The Effect of Breakfast Macronutrient Composition in Children Ages 7-17 Years Old as a Potential Method to Combat Childhood Obesity

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The Effect of Breakfast Macronutrient Composition in Children Ages 7-17 Years Old as a Potential Method to Combat Childhood Obesity

A thesis submitted in partial fulfilment of the requirements for the degree of Master of Science in Food Science

by

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May 2020
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ABSTRACT

The prevalence of childhood overweight and obesity is an ongoing concern. Currently, approximately 20% of children in the United States are obese. While obesity was once regarded merely as excessive adiposity within the body, it has emerged as a major risk factor for chronic diseases such as metabolic syndrome, type 2 diabetes, and cardiovascular disease. Obesity is multifactorial in nature. Weight gain can result from an energy imbalance in the body due to excess energy intake (calories in) and decreased energy expenditure (calories out). Identifying methods to combat obesity is essential. Nutritional intervention may be a strategy to help regulate energy balance and fight obesity. The benefits of high protein diets on body composition, energy expenditure, appetite and markers of metabolic health have been well studied in adults. In addition, there is evidence that supports regular breakfast intake is an important component in limiting the risk of developing obesity and other subsequent health-related diseases. However, over time, there has been a decline in the consumption of breakfast and the effects of higher protein intake, specifically at breakfast, in children is lesser known. Therefore, the objective of this thesis was to determine the effect that a higher protein breakfast consumption for 6-weeks can have on energy expenditure, substrate oxidation, appetite, and markers of metabolic health in normal weight and overweight children ages 7-17 years old.
ACKNOWLEDGEMENTS

I would like to thank Dr. Jamie Baum for her guidance and mentorship towards this degree, as it has given me an opportunity to grow both personally and professionally throughout my time here. I would also like to thank Aubree Hawley, Sam Walker, Hexirui Wu, Jamie McDermott, Caroline Ganoung, and Josey Moix. Without their collaborative efforts and dedication, these projects would not have been possible. Lastly, I would like to thank Dr. Elisabet Børsheim and Dr. Michelle Gray for serving on my thesis committee and their continued support and understanding throughout my degree.
DEDICATION

This thesis is dedicated to my parents, Michael & Giuseppina Tacinelli, and Michael Tuscano. Thank you for your unyielding support and confidence in all of my endeavors.
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INTRODUCTION

Currently, 1 in every 5 children in the United States is considered obese (1). More specifically, approximately 40% of Arkansas students in public schools are classified as either overweight or obese (2). The Centers for Disease Control and Prevention (CDC) classifies children between the ages of 2-19 years old based on their Body-mass-index-for age percentile. Overweight is a body mass index (BMI) between the 85\textsuperscript{th} and 94\textsuperscript{th} percentiles, whereas ≥ 95\textsuperscript{th} percentile is considered obese. Obesity leads to changes in metabolic health due to the shift in body composition (3-5). Obesity is associated with an increased risk of acquiring many chronic diseases such as type 2 diabetes, cardiovascular disease, and metabolic syndrome (6, 7), resulting in dysregulation of insulin response, blood glucose levels, and cholesterol (3). An increase in these chronic diseases and health complications among children is alarming given that they can lead to severe lifestyle limitations or premature death (8, 9).

While obesity is multifactorial in nature, energy imbalance caused by increased energy intake and decreased energy expenditure is a primary contributor to the onset of obesity (6, 10). Research suggests that adipose tissue may influence energy balance in the body (11). Therefore, to improve regulation of energy balance in overweight and obese children who have an excess of adipose tissue, it is important to understand how diet composition can potentially increase energy expenditure. Energy expenditure is comprised of three components: 1) resting metabolic rate (RMR), 2) activity and non-activity thermogenesis, and 3) thermic effect of food (12). Thermic effect of food is responsible for ~10\% of daily energy expenditure (DEE) and resting metabolic rate is responsible for ~60-75\% of DEE (13). Among the macronutrients, protein has the greatest thermic effect, comprising approximately 20-35\% of energy intake, compared to carbohydrates and fats (14). In addition, the consumption of high-protein diets positively influence appetite and markers of metabolic health (15-17). This suggests that increasing protein intake within the diet may increase thermic effect of food, subsequently
increase overall daily energy expenditure, and decrease energy intake following meal consumption.

The recommended dietary allowance (RDA) for protein intake in children is approximately 15% of daily energy intake, however, research in adults examining the benefits of higher protein diets shows that the recommendation should be increased to 30% of daily energy intake consisting of protein (18). Research suggests that, of their total calories in a 24-hour period, children and adolescents are consuming a maximum of 20% protein intake in the morning compared to a minimum of 40% protein intake in the evening (19). Higher protein intake at breakfast regulates appetite by increasing fullness and inhibiting hunger when compared to a normal protein or carbohydrate breakfast (20, 21); evident in both subjective and hormonal appetite assessments following a high protein breakfast (20, 22). In addition, a higher protein breakfast helps to regulate markers of metabolic health such as glucose (23).

Collectively, increasing protein within the diet at the breakfast meal, may help serve as a regulator of appetite which may decrease overall energy intake and increase overall energy expenditure via thermic effect of food. Taken together, increasing dietary protein at the breakfast meal may help improve energy balance within the body and thus serve as a potential method for combatting childhood obesity. Currently, the research literature primarily focuses on high protein diets in adults and the benefits of consuming breakfast in adolescents. However, a gap still remains with regards to the effects of a higher protein breakfast on metabolic health and appetite related to energy balance in overweight and obese school-aged children and adolescents.

Therefore, the objective of this thesis is to determine if consuming a higher protein intake at breakfast can serve as a potential method to combat childhood obesity by increasing energy expenditure, improving appetite and markers of metabolic health in overweight and obese children.
REFERENCES


LITERATURE REVIEW

Prevalence and Economic Impact of Childhood Obesity in the US

During recent decades, childhood obesity rates have continuously increased in developed and undeveloped countries causing an epidemic of worldwide concern (1-4). In the U.S., nearly 20% of children ages 2-19 years old are classified as obese (5). It has been estimated that approximately one-third of obese children will remain so as adults (1). Research has found that children who are obese have an increased chance of being obese and having health complications as an adult; therefore, childhood and adolescence serves as a critical period for both their current and future health status (6-8). The impact of rising obesity rates extends beyond merely health complications but can also have a significant economic impact (4, 9). These health complications associated with obesity, such as psychosocial, endocrine, and cardiovascular (4), contribute to significantly higher healthcare costs including an increase in doctor visits, drug costs, and healthcare resources including laboratory tests, medical staff, and short- and long-term medical care centers (10). It has been estimated that by 2030, 65 million more adults will be obese in the US requiring an estimated $48-66 billion/year in treatment of diseases associated with obesity (10, 11).

Defining Childhood Obesity

Obesity is generally defined as an excess of body weight (12). The Centers for Disease Control and Prevention (CDC) classifies overweight and obesity in adults based on their body mass index (BMI). Adult BMI is calculated by dividing a person’s weight in kilograms by their height in meters squared (kg/m²), where overweight is a BMI of 25-29.9 kg/m² and a BMI ≥ 30 kg/m² is obese (13). However, overweight/obesity in children is classified differently. Children between the ages of 2-19 years old are classified as overweight or obese based on their BMI percentile using the CDC’s BMI-for-age growth chart (14). The growth chart takes into
consideration a child’s height and weight to determine their BMI, but then additionally factors in their age (in months) to determine the percentile that the BMI falls within on the growth chart. Children with a BMI-for-age that falls between the 85th and 94th percentile are categorized as overweight, while those that are ≥ 95th percentile are considered obese.

BMI serves as a tool to indirectly estimate the amount of fat within the body (15, 16), however other methods may be more accurate in directly quantifying body composition, i.e. the amount of fat free mass (FFM) and fat mass (FM), within the body of children and adolescents (17, 18). FFM and FM have been speculated to be regulators of daily energy intake (DEI), however these findings remain inconsistent and require further research (19-21). Although both FFM and FM increase with obesity (22), body composition of overweight and obese individuals is comprised of a higher FM to FFM ratio (19, 20), where as much as 30-50% of weight in obese children is FM (22). Obese individuals undergo metabolic changes as a result of changes in body composition (19, 20), and the excessive adiposity characteristic of obesity is linked with metabolic disorders (23) and health complications (24-27).

Health & Metabolic Consequences Associated with Childhood Obesity

Obesity increases the risk of developing chronic diseases including cardiovascular disease, type 2 diabetes, dyslipidemia, or hypertension (28-31). The prevalence of childhood obesity increases the risk for premature development of chronic diseases (4, 32). As a result, chronic diseases that were once associated only with adulthood, are now commonly being seen in children and adolescents (4, 32-35). Long-term consequences may results from the early onset of these diseases in childhood and adolescence, collectively leading to increased risk of morbidity and premature mortality (36).
Contributors to Childhood Obesity

Childhood obesity is a multifactorial issue. Several factors have been identified as being influential towards the development of obesity including genetics, lifestyle, environmental, socioeconomic, and energy balance (30, 37). Therefore, it is impossible to identify just one factor as the cause for the increase in overweight and obesity in children.

**Genetics.** Ongoing research aims to map genes that may be associated with the onset of human obesity and obesity-related diseases (4, 38). While obesity has been found to be partly related to genetics, BMI is only 20-40% heritable and BMI genetics contribute to less than 5% of childhood obesity (30, 39, 40). Genetic mutations have been found to have a role in the onset of childhood obesity (41). The first obesity susceptibility gene, fat mass and obesity associated (FTO) gene, was initially discovered in genome-wide association studies in 2007 (42-44). The FTO gene was found to be associated with an overweight BMI and an increased risk of childhood obesity as early as 7 years old and continuing into puberty (43). However, as many as 250 quantitative trait loci, or sections of DNA, have been found to be associated with human obesity phenotypes, suggesting that it is not a single gene associated with obesity (38). Genetic links have also been observed between carbohydrate metabolism and BMI as a predictor for obesity (45), as well as postprandial lipid metabolism (46). Maternal weight status, health conditions, and diet during pregnancy has also been correlated with risk for weight gain and disease in childhood (47-49). Given that children are predisposed to the onset of obesity as early as in utero, specific research focuses on continuing to understand the influential role that genetics and maternal health have towards the risk for obesity and disease of the child.

**Lifestyle, Environmental, and Socioeconomic Factors.** Environmental factors and socioeconomic status are both contributors towards increasing rates of childhood obesity (4, 16, 30). Sedentary time spent watching television, sitting at a computer, or doing homework has increased compared to physical activity time (50, 51); in turn, sedentary behavior has been found to be associated with obesity among children and adolescents (52). In addition to
technology advancements leading to increased sedentary behavior, marketing tactics and advertisements encompassed within the screen time also serve as influential factors towards poor dietary choices made by kids (53, 54). An increase in accessibility of low-quality foods and beverages, including sugary beverages and energy dense junk foods, due to the presence of vending machines and school stores within the academic environment provides additional opportunity for those marketing strategies to take effect (55, 56). As much as 13% of elementary schools, 67% of middle schools, 85% of high schools reported having vending machines (55). The persistence of high fat, high calorie foods and beverages, including sugary beverages and energy dense junk foods, due to the presence of vending machines (57) provides poor nutritional value and enforces poor dietary habits that may influence children and adolescent obesity risk. In addition, it has been found that the frequency of eating out is associated with an increase in total energy intake, fat intake and BMI (58) likely due to the increased portion sizes and energy density of the food choices. Specifically, eating fast food has been found to be correlated with obesity (59), and consumption of food not prepared at home (i.e. meals from restaurants, fast food, or grocery store) has been found to correlate with increased BMI (60, 61) and increased body percentage (61). Along with an increase in consumption of low-quality food outside of the home, household family mealtimes and sociocultural factors also influence the risk for obesity (30). Studies have also shown that the household dynamic can affect a child’s access to healthy meals and physical activity leading to a greater risk for becoming overweight or obese (62). Over time, an increase in BMI within childhood tracking into adulthood has been observe among racial and ethnic minorities (63, 64). Additionally, obesity rates have been found to be inversely related to level of education (65, 66) and income (66), possibly due to low-income households selecting lower-quality food items to maximize cost, especially when on food assistance programs (67, 68).

