Mount Saint Vincent University

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Effect of Sedentary Video Game Playing on Subjective Appetite and Short-term Food Intake After a Glucose Preload in Normal Weight Boys

By

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Abstract

The rise in childhood obesity has coincided with increased use of screen activities. This relationship is hypothesized to result from decreased recognition of satiety signals because television viewing at mealtime delays satiation and increases food intake (FI) in children. However, it is unknown if other screen activities, such as video game playing (VGP), impact FI similarly or if the type of screen activity is an important predictor of FI. Therefore, this study was conducted to test the hypothesis that VGP for 30 min increases subjective appetite, reduces satiation, and diminishes satiety signals from a glucose preload at a test meal consumed immediately after in 9-14 year old normal weight (NW) boys. The objective of this study was to determine the effect of VGP for 30 min before a mixed meal on subjective appetite, satiation and satiety signals from a glucose preload in NW boys. On four test mornings and in random order, one-week apart, 19 NW (10^{th}-85^{th} BMI percentile) boys (mean ± SEM age: 12.0 ± 0.5 y) received equally sweetened drinks containing Sucralose® or 50 g of glucose in 250 mL of water, with or without subsequent VGP for 30 min, 2 h after a standardized breakfast of milk, cereal and orange juice. FI (mean kcal ± SEM) from an *ad libitum* pizza meal was measured immediately following VGP. Subjective appetite was measured at baseline (0 min), 15, 30, and post-meal at 60 min, and subjective emotions were measured at 0 and 30 min. FI was suppressed by the glucose preload (759.2 ± 65.8) and VGP (823.5 ± 63.0) compared to the Sucralose control preload (937.7 ± 58.4, ($p < 0.0001$), and the no VGP control (873.4 ± 60.0, $p = 0.05$), respectively. Caloric compensation (CC) was not affected by VGP (No VGP: 104% vs. VGP: 75%, $p = 0.25$). Body composition was positively associated with FI at the test lunch, but not CC. Change from baseline average appetite scores following glucose were significantly lower compared to the control ($p = 0.01$). Subjective frustration and aggression scores increased after VGP ($p < 0.05$), but change from baseline subjective emotion scores were not consistently associated with FI. In conclusion, VGP for 30 min before a meal increased satiation through a decrease in energy intake at mealtime, but did not diminish satiety signals from the glucose preload in 9- to 14- year-old NW boys.

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List of Abbreviations

A

AA	Average Appetite
AVG	Active Video Game
AVGs	Active Video Games
ANOVA	Analysis of Variance

B

BDNF	Brain-Derived Neurotrophic Factor
BG	Blood Glucose
BIA	Bioelectrical Impedance Analysis
BMI	Body Mass Index
BW	Body Weight

C

CC	Caloric Compensation
CCK	Cholesytokinin
CDC	Centres for Disease Control and Prevention
CNS	Central Nervous System

D

DEBQ	Dutch Eating Behaviour Questionnaire
DTE	Desire to Eat
DXA	Dual Energy X-Ray Absorptiometry

E

EE	Energy Expenditure

F

FFM	Fat-Free Mass
FI	Food Intake
FM	Fat Mass

G

GI	Glycemic Index
GIP	Glucose-Dependent Insulinotropic Peptide
GLP-1	Glucagon-Like Peptide-1

K

Kcal	Kilocalories

L

LEPR	Leptin Receptor

M

MC4R	Melanocortin-4 Receptor
METs	Metabolic Equivalents
MetS	Metabolic Syndrome
Min	Minutes

N

NW	Normal Weight

P

PA	Physical Activity
PFC	Prospective Food Consumption
POMC	Pro-opiomelanocortin

S

SEM	Standard Error of Mean
ST	Screen Time

T

TVV	Television Viewing

V

VAS	Visual Analogue Scale
VGP	Video Game Playing

Chapter 1. Introduction

1.1. General Introduction

One of the etiological factors identified as a contributor to the epidemic of obesity in children is sedentary behaviours and screen use, including television viewing (TVV), computer use, and video game playing (VGP) [1]. In Canada, 96% of households own a computer and 47% of households have at least one video game console [2]. Although TVV is the preferred screen time (ST) activity in children, time spent playing video games occupies approximately two hours of a typical 10- to 16-year-old Canadian's day [3]. There is a positive association between screen time activities and body fatness [4] but it is uncertain whether this effect is attributed to decreased energy expenditure, increased food intake (FI), or both.

Few studies have reported quantitative FI in children and adolescents while they are engaged in ST activities using a within-subject, repeated measures design. A design of this type is optimal for implementing a preload experiment, which enables the measurement of physiologic signals affecting both satiation (factors in a meal that bring eating to an end) and satiety (absence of hunger). In 9-14 year old boys, TVV at mealtime resulted in a 24% greater energy intake and reduced satiety response to a glucose drink consumed 30 min before an ad libitum pizza meal, illustrating that TVV diminishes normal FI regulating responses to food ingestion [5]. Using the same experimental design and test treatments, 45 min of TVV reduced caloric compensation following consumption of the glucose preload in peripubertal (16%) but not postpubertal (127%) girls, indicating TVV impaired sensitivity to satiety signals in peripubertal girls [6]. These studies confirm that the preload design can adequately assess how children's physiological control of FI is altered by environmental modifications during mealtime but it has not been applied using VGP as a stimulus in the pre-meal environment.

Video game playing is an activity that children typically engage in during the inter-meal interval [7] but its effect on FI at a later meal has not been investigated in children. Experimental evidence examining the impact of VGP on subsequent FI is limited to one study in older adolescent males, during which 1 h of VGP led to an energy surplus of 80 kcal in spontaneous FI at a later meal compared to 1 h of sitting (control) [8]. Although FI regulation was not measured, this provides evidence that VGP in the pre-meal environment may increase FI.

Therefore, the objective of this research is to determine the effect of VGP for 30 min before a mixed meal on subjective appetite, satiation and satiety signals from a glucose preload in 9-14 y old NW boys.

Chapter 2. Literature Review

2.1. Introduction

To provide a background for the research conducted, this literature review comprises six sections. The first section provides an introduction to the prevalence, consequence and causes of childhood obesity. In the second and third sections, the prevalence of common ST activities (VGP, computer use, and TVV) in relation to obesity and their effects on eating behaviour is described. Section four describes an overview of currently available experimental research on the effect of ST activities on FI regulation in adults and children. Section five describes short-term FI control in children, with a focus on carbohydrate source. The final section is a review of test methods used to measure satiety, short-term FI, and body composition in children.

2.2. Childhood Obesity

Obesity has become an increasingly common characteristic of Canadian children and adolescents. In 2009 to 2011, an estimated 19.8% of Canadian children and adolescents aged 5 to 17 were considered overweight, and 11.7% were obese [9]. Given the nature of the disease, and its impact, there is an urgent need for evidence-based prevention efforts.

2.2.1. Diagnosis

While there are several ways to assess body fatness, the body mass index (BMI; weight in kilograms divided by height in meters squared) is a practical tool that is considered a valid proxy for measuring body fat [10]. In adults, BMI correlates with total body fat [11] and other risk factors for obesity-related morbidity [12]. Until recently, obesity in children was commonly determined from age and sex specific BMI percentiles based on the Centers for Disease Control and Prevention (CDC) 2000 growth charts [13], which were developed using anthropometric data from a nationally representative reference population in the United States [14]. Several countries, including Canada, have now adopted the use of the 2006 World Health Organization (WHO) Child Growth Standards for monitoring growth in children (birth to five years) and the WHO Growth Reference 2007 for children and adolescents (5-19 years), which have been developed to reflect optimal growth in these populations [15].

In Canada, children and adolescents who fall between the 85th and 95th percentile are defined as “overweight” while “obese” is considered greater than the 95th percentile [16]. These cutoffs were recommended as acceptable assessment criteria in a collaborative statement released by the Dietitians of Canada, Canadian Pediatric Society, College of Family Physicians

of Canada and Community Health Nurses Association of Canada in 2004 [16]. This classification differed from the United States where a BMI at or above the 95th percentile of the 2000 CDC growth charts was defined as "overweight" and a BMI between the 85th and 95th percentiles was defined as "at risk for overweight". Recently, although the cut-off values have not changed, the terms "overweight" and "obese" were also adapted in the United States as a result of recommendations by the Institute of Medicine, and the American Medical Association [17].

