study – Longitudinal Study, what is required of my family if we participate, and the benefits and risks. I have had the opportunity to ask questions about the study and have received adequate answers. **I am making an informed decision for myself and on behalf of my children to participate in this study.**

Please provide us with the names of your children:

Child #1 \_\_\_\_\_ Child #2 \_\_\_\_\_ Child #3 \_\_\_\_\_

I understand that participation in this study is voluntary. I know that I can refuse to participate, refuse to answer questions, or withdraw myself or my children from the study at any time with no effect on our future healthcare or relationship with the University of Guelph. I understand that any information or samples I do not ask to be destroyed will remain with the study for future research.

I understand that the study team may withdraw my family from this research at their discretion.

I understand that there may be no clinical benefit to my family by participating in this study.

I understand that the results of my body fat and muscle analysis will be provided to me, if requested. I understand that the results my children's body fat and muscle analysis will not be provided to me since we do not yet have standards for level of fat and muscle for children.

I understand that our saliva samples will be used to extract DNA and study how the genes we were born with affect our behaviours and responses to food. I understand that the results of our saliva tests will not be provided to me. This is to protect my family from having to potentially provide genetic information to a third party, such as an insurance provider or employer.

I accept that if we provide blood samples, they will be tested for sugars, fats and hormones. I understand that some of my blood tests results will be provided to me if I request them. I understand that my children's blood test results will not be provided to me because we don't yet have standards for healthy blood ranges in children. I accept that if any of my blood tests are significantly abnormal, they will be reported to me for discussion with my doctor.

I understand that if we provide fecal samples, they will be tested for gut bacteria. The results of the fecal tests will not be provided to me, unless they are significantly abnormal results. Any of the material which is used in research may result in new products, tests or discoveries. In some instances, these may have potential commercial value and may be developed and owned by the researchers, University of Guelph and/or others. However, I understand that I do not retain any property rights to the materials. Therefore, I cannot share in any financial benefits from these products, tests or discoveries.

I understand that our tests will not be provided by a medical doctor and cannot be used to diagnose a disease or condition.

I accept that the results of my family's tests may be used in publications and at conferences for the purpose of learning, only after any information that can identify us has been removed.

I understand that since I could be receiving over \$500 worth of incentives for participating in this study, I may need to provide my Social Insurance Number (SIN). I understand my SIN will be kept confidential and will only be shared with the University of Guelph financial office.

I understand that my family will have the opportunity to participate in the Guelph Family Health Study – Intervention Program. This study program will provide 6-months of support to my family for healthy lifestyle choices. I will discuss this with the Study Coordinator during our home visit and I will be able to choose to participate at that time.

I understand that I will have the option to have my family's blood, fecal and saliva samples stored in the Guelph Family Health Study – BioBank. The samples will be used for future research to help better understand the human behaviours that affect health. I will discuss this with the Study Coordinator during our home visit and I will be able to choose to participate at that time.

Parent #1 Name \_\_\_\_\_

Parent #1 Signature \_\_\_\_\_ Date \_\_\_\_\_

Study Coordinator Signature \_\_\_\_\_ Date \_\_\_\_\_

## **APPENDIX B – Parental-Perceived Stress**

Source: Public Health Management Corporation Community Health Data Base's (2015)  
Southeastern Pennsylvania Household Health Survey

Using a scale from 1 to 10, where 1 means 'no stress' and 10 means 'an extreme amount of stress,' how much stress would you say you have experienced in the last year?

1  2  3  4  5  6  7  8  9  10

## APPENDIX C – Parenting Stress Index

Source: Parenting Stress Index; copyright 1990 and 1995 Psychological Assessment Resources, Inc

Please indicate how much you agree or disagree with each of these statements using the following options:

- a. Strongly disagree (1)
  - b. Disagree (2)
  - c. Neither Disagree nor Agree (3)
  - d. Agree (4)
  - e. Strongly agree (5)
- 
- 1) I often have the feeling that I cannot handle things very well
    - a. Strongly disagree
    - b. Disagree
    - c. Neither disagree nor agree
    - d. Agree
    - e. Strongly agree
  - 2) I find myself giving up more of my life to meet my children's needs than I ever expected
    - a. Strongly disagree
    - b. Disagree
    - c. Neither disagree nor agree
    - d. Agree
    - e. Strongly agree
  - 3) I feel trapped by my responsibilities as a parent.
    - a. Strongly disagree
    - b. Disagree
    - c. Neither disagree nor agree
    - d. Agree
    - e. Strongly agree
  - 4) Since becoming a parent, I have been unable to do new and different things.
    - a. Strongly disagree
    - b. Disagree
    - c. Neither disagree nor agree
    - d. Agree
    - e. Strongly agree

- 5) Since becoming a parent, I feel that I am almost never able to do things that I like to do.
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 6) I am unhappy with the last purchase of clothing I made for myself.
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 7) There are quite a few things that bother me about my life.
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 8) Having a child has caused more problems than I expected in my relationship with my spouse (or male/female friend)
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 9) I feel alone without friends.
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 10) When I go to a party, I usually expect not to enjoy myself.
  - a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 11) I am not as interested in people as I used to be.
  - a. Strongly disagree

- b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree
- 12) I don't enjoy things as I used to.
- a. Strongly disagree
  - b. Disagree
  - c. Neither disagree nor agree
  - d. Agree
  - e. Strongly agree

## APPENDIX D - Confusion, Hubbub, and Order Scale (CHAOS)

Source: Confusion, Hubbub, and Order Scale (CHAOS) (Matheny et al., 1995).

Available from:

[http://www.performwell.org/index.php?option=com\\_mtree&task=att\\_download&link\\_id=483&cf\\_id=24](http://www.performwell.org/index.php?option=com_mtree&task=att_download&link_id=483&cf_id=24)

Please indicate how much you agree or disagree with each of the statements below using the following options:

1 = Very much like your own home environment

2 = Somewhat like your own home environment

3 = A little bit like your own home environment

4 = Not at all like your own home environment

1) There is very little commotion in our home.

1  2  3  4

2) We can usually find things when we need them.

1  2  3  4

3) We almost always seem to be rushed.

1  2  3  4

4) We are usually able to stay on top of things.

1  2  3  4

5) No matter how hard we try, we always seem to be running late.

1  2  3  4

6) It's a real zoo in our home.

1  2  3  4

7) At home we can talk to each other without being interrupted.

1  2  3  4

8) There is often a fuss going on at our home.

1  2  3  4

9) No matter what our family plans, it usually doesn't seem to work out.

1  2  3  4

10) You can't hear yourself think in our home.

1  2  3  4

11) I often get drawn into other people's arguments at home.

1  2  3  4

12) Our home is a good place to relax.

1  2  3  4

13) The telephone takes up a lot of our time at home.

1  2  3  4

14) The atmosphere in our home is calm.

1  2  3  4

15) First thing in the day, we have a regular routine at home.

1  2  3  4

## APPENDIX E - Standard Operating Procedure for Height Measurement in Adults



Photography by Scott Schau, reproduced with permission

### Measuring Height Using a Stadiometer

1. The participant should be barefoot or in sock feet, and be wearing minimal clothing to facilitate the correct position during measurement. Any hair ornaments (barrettes, etc.) should be removed.
2. The participant should stand with heels together (the toes can be pointed outward slightly), arms to the side, legs straight, shoulders relaxed, head in Frankfort horizontal plane (“look straight ahead”)
3. There should be four points of contact between the participant and stadiometer/wall: heels, buttocks, scapulae, back of head.
4. Just prior to measurement, instruct the participant to inhale deeply & hold their breath with shoulders up, back and down to maintain an erect posture (“stand up tall”), while the headboard is lowered on the highest point of the head with enough pressure to compress the hair.
5. Measure to the nearest 0.1 cm. Read the measurement at the red line in the center of the ruler. Participant can exhale.
6. Reposition the participant and repeat steps 2 to 5 for a second measurement.
  - If the two values are within 0.5 cm, take the mean.

- If the two values diverge by more than 0.5 cm, take a third measurement, and GFHS will use the mean of the two closest values.