**Energy Balance.** Dietary intake and physical activity are both vital for maintaining energy balance within the body. A positive energy balance is when energy intake is greater than energy expenditure; therefore a negative energy balance is when energy expenditure is greater
than energy intake (69). If energy intake (calories consumed) is equal to energy expended (calories burned), then energy balance is achieved (69); however, a state of continuous energy imbalance, specifically positive energy balance, can cause weight gain, and eventually lead to obesity (30, 69, 70).

There are three components of energy expenditure: resting metabolic rate (RMR), activity thermogenesis and non-activity thermogenesis, and thermic effect of food (TEF) (71). Activity and non-activity thermogenesis, or the energy expended from exercise and non-exercise physical activity, can contribute as much as one-third of a person’s daily energy expenditure (71). Therefore, if physical activity recommendations are not being met, energy intake will outweigh energy expenditure and the resulting energy imbalance can lead to weight gain. Currently, it is recommended that children and adolescents ages 6-17 years participate in at least 60 minutes of physical activity every day (72). However, less than one quarter of children and adolescents are meeting this recommendation (73). A lack of physical activity, or energy expenditure (EE), can lead to an excess of energy intake, resulting in an energy imbalance in the body (30). The total caloric and macronutrient intake within the diet influences the body’s energy balance (70). If carbohydrate and fat oxidation are not stimulated, it may lead to weight gain (70). There is increasing evidence that dietary protein can help influence energy intake and energy expenditure, thereby controlling energy balance (74). A high-protein low-carbohydrate diet compared to a high carbohydrate low-protein diet was shown to help maintain energy balance (75). Therefore, dietary intervention may be a potential method for the prevention and treatment of childhood obesity.

**Dietary Protein and Obesity**

It is well-established that obesity causes negative body composition and metabolic health changes (7, 16, 76, 77). One possible mechanism for combatting obesity is through dietary intervention; however, the optimal macronutrient composition and distribution is
unknown. Continuous research aims at understanding the effect that quantity and quality of macronutrient consumption at specific meal times can have on appetite, body composition, metabolic health, and energy expenditure (4, 78).

High-protein diets have been shown to have promising effects in regulating or improving obesity-related health issues (79). High-protein diets have shown to improve blood glucose regulation, increase energy expenditure, reduce blood pressure, increase satiety, and promote weight loss (79-82). Proteins are comprised of essential and non-essential amino acid chains. The essential branched chain amino acids (leucine, isoleucine, valine), which must be acquired from the diet, are believed to be the primary drivers in the metabolic effects of a high-protein diet and has been extensively reviewed (81, 83-85).

As childhood obesity rates have increased, so have the cases of pediatric type 2 diabetes (T2D) (32, 86, 87). A characteristic of T2D is insulin resistance due to the imbalance between its secretion and action. As a result of insulin resistance, glucose homeostasis is dysregulated in the body, which can increase the risk of developing additional diseases (88). High protein diets may help regulate glucose homeostasis and prevent hyperglycemia, especially following meal consumption (79, 82, 89-91).

The consumption of protein has also been influential in regulation of satiety and gut hormones that regulate appetite (92-97). Peptide YY (PYY) and cholecystokinin (CCK) are two gut hormones that have an anorexigenic effect and therefore influence appetite (as reviewed by 98, 99). Research has shown that, following a meal, plasma levels of CCK and PYY can change in as little as 15 minutes or 1 hour, respectively (99, 100). Both PYY and CCK may reduce food intake (99). In normal and overweight patients, a protein preload caused an increased postprandial CCK response when compared to a glucose preload (92, 101). When comparing a protein beverage to a fructose beverage, the fructose beverage resulted in reduced CCK concentration (93, 101). In addition to appetite hormones, high protein diets may improve satiety (92-94, 97, 102) and therefore decrease subsequent energy intake (92-95). When lean women
were given a protein preload, subjects were less hungry and had lower energy intake compared to carbohydrate, fat, and alcohol preload (103). When preschool children were fed a high protein meal versus a high carbohydrate meal, greater energy intake was observed with the high carbohydrate meal (104). Collectively, protein in the diet can help modulate appetite by increasing satiety and decreasing energy intake.

Thermic effect of food, a component of energy expenditure, is directly influence by the macronutrient composition within the diet (105, 106). Adipose tissue is believed to be a main driver in appetite control and a predictor of energy balance (energy intake vs. energy expenditure) (107, 108). Daily energy expenditure is comprised of three components: 1) RMR, 2) activity and non-activity thermogenesis, and 3) TEF (71). RMR, or the energy required for basic bodily functions, is responsible for ~60-75% of daily energy expenditure. TEF is the increase in energy expenditure after meal consumption and is responsible for ~10% of daily energy expenditure. RMR of obese individuals have been found to be correlated with meal size and DEI (109). In addition, adults consuming higher protein diet have increased TEF compared to a low protein diet or a carbohydrate diet (110, 111). Therefore, given that TEF can be directly influenced by the meal compositions of the diet, it has been a primary target for influencing energy balance and combatting obesity.

The thermic effect of protein is as much as 20-35% of energy intake, carbohydrate is approximately 5-15% of energy intake, and fat is 0-3% of energy intake (112). Given that protein has the greatest thermic effect of the macronutrients, dietary interventions involving protein may serve as a potential method for increasing daily energy expenditure and combatting obesity. TEF was found to be higher by approximately 346 kJ/d following a high protein/carbohydrate diet (29% protein) when compared to a high fat diet (9% protein) (112). In a study comparing an isocaloric low protein/high carbohydrate meal-replacement shake (17% protein, 28% carb) to a high protein/low carbohydrate shake (62% protein, 28% carb), TEF was significantly higher in the high protein/low carbohydrate group (106). Another study involving obese adults with type 2
diabetes observed a significant increase in TEF 2 hours postprandial at week 0 and week 12 when consuming a high protein meal compared to a low protein meal (110).

In addition to dietary protein increasing energy expenditure via TEF, it may help improve body composition, promote weight loss and maintain muscle. Collectively, consumption of dietary protein may serve as a regulator for appetite, body composition, and metabolic health, and a stimulator of diet induced energy expenditure; however, the ideal meal time of protein intake within the diet still remains unclear, especially in children.

**Breakfast Intake, Dietary Protein and Obesity**

Breakfast is often referred to as the most important meal of the day, however the positive effects of breakfast intake do not align with the decrease in breakfast consumption patterns (113). From 1965-1991, breakfast consumption among US adults declined 86% to 75%. In a study examining adult breakfast consumption from 2001-2008 NHANES data, approximately 20% skipped breakfast and had a significantly higher BMI and prevalence of obesity than breakfast consumers (114). In addition to decreased breakfast consumption among adults, research has also observed a significant decrease in breakfast intake among children (115). Studies have shown that as much as 19% of the children ages 2-18 years old skipped breakfast (116), and that of the 7116 subjects ages 6-18, one in four overweight or obese children regularly skipped breakfast (117). In a 21-year longitudinally designed survey, a decrease in regular breakfast consumption was observed (113). An association was also observed between an increase in breakfast skipping and female, not male, adolescents who are dieting to lose weight (118), suggesting that the frequency of breakfast skipping may be more correlated with specific gender or body image motives.

Research has further supported the impact that breakfast consumption can have on both disease prevention and health intervention in children and adolescents. Breakfast consumption among children and adolescents have been shown to decrease adiposity (119-121), decrease
BMI (122-125), decrease risk of type 2 diabetes (126, 127), and positively influence subsequent energy consumption (126-129), satiety (28, 126, 130), and mood responses (126, 131).

In the diets of preschool children, as much as a ≥ 700 kcal increase in overall energy intake and a ≥ 100 g increase in carbohydrate intake was observed among breakfast skippers’ diets (132). Children who did not have breakfast had increased fasting insulin levels and insulin resistance, both risk markers for type 2 diabetes, compared to regular breakfast eaters (127). Research in adults has also shown that skipping the breakfast meal can have a greater metabolic and appetitive effect in regular breakfast consumers compared to regular breakfast skippers, possibly due to the metabolic response the body habitually expects (133). Similarly, in a 5-year longitudinal study, breakfast frequency among adolescents was found to be inversely related to BMI and weight-gain (134).

While there appears to be an overall agreement among scientific literature encouraging breakfast intake, there is a lack of established scientific consistency with regards to breakfast recommendations due to varying breakfast meal parameters such as timing, nutrient composition, and meal size (114). Despite the positive benefits of eating breakfast, the number of breakfast skippers has increased overall, and continues to increase with age (115, 135); and the decrease in breakfast eating habits share a negative correlation with increasing rates of childhood obesity; which may be a result of the change in dietary patterns (128).

Additionally, the ideal nutrient composition of the breakfast meal remains controversial (124, 126, 136-146); however, efforts such as the International Breakfast Research Initiative aims at working towards a standardized consensus (147, 148). Out of 7800 dietary intake records from children ages 2018 years old, nearly 83% of the reported breakfast meals were either bread-based or ready-to-eat-cereals (RTECs) (113)- both high-carbohydrate, low-protein food choices. Research from the NHANES 2013-2014 data set showed that, across eating times, protein and energy intake distribution is shifted toward evening meals (149). However, a majority of research, instead, suggests the optimal intake pattern is to consume low
carbohydrate, high protein meals earlier in the day, specifically at breakfast (96, 97, 102, 129, 139, 144, 150). Evidence further suggests that breakfast consumption within the overall diet of children and adolescents is influential in the quantity of macronutrient and total energy intake (151). Therefore, additional research is needed to understand the effect of protein at breakfast, specifically in children and adolescents.

While the recommended daily allowance (RDA) for protein is 0.95 g/kg/d or 0.85 g/kg/d in children ages 4-13 and 14-18 years old, respectively, this equates to less than 15% of total daily energy intake. Higher protein diets recommend as much as 30% of total daily energy intake come from protein (152). A normal protein breakfast has been shown to contribute to higher daily energy intake compared to a high protein breakfast (96). Independent of body weight, a high protein breakfast was found to suppress hunger and desire to eat, while stimulating more fullness compared with a high carbohydrate breakfast (102). Similarly, it was observed that when a protein rich breakfast (38% protein, 39% carb) was consumed compared to a normal protein breakfast (14% protein, 73% carb) or no breakfast at all, postprandial appetite was significantly inhibited (97). In addition, a high-protein breakfast (40% protein, 35 g) lead to lower peak glucose levels and reduced glucose variability when compared to a normal-protein breakfast (15% protein, 13 g) (153). High-protein diets have been found to increase TEF by ~1-22% compared to low-protein diets (112, 154-159). In addition, a high protein breakfast compared to a carbohydrate breakfast increased energy expenditure and fat oxidation in overweight/obese children (102). In a study that compared a high-protein based (boiled egg) or high-carbohydrate based (steamed bread) breakfasts, a decrease in subsequent energy intake at the following mealtime was observed following the high protein breakfast consumption (95).

It is believed that a high-protein breakfast can have positive effects in regulating energy expenditure, appetite and markers of metabolic health which are commonly dysregulated in overweight and obese individuals.
Gaps in the Literature

In conclusion, the prevalence of childhood obesity a primary public health concern. The presence of childhood obesity increases the risk for developing chronic disease prematurely and therefore requires attention. Finding potential methods to manage or reduce the rate of childhood obesity is imperative. One potential strategy is nutritional intervention. Increasing dietary protein within the diet has been found to promote weight loss, regulate markers of metabolic health, increase satiety, increase energy expenditure, and decrease energy intake. Current literature focuses on the effects of an overall high protein diet on health and aging in adults. However, further research is still needed to better understand the effect of dietary protein in school-aged children and at breakfast.

Therefore, the overall objective of this thesis was to determine the effect of protein intake at breakfast as a potential method to combatting childhood obesity.

The objectives of each study aim were:

Aim 1: Determine the postprandial effect of a higher protein breakfast on improving energy expenditure, substrate oxidation, markers of metabolic health, and appetite in school-aged children.

Aim 2: Determine the adaptation effect of a higher protein breakfast on resting energy expenditure, energy intake, and markers of metabolic health in normal versus overweight/obese school-aged children.