2.2.2. Prevalence

According to data from the 2004 Canadian Community Health Survey, 8% of Canadian children and adolescents aged 2 to 17 were obese, and the combined prevalence of overweight and obesity was 26%. Both are significantly higher than 1978/79 figures, where estimates were 3% and 15%, respectively. The prevalence of overweight/obesity has doubled in this age group while obesity alone has tripled during these three decades [18]. Furthermore, the prevalence increase of obesity in children has risen more quickly, and has been more widespread than the rate of increase in adults [19]. In the long term, obese children are more likely to be obese adults, especially if they have at least one obese parent [20], placing them at risk for increased morbidity and mortality in later life.

2.2.3. Consequences

There are numerous short-term and long-term psychosocial [21], physical [22] and economic [23] consequences that result from overweight and obesity. In the pediatric population, medical complications include type 2 diabetes [24], the development of early atherosclerosis [25] and a variety of orthopedic, respiratory and hepatic abnormalities [22]. A major predictor for cardiovascular disease is the development of metabolic syndrome (MetS), which is a clustering of risk factors including obesity, dyslipidemia, impaired glucose metabolism, and elevated blood pressure [26]. Metabolic syndrome was estimated to be prevalent in 11.5% of Canadian youth, and the probability of having at least three risk factors (high systolic blood pressure, impaired fasting glucose, elevated plasma triglycerides or low high-density lipoprotein cholesterol) was strongly associated with increased adiposity [27]. In the United States, the prevalence of MetS reached 50% in a multi-ethnic cohort of severely obese children and adolescents, and a higher degree of obesity severity increased the odds of MetS being present in an individual [28]. The

psychosocial consequences of obesity in children can lead to low self esteem [29], academic underachievement, drug and alcohol abuse, and risk taking behaviours [30]. In addition, there is evidence that obese children and adolescents are more likely to experience impaired quality of life, similar to that of children diagnosed with other chronic diseases [31].

2.2.4. Causes

Obesity develops when energy intake exceeds EE but the factors that contribute to a positive energy balance are numerous and complex. Physiological, genetic, behavioral and environmental factors all contribute to obesity [32-34].

The causes of obesity can be primary or secondary, with the latter being medical conditions that result in obesity. The major physiological causes of secondary obesity include endocrine disorders, disorders of the central nervous system (CNS), and genetic syndromes. Secondary causes of obesity are more rare, and most cases are primary, which cannot be explained by known genetic or metabolic defects [35]. However, in the future, a larger proportion of cases may be explained by single gene defects where obesity is presented as a primary clinical feature [35]. A detailed description of the physiological and genetic causes of both primary and secondary obesity is discussed below.

In rare cases, obesity may be associated with a spectrum of endocrine disorders caused by a hormonal imbalance and children diagnosed with these are distinguished as having a prominent short stature. These include hypothyroidism, Cushing's syndrome, Growth hormone deficiency and pseudoparahypothyroidism. The mechanisms for the development of obesity differ with each disorder and in some cases, correcting the underlying endocrinopathy will reverse the process [36]. Growth hormone deficiency can be congenital or acquired as the result of hypothalamic-pituitary damage and it is characterized by an increase in visceral fat. Acquired growth hormone deficiency is common after traumatic brain injury in children and adolescents [37], as well as injury to the hypothalamus due to tumors, irradiation, or surgery involving the CNS [38]. Irrespective of the cause, the obesity associated with growth hormone deficiency is reversible with growth hormone replacement therapy. Relative hypercortisolemia is characteristic of obese children [39, 40], and hypercortisolemia in Cushing's syndrome is associated with truncal adiposity [41, 42]. Likewise, successful treatment of hypercortisolism reverses symptoms [43].

Obesity during childhood increases the risk of adult obesity [44], and its persistence is related to age of the child [45], parental obesity [22], and the severity of obesity [46]. The

observation that obesity tends to aggregate in families, and the congruence of body weight (BW) for monozygotic twins are both arguments that reinforce a genetic basis for obesity. Having obese relatives increases one's risk for obesity, even if the family members do not live together or share the same patterns of exercise and FI [47]. Furthermore, findings from twin studies have provided reliable estimates that BMI is highly heritable. The heritability of a trait is defined as the percent of inter-individual variation in that trait that can be explained by inherited factors [48]. Bouchard and Tremblay et al. (1990) found that overfeeding monozygotic twins resulted in significant within-pair similarity in respect to BW and fat distribution, suggesting that individual differences in BW changes are related to genetic factors [49]. Evidence from twin, adoption, and family studies have established that heritability, and genetic factors may account for between 16 to 85% of variance in BMI [50-54]. Obesity is classified into three main categories on the basis of genetic etiology: monogenetic, syndromic and polygenic [55]. An overview of this classification is provided below.

Obesity is commonly considered to be a polygenic disease but in rare cases, monogenic forms do exist and they are defined as obesity resulting from mutation or deletion of a single gene [56]. The main genes affected in monogenic disorders include leptin, leptin receptor (LEPR), pro-opiomelanocortin (POMC), prohormone convertase, melanocortin-4 receptor (MC4R), brain-derived neurotrophic factor (BDNF), and neurotrophic tyrosine kinase receptor type 2. Mutations of MC4R and LEPR are most common, having been reported in 2.5% [57] and 1.5% [58] of children with severe obesity. Recently, a highly penetrant form of obesity has been discovered due to deletions on chromosome 16p11.2 [59]. The SH2B adaptor protein-1 gene has been identified as a significant gene in this region, and is known to be involved in insulin signaling [60]. Its absence affects the brain’s ability to respond to leptin and as a result, children who are deletion carriers exhibit extreme hyperphagia and insulin resistance [61].

Syndromic obesity is also a rare cause of obesity characterized by severe obesity plus a wide array of phenotypic characteristics [62, 63]. Obesity is a component of over 30 Mendelian syndromes, which can be inherited in either an autosomal or X-linked pattern [64]. Syndromic obesity is complex; it may be monogenic, or oligogenic, and obesity may not have to be present for diagnosis of the syndrome. For instance, Bardet-Biedel syndrome, a rare autosomal recessive disorder, requires the presence of four of the five cardinal features for clinical diagnosis [65]. The principal manifestations are rod-cone dystrophy, postaxial polydactyly, central obesity,

mental retardation, hypogonadism in males, and renal dysfunction [66]. Affected infants commonly have a BW that exceeds the 90th percentile (38% of infants) and if increased BW isn't present at birth, it usually develops during the first year of life [67]. Prader-Willi Syndrome is the most common syndromic form of obesity, with prevalence rates in the United States ranging from one per 15,000 [68] to one per 25,000 births [69]. Classic components of this syndrome include hypotonia, obesity, and hypothalamic dysfunction [70]. Unlike other forms of obesity, Prader-Willi Syndrome is distinguished by the presence of elevated ghrelin levels, which may be partly responsible for the hyperphagia observed in this population [71]. Another common disorder is Alström syndrome, which is characterized by normal development and the absence of dysmorphism, although retinal dystrophy, neurosensory deafness and diabetes may be present [72]. Obesity, which normally starts to develop within the first year of life, is almost always a component of the disorder and is thought to have a hypothalamic origin, but the cause is still unknown [73].

For the majority of individuals, the genetic predisposition to obesity cannot be explained by a single gene but rather, is the result of multiple genes interacting with a changing environment. Polygenic or common obesity arises when an individual's genetic makeup is susceptible to an environment that promotes energy consumption over EE. Knowledge of the polygenic basis for obesity has been accelerated by the use of candidate gene, genome-wide linkage and genome-wide association studies [74]. Of particular significance is the observation that a partially overlapping continuum exists between monogenic and polygenic forms of obesity. Currently it is thought that at least five genes causing monogenic obesity also increase the risk for polygenic obesity. They include LEPR, MC4R, POMC, single-minded 1 transcription factor and proprotein convertase subtilisin/kexin type 1 [75].

A variety of behavioural responses to changes in the food supply and home environment have been hypothesized to contribute to increased BW. These include increased availability of added sugars in the food supply [76], fast food consumption [77], food consumption away from home [78], increased portion sizes of inexpensive, highly palatable foods [79], increased snack consumption [80], decreased physical activity (PA) [81], and increased ST. An overview of these etiological contributions is discussed briefly below.