## References

1. Frisancho AR. Anthropometric Standards for the Assessment of Growth and Nutritional Status. 2004. The University of Michigan Press, Ann Arbor. Chp 2.

## **APPENDIX F - Standard Operating Procedure for Weight Measurement**

To measure weight using the BODPOD:

1. Instruct the participant to remove his/her footwear and outer garments (e.g. jackets, hats, heavy sweaters, etc).
2. Turn on POD (green button on white box and computer).
3. Login to the BODPOD software
4. Be sure the scale has been calibrated in the last 2 weeks.
5. Click on PRACTISE>MASS. The scale will tare and then prompt you to ask participant to stand on the scale for measurement.

If a child participant is too young or too nervous to stand on the scale, measure the mass of one parent holding the child and then measure the parent alone. The child's mass can be calculated from these two measurements.

Weight only needs to be measured once as reliability testing has been done and is very high.

If the BODPOD scale is unavailable, use the Seca scale in the back room to take a manual weight measurement.

## APPENDIX G - Standard Operating Procedure for Waist Circumference Measurement



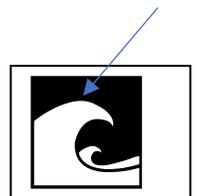
Requires **TWO** technicians:

- One to do the landmarking and measurement
- The other to ensure measuring tape is parallel with the floor, and to record the values



Measuring waist circumference at the top of the iliac crest:

1. Participant should clear abdomen of all clothing.
2. Participant should stand with feet shoulder-width apart and arms crossed over chest.
3. On the **RIGHT** side of the participant, on **BENDED KNEE**, palpate the **TOP** of the right iliac crest (think of the crest of a wave). You are aiming for the uppermost lateral border of the right hipbone.
4. Draw a horizontal line at this landmark, using eyeliner.
5. Wrap a Gulick II measuring tape around the abdomen. The bottom of the measuring tape should be level with the line. It should be **horizontal** all the way around.



6. Apply tension to the tape to make sure the tape is snug, but not so tight as to compress tissue. One of the two red balls of the spring mechanism should be visible. See “How to take a measurement” in the Gulick II Instruction Manual.
7. At the end of normal expiration (**after expiration, before inspiration**), take the measurement to the nearest 0.1 cm.
8. Repeat steps 5 to 7 for a second measurement.
  - If the two values are within 0.5 cm, take the mean.
  - If the two values diverge by more than 0.5 cm, take a third measurement, and GFHS will use the mean of the two closest values.

## References

2. Canadian Society for Exercise Physiology  
<http://www.csep.ca/english/view.asp?x=724&id=84>
3. Patry-Parisien J, Sheilds M, Bryan S. Comparison of waist circumference using the World Health Organization and National Institutes of Health protocols. Sept 2012. Statistics Canada, Catalogue no. 82-003-XPE. Health Reports, 23(3): 1-10. *This is the CHMS method.*

## **APPENDIX H - Standard Operating Procedure for BOD POD Body Composition Measurement**

### **BOD POD™ Warm-up Protocol for Guelph Family Health Study**

1. Turn on the BOD POD™ upon arrival to the lab
2. **Wait 30 minutes after turning on BOD POD before running QC**
3. Run an ANALYZE HARDWARE test from the QC menu
  - a. If anything fails, call the BOD POD™ technicians (techsupport@bodpod.com)
4. Run a CHECK SCALE test from the QC menu
  - a. Calibrate the scale if it has been more than two weeks since last calibration. If in doubt as to when calibration was last done, proceed to calibration.
5. Run one AUTO RUN tests from the QC menu
  - a. Passing Standard deviations are lower than 60
6. Run a VOLUME CHECK test from the QC menu

The BOD POD™ is ready for a body composition test.