We hypothesized:

Aim 1: Increasing dietary protein intake at breakfast will increase postprandial energy expenditure, increase postprandial substrate oxidation, improve postprandial markers of metabolic health and appetite in school-aged children.
**Aim 2:** Adaptation to higher dietary protein intake at breakfast will increase resting energy expenditure, improve markers of metabolic health and decrease energy intake in overweight/obese school-aged children.
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CHAPTER 1
6-weeks of Higher Protein Intake at Breakfast Does Not Change Postprandial Meal Response in 7-17 Year Old Children who Regularly Consume Breakfast

Abstract

Objective: The objective of this study was to determine if increased protein intake at breakfast for 6 weeks influences energy balance by increasing energy expenditure and decreasing energy intake through changes in postprandial appetite response in 7-17 year old children.

Methods: This study was a 6-week, double-blind, randomized controlled dietary intervention in 7-17 year old children. A total of 24 participants completed the study (11 males, 13 females). Participants were randomly assigned to either a protein-based breakfast (PRO; 30 g protein; n=13; 7 male; 6 female) or carbohydrate-based breakfast (CHO; 13 g protein; n=11; 5 males, 6 females). Participants arrived fasted on day 1 and day 42 to complete two laboratory visit test days. Anthropometrics were measured at each visit. Energy expenditure, thermic effect of food (TEF), substrate oxidation, and plasma markers were measured at baseline, 30, 60, 120, 180, and 240 minutes postprandial. Appetite was measured at baseline, 15, 30, 60, 90, 120, 180, and 240 minutes postprandial.

Results: After controlling for body weight, there was a significant effect of time ($P < 0.05$) and diet intervention x time interaction ($P < 0.01$) on resting energy expenditure. There was a significant effect of time ($P < 0.0001$) and diet intervention ($P < 0.01$) on substrate oxidation. There was no effect of diet on perceived hunger, perceived fullness, perceived desire to eat, and prospective food consumption. There was no effect of diet on plasma glucose. There was an effect of diet ($P < 0.05$) on plasma cholecystokinin and plasma leucine ($P < 0.01$).

Conclusions: Increasing protein intake at breakfast for 6-weeks does not change postprandial meal response in 7-17 year old children who regularly consume breakfast.
Approval for this study was obtained by the Institutional Review Board at the University of Arkansas for Medical Sciences (Little Rock, AR) and registered as a clinical trial on ClinicalTrials.gov (NCT03602144).

Introduction

The increasing prevalence of childhood obesity is a global epidemic (1, 2). Therefore, identifying methods of treatment and/or prevention is essential for the future health of children and adolescents. Two factors that contribute towards the onset of obesity include the increase in calorie intake and decrease in energy expenditure (3, 4). This energy imbalance then leads to storage of fat and potential weight gain (5). Therefore, regulation of energy balance through dietary intervention may be a potential method for combatting childhood obesity (6).

Current literature suggests that breakfast consumption may effect daily food and nutrient intake, serving as an indicator of overall diet quality (7). However, nearly 25% of adults do not consume breakfast (7). In parallel, there has been a 9-20% decline of breakfast consumption in children and adolescents (8). Positive associations have been found between eating breakfast and appetite response (9), cognitive performance (10-12), body composition (11-13), risk for developing chronic diseases (14), mental and emotional health (2) in children and adolescents. In addition, breakfast consumption frequency is associated with adiposity and body mass index (BMI) (15-21). While scientific literature has recognized the many benefits of eating breakfast, a lack of research still remains in defining what the best macronutrient composition is for a quality breakfast (7, 12, 22).

Almost 25% of adults (7) and 19% of children (22) do not eat breakfast. In addition to a decline in reported breakfast consumption (23) the breakfast meal for children is predominantly comprised of carbohydrates (24). Previous research has found that as much as 90% of children ages 4-12 years old consume ready-to-eat-cereals (RTEC) for breakfast at least once in 14 days (25); and of the children and adolescents who regularly consume breakfast, approximately
50% consume RTEC for breakfast (26). Though they may serve as a good source of micronutrients, it offers an imbalanced profile of macronutrients for the breakfast meal: high carbohydrate and low protein composition (25, 27).

There are several beneficial health effects associated with consuming quantities of protein (1.2-1.6 g/kg or 25-30 g/meal) higher than the recommended dietary allowance (RDA; 0.8 g/kg/d), including reduced body weight and fat mass and conserved lean mass, improved appetite and satiety, increased fullness, improved cardiometabolic risk factors, and increased substrate oxidation, TEF and resting metabolic rate (28-31). Previous research also suggests that protein intake can influence appetite hormones such as CCK (32, 33) and PYY (34), and markers of metabolic health such as glucose regulation (35). Despite this knowledge in adults, less attention has been given towards understanding the effect of higher protein intake in children and adolescents. Although limited research has been done in this age group, the focus has primarily been done in breakfast skipping adolescents and the results suggest that a high-protein breakfast can aid in weight management, reduced energy intake, glycemic control and appetite regulation (30, 36-40).

Protein has a higher effect on TEF compared to other macronutrients (41). By increasing protein intake at breakfast, TEF may drive an increase in postprandial energy expenditure (EE). In addition, a higher protein breakfast may decrease hunger and increase fullness to reduce subsequent energy intake. Following the consumption of a high protein breakfast in breakfast skipping adolescents, an increase in energy expenditure, fat oxidation, and reduction in hunger has been observed (40, 42). However, current evidence shows that protein intake in children is skewed away from the breakfast meal (43).

Collectively, increasing protein at breakfast to target TEF and appetite may serve as an effective method for obesity prevention. To our knowledge, energy expenditure and appetite response to a high protein breakfast over time has not been explored in children and adolescents. Therefore, the objective of this study was to determine if increased protein intake
at breakfast for 6 weeks influences energy balance by increasing postprandial energy expenditure and decreasing energy intake through changes in postprandial appetite response in 7-17 year-old children. We hypothesized that children who consumed a higher protein breakfast would have increased postprandial energy expenditure, improved appetite response, and decreased energy intake compared to children consuming a higher carbohydrate breakfast.

Methods

Participants and Screening. Male and female children between 7-17 years of age were recruited to participate in this study. Participants were recruited through the daily University of Arkansas e-newsletter, local blogs, local after-school camps, and flyers posted throughout the community. An initial phone screening was conducted with the parents or legal guardians of interested participants to determine if they met the inclusion and exclusion criteria. Approximately 150 initial phone screenings were conducted. To qualify, participants had to reside in Northwest Arkansas, be 7-17 years of age, and have a BMI > 5th percentile. Participants who regularly skipped breakfast (> 5 times per week), regularly consumed protein at breakfast (> 25 grams of protein at breakfast > 4 time per week), had allergies or dietary restrictions, were classified as a picky eater by parent/guardian, had a fear of needles, were claustrophobic, or were on prescription medication were excluded from the study. A total of 24 subjects completed the study. Eleven subjects dropped out due to lack of protocol compliance or not being able to collect blood prior to the start of the diet intervention. A diagram of the recruitment, screening and enrollment process can be found in Figure 1. Approval for this study was obtained by the Institutional Review Board at the University of Arkansas for Medical Sciences (IRB Protocol # 207201; Little Rock, AR) and registered at ClinicalTrials.gov at NCT03602144.

Study Design. Once qualified, subjects underwent an in-person screening visit in which they were first presented with an overview of the protocol. Parents and legal guardians of the
children provided written consent and children provided written assent before beginning participation in the study. Prior to the start of the intervention, baseline height, weight, and body composition were collected. To aid with compliance throughout the study, parents and participants were provided with a welcome bag upon initial enrollment. The welcome bag contained measuring cups/spoons and food scale for reporting their food intake and an informational booklet containing their study schedule, dietary food log examples and reference sheets (i.e. estimating portion size with your hands), and instructions for consuming the breakfast shake. A double-blinded, randomized study design was used to assign participants (n=24) to one of two dietary interventions: 1) a protein-based breakfast beverage (PRO; n=13), or 2) a carbohydrate-based breakfast beverage (CHO; n=11) intervention for six weeks. Participant characteristics can be found in Table 1. Participants completed two laboratory visits at the Center for Human Nutrition at the University of Arkansas on day 1 and day 42 of the dietary intervention. Parents/guardians we instructed that participants should fast 8-10 hours overnight and avoid any vigorous physical activity the day before each laboratory visit. On each testing day, height, weight, and resting energy expenditure (REE) were measured, followed by a fasted blood draw and baseline appetite assessment. Participants were then provided with their assigned test breakfast beverage (PRO or CHO) and given 10 minutes to consume the entire beverage (295.7 ml). Blood was drawn at 30, 60, 120, 180, and 240 minutes postprandial. TEF was measured at baseline, 30, 60, 120, 180, and 240 minutes postprandial. Appetite response was assessed at baseline, 15, 30, 60, 90, 120, 180, and 240 minutes postprandial. Movies were shown during laboratory visits to reduce physical movement during indirect calorimetry testing periods. Previously reported research has also used this method (44). A study day timeline can be found in Figure 2. At the end of the first test day, participants were given test beverages for the first 21 days of the intervention as well as a 24-hour food intake record to complete for the remainder of the day. Parents/guardians returned on day 21 of the intervention to pick up the
remaining 3 weeks supply of test beverages on behalf of the participants. Parents/guardians and participants returned on day 42 to repeat the protocol described above.

**Test Breakfasts.** Participants were randomly assigned to one of two test breakfasts: protein-based breakfast (PRO; 30 g protein, 31 g carbohydrate, 11 g fat) or carbohydrate-based breakfast (CHO; 13 g protein, 48 g carbohydrate, 11 g fat). Participants were instructed to consume their assigned test breakfast beverage each day of the intervention period prior to 10:00 am (45) and provided various flavors for options including chocolate, strawberry and vanilla. PRO and CHO were isocaloric, matched for fat and fiber (Table 2).

**Anthropometrics.** Anthropometrics were measured at baseline and on day 42 of the dietary intervention. Body height was measured barefoot in free-standing position to the nearest 0.01 cm using a stadiometer (Detecto, St. Louis, MO). Body weight was measured in the fasted state to the nearest 0.01 kg using a calibrated balance-beam eye level scale (Detecto, St. Louis, MO). BMI was calculated as weight (kg) divided by height squared (m²). BMI-for-age percentile was calculated using the Center for Disease Control’s (CDC) BMI Percentile Calculator for Child and Teen (46). Body composition was assessed using dual energy x-ray absorptiometry (DXA; Lunar Prodigy, GE Healthcare, Belgium) in the Exercise Science Research Center at the University of Arkansas.

**Energy Expenditure and Substrate Oxidation.** REE (kcal/day) and energy expenditure (EE; kcal/day) were measured with a TrueMax 2400 metabolic cart (Parvomedics, Sandy, UT) via indirect calorimetry using the ventilation hood technique (47). Measurements were taken every 30 seconds during the data collection period in a relaxed, supine position. REE was measured in the fasted stated at 0 minutes for a 30-minute collection period. Only the last 20 minutes of data collection was used for analysis. TEF was calculated from EE measured at 30, 60, 120, 180, and 240 minutes for a 20-minute collection period minus the baseline REE measurement. Only the last 15 minutes of data collection was used for analysis. Respiratory
quotient (48) was determined based on the ratio of oxygen inhalation to carbon dioxide exhalation; values were used to define substrate oxidation.

**Sample Analysis.** Blood draws were performed by licensed phlebotomists. Blood was collected in EDTA vacutainer tubes (10ml/tube/timepoint). Blood samples were collected at 0, 30, 60, 120, 180, 240 minutes following breakfast consumption. Immediately following collection, samples were centrifuged at 4°C for 10 minutes at 1800 x g. The plasma was extracted and aliquoted into sterile 2 ml cryovial tubes (Corning, Tewksbury, MA) and stored at -80°C for future analysis. Glucose was measured using a clinical analyzer (Randox Laboratories Ltd, Kearneysville, WV) at University of Arkansas for Medical Sciences. Cholecystokinin (CCK) was measured using a commercially available enzyme immunoassay kit (RayBiotech, Peachtree Corners, GA). Leucine was measured using a commercially available amino acid kit (Phenomenex EZ:faast, Torrance, CA) and Shimadzu QP-2010 GCMS (Shimadzu Scientific Instruments, Columbia, MD).

**Appetite and Palatability.** Appetite and palatability were assessed using a 100mm visual analog scale (VAS) with opposing anchors (i.e. “very hungry” or “not at all hungry”) (49). Appetite was assessed at 0, 15, 30, 60, 90, 120, 180, and 240 minutes using a series of 7 questions. Participants were asked to indicate how hungry, how full, how strong their desire to eat, how much food they could eat, desire for something salty, desire for something sweet, and their desire for a snack. Palatability was assessed at 15 minutes after consuming the test breakfast.