An increase in the availability of sugars in the United States food supply has been used to support an indirect relationship between sugar and obesity but this observation is based on

unreliable data that overestimates actual consumption of this nutrient. Food disappearance (availability) data reflects the total amount of food or commodity entering the market. It is calculated as the difference between available commodity supplies (the sum of production, beginning inventories, and imports) and non-food use (exports, farm use, and industrial consumption) [82]. Based on food disappearance data, there has been a 30% increase in the availability of sugars in the United States from 1971 to 1997 [76] and this has been hypothesized to contribute to a positive energy balance [83]. However, when other components of the food supply are considered, there is little justification for singling out this one change. During this same time, there has also been a proportional increase in per capita availability of poultry (84%), fats and oils (47%), dairy products, especially milks (423%) and yogurts (111%), fruit (28%) and vegetables (72%) and energy (15%) [84]. It should be considered that these are not accurate indicators of food consumption because an estimated 40% of national food availability is wasted or spoiled rather than eaten [85]. Furthermore, observation studies, which cannot be used to determine a causal relationship, have yielded inconsistent results [83, 86, 87], and further discredits the sugars-obesity hypothesis.

Fast food consumption has been positively associated with overweight or obesity in several observational studies [88-92]. Fast food may promote increased energy intake and overweight because of its larger portion sizes, high energy density, palatability, high content of saturated and trans fat, high glycemic load, and low content of fiber [77]. A cause and effect relationship between fast food consumption and BW has not been established. Energy consumed from fast food in a naturalistic setting accounted for a large percentage (61.6%) of estimated daily energy requirements in both overweight and lean participants aged 13 to 17 years and the effect was more pronounced in the overweight group [77]. While lean individuals were able to compensate for excess energy from fast food by eating less at later meals, overweight individuals consumed significantly more total energy on days where fast food was consumed than non fast food days ($p = 0.02$) [77]. The inability for overweight adolescents to compensate for the energy in fast food suggests that fast food consumption serves to maintain or exacerbate obesity in susceptible individuals.

The decreased frequency of planned family meals is a hypothesized contributor to the obesity epidemic. While this meal pattern has become less common, there has been an increase in foods purchased outside the home, which encompasses meals and single ready-to-eat items

(including takeaway foods) purchased at restaurants, prepared-food counters at grocery stores, and other outlets [93]. On a monthly basis, obese children consume food away from home significantly more often than non-obese children aged 4-16 years [78]. In addition, food away from home was positively correlated with percent body fat ($p = 0.02$), and had a negative effect on the total energy and diet quality of the children and adolescents [78]. A recent study found that an association between food source and weight status among adolescents is also supported when the food is purchased away from home for a family meal; the odds of overweight/obesity were considerably greater in youth aged 10-16 years when families reported at least one away-from-home dinner purchase in the past week (odds ratio = 1.5 to 2.0) [94]. The increased portion sizes and caloric content of food offered at away-from-home sources may explain the association with overweight status but it cannot be determined which one was the contributing factor or if they have a combined effect on the relationship.

Larger portion sizes are hypothesized to adversely affect BW because they encourage the overconsumption of food, which increases energy intake [95]. North America portion sizes have increased since the 1970s and almost all commonly available food portions exceed USDA and FDA standard servings [96]. Observations from a cross-cultural study indicate children's exposure to large portions is also likely to include foods prepared within the home as recipe portions of the same dishes from popular cookbooks were 25% larger in the United States than in France [97]. Laboratory-based studies have shown that providing adults and children (aged 4 years or more) with larger food portions can lead to significant increases in energy intake although it is unclear in children whether the increases in energy consumed persists at later meals [95, 98-100]. More recently, adult studies have shown that this positive relationship between portion size and energy intake is sustained in the long term when portion sizes of all food and beverages are increased over 2 days [79] and 11 days [101]. While the effect of portion size on energy intake is an important consideration, it is more specifically increased intake of energy-dense foods that is contributing to this trend [102, 103]. A cross-sectional study in France found that overweight in children aged 3-6 years was positively correlated to portion sizes of sweetened pastries, but portion sizes of less energy-dense foods were either unrelated or inversely related to weight status [104]. These observations suggest that the combined effect of increased portion size and energy density is a risk factor for childhood overweight and obesity.

Snacking is a common practice included in the dietary routine of 98% of children age 2-18 [80], that may impact BW because it is an opportunity for children to consume food that is energy dense. Children in the United States consume three snacks per day, and more than 27% of daily caloric intake comes from snacks [80]. While these snacks are important nutritionally, the largest increases have been in salty snacks and candy. Desserts and sweetened beverages remain the major sources of calories from snacks [80]. An increase in snack consumption is thought to contribute to weight gain through a daily increase in energy-dense food. The total daily energy intake from snacks has risen from 18% to 25%. At the same time, the energy density of snacks has changed from 1.25 to 1.54 kcal per gram, a statistically significant increase [105]. This is important because small increases in the energy density of foods consumed can lead to large increases in total energy intake over the day [106].

A decline in PA is thought to be another contributor to the increase in childhood overweight and obesity. The Canadian Society for Exercise Physiology currently recommends that for health benefits, children (age 5 to 11) and youth (age 12 to 17) should get at least 60 minutes of moderate to vigorous PA daily [107]. Results from the 2007-2009 Canadian Health Measures Survey indicate that only 7% of children and youth met activity guidelines [108] and activity levels have been reported to drop in adolescence in cross-sectional [109] and longitudinal studies [110]. Participation in organized or unorganized sport was found to be negatively associated with obesity ($p < 0.001$) in Canadian children aged 7-11 [81]. This protective effect of activity has also been observed in youth aged 7-11 where increasing PA participation decreased the odds of overweight and obesity ($p < 0.05$ trend) independent of gender [111]. Despite these findings, studies that used PA as an intervention were unsuccessful at having a significant effect on BMI [112, 113], which suggests that children compensate for exercise energy expenditure (EE) by increasing FI.

Children may be spending more time being sedentary because they frequently engage in electronic forms of entertainment, which require minimal or no activity. The estimated number of Canadian households with a television set has reached 99% [114]. Other forms of electronic entertainment that are widely available include computers and video game consoles, which are present in 96% and 47% of households, respectively [2]. One concern is the sedentary nature of these activities, which may prevent children from meeting optimal levels of PA. According to self-reported data from the 2005 to 2006 Health Behaviour in School-aged Children Survey, time

spent in front of a screen in Canadian youth is 6 hours a day on weekends and 7 hours a day on weekends, not including time dedicated to schoolwork [115]. This is well in excess of current guidelines set by the Canadian Society for Exercise Physiology, which recommend limiting this recreation activity to 2 hours a day in children [116].

2.3. Overweight and Obesity in Relation to Screen Time Activities

The following section will define screen time (ST) and separate it into three main activities: video game playing, computer use, and TVV. The frequency of their separate and combined use and their relationship to overweight and obesity is described.

Cross-sectional studies suggest ST is associated in a dose dependent manner with adverse effects on body adiposity [1, 4]. In Canadian children who report more than 2 hr a day of ST, the risk of being overweight or obese doubled, compared to those who report less than 1 hr a day [53]. Similarly, in adolescents, higher ST (>30 hrs/week) is associated with an increased proportion of overweight/obese individuals [1]. A positive association also exists between daily ST and risk of MetS in adolescents [4]. Furthermore, several randomized controlled trials support a possible causal relationship by demonstrating that reducing ST is an intervention that can decrease levels of adiposity [117], and obesity/overweight prevalence in children [118, 119].

2.3.1. Definition of Screen Time

Sedentary activities such as television viewing, video game playing and computer use are collectively referred to as screen time [37, 120]. This term is often used as an indicator of overall media or screen exposure. .

2.3.1.1. Demographic Characteristics of Video Game and Computer Users

TVV is the largest contributor to ST use. It has been suggested that more interactive ST activities such as video games and computers may replace television in terms of popularity, as they become more affordable and readily available [121]. In a nationally representative 2004 survey of U.S. children aged 8 to 18, exposure to video games and computers were the only types of individual media to increase significantly from the previous survey conducted in 1999 by 23 and 35 minutes, respectively [122]. This increase in exposure is likely reflective of advancements in technology, which created a more expansive selection of interactive ST activities. For example, computer use currently refers to a range of recreational activities, including gameplay, Web browsing, communication, and graphic design. Video games can be

played on both television-based consoles and several hand-held devices. During this same time period, total media use, an estimate that adjusts for time that youth spend using two or more media simultaneously, only increased by two minutes [122]. This suggests that while the total amount of time that youth devote to media has not changed, a larger proportion of it is now spent with video games and computers.