### **BOD POD™ Body Composition Test Protocol for Guelph Family Health Study**

1. Begin a new body composition test and enter the information for the participant. Participant ID is entered for the first name (PXXXX) and GFHS for the last name
2. Confirm birthdate (dd/mm/yyyy) with data sheet and participant and enter into the computer
3. Enter gender for the participant
4. Enter average height from two measurements following GFHS height measurement SOP, including units (cm).
5. Select the appropriate ethnicity classification (general population vs. African American)

**\*NOTE:** If prompted, choose an existing participant from within the BODPOD software – this is only applicable for follow-up visits

6. Continue to the next screen
7. Select the Siri equation to use as the density model and measured thoracic gas volume
8. Continue to the next screen and follow the prompts to begin the body composition test
9. Install breathing tube and perform calibration, as prompted by the BODPOD software. Ensure participant is wearing cap and has removed glasses, jewellery.

Check to see if participant has anything to eat or drink or exercised in the past 2 hours.

10. Weigh participant on BOD POD scale following on screen prompts
11. BOD POD is now ready for volume measurements
12. Two volume measurements will be taken (a third measurement may be needed if these two measurements are significantly different – the computer will instruct you if a third measurement is needed)
13. Instruct participant to sit still and breath normally for the duration of the 30 second measurement. Inform the participant that they may hear clicking or feel pressure but it is all a normal part of the test. Instruct the participant about the emergency stop button behind their left knee.

### **BOD POD Thoracic Gas Volume Measurement Protocol for Guelph Family Health Study**

**\*NOTE:** Participants can wear glasses for this portion of the test if needed.

1. We will now begin the lung volume measurement part of the test. This measurement is used to determine how much air is within your lungs.
2. The screen will instruct you when to breathe by moving bars up and down. The bars read “breath in” and “breath out”. The test will begin with a short calibration period where you will hold the tube in your hand and breath normally before putting the tube in your mouth. Then the screen will prompt you to “prepare to put the tube in your mouth and plug your nose”, and then will say “put the tube in your mouth and plug your nose”. You will then breathe in and out normally through the tube with your lips sealed all the way around the tube.
3. Near the end of the breathing test, the screen will prompt you to “prepare to huff” and then “huff, huff, huff”. When you are instructed to “huff, huff, huff” pant into the tube as if you were fogging your glasses or gently blowing a feather off the palm of your hand. Do not try to blow hard while doing this, it is a gentle huff.
4. There will be clicking heard during the test but this is a normal part of the test. You may feel resistance when breathing into the tube, but do not try to blow harder to overcome this resistance, just continue to breathe normally.

### **Completing the Test**

1. Upon completion of the volume measurements, ask the participant to exit the BOD POD.
2. Enter participant’s activity level as “Active” on BOD POD software.
3. Follow the prompts on screen to print the data sheet in kg/cm.
4. Attach BOD POD results to data collection form.

## Note to Operator

Please note the following instructions:

- Operator will leave the room and participants can get changed. Be sure to offer the participant a hospital gown to wear on top of their bathing suit.
- Instruct the participant not to take off the hospital gown until just before stepping into the BOD POD™. This will minimize the participant's exposure while wearing a bathing suit.
- *Above all, remember that research participants are volunteers. Treat them respectfully, in the same manner as you would want to be treated if you were in their shoes.*

The entire BOD POD™ test takes approximately 15 minutes. Of these 15 minutes, participants are in the chamber for perhaps a total of 5 minutes. There is additional calibration time (approx. 5 to 10 minutes), but for this the participant is standing outside the chamber.

## APPENDIX I – Block Fat Screener

Think about your eating habits over the past year or so. About how often do you eat each of the following foods? Remember breakfast, lunch, dinner, snacks and eating out. Mark one box for each food.

1. Beef or pork, such as steaks, roasts, ribs, or in sandwiches

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

2. Fried chicken

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

3. Hot dogs, or Polish or Italian sausage

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

4. Cold cuts, lunch meats, ham (not low-fat)

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

5. Bacon or breakfast sausage

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

6. Salad dressings (not low-fat)

- 1/MONTH or less
- 2-3 times a MONTH

- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

7. Margarine, butter or mayo on bread or potatoes

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

8. Margarine, butter or oil in cooking

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

9. Eggs (not Egg Beaters or just egg whites)

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

10. Pizza

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

11. Cheese, cheese spread (not low-fat)

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

12. Whole milk

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

13. French fries, fried potatoes

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

14. Corn chips, potato chips, popcorn, crackers

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

15. Doughnuts, pastries, cake, cookies (not low-fat)

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

16. Ice cream (not sherbet or non-fat)

- 1/MONTH or less
- 2-3 times a MONTH
- 1-2 times a WEEK
- 3-4 times a WEEK
- 5+ times per WEEK