**Dietary Assessment.** The 24-hour food records from day 1 and day 42 of the intervention were analyzed using the Nutrition Data System for Research software (NDSR; NDS version 2018, Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN) to determine average energy and macronutrient intake.

**Statistical Analysis.** Summary statistics were calculated and reported as sample means and standard deviation. Net incremental area under the curve (niAUC) was calculated.
for energy expenditure, substrate oxidation, appetite ratings, and plasma markers. Paired and unpaired t tests were used to analyze initial differences between day 1 and day 42 measurements. One-way ANOVA was used to analyze differences in day 1 versus day 42 within diet groups. Two-way ANOVA were used to compare differences between day 1 and day 42 between diet groups. Results are reported as means ± standard deviation. All analyses were conducted using GraphPad Prism Software, version 8.3.1. Statistical significance was defined as $P < 0.05$.

Results

**Participant Characteristics.** Participant characteristics are presented in Table 1. There was no significant difference in age, height, weight, BMI, BMI percentile, fat mass, lean mass, or fat free mass between diet groups.

**Energy Expenditure and Substrate Oxidation.** Energy expenditure, substrate oxidation, and respiratory quotient in response to the breakfast shake over 240 minutes (line graphs) and net incremental area under the curve (niAUC; bar graphs) on day 1 and day 42 are presented in Figure 3. After controlling for body weight (kg), there was a significant effect of postprandial response time (time, $P < 0.05$) and time x diet intervention interaction ($P < 0.01$) on energy expenditure. There was a significant effect of time ($P < 0.0001$), diet intervention ($P < 0.01$) and time x diet intervention interaction ($P < 0.0001$) on fat oxidation. PRO on day 1 of the intervention had significantly higher fat oxidation than CHO on day 42 of the intervention ($P < 0.01$). There was also a significant effect of time ($P < 0.0001$), diet intervention ($P < 0.01$) and time x diet intervention interaction ($P < 0.001$) on carbohydrate oxidation. PRO had a significant decrease in carbohydrate oxidation on day 1 ($P < 0.01$) and on day 42 ($P < 0.05$) of the intervention compared to CHO on day 42. There was no effect of diet on thermic effect of food.

**Appetite and Palatability.** There was no significant difference in palatability between diet groups on day 1 or day 42 of the intervention, however, there was a significant decrease in
palatability within the PRO group from day 1 to day 42 of the intervention (Table 2). Figure 4 presents results for perceived hunger, perceived fullness, perceived desire to eat, and prospective food consumption in response to the breakfast shake over 240 minutes (line graphs) and niAUC (bar graphs) on day 1 and day 42. There was a significant effect of time ($P < 0.0001$) on perceived hunger, perceived fullness, perceived desire to eat, and prospective food consumption. There was no significant effect of diet intervention or time x diet intervention interaction on perceived hunger, perceived fullness, perceived desire to eat, or prospective food consumption.

**Plasma Biomarkers.** Glucose and CCK in response to the breakfast shake over 240 minutes (line graphs) and niAUC (bar graphs) on day 1 and day 42 of the intervention are presented in Figure 5. There was a significant effect of time ($P < 0.0001$) and time x diet intervention interaction ($P < 0.05$) on plasma glucose. There was no significant effect of diet intervention on plasma glucose. There was a significant effect of time ($P < 0.0001$) and diet intervention ($P < 0.05$) on plasma CCK. There was no significant effect of time x diet intervention interaction on plasma CCK.

**Plasma Leucine.** Figure 6 presents plasma leucine in response to the breakfast shake over 240 minutes (line graph) and niAUC (bar graph) on day 1 and day 42 of the intervention. There was a significant effect of time ($P < 0.0001$) and diet intervention ($P < 0.01$) on plasma leucine. There was no significant effect of time x diet intervention interaction on plasma leucine.

**Energy Intake.** Average 24-hour energy and macronutrient intake on day 1 and day 42 of the intervention is presented in Table 3. There was no significant difference in total energy (kcal), carbohydrate (g), or fat (g) intake between diet groups on day 1 or day 42. There was no significant difference in protein (g) intake on day 1 between groups. PRO had a significantly higher intake of protein (g) on day 42 compared to CHO ($P < 0.01$).
Discussion

To our knowledge, this is the first study to examine the role of higher protein intake at breakfast for 6-weeks on postprandial energy expenditure and appetite in children ages 7-17 years old. Our results indicate that the consumption of a higher protein breakfast (30 g protein, 31 g carbohydrate, 11 g fat) compared to a higher carbohydrate breakfast (13 g protein, 48 g carbohydrate, 11 g fat) led to an increase in fat oxidation following the breakfast meal. There was no effect of diet on appetite or energy intake, despite PRO having a significantly higher plasma CCK following the breakfast meal. Collectively, this data suggests that children who consume a higher protein breakfast, compared to a carbohydrate breakfast, for 6-weeks may have increased fat oxidation and increased CCK following the meal; however, an overall effect on postprandial meal response is not observed. Therefore, a longer intervention period is needed to determine if a higher protein breakfast could potentially serve as a method for improving energy balance in children.

It is well understood the beneficial effects higher protein diets can have on improving body composition (29, 50-53), improving glycemic control (13, 54-58), improving appetite (32, 59, 60), and regulating energy balance (31, 61-65) in adults. However, limited research on the effects of high protein diets have been done in children and adolescents. A majority of studies that have been done regarding breakfast intake have focused on its effects on glycemic control and appetite in breakfast skipping children and adolescents (30, 36, 37, 39, 40); whereas this study focuses on children and adolescents who already regularly consume breakfast. In this study, PRO was composed of 30 grams of protein compared to CHO which consisted of only 13 grams of protein. This aligns similarly with previous dietary intervention studies (30, 37, 39, 40).

Although the results from this study show perceived hunger is decreased and perceived fullness and plasma CCK are increased following the PRO meal, no change in total energy intake was observed throughout the remainder of the day. This may be due to appetite levels returning back to baseline after 240 minutes, or complete digestion, following the meal.
consumption. Compared to CHO, PRO had increased postprandial fat oxidation, which has been shown to be associated with long-term weight changes (66). At 30 minutes postprandial, plasma glucose is blunted in PRO versus CHO, despite both groups returning back to baseline at 240 minutes postprandial. This aligns with previous observations that a diet higher in protein can aid in glycemic control (35, 36, 42, 55, 56, 58). Leucine has been shown to influence lipid and glucose metabolism (67); therefore, the increase in plasma leucine of PRO compared to CHO may explain the increase in fat oxidation and regulation of glucose metabolism after PRO.

To our knowledge, this is the first study demonstrating the postprandial effect of a PRO versus CHO breakfast on energy expenditure, appetite, and markers of metabolism in breakfast eating 7-17 year old children for 6-weeks. Previous studies have looked at longer intervention periods (i.e. 12-weeks), however they have focused on breakfast skipping adolescents (36, 39). Additionally, after 12-weeks, these previous studies have mostly observed differences between breakfast skippers and high-protein breakfasts, but not between normal- and high-protein breakfast; suggesting that the effect of protein intake at breakfast dissipates with adaptation to regular breakfast consumption habits. Therefore, future studies should aim at further understanding the long-term effects of higher protein breakfasts in regular breakfast consumers. In a previously published paper, there was a reduced effect of diet on postprandial energy expenditure in normal weight compared to overweight/obese children (42). A limitation of this study is that, while both normal weight and overweight/obese children were recruited, the average BMI percentile of this study is normal weight for both PRO and CHO groups, which may account for the lack of diet effects observed. A second limitation is that fiber content was low in each of the breakfasts so that any effects observed could be attributed to the protein or carbohydrate within the diet; however, research shows that a low glycemic profile may be responsible for appetite and glucose regulation (68, 69). Participants recruited for this study had to meet strict inclusion and exclusion criteria. Compliance was closely monitored, and the various shake flavors provided as well is believed to have helped aid with compliance.
throughout the intervention. As well, the observed levels of plasma leucine in PRO versus CHO suggest compliance was upheld by participants. The sample size was small and not diverse, therefore limiting its potential application to other populations. Although subjects were provided with measuring cups, measuring spoons and food scales to improve accuracy, the study did rely on self-reported 24-h dietary intake records and therefore may have provided inaccurate measurements of energy intake (70).

In conclusion, compared to a CHO, PRO increased postprandial fat oxidation and postprandial fullness. PRO also showed improved postprandial glucose regulation and increased plasma CCK. These data suggest that increasing protein intake at breakfast for 6-weeks does not change the postprandial meal response in 7-17 year old children who regularly consume breakfast. Therefore, additional research is still needed to determine effective methods for combatting obesity.
REFERENCES


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42. Baum JI, Gray M, Binns A. Breakfasts Higher in Protein Increase Postprandial Energy Expenditure, Increase Fat Oxidation, and Reduce Hunger in Overweight Children from 8 to 12 Years of Age. J Nutr. 2015;145:2229-35.


Inquires Received  
(n = 250)

Phone Screenings  
(n = 114)

Qualified & Randomized  
(n = 34)

Met one or more exclusion criteria  
(n = 56)

Qualified but no longer interested  
(n = 24)

Enrolled in PRO  
(n = 19)

Enrolled in CHO  
(n = 16)

Completed PRO Intervention  
(n = 13)

Completed CHO Intervention  
(n = 11)

Male  
(n = 7)

Female  
(n = 6)

Male  
(n = 5)

Female  
(n = 6)

Dropout  
(n = 6)

Dropout  
(n = 5)