Children's choice of game platform is related to age and gender. Boys spend more time playing computer games (25 min) and video games on consoles (56 min) on an average day than girls (8 min and 14 min respectively) [123]. This finding may be due to gender differences in video game ownership and use. For instance, boys are 18% more likely to own video game equipment than girls [124]. In addition, video game content is often male oriented because it features violence, and under represents female characters [125]. Half of the time children devote to playing video games takes place on a console and this activity peaks in duration between the ages of 11 and 14 [123].

2.3.1.2. Video Game Playing and Obesity

Despite the finding the VGP is a distinct behaviour from TVV and computer use, the majority of studies have analyzed VGP in combination with other ST activities for the purpose of examining their combined effect on obesity [81, 126-137] and the results have been inconsistent. One problem with the available research is that video game use can be a general term that refers to several platforms (handheld, console-based, arcade, and computer) and therefore, studies that measure this behavior are subject to variability. Obtaining an accurate measure of children's TVV and computer use is also challenging because they often multitask with other media devices while engaging in these ST activities [138]. In order to determine if VGP has an independent effect on obesity, it must be measured separately from other ST activities.

Children predominately play video games on consoles and handheld devices and both increased frequency and duration of usage of these platforms have been associated with increased risk of overweight [139-141]. Playing video games on consoles and handheld devices every day increased the odds of excess weight gain in 9-11 year old girls at a 1-yr follow up (OR: 1.48) compared to those who played video games less often [139]. In addition, the prevalence of overweight and obesity was higher for boys and girls who played video games on these devices for a longer period of time, which varied among studies [140, 141]. The relationship between playing video games on multiple platforms, including computers, and

obesity is more inconsistent with a positive association found in most studies [81, 142-145] and a null effect in others [146, 147]. If a true lack of association is the reason for results in the later studies, it may be because video games do not leave the hands free for eating. It has also been reported that the average EE during seated video game playing (2.08 kcal·h−1·kg−1) is 52% higher than during rest in 7-10 year old boys [148]. However, it is more likely that the inconsistent results can be explained by differences in sample sizes and techniques used to measure video game use. Collectively, these studies support an independent association between VGP and obesity risk but they are mostly cross-sectional, which cannot determine the temporal sequence between these two variables. More longitudinal research is needed to clarify if VGP is a potential cause for obesity or whether these observations are because obese children play more video games.

2.3.1.3. Computer Use and Obesity

There is weak evidence that increased computer use is independently related to obesity in older children and adolescents. Limited studies have found a significant positive association between overweight or weight status and computer use in both sexes [141, 149-151] and a gender specific effect in girls [147, 152, 153]. For instance, adolescents who used computers more than 4 hours per day on weekdays were five times more likely to be overweight or obese [151]. However, an equal amount of studies have found a null effect [140, 144, 150, 153-156]. This inconsistency may be reflective of the average age of participants because a positive association was mainly present in older children. Younger children spend the least amount of time on the computer [123], and it is possible that the duration of computer use in the samples used was not high enough to detect a difference in weight status.

Several studies have relied on weak measures of computer use, which could explain their failure to find a relationship between computer use and obesity. Surveys that do not investigate every aspect of recreational computer use (entertainment, communication, and web browsing) are likely not as accurate. For instance, increased time spent on the computer for email, writing, and surfing was associated with overweight prevalence in 14 y old Finnish girls, but there was no relationship in boys or older and younger girls [147]. Similarly, an increase in time spent on the computer for non-gaming purposes or the Internet was not predictive of a higher weight status in American children [144, 156]. This could indicate that children who use the computer for information seeking purposes are more likely to engage in other behaviours that favor a healthy

weight status. However, American children mainly use the computer for entertainment purposes [123], and it is possible that the time they allocate for other computer activities is not substantial enough to influence weight status. Research indicates that computer use in general favors weight gain in children but more rigorous research is needed to determine if the type of computer activity, such as gaming, is an important factor in the relationship.

2.3.1.4. Television Viewing and Obesity

Television viewing is the most common ST activity, both in terms of time spent engaged in it and the proportion of children who watch it on any given day (80% of kids) [122]. On average, television exposure averages 3.5 hours daily for children and teens [116], and time spent with this medium is similar between boys and girls [157]. Television viewing is cross-sectionally and prospectively related to obesity in children [158-163].

Television viewing has been shown to lower resting EE (metabolic rate) [164]. Energy expenditure during TVV is lower than during any other sitting activity, such as reading [165]. It is also significantly lower than rest in both normal weight (NW) and obese children [164]. In all subjects, EE during TVV was 1306 kcal/d and this was significantly lower than both baseline (1517.32 kcal/d) and the no television condition (1469.74 kcal/d) [164].

According to the "displacement hypothesis", the inactive nature of TVV decreases the time that children could spend doing PA. Robinson et al. proposed a relationship where TVV may lead to obesity because it replaces time that could be spent in more energy expending activities [166]. However, a lack of consistency from cross-sectional [122, 161, 162, 167-169], longitudinal, [169, 170], and experimental [171] studies undermines the strength of this association. For example, a meta-analysis of cross-sectional and prospective studies found that only vigorous PA was significantly and inversely associated with time spent in TVV [172]. There is weak evidence to conclude that a strong relationship exists between TVV and low PA levels. However, the two variables are likely independently related to weight change and simply restricting TVV may not be effective in increasing PA [173]. It is evident that TVV and PA are likely not functional opposites because 1 hr/week increases in TVV were not related to changes in time spent in moderate and vigorous physical activities outside of school (0.03 hrs/week) during 1 year periods of follow up in 10-15 y old adolescents [170]. A similar observation was reported in a longitudinal study on female adolescents where baseline hours of after-school TVV was not significantly associated with change in PA over follow up periods across two years (r=

0.04, p = 0.48) [169]. These studies suggest that TVV does not function by acting as a hindrance to PA, but it cannot be excluded that children who engage in more TVV have a tendency to be more sedentary in general. If TVV is directly related to PA, these studies may have failed to detect it because they relied on self-reports of these behaviours. It cannot be verified that an increase in TVV automatically results in a decrease in PA, which suggests that increasing EE during ST activities may be more a more effective solution for preventing obesity.

2.3.2. Active Video Games

'Exergaming' is a term used by researchers to describe physically active gaming. This type of entertainment has also been referred to in the literature as exertainment, dance simulation video game, activity promoting video game, active video game (AVG), physical gaming, (kin)aesthetic video game, physical activity-change game and interactive video game [174]. Traditional video games typically involve the manipulation of joysticks and game control buttons as a means of interacting. Active video games (AVGs), however, require physical movement or exertion to activate the input device.

Active video games have been positioned as an entertaining and convenient means for children to replace sedentary ST for time that is spent being physically active. They may also be an appealing alternative to sports and traditional exercise for children with low self esteem or functional and/or coordination problems [175]. For instance, participation in traditional video games increased perceptual-motor skills, including, hand–eye coordination, dexterity, and fine motor ability in 61-78 y old men and women [176]. These benefits could be extended to exergaming because games such as Wii Sports tennis require rapid hand–eye or foot–eye coordination, and therefore, they may improve motor control in children as well [177].

Active video game playing leads to low to moderate activity and increased EE in children and adolescents [178-181]. For instance, 10-14 y old children had significantly higher EEs (range = 2.9 ± 0.7 kcal/min to 6.5 ± 1.7 kcal/min) while playing a variety of active simulation games compared with rest (1.3 ± 0.2 kcal/min) and non-active gaming (1.6 ± 0.2 kcal/min) conditions [182]. A recent study compared the EE involved with operating a handheld game console and a lower limb operated game console, playing boxing on the Nintendo Wii, cycling on a stationary bike, and exercising on a treadmill in adult males [183]. Using the lower limb operated game console for 12 min resulted in a mean EE of 7.2 ± 1.2 kcal/kg^{-1}/h^{-1}, and this was significantly higher than the handheld game console, walking, and Wii gaming [183]. These

results can likely be attributed to the fact that a lower limb operated game requires continuous stimulation of larger muscle groups where the Wii and hand operated games rely on smaller muscle groups, which expend less energy that is needed to favor a negative energy balance.