## APPENDIX J – International Physical Activity Questionnaire (Short-Form)

Source: The International Physical Activity Questionnaire (IPAQ). Available from: [https://sites.google.com/site/theipaq/questionnaire\\_links](https://sites.google.com/site/theipaq/questionnaire_links)

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The following questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

\*Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

- 1) During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?
  - a. \_\_\_\_ days per week
- 2) Skip if Q\* = 0 How much time did you usually spend doing vigorous physical activities on one of those days?
  - a. \_\_\_\_\_ hours per day
  - b. \_\_\_\_\_ minutes per day
  - c. Don't know

\*\*Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

- 3) During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.
  - a. \_\_\_\_ days per week
- 4) Skip if Q#\*\* = 0 How much time did you usually spend doing moderate physical activities on one of those days?
  - a. \_\_\_\_\_ hours per day
  - b. \_\_\_\_\_ minutes per day
  - c. Don't know

\*\*\*Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you have done solely for recreation, sport, exercise, or leisure.

- 5) During the last 7 days, on how many days did you walk for at least 10 minutes at a time? \_\_\_\_\_ days per week
- 6) Skip if Q#\*\*\* = 0 How much time did you usually spend walking on one of those days?
  - a. \_\_\_\_\_ hours per day
  - b. \_\_\_\_\_ minutes per day
  - c. Don't know

This question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

- 7) During the last week, how much time did you spend sitting on a week day?
  - a. \_\_\_\_\_ hours per day
  - b. \_\_\_\_\_ minutes per day
  - c. Don't know
- 8) During the last week, how much time did you spend sitting on a weekend day?
  - a. \_\_\_\_\_ hours per day
  - b. \_\_\_\_\_ minutes per day
  - c. Don't know
- 9) Is there a children's playground within 10-minutes walking distance of your house?
  - a. Yes
  - b. No

## APPENDIX K – Pittsburgh Sleep Quality Index

Source: The Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989) Available from: <http://www.sleep.pitt.edu/includes/showFile.asp?fltype=doc&flID=2532>

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

- 1) During the past month, what time have you usually gone to bed at night?
- 2) During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
- 3) During the past month, what time have you usually gotten up in the morning?
- 4) During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)
- 5) During the past month, how would you rate your sleep quality overall?
  - a. Very good
  - b. Fairly good
  - c. Fairly bad
  - d. Very bad
- 6) \*During the past month, have you had a bed partner or roommate?
  - a. Yes, we sleep in the same bed
  - b. Yes, we sleep in the same room, but not in the same bed
  - c. Yes, but we sleep in different rooms
  - d. No, I do not have a bed partner or room mate
- 7) (Skip if prev Q\* = No or if sleep in different rooms) Please ask your bed partner or roommate the following question: During the past month, have you heard me snoring loudly?
  - a. No
  - b. Yes, less than once per week
  - c. Yes, once or twice per week
  - d. Yes, three or more times per week
- 8) (Skip if prev Q\* = No, or if sleep in different rooms) Please asked your bed partner or roommate the following question: During the past month, have you heard me take any long pauses between breaths while asleep?
  - a. No
  - b. Yes, less than once per week
  - c. Yes, once or twice per week
  - d. Yes, three or more times per week

## APPENDIX L – Additional Statistical Analyses

**Table 3** Available analytic sample sizes for all variables

	Mothers (n = 64)	Fathers (n = 61)	Total (n = 125)
<b>General Stress</b>	63	58	121
<b>PSI</b>	62	58	120
<b>CHAOS</b>	61	59	120
<b>BMI</b>	64	60	120
<b>WC</b>	63	61	124
<b>WHtR</b>	63	61	124
<b>%FM</b>	64	60	124
<b>Sleep Efficiency</b>	61	54	115
<b>Dietary Fat Intake</b>	23	29	52
<b>Physical Activity</b>	61	51	112
<b>Cortisol</b>	47	39	86

**Table 4** Unadjusted regression results using generalized estimating equations of body composition on stress. Significant associations are shown in bold.