**Figure 1.** Flowchart visualizing recruitment, screening and enrollment process of the study intervention. Inquiries were received via phone & email. CHO, carbohydrate-based breakfast intervention; PRO, protein-based breakfast intervention.
Figure 2. Study day timeline for day 1 and day 42 of the intervention. Subjects arrived fasted 8-10 hours at the Center for Human Nutrition to complete their laboratory visit. Resting energy expenditure at baseline, energy expenditure, and substrate oxidation were measured using indirect calorimetry. Baseline indirect calorimetry, appetite, and blood draw were measured and collected before consumption of the breakfast shake. Indirect calorimetry was measured at 30, 60, 120, 180, and 240 minutes postprandial. Blood was drawn at 30, 60, 120, 180, and 240 minutes postprandial. Appetite was assessed via visual analog scale (VAS) at 15, 30, 60, 90, 120, 180, and 240 minutes postprandial.
Figure 3. Energy expenditure and substrate oxidation after consumption of the breakfast shake. (A) Energy expenditure and niAUC after controlling for body weight on day 1 and day 42 of the intervention. (B) Fat oxidation and niAUC on day 1 and day 42 of the intervention. (C) Carbohydrate oxidation and niAUC on day 1 and day 42 of the intervention. (D) Respiratory quotient on day 1 and day 42 of the intervention. Means without a common letter are statistically significant ($P < 0.05$). CHO, carbohydrate-based breakfast; niAUC, net incremental area under the curve; ns, not significant; PRO, protein-based breakfast.
Figure 4. Ratings of perceived appetite assessment using visual analog scales after consumption of the breakfast shake. (A) Perceived hunger over time and niAUC on day 1 and day 42 of the intervention. (B) Perceived fullness over time and niAUC on day 1 and day 42 of the intervention. (C) Perceived desire to eat over time and niAUC on day 1 and day 42 of the intervention. (D) Percieved prospective food consumption over time and niAUC on day 1 and day 42 of the intervention. CHO, carbohydrate-based breakfast; niAUC, net incremental area under the curve; ns, not significant; PRO, protein-based breakfast.
Figure 5. Changes in plasma biomarkers after consumption of the breakfast shake. (A) Glucose changes over time and niAUC on day 1 and day 42 of the intervention. (B) Changes in cholecystokinin and niAUC on day 1 and day 42 of the intervention. (C) Changes in plasma leucine on day 1 and day 42 of the intervention. Means without a common letter are statistically significant \(P < 0.05\). CHO, carbohydrate-based breakfast; niAUC, net incremental area under the curve; ns, not significant; PRO, protein-based breakfast.
Figure 6. Changes in plasma leucine after consumption of the breakfast shake. Means without a common letter are statistically significant ($P < 0.05$). CHO, carbohydrate-based breakfast; niAUC, net incremental area under the curve; ns, not significant; PRO, protein-based breakfast.
### Table 1
**Baseline participant characteristics by diet**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>PRO</th>
<th>CHO</th>
<th>p-value</th>
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<tbody>
<tr>
<td><strong>Total</strong></td>
<td>13</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Female, n</td>
<td>6</td>
<td>6</td>
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<tr>
<td>Male, n</td>
<td>7</td>
<td>5</td>
<td></td>
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<tr>
<td><strong>Age, years</strong></td>
<td>11.8 ± 2.2</td>
<td>12.0 ± 2.5</td>
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<tr>
<td><strong>Anthropometrics</strong></td>
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<tr>
<td>Height, cm</td>
<td>156.2 ± 15.3</td>
<td>154.2 ± 11.9</td>
<td>0.8310</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>59.8 ± 30.0</td>
<td>50.7 ± 13.0</td>
<td>0.8755</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>21.8 ± 6.9</td>
<td>21.1 ± 4.6</td>
<td>0.7881</td>
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<tr>
<td>BMI Percentile, %</td>
<td>64 ± 33</td>
<td>72 ± 19</td>
<td>0.6183</td>
</tr>
<tr>
<td><strong>DEXA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat Mass, kg</td>
<td>17.4 ± 15.0</td>
<td>12.9 ± 9.3</td>
<td>0.5691</td>
</tr>
<tr>
<td>Lean Mass, kg</td>
<td>35.0 ± 9.9</td>
<td>35.1 ± 7.7</td>
<td>0.9095</td>
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<tr>
<td>Fat-free Mass, kg</td>
<td>37.0 ± 10.6</td>
<td>36.9 ± 8.0</td>
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<tr>
<td><strong>Ethnicity</strong></td>
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</tr>
<tr>
<td>Other</td>
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<td>0</td>
<td></td>
</tr>
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*Values are means ± standard deviation. PRO, protein-based breakfast group; CHO, carbohydrate-based breakfast group; BMI, body mass index; DEXA, dual energy x-ray absorptiometry.*
<table>
<thead>
<tr>
<th></th>
<th>PRO</th>
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</thead>
<tbody>
<tr>
<td>Energy content, kcal</td>
<td>360</td>
<td>360</td>
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<tr>
<td>Total protein, g</td>
<td>30</td>
<td>13</td>
</tr>
<tr>
<td>Total carbohydrate, g</td>
<td>31</td>
<td>48</td>
</tr>
<tr>
<td>Total fat, g</td>
<td>11.7</td>
<td>11.7</td>
</tr>
<tr>
<td>Total fiber, g</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Breakfast Palatability&lt;sup&gt;2&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 1, mm</td>
<td>89.2 ± 12.7</td>
<td>76.7 ± 20.2</td>
</tr>
<tr>
<td>Day 42, mm</td>
<td>56.0 ± 37.9</td>
<td>68.7 ± 22.4</td>
</tr>
</tbody>
</table>

<sup>1</sup> PRO, protein-based breakfast; CHO, carbohydrate-based breakfast. Values are means ± standard deviation. <sup>2</sup> Units are in millimeters according to a traditional 100 mm visual analog scale. PRO within group difference (P< 0.01) from day 1 to day 42. No between group differences were observed on day 1 or day 42.
Table 3
Average 24-hour energy and macronutrient intake on day 1 and day 42 of intervention.

<table>
<thead>
<tr>
<th></th>
<th>PRO</th>
<th>CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 42</td>
</tr>
<tr>
<td><em>n</em></td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Energy, kcal</td>
<td>1958 ± 824</td>
<td>2079 ± 793</td>
</tr>
<tr>
<td>Protein, g</td>
<td>95.5 ± 38.2</td>
<td>92.5 ± 27.8&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Carbohydrate, g</td>
<td>220.6 ± 94.9</td>
<td>240.5 ± 69.9</td>
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<tr>
<td>Fat, g</td>
<td>78.8 ± 45.9</td>
<td>85.2 ± 53.7</td>
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</table>

<sup>1</sup> Values are mean ± standard deviation or n. PRO, protein-based breakfast group; CHO, carbohydrate-based breakfast group. Data obtained from 24-hour weighed dietary intake records. <sup>2</sup> Letters indicates significant difference between protein intake on day 42 between PRO and CHO groups (*P* < 0.01). Data obtained from 24-hour weighed dietary intake records. *P* value calculated based on Welch’s t test.
CHAPTER 2

Effects of Higher Protein versus Higher Carbohydrate Breakfast for 6-weeks in Overweight Children Ages 7-17 Years Old

Abstract

Objective: The purpose of this study was to determine if a higher protein breakfast compared to a normal protein breakfast improves resting energy expenditure, resting substrate oxidation, markers of metabolic health, and dietary intake after a 6-week dietary adaptation in normal weight and overweight/obese school-aged children.

Methods: This study was a 6-week, double-blind, randomized controlled design dietary intervention in 7-17 year-old children. A total of 71 participants (female and male) completed the study. Participants were classified as either normal weight (NW) or overweight/obese (OW) and then randomly assigned to either a protein-based breakfast (PRO; 30 g protein; NW PRO, n=19; OW PRO, n=18) or carbohydrate-based breakfast (CHO; 13 g protein; NW CHO, n=16; OW CHO, n=18). Anthropometrics, resting energy expenditure, resting substrate oxidation, and markers of metabolic health were collected Pre (day 1)- and Post (day 42)-dietary intervention. Energy intake was also collected at baseline, Pre- and Post-dietary intervention.

Results: There were significant differences in REE between NW PRO and OW PRO at Pre- (P < 0.001) and Post-intervention (P < 0.01). There were no significant differences within or between groups for plasma glucose, cholesterol, and total protein. There was no significant difference in total energy intake for all groups from baseline to post-intervention.

Conclusions: Increasing protein intake at breakfast for 6-weeks does not have an overall effect on energy expenditure, metabolic marker, or energy intake adaptation change in NW versus OW 7-17 year old children who regularly consume breakfast. Approval for this study was obtained by the Institutional Review Board at the University of Arkansas for Medical
Introduction

The rising rate of obesity is a worldwide public health concern with a rate that has tripled since 1975 (1, 2). It is estimated that approximately one in every five children between the ages of 2-19 years old is overweight or obese (3). While obesity is often described simply as excess body fat (4, 5), its effects go far beyond physical implications. Obesity increases the risk of developing chronic diseases including metabolic syndrome, type 2 diabetes, cardiovascular disease, polycystic ovarian syndrome (PCOS), and psychological problems leading to an increased chance of premature morbidity (6-9). Although many factors contribute to the onset of obesity (10, 11), the balance between energy intake and energy expenditure (i.e. energy balance) is a critical nutritional focal point for prevention and treatment of obesity (12, 13).

Regulation of energy balance is largely influenced by the macronutrient composition of the diet (14). Increasing protein intake has been suggested as a potential approach towards preventing obesity and therefore improving biomarkers of metabolic health (15). Research has shown that protein intake beyond the recommended dietary allowance (RDA) is a key driver in maintaining muscle mass for better body composition and improving overall health in aging adults (16-18). Resting metabolic rate has been found to be correlated with energy intake and appetite in overweight and obese individuals (19). Higher protein diets have also been shown to maintain resting energy expenditure (REE), increase thermic effect of food (TEF) following the meal, and decrease hunger and increase fullness for improved satiety; thus regulating appetite and subsequent energy intake (15).

Studies have found that consuming higher protein breakfasts (30% of energy intake) compared to skipping breakfast or a normal/lower protein (15% of energy intake) breakfast led
to reductions in subsequent energy intake and reductions in perceived appetite following the breakfast meal in adolescents (20-22).

The beneficial health effects of a higher protein diet are well understood in adults; however, a gap remains in the scientific literature to adequately understand the effect of higher protein intake on normal weight (NW) and overweight/obese (OW) children. Therefore, the objective of this study was to determine if consuming a protein-based breakfast for 6-weeks would have an adaptation effect on resting energy expenditure, markers of metabolic health, and energy intake in NW versus OW compared to a carbohydrate-based breakfast. We hypothesized that consuming a protein-based breakfast compared to a carbohydrate-based breakfast for 6 weeks would increase resting energy expenditure and substrate oxidation, improve markers of metabolic health, and decrease energy intake in OW compared to NW school-aged children.

Methods

Participants and Screening. NW and OW male and female children between 7-17 years of age were recruited to participate in the study. Participants were recruited through the University of Arkansas newsletter, local website blogs, local after-school camps, and flyers. An initial phone screening was conducted with the parents or legal guardians of interested participants to determine if they met the inclusion and exclusion criteria. Participants who did not reside in Northwest Arkansas, had a BMI < 5th percentile, regularly skipped breakfast (> 5 times per week), regularly consumed protein at breakfast (>25 grams of protein at breakfast > 4 time per week), were classified as a picky eater by their parent/guardian, had allergies or dietary restrictions, had a fear of needles, were claustrophobic, or were on prescription medication were excluded from the study. A total of 71 subjects completed the study. Eighteen subjects dropped out due to lack of protocol compliance or inability to collect blood samples. A diagram of the recruitment, screening and enrollment process can be found in Figure 1.
**Study Design.** Parents and legal guardians provided written consent and children provided written assent prior to enrolling in the study. Following an initial phone screening, participants were classified as either NW or OW based on their BMI-for-age percentile (23). A double-blinded randomized, controlled study design was used to assign participants (n=71) to either the protein-based breakfast (PRO; 30 g protein) or carbohydrate-based test breakfast (CHO; 13 g protein) for six weeks (Table 1). To aid with compliance, parents and participants were provided with a welcome booklet upon enrollment into the study. The welcome booklet contained their study schedule, dietary food record examples and reference sheets (i.e. estimating portion size with your hands), and instructions for consuming the breakfast shake. All participants underwent anthropometric measurements and a body composition scan at baseline and at the end of the dietary intervention period (Tables 2, 3). Participants completed two laboratory visits at the Center for Human Nutrition at the University of Arkansas (Fayetteville, AR) on day 1 (Pre) and day 42 (Post) of the dietary intervention. Participants were instructed to fast for 8-10 hours overnight and avoid any vigorous physical activity the day before their laboratory test day. Participants were provided with a 24-hour dietary intake record to complete prior to the start of the intervention to assess baseline assessment of energy and macronutrient intake. Upon arrival on day 1 of the dietary intervention, height and weight were collected. A fasted blood draw and resting energy expenditure (REE) were then collected. Participants were given their assigned test breakfast (PRO or CHO) to consume. At the conclusion of the first test day, participants were provided with 21 days of test beverages. Participants were instructed to consume the test beverage daily prior to 10:00am. Participants were also provided with a 3-day food intake record to complete (2 weekdays, 1 weekend) during the first week of the intervention. To improve reporting accuracy, participants were given measuring cups, measuring spoons, and a food scale. After 3 weeks, parents/guardians returned to the Center for Human Nutrition on behalf of the participants to pick up the remaining 21-day supply of test beverages and a second 3-day food intake record to complete during
week 6 of the intervention. Parents and legal guardians returned with participants to complete another laboratory test day on the last day of the intervention (day 42). To help increase compliance throughout the study, parents and participants were provided with a welcome booklet upon enrollment into the study. The welcome booklet contained their study schedule, dietary food log examples and reference sheets (i.e. estimating portion size with your hands), and instructions for consuming the breakfast shake. Approval for the study was obtained by the Institutional Review Board at the University of Arkansas for Medical Sciences (IRB Protocol # 207201; Little Rock, AR) and registered at ClinicalTrials.gov as NCT03602144.

**Test Breakfasts.** Participants were randomly assigned to one of two breakfast interventions: protein-based breakfast shake (PRO; 30 g protein, 31 g carbohydrate, 11 g fat) or carbohydrate-based breakfast shake (CHO; 13 g protein, 48 g carbohydrate, 11 g fat). Participants were instructed to consume the test breakfast each day prior to 10:00am (24) throughout the intervention. To prevent boredom and to help with compliance, participants were provided three flavor options to select from including chocolate, strawberry and vanilla. All PRO and CHO shakes were isocaloric and matched for fat and fiber (**Table 5**).