Energy expenditure during AVG play is highly variable in adolescents, but most exergames can provide PA of a moderate intensity [184]. A meta-analysis of nine studies estimated that the average EE during AVG playing in youth (18 years or younger) was 3.2 metabolic equivalents (METs), which met the definition of moderate intensity (3.0 METs) that is suggested in PA guidelines [184]. Under naturalistic settings, providing children with AVGs does not increase the amount of time that they spend in moderate to vigorous PA [185]. Normal weight, overweight, and obese children (9-12 y old) who were given a Wii console and two AVGs logged similar amounts of moderate to vigorous PA (treatment group range: 25.3 ± 14 min to 27.9 ± 14.7 min) during a 12 week period as children who were provided with the same console and two sedentary video games (control group range: 25.1 ± 13.9 min to 29.5 ± 19.5 min) [185], and both groups did not differ from baseline levels. This indicates that children do not typically acquire the optimal amount of PA, whether they have access to AVGs or not [185]. Exergames have the potential to increase the amount of time that children are physically active, but improvement in their design may be needed to entice children's regular use of them.

2.4. Mechanisms of Screen Time Activities on Eating Behavior and Food Choices in Children and Adolescents

The following section describes the different forms of food advertising in the media and how they affect food choices and behaviour, as well as the influence of time spent engaged in ST activities on dietary selection and eating patterns.

2.4.1. Impact of Food Advertising

While the majority of research has been conducted on the influence of television on children's food preferences and eating [186, 187], it has only been recently that the influence of other forms of media has been explored. There is a growing potential for newer media influences such as computers to create other avenues for marketers to sell products [2, 188]. On the Internet, these come in the form of "advergames", which are online computer games developed for the purpose of promoting a brand [189]. The use of advergames have proliferated on food and beverage Web sites, with one study finding they were featured in 73% of Web sites sampled

[190]. In addition, they are often present on areas specifically designed for children [191]. Foods and beverages promoted through advergames often have a low nutrient density and contain high levels of sugar, sodium, and/or fat [192].

Children's food and beverage preferences are heavily influenced by the persuasive content featured in advergames. For instance, 5-8 year old children who played a Froot Loops cereal advergame had a significantly higher preference for the brand over other cereals and food types [193]. In addition to increasing brand recognition, advergames also affect children's actual consumption of both healthy and unhealthy foods. Children (9-10 y old) ate a greater number of nutritious snacks after playing a Pac-Man advergame where they were rewarded for selecting nutritious snacks for their character compared to children who were rewarded for selecting snacks that were less nutritious [194]. Similarly, after 12 min of exposure to an advergame that promoted sweet snack foods, 7-12 y old children ate 56% more of foods that were energy-dense, and 50% less of foods that were lower in energy density than children who played advergames that promoted fruit and vegetable consumption [195]. This indicates that advergames may contribute to increased consumption of nutritionally poor foods in children but if they are used to promote nutritious eating habits, they can also be part of a strategy for preventing obesity.

Another way of reaching consumers through electronic games is the use of product placement, which involves inserting a product or brand into a movie, television show, book or video game. Product placement is commonly used in television to increases sales of a featured product but its application to video games is relatively recent and in the later case it is ideally targeted towards 18-34 yr old men. Due to the interactive nature of games, participants are much more involved with the products presented [196]. A study involving undergraduate students showed that after playing a video game featuring a branded product, they were more likely to have a positive attitude towards the brand than equivalently rated products [197]. The ability for video games to change participants' brand attitudes suggests that they have the potential to adversely effect eating habits if they are used to market nutrient-poor products but this hypothesis needs to be explored further.

2.4.2. Screen Time Activities and Dietary Quality

Increased participation in ST activities has been identified as a risk factor for children having a diet that is less balanced and nutritionally adequate [198]. Several epidemiological studies have correlated TVV with consumption of higher dietary energy or dietary fat [92, 199,

200], increased sweetened drinks [201], foods of low nutritional value [202] and decreased fruit and vegetable consumption [203-205]. In addition to TVV, there is consistent evidence that the overall time spent in ST activities negatively impacts diet quality. Less cumulative time spent TVV, and playing computers and video games has been associated with a diet that more closely adheres to the Mediterranean diet in 3-12 y old children ($p = 0.001$) and 13-18 y old adolescents ($p = 0.002$) [206]. Similarly, more TVV and VGP has been associated with higher consumptions of salty snacks in 10-12 year old children [207] and energy-dense snacks in 8-12 year old girls [208]. There is also evidence that children alter their macronutrient and energy intakes in response to changes in their ST exposure. For instance, an experimental intervention that decreased VGP and TVV by approximately 100 min per day reduced energy intake by >450 kcal/d and fat intake by almost 300 kcal/d in 12-16 year old youth [209].

Limited evidence suggests that time spent playing video games are associated with high calorie and low nutrient intakes from food and beverages in children [210]. In experimental settings, the type of video game console impacts food selection in the short term. When children had access to three types of snacks, that ranged in nutrient density during a session of VGP, they ate more of the snacks that were higher in nutrient density while they played an AVG compared to a traditional video game [211]. Several variables, including, weight status, and game genre during the sedentary condition may have contributed to this observation, and therefore, more investigation is needed to understand the factors that influence food choice during varying types of video game play.

2.4.3. Screen Time Activities and Meal Frequency

Screen time activities increase the frequency of eating episodes and promote irregular meal patterns in children. More than 50% of children report eating during ST activities [212], and food consumption during TVV alone accounts for 18% and 26% of total daily energy on weekdays and weekends in schoolchildren [213]. Eating during VGP, however, is not a common occurrence. For instance, food consumption during both VGP and other non-ST recreational activities accounts for less than 3% of daily energy intake in children [213]. Another study was not able to identify any occasions where VGP and eating episodes occurred during the same time in children [7]. It is plausible that VGP is not conducive to eating because typically the player's hands are occupied, which restricts the ability to consume food [214]. The possibility of the controller becoming dirty or greasy from handling food could also be a deterrent. Snacking may

be easier during computer game playing because games move at a slower pace, and only requires the use of one hand [215] but there is no research on the co-occurrence of these two behaviours.

There is evidence that consuming meals during ST may be a predictor of increased meal skipping. Meal skipping is more prevalent in children and adolescents who participate in ST activities more often [216] and those who commonly eat and engage in ST activities at the same time [217]. In addition, adolescents report eating faster in order to watch television or play a computer game, and this practice is more common in heavy users of these two activities [216]. This observation is relevant to the relationship between ST and obesity because meal skipping is linked to increased fat mass [218, 219] and obesity [219, 220] in children and adolescents.

2.5. Effect of Screen Time Activities on Food Intake Regulation

There are two primary mechanisms that have been suggested in explaining the relationship with obesity: decreased EE and increased FI. The former hypothesis suggests this activity displaces time spent being physically active, potentially reducing total EE. The latter suggests that children eat more during this activity or consume more energy outside the activity as a result of exposure to food advertisements. The following section will be an overview of how screens affect FI.

2.5.1. Television Viewing and Food Intake

Television viewing is an environmental stimulus that requires attending to both auditory and visual cues [221]. This activity has the potential to increase eating by limiting an individual's capacity to monitor signals associated with hunger and satiety and/or divert them from their deliberate habitual control over FI [222]. Few studies have examined the effect of TVV on the regulation of FI.

Television viewing has the potential to either decrease or increase meal intake in preschool children. Using a within-subject design, parental reports of 3-5 year old children's TVV behaviours were associated with FI during exposure to television, but it did not influence FI when television was absent. Children, who habitually watched more television or frequently ate during TVV at home, had higher energy intakes during the 22-min lunch session when television was present [223]. An explanation for this is that they ignored internal satiety cues and satiation was delayed. Overall, children consumed significantly fewer calories from the same lunch meal and a snack during the presence of television [223].

In older children, TVV is a distracter that enables overeating. On four separate test days, boys between the ages of 9 and 14 received equally sweetend preloads containing Splenda sucralose or glucose [1.0 g/kg BW] in 250 mL of water. After consumption of the preload, the boys were engaged in child appropriate games of a sedentary nature (i.e. chess) for 30 min followed immediately by a test meal with or without TVV. Television viewing compromised 9-14 year old boys' ability to compensate for previously consumed calories from a glucose preload, which increased lunchtime intakes by 228 kcal ($p < 0.001$) compared to the no TVV condition. This supports the finding that TVV has the ability to delay satiation and diminish satiety signals from previously consumed calories. A similar observation was also seen in peripubertal girls but in postpubertal girls, glucose reduced FI (~ 30%, $p < 0.001$), with or without TVV, which suggests pubertal status has an influence on physiological signals involved in FI regulation [6].