Body Composition		General Stress $\hat{\beta}$ (95% CI)	Parenting Stress $\hat{\beta}$ (95% CI)	Household Chaos $\hat{\beta}$ (95% CI)
<b>Mothers</b>	<b>BMI</b>	0.39 (-0.27, 1.06) N = 63	0.11 (-0.07, 0.28) N = 62	<b>0.22 (0.04, 0.39)</b> N = 61
	<b>WC</b>	0.89 (-0.32, 2.11) N = 62	0.33 (-0.02, 0.67) N = 61	<b>0.37 (0.05, 0.68)</b> N = 60
	<b>WHtR</b>	0.001 (-0.001, 0.001) N = 62	0.002 (-0.001, 0.004) N = 61	<b>0.002 (0.0001, 0.004)</b> N = 60
	<b>%FM</b>	0.11 (-0.952, 1.173) N = 63	0.13 (-0.11, 0.37) N = 62	<b>0.32 (0.10, 0.55)</b> N = 61
<b>Fathers</b>	<b>BMI</b>	0.31 (-0.43, 0.04) N = 58	-0.01 (-0.19, 0.20) N = 58	0.10 (-0.06, 0.25) N = 58
	<b>WC</b>	0.64 (-1.52, 2.80) N = 58	-0.06 (-0.48, 0.60) N = 58	0.16 (-0.22, 0.54) N = 59
	<b>WHtR</b>	0.005 (-0.007, 0.016) N = 58	0.001 (-0.002, 0.004) N = 58	0.001 (-0.001, 0.004) N = 59

	<b>%FM</b>	0.16 (-1.16, 1.47) N = 58	-0.08 (-0.25, 0.40) N = 58	0.08 (-0.18, 0.34) N = 58
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**Table 5** Means (SD) and regression coefficients (95% CI) of potential mediators with predictors and outcomes in mothers' data, adjusted for age, family size, and annual household income. Significant associations are shown in bold.

	<b>Cortisol</b>	<b>Sleep</b>	<b>Dietary Fat Intake</b>	<b>PA</b>
<b>N</b>	47	61	23	61
<b>Mean (SD)</b>	313.62 (91.08)	0.88 (0.11)	39.74 (6.40)	1948.09 (1839.04)
<b>Regression of BMI on mediator</b>	0.001 (-0.028, 0.027) N = 46	-4.66 (-19.96, 10.65) N = 58	0.11 (-0.35, 0.57) N = 23	-0.0003 (-0.001, 0.001) N = 58
<b>Regression of WC on mediator</b>	-0.003 (-0.07, 0.06) N = 45	-11.70 (-42.06, 18.65) N = 57	-0.05 (-0.74, 0.65) N = 22	-0.001 (-0.003, 0.001) N = 57
<b>Regression of WHtR on mediator</b>	-0.0000 ± 0.0002 (-0.0004, 0.0003) N = 45	-0.10 (-0.29, 0.09) N = 57	-0.001 (-0.005, 0.003) N = 22	-0.000 ± 0.000 (-0.000, 0.000) N = 57
<b>Regression of %FM on mediator</b>	1.38 (-0.84, 3.60) N = 46	-0.002 (-0.005, 0.001) N = 58	-0.04 (-0.27, 0.20) N = 23	-35.65 (-99.96, 28.67) N = 58
<b>Regression of mediator on PSI</b>	-1.18 (-3.77, 1.41) N = 45	<b>0.003</b> <b>(0.0001, 0.006)</b> <b>N = 56</b>	0.07 (-0.44, 0.58) N = 21	7.49 (-76.85, 91.83) N = 56
<b>Regression of mediator on CHAOS</b>	2.65 (-0.39, 5.69) N = 43	0.0003 (-0.003, 0.004) N = 55	<b>0.59</b> <b>(0.27, 0.91)</b> <b>N = 20</b>	-26.01 (-94.45, 42.43) N = 55