**Anthropometrics.** Body height was measured barefoot in free-standing position to the nearest 0.01 cm using a stadiometer (Detecto, St. Louis, MO). Body weight was measured in the fasted stated to the nearest 0.01 kg using a calibrated weigh beam eye level scale (Detecto, St. Louis, MO). BMI was calculated as weight (kg) divided by height squared (m²). BMI-for-age percentile was calculated using the Center for Disease Control’s (CDC) BMI Percentile Calculator for Child and Teen (23). Body composition was assessed at the Human Performance Laboratory at the University of Arkansas using dual energy x-ray absorptiometry (DXA; Lunar Prodigy, GE Healthcare, Belgium).

**Energy Expenditure and Substrate Oxidation.** REE (kcal/day) was measured with a TrueMax 2400 metabolic cart (Parvomedics, Sandy, UT) via indirect calorimetry using the ventilation hood technique (25). Measurements were taken every 30 seconds during the data
collection period in a relaxed, supine position. REE was measured in the fasted state at 0 minutes for a 30-minute collection period. Only the last 20 minutes of data collection was used for REE and substrate oxidation (fat and carbohydrate) analysis.

**Blood Collection and Biomarkers.** Blood draws were performed by licensed phlebotomists. Blood was collected in EDTA vacutainer tubes (10ml/tube; 20ml total). Blood samples were collected in the fasted state prior to consuming the test breakfast. Immediately following collection, samples were centrifuged at 4ºC for 10 minutes at 1800 x g. The plasma was extracted and aliquotted into sterile 2 ml cryovial tubes (Corning, Tewksbury, MA) and stored at -80ºC for future analysis. Glucose (mmol/l), triglycerides (mmol/l), cholesterol (mmol/l), creatinine (mg/dl), and total plasma protein (g/l) were measured using a clinical analyzer (Randox Laboratories Ltd, Kearneysville, WV) at the University of Arkansas for Medical Sciences (Little Rock, AR).

**Dietary Assessment.** The 24-hour baseline record and 3-day food records were analyzed using the Nutrition Data System for Research software (NDSR; NDS version 2018, Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN) to determine average energy and macronutrient intake.

**Statistical Analysis.** Summary statistics were calculated and reported as sample means and standard deviation. Wilcoxon non-parametric paired t-tests were used to compare within-group differences between Pre- and Post-intervention. One-way ANOVA was used to analyze differences in day 1 versus day 42 between intervention groups. Results are reported as means ± standard deviation. All analyses were conducted using GraphPad Prism Software, version 8.3.1. Statistical significance was defined as $P < 0.05$.

**Results**

**Participant Physical Characteristics.** Participant characteristics are presented in Tables 2, 3, and 4. There was no significant difference in height, weight, BMI, BMI percentile,
fat mass, lean mass, and fat free mass Pre- and Post-intervention within groups. There were no significant differences between groups for changes in weight, BMI, or fat mass. Compared to OW CHO, NW PRO ($P < 0.01$) and NW CHO ($P < 0.05$) had a significantly greater change in BMI percentile. NW PRO ($P < 0.01$) and NW CHO ($P < 0.05$) had a significantly lower change in fat free mass compared to OW CHO.

**Energy Intake.** Average energy and macronutrient intake at baseline, Pre- (week 1) and Post (week 6) of the intervention are presented in Table 6. There was no significant difference within group for all groups for total energy intake and fat intake from baseline to Post-intervention. There was a significant difference within group in protein intake from baseline to Post-intervention for NW PRO, NW CHO, OW CHO ($P < 0.05$) and OW PRO ($P < 0.01$). There was no significant difference in carbohydrate intake within group for NW PRO, OW PRO, and OW CHO. There was a significant difference between baseline and Post-intervention carbohydrate intake for NW CHO ($P < 0.05$).

There was no significant difference between groups for energy intake, carbohydrate intake, or fat intake at baseline, Pre- or Post-intervention. There was no significant difference between groups for protein intake, at baseline or Pre-intervention. There was a significant difference in protein intake between OW PRO and NW CHO ($P < 0.01$) and OW CHO ($P < 0.01$) Post-intervention. NW PRO had a significant decrease in energy intake compared to all other groups. OW PRO had a significant increase in protein intake compared to all other groups. NW PRO had a significant decrease in fat intake and carbohydrate intake compared to all other groups.

**Resting Energy Expenditure and Substrate Oxidation.** As shown in Table 7, there were no significant differences within groups for REE, fat or carbohydrate oxidation. There was no significant difference between groups for the change in fat or carbohydrate oxidation at Pre-, Post, or change from Pre- to Post intervention ($\Delta$). There was a significant difference in REE before controlling for body weight Pre-intervention between NW PRO and OW PRO ($P < 0.001$),
NW PRO and OW CHO ($P < 0.05$), and NW CHO and OW PRO ($P < 0.01$). There was a significant difference in REE before controlling for body weight Post-intervention between NW PRO and OW PRO ($P < 0.01$), NW PRO and OW CHO ($P < 0.01$), NW CHO and OW PRO ($P < 0.01$), and NW CHO and OW CHO ($P < 0.01$). After controlling for body weight, there were significant differences in REE between the NW and OW diet groups Pre-intervention. There was no significant difference between groups for the change in REE, resting fat oxidation, or resting carbohydrate oxidation from Pre- to Post intervention ($\Delta$).

**Plasma Biomarkers.** Concentrations of plasma biomarkers Pre- and Post-intervention are presented in Table 8. There was no significant within group differences from Pre- to Post-intervention ($\Delta$) in all groups for plasma glucose, cholesterol, creatinine, or triglycerides. There were no significant differences for total protein within NW PRO, NW CHO, or OW PRO; however, there was a significant difference within OW CHO ($P < 0.05$). There were no significant differences between groups for changes ($\Delta$) in plasma glucose, cholesterol, creatinine, total protein, and triglycerides. There was no significant difference between groups for plasma glucose, cholesterol, and total protein at Pre- or Post-intervention. Pre-intervention (day 1), there was a significant difference in plasma triglycerides between NW PRO and OW CHO ($P < 0.05$). Post intervention (day 42), there was a significant difference in plasma triglycerides between OW CHO and NW PRO ($P < 0.05$), and NW CHO and OW CHO ($P < 0.05$). There was no significant difference in plasma creatinine Pre-intervention between NW CHO and OW PRO ($P < 0.05$). There was no significant difference in plasma creatinine between groups Post-intervention.

**Discussion**

To our knowledge, this is the first study to examine the effect of a PRO breakfast, compared to a CHO breakfast, for 6-weeks on energy expenditure, markers or metabolic health, and appetite in NW and OW 7-17 years old children. Our results indicate that PRO compared to
a CHO breakfast does not lead to adaptive changes in REE, resting substrate oxidation, energy intake, or plasma glucose within groups after 6-weeks of consumption. This data suggests that in OW children who already regularly consume breakfast, a 6-week dietary PRO intervention is not of sufficient duration for potential metabolic changes. Therefore, additional research is necessary to determine effective nutritional interventions that might lead to metabolic changes in OW children and help combat obesity.

In adults, higher protein diets have been found to promote weight loss (26, 27), preserve body composition (28, 29), and regulate energy balance (30-32) and circulating glucose concentration (33, 34). However, these effects of higher protein diets are lesser known in children, especially those that are OW. In the current study, OW PRO continued to have higher REE at Pre- and Post-intervention compared to OW PRO, however no differences were observed in resting substrate oxidation between the groups. Previously published data aligns with this, in which OW participants also had higher EEs compared to NW (35).

While other studies have looked at the effect of a low-glycemic diet in regulating blood glucose of overweight and obese children with type 2 diabetes (36), this study looks at the effect of high protein breakfast in healthy overweight and obese children. Despite the lack of evidence this study provides for changes in blood glucose among any of the groups, research suggests that the macronutrient composition of breakfast is an important factor in plasma glucose regulation (37). Given that there was no change observed in whole-diet carbohydrate intake for each group from the start to the end of the intervention, this may have mitigated any effect the breakfast had on improving blood glucose levels.

To our knowledge, this is the first study to look at the effects of PRO versus CHO intake for 6-weeks on REE, resting substrate oxidation, energy intake, and markers of metabolic health in both normal and overweight children ages 7-17 years old. Although some previous studies have been conducted in children and adolescents, they have been focused either in breakfast skippers (20-22, 38-40), or for shorter (crossover design or one-time testing days) (21, 22, 35)
and longer (i.e. 12-weeks) (38, 40) intervention periods. Although no changes in body composition was observed within the intervention groups of this study, this may be attributed to too short of an intervention period given that diet-induced weight loss, and changes in fat mass and fat free mass associated with weight loss, require 4-6 months (41). However, the aim of this study was not to directly observe weight loss as an effect of PRO. Although recruitment aimed to balance genders between intervention groups, NW PRO was largely comprised of females and OW CHO was largely comprised of males. In addition, although a component of the screening process included puberty, tanner stages were not confirmed and steroid hormones such as estrogen and testosterone were not measured. Therefore, these could have served as confounding variables in energy intake or biomarker variations.

Another limitation of this study is that each of the breakfasts had a small amount of fiber. This was done intentionally so that any effects observed could be attributed to the protein or carbohydrate content within the breakfast composition. However, this low glycemic profile may have an effect on glucose regulation (42, 43). While participants in this study were required to meet strict inclusion and exclusion criteria, compliance was monitored throughout the intervention. The various shake flavors provided are believed to have helped with compliance. An added limitation of this study is that beyond the breakfast meal, no other dietary components were controlled. This may have been a contributing factor in limited observable effects between the diets and weight groups. Despite subjects being provided with tools to improve reporting accuracy (i.e. measuring cups, measuring spoons and food scales) the study relied on self-reported 24-h dietary intake records for estimating energy intake, and therefore may have led to inaccuracies (44). In addition, although the participant’s large age range (7-17 years old) fell within the CDC’s range for defining childhood (23), it could have also contributed to the lack of observational changes in body composition. This study administered a shake as the breakfast meal, however, a previously published study suggested that the form of the meal (solid versus beverage) can have an effect on ad libitum food intake following the meal (39). Therefore, future
research should focus on whether the breakfast meal form has the same effect on energy intake after consumption for a longer intervention period, or whether it also effects energy expenditure and plasma biomarkers. The study aimed to enroll an additional 10-20\% of participants as recommended for human dietary intervention studies (45) to account for the 20\% participant dropout within this study.

In conclusion, consumption of PRO compared to CHO for 6-weeks did not show an overall improvement in energy expenditure, energy intake, or plasma biomarkers in NW versus OW. Future research should focus on controlling energy intake of the whole diet, not just breakfast, and a longer intervention period. Collectively, additional research is needed to understand the effects of protein intake at breakfast in OW children.
REFERENCES


**FIGURES**

**Figure 1.** Flowchart visualizing recruitment, screening and enrollment process of the study intervention. Inquiries were received via phone & email. CHO, carbohydrate-based breakfast intervention; NW, normal-weight participants; OW, overweight and obese participants; PRO, protein-based breakfast intervention.
Table 1
Participant demographics by weight and diet group.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>19</td>
<td>16</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Female, n</td>
<td>5</td>
<td>8</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Male, n</td>
<td>14</td>
<td>8</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>Age, y</td>
<td>11.5 ± 2.5</td>
<td>11.1 ± 2.3</td>
<td>13.0 ± 2.2</td>
<td>12.9 ± 2.6</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>15</td>
<td>14</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Asian</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>African American</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic/Latino</td>
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<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Non-Hispanic/Latino</td>
<td>18</td>
<td>15</td>
<td>17</td>
<td>17</td>
</tr>
</tbody>
</table>

1 Values are expressed as means ± standard deviation. NW, normal weight subjects; OW, overweight/obese subjects; PRO, protein-based breakfast; CHO, carbohydrate-based breakfast.
Table 2

Participant physical characteristics pre-intervention by weight and diet group.\(^1\)

<table>
<thead>
<tr>
<th></th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometrics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>150.2±14.4</td>
<td>147.7±14.3</td>
<td>161.0±11.7</td>
<td>159.7±12.0</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>43.0±20.2</td>
<td>28.9±11.2</td>
<td>69.4±17.1</td>
<td>68.1±17.5</td>
</tr>
<tr>
<td>BMI, kg/m(^2)</td>
<td>17.3±2.1</td>
<td>17.4±1.9</td>
<td>26.5±4.6</td>
<td>26.3±4.0</td>
</tr>
<tr>
<td>BMI Percentile, %</td>
<td>43±21</td>
<td>47±23</td>
<td>93±5</td>
<td>93±6</td>
</tr>
<tr>
<td><strong>Body Composition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat Mass, kg</td>
<td>15.0±18.2</td>
<td>8.6±5.6</td>
<td>25.1±11.8</td>
<td>25.2±8.9</td>
</tr>
<tr>
<td>Lean Mass, kg</td>
<td>30.3±7.7</td>
<td>29.6±8.3</td>
<td>41.3±11.3</td>
<td>39.4±11.1</td>
</tr>
<tr>
<td>Fat-free Mass, kg</td>
<td>31.9±8.1</td>
<td>31.1±8.7</td>
<td>43.8±11.8</td>
<td>41.7±11.9</td>
</tr>
</tbody>
</table>

\(^1\) Values are expressed as means ± standard deviation. Values are day 1 data. CHO, carbohydrate-based breakfast; BMI, body mass index. NW, normal weight subjects; OW, overweight/obese subjects; PRO, protein-based breakfast.
Table 3  
*Participant physical characteristics post-intervention by weight and diet group*.