Television can affect FI through disruption of habituation to food cues [224]. Habituation is a decrease in response after repeated presentation of a stimulus. This process normally occurs during eating because there is continuous exposure to sensory cues from food [225] which humans respond to by salivating [226]. Slower salivary habituation to food stimuli is related to greater energy intakes in children [227], which suggests that habituation is a mechanism for the cessation of eating [228]. Both food-related [229] and nonfood-related stimuli, such as the presence of TVV [224] can cause dishabituation to occur, which is the recovery of response to a habituating stimulus [225]. For instance, in 9-12 year old children, TVV recovered the motivation to eat after the motivation to eat had reduced [224]. Additionally, children who were provided with an *ad libitum* snack during ongoing TVV consumed significantly more energy ($p = 0.007$) and spent more time eating ($p < 0.0001$) than either children who were exposed to a repeated segment of a television program or no TVV [224]. It is hypothesized that television is a distracter that disrupts or delays satiety through activation of new memory nodes, which removes information about food from short-term memory [225]. Thus, a potential mechanism for increased FI during TVV is the ability for television to allocate attention away from eating, which leads to the disruption of habituation to food cues.

2.5.2. Computer Based Work and Food Intake

Evidence on the effects of computer related activities on appetite control and energy balance is limited. It is hypothesized that the cognitively demanding aspect of these activities

contributes to a positive energy balance through unique physiological mechanisms that affect control of FI [8]. Unlike physical work, mental work exercises the brain, which is primarily dependent on glucose for its energy metabolism [230]. The metabolic demand associated with mental work depletes the limited stores of cerebral carbohydrate available for neurons. In response to this, a compensatory increase in energy intake may be triggered by the brain with the goal of restoring glucose homeostasis [8]. It is therefore hypothesized that a reduced availability of glucose caused by its brain utilization during mental work could lead to the perception and expression of hunger and FI.

Mental work may promote a positive energy balance [231, 232] because the cognitive demand associated with this activity represents a neurogenic stress, which increases food consumption. Cognitive tasks may favor an increase in caloric intake through activation of the hypothalamo–pituitary–adrenal (HPA) axis, which leads to the release of stress-induced cortisol [8]. This effect has been demonstrated in women who were engaged in a reading-writing task, which resulted in an increase in energy intake that exceeded the control condition of sitting quietly by 959 kJ and was not accompanied by increased appetite sensations [232]. Similarly, the first study to report FI after computer use in young women observed that completion of a battery of computerize tests for 45 min resulted in an approximate increase of 250 kcal at a later buffet meal, as well as plasma glucose and insulin instability, and increased cortisol levels [233]. It is thought that alterations to blood glucose (BG) concentrations from computer-based work might have important implications over the long term as glucose homeostasis is a predictor of weight gain [234]. This response may be relevant to other sedentary activities such as TVV and VGP because they have a similar ability to invoke mental fatigue and neurogenic stress.

Playing computer games during lunch decreases satiety and increases FI immediately afterwards in young adults. Men and women who were distracted by a computer game during a fixed lunch (distraction condition) consumed 100% more biscuit (mean intake: 52.1 g) 30 min later than participants who ate lunch without the distracter (mean intake: 27.1 g) [235]. Adults who played the computer game also reported lower fullness immediately after the lunch, and having a poorer memory for the lunch meal, as assessed by a serial-order recall. This supports that ST behaviors have the capacity to increase FI after their termination, most likely via an impairment of memory for recent eating [235].

2.5.3. Video Game Playing and Food Intake

Experimental studies on how electronic games affect FI are limited to six reports, with only four in children [211, 236-240]. The majority of studies have looked at snacking behaviour during simultaneous video game play. Regardless of the genre of video game played, it has been demonstrated that youth underestimate the amount of food they consume during 30 min of VGP compared to sitting when snacks are provided [236]. However, the type of console may have an effect on the quality and quantity of FI because youth were more likely to choose nutritious snacks that were provided [211], and eat less overall [211, 237] during active video game play than traditional video game play. Conversely, the addition of a motor component to a video game does not lower energy consumption [238]. For instance, 9-13 y old children's ad libitum snack consumption during video game play on the X-BOX®360 did not differ from playing the same game while walking on an adapted treadmill but a considerable amount of energy was consumed (seated: 374 kcal compared to video game condition: 383 kcal) [238].

Adults consume a substantial amount of energy during both traditional and AVG play when palatable food is provided, which contributes to a net positive energy balance in the short term. For instance, 18-35 y old men and women played from a selection of either sedentary or active video games for 1 hr while snacks and beverages were provided. The type of video game did not have an effect on total energy intake (sedentary: 747 kcal compared to active: 553 kcal) but EE during active video game play (2.50 $kcal/kg^{-1}/h^{-1}$) exceeded the sedentary condition by 1.20 $kcal/kg^{-1}/h^{-1}$ [240]. Thus, adults did not adjust their energy intake according to the energy they expended, but an overall positive energy balance was more pronounced after playing sedentary video games. Since a variety of games were used in the study, it remains unclear whether the genre of video game or console used has the most influence on FI. From an energy balance perspective, these studies provide evidence that AVGs are most beneficial when snacks are not provided in the video game environment but it is also important to consider how FI is affected after the video game session has ceased.

Limited evidence suggests that video games increase FI outside of the gaming environment in older adolescents and young adults. Using a within-subject experimental design, males between the ages of 15 and 19 were randomly selected in two separate conditions: playing a soccer video game on the Xbox 360 or sitting quietly for 1 hr followed by a test meal. *Ad libitum* energy intake after video game play exceeded the resting condition by 335 KJ, and this

occurred without increased sensations of appetite [239]. The disconnect between subject appetite sensations and measured energy intake emphasizes the role of reward-driven or hedonic feeding behaviour, which has the potential to override the metabolic control mechanisms of energy balance. This study provides evidence that VGP delays normal mealtime satiation and likely increases FI because satiety signals become compromised.

2.6. Food Intake Regulation in Children

The regulation of FI is achieved by a complex neuronal network that integrates several stimulatory and inhibitory signals from the central and peripheral nervous system. These signals function to determine how often we eat (satiety) and how much we eat at each meal (satiation). Most of the knowledge on the physiological control of FI pertains to adults, and research is limited in children.

Food intake is regulated in both the short and long term by physiological systems. Satiety and regulatory hormones, acting in shorter term, transmit information in regards to the size and duration of the meal (satiation), as well as the interval to next meal (satiety) [241]. These signals interact with leptin and insulin, which are long-term regulators of FI control [242] and which are secreted in proportion to the amount of adipose tissue of the individual [243].

Short-term satiety is modulated by several dietary factors such as energy intake, macronutrient composition, energy density, physicochemical food properties and the physical state of the meals [244-248]. The effect of macronutrients on energy intake cannot simply be predicted by their energy density because both experimental animals and humans consume excess energy when provided with highly palatable, energy-dense foods or a high fat diet [249]. A review by Stubbs and colleagues revealed that under normal circumstances in which fat contributes disproportionately to energy density, macronutrients exert hierarchical effects on satiety in the order of protein > carbohydrate > fat [250].

2.6.1. Carbohydrate Induced Satiety

Carbohydrate provides the main source of energy in the diet, and accounts for between 40 and 80% of total energy intake [141]. Carbohydrates are classified according to their chemical structure and this divides them in three main groups: sugars, oligosaccharides (short-chain carbohydrates), and polysaccharides. The term “sugars” is conventionally used to describe all mono- and disacchairdes that are naturally occurring or added to food. The most common sugars

in the human diet are the disaccharides sucrose and lactose, which are comprised of two monosaccharides: glucose and either fructose or galactose [251].

The glucostatic theory proposed that low BG concentrations trigger the onset of feeding, and high BG concentrations signal satiety and the termination of feeding [252]. This was based on the observation in rodents that hypothalamic glucoreceptors respond to the level of glucose in the blood. A fall in glucose levels would signal hunger, whereas a rise would activate hypothalamic centers that regulate eating and suppress FI. Acute changes in the BG concentrations have a major impact on gastrointestinal motor function. Glucagon-like peptide-1 (GLP-1) has received considerable attention as a putative satiety peptide involved in regulating carbohydrate-induced satiety [253] and is released when glucose comes into contact with the L cells of the lower small intestine. Hyperglycemia slows the rate of gastric emptying in healthy individuals [254], and consequently contributes to fullness and short-term satiety.