<table>
<thead>
<tr>
<th></th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometrics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>150.2±13.7</td>
<td>148.4±13.9</td>
<td>161.4±11.4</td>
<td>160.4±20.9</td>
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<tr>
<td>Weight, kg</td>
<td>40.1±10.9</td>
<td>40.0±11.5</td>
<td>71.2±17.1</td>
<td>26.3±4.0</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>17.5±2.3</td>
<td>17.7±2.1</td>
<td>27.1±4.6</td>
<td>25±6</td>
</tr>
<tr>
<td>BMI Percentile, %</td>
<td>48±20</td>
<td>51±25</td>
<td>94±4</td>
<td>93±6</td>
</tr>
<tr>
<td><strong>Body Composition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat Mass, kg</td>
<td>16.1±20.4</td>
<td>8.1±4.0</td>
<td>25.5±11.9</td>
<td>25.0±8.3</td>
</tr>
<tr>
<td>Lean Mass, kg</td>
<td>30.5±7.7</td>
<td>30.0±8.3</td>
<td>42.3±11.5</td>
<td>41.1±11.8</td>
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<tr>
<td>Fat-free Mass, kg</td>
<td>32.1±8.1</td>
<td>31.6±8.7</td>
<td>44.9±12.0</td>
<td>43.3±12.4</td>
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</table>

* Values are expressed as means ± standard deviation. Values are day 42 data. CHO, carbohydrate-based breakfast; BMI, body mass index. NW, normal weight subjects; OW, overweight/obese subjects; PRO, protein-based breakfast.
Table 4
Change in participant physical characteristics from pre- to post-intervention by weight and diet group\textsuperscript{1,2}.

<table>
<thead>
<tr>
<th></th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometrics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>-0.07±2.53</td>
<td>0.67±0.77</td>
<td>0.40±1.23</td>
<td>0.44±1.38</td>
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<tr>
<td>Weight, kg</td>
<td>0.41±2.17</td>
<td>1.33±0.71</td>
<td>1.89±1.45</td>
<td>-1.24±7.62</td>
</tr>
<tr>
<td>BMI, kg/m\textsuperscript{2}</td>
<td>0.16±0.84</td>
<td>0.35±0.32</td>
<td>0.61±0.63</td>
<td>-1.52±5.07</td>
</tr>
<tr>
<td>BMI Percentile, %</td>
<td>4.26±5.77\textsuperscript{a}</td>
<td>3.50±3.83\textsuperscript{a}</td>
<td>1.16±1.04\textsuperscript{ab}</td>
<td>0.17±1.58\textsuperscript{b}</td>
</tr>
<tr>
<td><strong>Body Composition</strong></td>
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</tr>
<tr>
<td>Fat Mass, kg</td>
<td>1.10±2.36</td>
<td>0.66±0.53</td>
<td>0.46±1.61</td>
<td>-0.25±1.73</td>
</tr>
<tr>
<td>Lean Mass, kg</td>
<td>0.19±0.58\textsuperscript{a}</td>
<td>0.47±0.58\textsuperscript{a}</td>
<td>1.06±1.47\textsuperscript{ab}</td>
<td>1.72±1.67\textsuperscript{b}</td>
</tr>
<tr>
<td>Fat-free Mass, kg</td>
<td>0.20±0.58\textsuperscript{a}</td>
<td>0.59±0.52\textsuperscript{a}</td>
<td>1.39±1.11\textsuperscript{ab}</td>
<td>1.59±1.47\textsuperscript{b}</td>
</tr>
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</table>

\textsuperscript{1} Values are expressed as means ± standard deviation. Values are change between day 1 and day 42 data. CHO, carbohydrate-based breakfast; BMI, body mass index. NW, normal weight subjects; OW, overweight/obese subjects; PRO, protein-based breakfast. \textsuperscript{2} No within group differences observed from pre- to post intervention for all measurements. Between group comparisons determined using Δ values and one-way ANOVA. Δ without a common letter are statistically significant (\( P < 0.05 \)).
Table 5
*Dietary characteristics of test breakfasts* 1.

<table>
<thead>
<tr>
<th></th>
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<th>CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy content, kcal</strong></td>
<td>360</td>
<td>360</td>
</tr>
<tr>
<td><strong>Total protein, g</strong></td>
<td>30</td>
<td>13</td>
</tr>
<tr>
<td><strong>Total carbohydrate, g</strong></td>
<td>31</td>
<td>48</td>
</tr>
<tr>
<td><strong>Total fat, g</strong></td>
<td>11.7</td>
<td>11.7</td>
</tr>
<tr>
<td><strong>Total fiber, g</strong></td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

1 Values are expressed as means ± standard deviation. PRO, protein-based breakfast; CHO, carbohydrate-based breakfast.

2 Units are in millimeters according to a 100 mm visual analog scale.
Table 6
Average energy and macronutrient intake at baseline, pre- and post-intervention. 1,2,3.

<table>
<thead>
<tr>
<th></th>
<th>Normal Weight (n=19)</th>
<th>Overweight/Obese (n=16)</th>
<th>Overweight/Obese (n=16)</th>
<th>Overweight/Obese (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy Intake, kcal</td>
<td>PRO</td>
<td>CHO</td>
<td>PRO</td>
<td>CHO</td>
</tr>
<tr>
<td>Baseline</td>
<td>2276.5 ± 1274.9</td>
<td>1756.3 ± 674.8</td>
<td>2043.5 ± 843.5</td>
<td>1712.6 ± 542.2</td>
</tr>
<tr>
<td>Pre</td>
<td>1849.1 ± 764.8</td>
<td>2046.8 ± 1231.8</td>
<td>2126.4 ± 789.4</td>
<td>2020.0 ± 1093.7</td>
</tr>
<tr>
<td>Post</td>
<td>2018.9 ± 782.8</td>
<td>2018.9 ± 782.8</td>
<td>2359.5 ± 714.7</td>
<td>2046.1 ± 672.5</td>
</tr>
<tr>
<td>Δ</td>
<td>-257.7 ± 492.1 a</td>
<td>262.6 ± 108.0 b</td>
<td>316.0 ± 129.0 b</td>
<td>333.5 ± 130.5 b</td>
</tr>
<tr>
<td></td>
<td>**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein Intake, g</td>
<td>PRO</td>
<td>CHO</td>
<td>PRO</td>
<td>CHO</td>
</tr>
<tr>
<td>Baseline</td>
<td>82.5 ± 55.3</td>
<td>63.8 ± 20.2</td>
<td>76.5 ± 37.7</td>
<td>60.6 ± 19.4</td>
</tr>
<tr>
<td>Pre</td>
<td>85.3 ± 31.3</td>
<td>79.7 ± 36.6</td>
<td>97.1 ± 38.3</td>
<td>73.4 ± 35.3</td>
</tr>
<tr>
<td>Post</td>
<td>84.6 ± 25.5 abc</td>
<td>73.0 ± 22.9 ac</td>
<td>105.9 ± 31.9 b</td>
<td>72.7 ± 29.7 c</td>
</tr>
<tr>
<td>Δ</td>
<td>2.1 ± 29.9 a, *</td>
<td>9.3 ± 2.7 a, *</td>
<td>29.4 ± 5.8 b, **</td>
<td>12.1 ± 10.4 ac, *</td>
</tr>
<tr>
<td>Carbohydrate Intake, g</td>
<td>PRO</td>
<td>CHO</td>
<td>PRO</td>
<td>CHO</td>
</tr>
<tr>
<td>Baseline</td>
<td>288.4 ± 169.7</td>
<td>205.9 ± 71.4</td>
<td>260.9 ± 111.4</td>
<td>220.6 ± 88.9</td>
</tr>
<tr>
<td>Pre</td>
<td>222.1 ± 100.5</td>
<td>255.0 ± 190.9</td>
<td>243.2 ± 88.5</td>
<td>248.8 ± 119.0</td>
</tr>
<tr>
<td>Post</td>
<td>222.6 ± 67.7</td>
<td>259.9 ± 111.1</td>
<td>282.7 ± 96.1</td>
<td>254.6 ± 87.0</td>
</tr>
<tr>
<td>Δ</td>
<td>-65.9 ± 102.0 a</td>
<td>54.0 ± 40.0 a</td>
<td>21.7 ± 15.2 b</td>
<td>34.0 ± 2.0 b</td>
</tr>
<tr>
<td>Fat Intake, g</td>
<td>PRO</td>
<td>CHO</td>
<td>PRO</td>
<td>CHO</td>
</tr>
<tr>
<td>Baseline</td>
<td>91.1 ± 49.9</td>
<td>77.7 ± 39.0</td>
<td>79.9 ± 35.9</td>
<td>67.1 ± 26.3</td>
</tr>
<tr>
<td>Pre</td>
<td>71.5 ± 33.9</td>
<td>81.3 ± 47.4</td>
<td>87.7 ± 44.4</td>
<td>84.3 ± 58.7</td>
</tr>
<tr>
<td>Post</td>
<td>73.5 ± 26.4</td>
<td>79.4 ± 38.4</td>
<td>92.3 ± 35.5</td>
<td>84.5 ± 36.1</td>
</tr>
<tr>
<td>Δ</td>
<td>-17.6 ± 23.6 a</td>
<td>1.7 ± 0.6 a</td>
<td>12.4 ± 4.0 bc</td>
<td>17.4 ± 9.8 c</td>
</tr>
<tr>
<td>Protein Intake, g/kgBW</td>
<td>PRO</td>
<td>CHO</td>
<td>PRO</td>
<td>CHO</td>
</tr>
<tr>
<td>Baseline</td>
<td>63.0 ± 53.9</td>
<td>45.5 ± 13.1</td>
<td>33.7 ± 21.2</td>
<td>28.3 ± 13.7</td>
</tr>
<tr>
<td>Pre</td>
<td>48.9 ± 15.8</td>
<td>53.9 ± 19.8</td>
<td>30.5 ± 13.5</td>
<td>33.3 ± 20.0</td>
</tr>
<tr>
<td>Post</td>
<td>48.8 ± 16.2</td>
<td>54.0 ± 15.8</td>
<td>35.4 ± 10.4</td>
<td>32.1 ± 11.7</td>
</tr>
</tbody>
</table>

1 Values expressed as mean ± standard deviation. CHO, carbohydrate-based breakfast; PRO, protein-based breakfast. Pre, week 1 of intervention; Post, week 6 of intervention. Baseline data obtained from 24-hour food record. Pre- and post data obtained from 3-day food records. Δ calculated as difference between baseline and post data. 2 Within group comparisons determined using Δ values and paired nonparametric t test. Upper case letters indicate within group differences. Differences were not calculated for protein intake (g/kgBW). Statistically significant Δ values within group indicated by asterisk. *, P < 0.05; **, P < 0.01. 3 Between group differences determined at baseline, pre- and post-breakfast using one-way ANOVA. Lower case letters indicate between group differences. Values without a common letter are statistically significant (P < 0.05).
### Table 7

*Resting energy expenditure and substrate oxidation pre- and post-intervention*<sup>1, 2</sup>.