The type and amount of carbohydrate consumed has an effect on postprandial glycemic response, which refers to an elevated BG concentration after a meal. Rapid digestion and absorption of glucose stimulates an increased glycemic response, and this rate is largely determined by the structure of dietary carbohydrates. Rapidly and slowly digestible carbohydrates also vary in their ability to secrete incretin hormones such as GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) [255]. Several methods have been used to quantify the glycemic response, including the glycemic index (GI), the relative glycemic index and the glycemic load. Since the glycemic response depends on both the amount of carbohydrate and its GI, the glycemic load has recently been developed to mathematically account for a food's GI and its total amount of available carbohydrate. In young adults, increases in glycemic load were found to be predictive of increases in both glycemia and insulinemia [256], and it is a stronger predictor of these metabolic responses after isoenergetic portions of single foods and mixed meals than carbohydrate content alone [257]. Glycemic load can therefore be considered physiologically valid at estimating postprandial glycemia and insulin demand in healthy individuals.

It has been well documented that carbohydrates mediates satiety through other mechanism unrelated to BG, including the effect on satiety signals. A satiety signal is defined as an endogenous factor that causes a sensation of fullness and reduces the size of an ongoing meal [242]. Several satiety signals are secreted in response to carbohydrate including cholesytokinin

(CCK), GLP-1, and amylin.

The satiety hormone that is the most well know is CCK, a gastrointestinal peptide that is secreted from the "I" cells of the proximal duodenum. Cholesytokinin responds rapidly to the presence of nutrients in the lumen, as evidenced by the observation that CCK levels are elevated within 15 min of meal initiation [258]. Compared to protein and fat, carbohydrate is the least potent stimulant of CCK, and the increase in CCK after carbohydrate is only sustained temporarily before returning to baseline. Exogenous peripheral CCK administration inhibits FI by reducing meal size and duration in animals and humans [259]. The satiating effect of CCK can be explained by several mechanisms including inhibition of gastric emptying and activition of neurons in brain regions such as the hypocampus, hypothalamus, and amygdala, which suppress hunger and FI.

The two main incretins in humans that induce insulin secretion are GIP and GLP-1. The later is an anorexigenic peptide that is secreted in response to nutrients by the enteroendocrine L cells located primarily in the small intestine [260]. Exogenous administration of GLP-1 is known to slow gastric emptying and inhibit gastrointestinal motor activity. Appetite suppression by GLP-1 also occurs through indirect mechanisms including, enhancement of of pancreatic β-cells survival, increased insulin secretion and improved insulin sensitivity of peripheral tissues in rats and humans [261]. These incretins have opposing effects on glucagon secretion with GLP-1 suppressing glucagon secretion when plasma glucose levels are above fasting levels while GIP stimulates glucagon release. After oral administration of glucose in humans, plasma levels of GLP-1 and GIP peak between 30-60 min and 4-5 min, respectively [260] and return to baseline within 3-4 hours. This incretin effect mediates approximately 60% of the overall postprandial insulin responses in non- diabetic individuals [262].

Amylin is secreted from pancreatic cells [263] and this hormone may have an anorexogenic effect in humans. Although knowledge of its effect on FI is mostly limited to rodents, it is thought be implicated in the regulation of FI in humans. In rodents, peripheral administration of amylin reduces FI and meal size and intraperitoneal administration of a specific amylin receptor antagonist abolishes amylin's anorexigenic effect [264]. Furthermore, a synthetic amylin analog, which has been approved for the treatment of diabetes, induces weight loss in both diabetic and non-diabetic individuals [265].

When carbohydrates are given as a preload or experimental meal, they have the ability to suppress appetite and FI in the short term by an amount equivalent to their energy content. Their time course of suppressive action on subsequent FI will depend on the form, source, and amount consumed. This is particularly evident in young children when sugars in solution are provided as preloads before meals. For example, NW preschool children given an average of 22 grams (average of 1.25 g/ kg of BW) of either sucrose or low glucose maltodextrin or a combination of both, demonstrated almost perfect caloric compensation (CC) in the test meal after the preloads [266-269]. These results suggest that children are sensitive to internal, physiological signals and dispute the notion that calories in the form of sugar are undetected in the body, and bypass FI control mechanisms. In this age group, the interval between administration of the preload and consumption of the test meal does not seem to be a factor when a relatively short delay is employed; young children demonstrated accurate CC for a sucrose drink (90 kcal) given 0, 30, and 60 min before a test meal. However, when a 90 min interval was used in design assessing the effect of a high carbohydrate yoghurt preload on subsequent FI, young children (4-6 y) demonstrated incomplete CC [270]. Similarly, no difference in FI was observed 90 min after older children (9-10 y) consumed either a sucrose or aspartame sweetened beverage, although the two treatments differed in caloric density (200-240 kcal).

A larger body of evidence shows sugars consumption suppress FI in adults and is dependent on the source, dose, and timing of the preload in relation to measurement of FI. For example, in young men, consumption of 75 g [271] or 50g [272] of glucose in a drink or 50g in yoghurt [273] reduces FI 60 min later. When fructose (50 g) is the sole source of sugar in a drink, the suppressive effect is even more pronounced at a test meal given both 38 min [274] and 145 min later [275-277]. Contrarily, a glucose 80, fructose 20 preload (G80:F20) was effective at suppressing FI in young males (18-35 y) 80 min later while a G20:F80 solution did not reduce FI [278]. The former observations can be explained by the slow absorption of fructose when it is consumed in the absence of glucose. When carbohydrate is administered in the form of a starch, it is less effective at reducing FI; sweetened drinks containing 75 g of high amylose cornstarch or high amylopectin cornstarch failed to suppress FI 60 min later in young men [271]. This suggests that rapidly digested carbohydrates are more effective in decreasing FI. Their effect on BG is an important consideration because high GI carbohydrates are associated with greater satiety and reduced FI in short-term studies of approximately 1 hour [279]. For example, when pure

isovolumetric preloads of 75 g polycose, sucrose, glucose or fructose/glucose mixture were consumed by young men, the greater the glycemic response, the greater the decrease in reported appetite and FI an hour later [271].

2.6.2. Mechanisms of Carbohydrate Induced Satiety

The ingestion of carbohydrate triggers FI regulatory systems through the secretion of satiety signals. It is well established that satiety is primarily the result of an interaction with receptors in the gastrointestinal tract as indicated by the observation that intraduodenal administration of glucose reduces subsequent FI more than glucose infused intravenously [280].

Physiologic responses to the ingestion of carbohydrate include increases in BG concentrations, and the release of a number of gastrointestinal hormones, including insulin, glucagon, GLP-1, amylin, ghrelin and CCK. These hormones are responsible for activating satiety signals in the CNS, and a variety of gastrointestinal functions including gastric emptying, intestinal transit as well as the perception of gastric and intestinal distention [281-283]

2.6.3. Hormonal Regulation of Food Intake

Insulin and leptin are long-term regulators of FI and energy balance. Woods and colleagues first proposed that insulin plays a role in the long-term regulation of energy balance [242, 284, 285]. Insulin secretion from islet β cells of the endocrine pancreas is stimulated by food ingestion. Insulin transport into the CNS occurs over a period of hours after circulating insulin concentrations increase, consistent with a role for insulin in the long-term regulation of body adiposity rather than as a short-term satiety signal [286]. Insulin signaling in the brain limits FI and over the long term, insulin secretion functions as a negative feedback signal of recent energy intake and body adiposity [286]. In the absence of elevated glucose, basal insulin as well as every increment of insulin above baseline during meals, is in direct proportion to adiposity [285, 287]. Insulin has a suppressant effect on FI as demonstrated by the observation that continuous infusion of insulin over a 20 day period into free-feeding baboons induced a sustained suppression of FI [288]. To inhibit FI, insulin interacts with CCK and several other hypothalamic neuropeptides also involved in the regulation of feeding behaviour [33, 243].

Leptin, the endocrine product of the ob gene in adipose cells, has an anorexic effect on energy balance that is mediated by the CNS. It is secreted in direct proportion to body adiposity [289-291]. It is well known that obesity is related to increased peripheral levels of leptin

indicating a decreased biological effect of the hormone, or resistance. This is consistent with the hypothesis that the biological impact of leptin is more pronounced when leptin levels are decreasing than when circulating leptin concentrations are elevated. For example, when endogenous leptin levels were chronically decreased in women during prolonged consumption of a moderately energy-restricted diet, their increased sensations of hunger correlated with reduction of plasma leptin levels.