<table>
<thead>
<tr>
<th></th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Resting Energy Expenditure, kcal/d</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>1386.9 ± 214.5&lt;sub&gt;a&lt;/sub&gt;</td>
<td>1424.3 ± 226.5&lt;sub&gt;ac&lt;/sub&gt;</td>
<td>1799.8 ± 437.8&lt;sub&gt;b&lt;/sub&gt;</td>
<td>1694.1 ± 274.3&lt;sub&gt;c&lt;/sub&gt;</td>
</tr>
<tr>
<td>Post</td>
<td>1422.1 ± 222.4&lt;sub&gt;a&lt;/sub&gt;</td>
<td>1395.8 ± 212.9&lt;sub&gt;ab&lt;/sub&gt;</td>
<td>1719.4 ± 304.3&lt;sub&gt;c&lt;/sub&gt;</td>
<td>1732.0 ± 271.8&lt;sub&gt;cd&lt;/sub&gt;</td>
</tr>
<tr>
<td>∆</td>
<td>35.2 ± 140.6</td>
<td>-28.5 ± 175.0</td>
<td>-54.0 ± 295.2</td>
<td>37.9 ± 161.3</td>
</tr>
<tr>
<td><strong>Resting Energy Expenditure, kcal/d/kgBW</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>36.2 ± 5.7&lt;sub&gt;a&lt;/sub&gt;</td>
<td>38.5 ± 8.3&lt;sub&gt;a&lt;/sub&gt;</td>
<td>26.5 ± 5.2&lt;sub&gt;b&lt;/sub&gt;</td>
<td>27.1 ± 6.5&lt;sub&gt;b&lt;/sub&gt;</td>
</tr>
<tr>
<td>Post</td>
<td>37.0 ± 7.6&lt;sub&gt;a&lt;/sub&gt;</td>
<td>36.7 ± 8.0&lt;sub&gt;a&lt;/sub&gt;</td>
<td>25.4 ± 5.4&lt;sub&gt;bc&lt;/sub&gt;</td>
<td>30.3 ± 14.8&lt;sub&gt;sc&lt;/sub&gt;</td>
</tr>
<tr>
<td>∆</td>
<td>0.8 ± 4.7</td>
<td>-1.8 ± 4.8</td>
<td>-4.0 ± 8.3</td>
<td>3.2 ± 12.2</td>
</tr>
<tr>
<td><strong>Resting Fat Oxidation, kcal/min</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>0.62 ± 0.22</td>
<td>0.59 ± 0.17</td>
<td>0.78 ± 0.28</td>
<td>0.73 ± 0.25</td>
</tr>
<tr>
<td>Post</td>
<td>0.56 ± 0.24</td>
<td>0.53 ± 0.21</td>
<td>0.69 ± 0.21</td>
<td>0.67 ± 0.26</td>
</tr>
<tr>
<td>∆</td>
<td>-0.06 ± 0.31</td>
<td>-0.06 ± 0.27</td>
<td>-0.06 ± 0.22</td>
<td>-0.06 ± 0.29</td>
</tr>
<tr>
<td><strong>Resting Carbohydrate Oxidation, kcal/min</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>0.34 ± 0.15</td>
<td>0.40 ± 0.11</td>
<td>0.47 ± 0.16</td>
<td>0.45 ± 0.20</td>
</tr>
<tr>
<td>Post</td>
<td>0.43 ± 0.21</td>
<td>0.44 ± 0.19</td>
<td>0.51 ± 0.22</td>
<td>0.54 ± 0.20</td>
</tr>
<tr>
<td>∆</td>
<td>0.09 ± 0.25</td>
<td>0.04 ± 0.20</td>
<td>0.02 ± 0.23</td>
<td>0.09 ± 0.28</td>
</tr>
</tbody>
</table>

| **Resting Respiratory Quotient** |                 |                |                 |                 |
| Pre                  | 0.81 ± 0.05      | 0.82 ± 0.03    | 0.81 ± 0.04     | 0.82 ± 0.05     |
| Post                 | 0.83 ± 0.05      | 0.84 ± 0.06    | 0.83 ± 0.05     | 0.83 ± 0.05     |
| ∆                   | 0.02 ± 0.0<sub>abc</sub> | 0.01 ± 0.03<sub>abc</sub> | 0.01 ± 0.0<sub>b</sub> | 0.02 ± 0.0<sub>c</sub> |

<sup>1</sup> Values expressed as mean ± standard deviation. CHO, carbohydrate-based breakfast; PRO, protein-based breakfast.

<sup>2</sup> Pre, week 1 of intervention; Post, week 6 of intervention. Baseline data obtained from 24-hour food record. Pre- and post-breakfast data obtained from 3-day food records. ∆ calculated as difference between baseline and post data. No within group differences observed from pre- to post intervention for all measurements. Between group differences determined using one-way ANOVA. Values without a common letter are statistically significant (P < 0.05).
Table 8
Plasma biomarkers pre- and post-intervention\textsuperscript{1,2,3}.

<table>
<thead>
<tr>
<th></th>
<th>NW PRO</th>
<th>NW CHO</th>
<th>OW PRO</th>
<th>OW CHO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mmol/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>5.38 ± 0.57</td>
<td>5.24 ± 0.67</td>
<td>5.48 ± 0.56</td>
<td>5.71 ± 0.48</td>
</tr>
<tr>
<td>Post</td>
<td>5.44 ± 0.45</td>
<td>5.29 ± 0.53</td>
<td>5.50 ± 0.34</td>
<td>5.67 ± 0.48</td>
</tr>
<tr>
<td>Δ</td>
<td>-0.23 ± 1.48</td>
<td>-0.03 ± 0.26</td>
<td>0.43 ± 1.50</td>
<td>-0.03 ± 0.47</td>
</tr>
<tr>
<td>Cholesterol, mmol/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>4.42 ± 0.85</td>
<td>4.53 ± 0.98</td>
<td>4.76 ± 0.75</td>
<td>5.00 ± 0.73</td>
</tr>
<tr>
<td>Post</td>
<td>4.52 ± 0.92</td>
<td>4.67 ± 0.94</td>
<td>4.69 ± 0.79</td>
<td>4.95 ± 0.70</td>
</tr>
<tr>
<td>Δ</td>
<td>-0.13 ± 1.08</td>
<td>0.14 ± 1.12</td>
<td>-0.06 ± 1.61</td>
<td>-0.33 ± 1.43</td>
</tr>
<tr>
<td>Triglycerides, mmol/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>0.80 ± 0.31\textsubscript{a}</td>
<td>0.85 ± 0.40\textsubscript{ab}</td>
<td>1.01 ± 0.45\textsubscript{ab}</td>
<td>1.22 ± 0.52\textsubscript{b}</td>
</tr>
<tr>
<td>Post</td>
<td>0.83 ± 0.41\textsubscript{a}</td>
<td>0.80 ± 0.37\textsubscript{a}</td>
<td>1.03 ± 0.50\textsubscript{ab}</td>
<td>1.28 ± 0.45\textsubscript{b}</td>
</tr>
<tr>
<td>Δ</td>
<td>-0.01 ± 0.49</td>
<td>-0.03 ± 0.44</td>
<td>-0.05 ± 0.40</td>
<td>-0.01 ± 0.50</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>0.90 ± 0.19\textsubscript{ab}</td>
<td>0.81 ± 0.09\textsubscript{a}</td>
<td>1.01 ± 0.23\textsubscript{b}</td>
<td>0.92 ± 0.18\textsubscript{ab}</td>
</tr>
<tr>
<td>Post</td>
<td>0.89 ± 0.17</td>
<td>0.83 ± 0.15</td>
<td>1.00 ± 0.21</td>
<td>0.91 ± 0.20</td>
</tr>
<tr>
<td>Δ</td>
<td>-0.06 ± 0.24</td>
<td>0.02 ± 0.09</td>
<td>0.05 ± 0.19</td>
<td>-0.06 ± 0.24</td>
</tr>
<tr>
<td>Total Protein, g/l</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>73.75 ± 7.85</td>
<td>75.08 ± 7.71</td>
<td>78.88 ± 8.85</td>
<td>79.13 ± 4.95</td>
</tr>
<tr>
<td>Post</td>
<td>74.68 ± 4.95</td>
<td>75.88 ± 4.89</td>
<td>75.68 ± 5.38</td>
<td>77.19 ± 4.66</td>
</tr>
<tr>
<td>Δ</td>
<td>-3.00 ± 20.47</td>
<td>0.81 ± 8.38</td>
<td>1.73 ± 19.65</td>
<td>-6.23 ± 18.88*</td>
</tr>
</tbody>
</table>

\textsuperscript{1} Values expressed as mean ± standard deviation. \textsuperscript{*}p < 0.05; \textsuperscript{**}p < 0.01. Δ calculated as difference between pre- and post data. CHO, carbohydrate-based breakfast; NW, normal-weight subjects; OW, overweight/obese subjects; PRO, protein-based breakfast. Pre, day 1 of intervention; Post, day 42 of intervention. \textsuperscript{2} Within group differences determined using Δ values and paired nonparametric t test. Statistically significant Δ values within group indicated by asterisk (P < 0.05). \textsuperscript{3} Between group differences determined for pre-, post, and Δ values using one-way ANOVA. Lower case letters indicate between group differences. Values without a common letter are statistically significant (P < 0.05).
CONCLUSION

In conclusion, consumption of a higher protein breakfast, compared to a higher carbohydrate breakfast, for 6-weeks did not show an overall postprandial effect or adaptation effect on energy expenditure, appetite, or markers of metabolic health in normal or overweight/obese children ages 7-17 years old who regularly consume breakfast. Dietary intervention still serves as a potential intervention method, however future research may require macronutrient regulation of the whole diet, not just at breakfast, and a longer intervention period. Altogether, additional research is necessary to determine effective strategies to help combat childhood obesity.
Institutional Review Board
4301 West Markham, #636
Little Rock, AR 72205-7199
501-686-5667
501-686-7265 (fax)
http://irb.uams.edu/

FWA00001119

03/05/2018

PI Name: Baum, Jamie

PI Department: Food Science; University of Arkansas

Protocol Number: 207201
Protocol Title: Breakfast, Energy Metabolism, and Skeletal Muscle Health in Children
Original Review Date:

NEW SUBMISSION APPROVED, MINOR CONTINGENCIES MET

The Institutional Review Board approved your minor revisions to this study by Expedited review on 03/05/2018.

The approval period for this study runs from to .

Next review date: .

The IRB determined the risk for adults who enter this study to be N/A.

The IRB determined the risk for children who enter this study to be 1.

The IRB determined the risk for children who enter this study’s control group to be risk_ped_na.

The IRB determined the risk for the study device to be na.

The IRB determined obtaining the permission of one parent is required.
The IRB determined obtaining assent is required.

Committee Notes/Comments:

The following documents were received:

- HIPAA v3 dated 03042018 TRACKED (Type: HIPAA Authorization)
- HIPAA v3 dated 03042018 CLEAN.docx (Type: HIPAA Authorization)
- IRB 207201 Consent Form v4 dated 02272018 TRACKED (Type: Consent/Assent/Parental Permission Form (NOT including HIPAA authorization))
- IRB 207201 Consent Form v4 dated 02272018 CLEAN (Type: Consent/Assent/Parental Permission Form (NOT including HIPAA authorization))
- IRB 207201 Protocol v5 dated 02262018 TRACKED (Type: Protocol)
- IRB 207201 Protocol v5 dated 02262018 CLEAN (Type: Protocol)
- IRB 207201 Assent Form v3 dated 02262018 CLEAN (Type: Consent/Assent/Parental Permission Form (NOT including HIPAA authorization))
- IRB 207201 Assent Form v3 dated 02262018 TRACKED (Type: Consent/Assent/Parental Permission Form (NOT including HIPAA authorization))
- IRB 207201 Grant (Type: Other (please specify))
- IRB 207201 VAS palatability assessment v1 dated 10232017 (Type: Evaluation Instrument)
- IRB 207201 VAS appetite and cravings assessment v1 dated 10232017 (Type: Evaluation Instrument)
- IRB 207201 Height Weight Collection Form v1 dated 10232017 (Type: Evaluation Instrument)
- IRB 207201 3-Day Food Record v1 dated 10232017 (Type: Evaluation Instrument)

If you have any questions, please contact an IRB administrator at 501-686-5667.

If you would like to have research compliance feedback on your proposed study documentation and processes, before you enroll subjects, you may contact the Office of Research Compliance for a New Investigation Consult and Education (NICE) review. Call 501-526-6270 to schedule a NICE review.

Click here to access study.

Allen Sherman, PhD
UAMS IRB Chair