It is known that stress has the capability of both increasing and decreasing FI [292, 293] but the underlying physiological mechanisms influencing eating behaviour remain unclear. Glucocorticoids (cortisol in humans, corticosterone in animals) are hormones released under stressful situations as a result of stimulation of the hypothalamic–pituitary–interrenal (HPI) axis. They enhance availability of glucose through protein breakdown, gluconeogenesis and lipolysis [294]. The effect of cortisol on lipolysis has received little investigation in children. One study found that alterations in salivary cortisol and lipolysis were not related in NW and obese children aged 8 to 10. However, a group difference did exist where the salivary cortisol increased and decreased in response to a breakfast meal and exercise, respectively, while no change was seen in the lean subjects [295]. This suggests that salivary cortisol and lipolytic responses are not necessarily linked, but are altered in obesity. Glucocorticoids have an anabolic effect in the CNS, where they act by increasing FI through working in opposition to changes in hypothalamic neuropeptide systems mediated by leptin and insulin that inhibit FI [296]. The cortisol response to FI is greater and more prolonged in obese adults than lean adults, particularly after lunch [297]. Infusing healthy adults with corticotropin-releasing hormone, which triggers a strong cortisol response, led to increased FI (597.5 ± 233.8 kcal) compared to a placebo (456.5 ± 213.0 kcal), which suggests that cortisol directly stimulates FI in humans [294].

2.7. Measurement of Satiety, Food Intake, and Body Composition in Children

This section will explain some of the common study definitions as well as the instruments used to measure short-term FI, appetite, and body composition and dietary restraint in children. The accuracy and validity of these methods have been demonstrated among adults although there is less evidence surrounding their accuracy in children.

2.7.1. Hunger, Satiety, and Satiation

Hunger describes the sensations that promote food consumption, and it is motivated by both biological and psychological factors. Satiation, sometimes called short-term or intra-meal satiety [298] refers to the events during the course of eating that lead to the termination of eating. Conversely, satiety (post-ingestive or inter-meal satiety) is the feeling of fullness that persists after a meal has ended and potentially serves to suppress further FI [298]. Satiation is important in controlling the amount of energy consumed at each of these eating occasions, while satiety affects the period of time between eating occasions and potentially the amount consumed at the next meal. The factors affecting satiety and satiation can be represented by the satiety cascade, which provides a conceptual framework of how four distinct mechanisms (sensory, cognitive, post-ingestive, and post-absorptive) operate in an overlapping manner to influence FI. Sensory and post absorptive factors serve first to stimulate and then to inhibit FI. Pre-absorptive and post-absorptive factors are generated postingestively to terminate the meal and inhibit postmeal intake. Operating together, and in interaction, the early and late stages of the satiety cascade determine the amount, duration, and frequency of eating. Their interaction is also a determinant of the duration and strength of the satiety and satiation.

2.7.2. Measurement of Subjective Appetite

Visual Analogue Scales (VAS) can be used to capture a range of subjective sensations related to appetite. Visual Analogue Scales are most often composed of lines (100 mm) with opposing statements anchored at each end. Subjects are asked to make a mark across the line corresponding to their feelings. This mark is quantified by measuring the distance from the left end of the line to the mark. Hill and Blundell (1982) [299] originally developed a questionnaire, using six motivational questions: ‘How strong is your desire to eat?’ (very weak/very strong); ‘How hungry do you feel?’ (not at all hungry/as hungry as I’ve ever felt); ‘How full do you feel?’ (not at all full/as full as I’ve ever felt); ‘How much do you think you could eat?’ (nothing at all/a large amount); ‘Urge to eat’ (no urge to eat/strong want to eat now, waiting is very uncomfortable); ‘Preoccupation with thoughts of food’ (no thoughts of food/very preoccupied, difficult to concentrate on other things). These questions and slight variations of them have been applied to a large body of appetite research [300].

Reproducibility in adult subjective appetite scores measured consecutively on the same day show high reproducibility [301], but are lower when measured on different days [302, 303].

There are few studies that report the use of VAS as a measure of subjective appetite in children. It has been demonstrated that subjective appetite scores are predictive of lunchtime FI in 9-14 year old boys [5, 304, 305] and meal consumption reduces subjective appetite [304, 306] which indicates that children understand the scales and VAS can accurately capture their feelings. Further supporting its use in children is the observation that ratings of hunger, desire to eat (DTE) and fullness fluctuated with time and nutrition state ($p < 0.01$) after young children were given caloric and non-caloric preloads [306]. While reproducibility of baseline appetite scores is lower in children than adults, a recent study found that the composite appetite score and prospective food consumption (PFC) is highly reproducible 30 min after 9-14 year old boys receive a 50 g glucose preload [307]. On two separate days, boys significantly differed in their within-subject subjective ratings of average appetite when they first arrived at the laboratory after receiving a standardized breakfast. Although this variation is expected, it is unknown if this is because the VAS was able to detect a true biological variation or if methodological issues related to using VAS in children are the reason for the difference between days [307].

2.7.3. Measurement of Short-term Food Intake

There are various protocols for measuring short-term FI. Food intake can be self reported from participants using food diaries, or a more precise quantitative measure can be obtained by weighing food consumed at an accurately monitored test meal. Studies that assess the effects of a specific dietary intervention on the short-term regulation of FI generally follow a preload paradigm [308]. The preload/test-meal paradigm is designed to assess an individuals' ability to detect the caloric load of a preload, and to accurately adjust their energy intake in the following meal (i.e. compensate). Since there is considerable inter-individual variability in FI behaviour, it is most beneficial to follow a within-subject repeated-measures design, in which subjects serve as their own controls [309]. A preload is most commonly defined as an eating occasion (smaller than a test meal and often about 1 MJ in energy value) that is given at a particular interval before the presentation of a test meal [309]. Theoretically, an individual who is responsive to internal cues of satiety will adjust their intake at the meal according to the energy content of the preload. The effect of the test preload is compared with a control preload, which may differ in energy content, macronutrient or ingredient composition, depending on the hypothesis to be tested. Subjective measures of appetite are usually taken prior to, and at regular intervals after the preload and the test meal using VAS. Satiety is also measured by calculating the intake of food at

an *ad libitum* test meal, and this is generally reported in terms of energy [271, 310].

One way to investigate the physiological effect of the preloads is to measure caloric compensation which is a measure of how well individuals reduce their energy intake at a test meal after a treatment preload relative to the control intake [311]. It is calculated by subtracting the calories consumed after the preload from those consumed after the control, dividing by the number of calories in the preload and multiplying by 100 [279, 312]. This generates a percentage where a score of 100% reflects precise (calorie for calorie) compensation. Values less than 100% reflect undercompensation or partial compensation, and values over 100% reflect overcompensation, indicating a reduction in energy at the test meal by an amount that is greater than the energy content of the preload. A negative value would be representative of no compensation (over eating).

2.7.4. Measurement of Body Composition

The accurate assessment of body composition (distinguishing fat from fat-free mass (FFM)) is an important component of obesity and nutrition related research. Direct methods to determine body composition such as cadaver analysis are considered the gold standard for body composition analysis [313]. Among the many techniques available for in vivo applications, DXA is closest to a gold standard. However, for screening purposes, skinfolds, and bioelectrical impedance analysis (BIA) are most desirable because they offer good feasibility and cost and reasonable accuracy [314]. Furthermore, estimates of FM obtained by DXA, BIA, and skinfold thickness are all highly intercorrelated and have high test-retest reliability in children [315]. A description of these three methods, as well as their respective strengths and weaknesses is discussed below.

2.7.4.1. Dual Energy X-Ray Absorptiometry

Dual energy x-ray absorptiometry (DXA) is considered to the clinical standard in randomized controlled trials for assessing body composition [316]. This technique relies on the attenuation of two distinct photon energies as different body tissues absorb them. Various body tissues differ in their attenuation properties and this enables quantification of the three main body compartments: fat mass (FM), bone mineral mass, and fat-free soft tissue; the latter two components can be combined to represent FFM. In addition to determining total body composition, DXA is particularly useful for assessing regional body composition, and it is

considered suitable for use in children as young as 4 years, and infants [313]. This technique is relatively unaffected by hydration status and can be completed quickly, and non-invasively with minimal radiation exposure (1 mSv or < 1/100th of the equivalent radiation exposure of a chest x-ray). Unfortunately DXA technology is expensive, and accuracy is device and software dependent. Interpretation of results is also limited by the lack of quality reference data.

2.7.4.2. Skinfolds

The measurement of skinfolds at selected sites is a widely used anthropometric technique, which allows for the quick determination of body composition without the use of specialized, expensive equipment. In this technique, the thumb and forefinger are used to pull a pinch of skin away from the body, and the width of thickness of the two layers of skin and subcutaneous fat underneath are measured by placing calipers on the fold. Its use depends on two assumptions: that selected skinfold thicknesses are representative of subcutaneous fat, and that there is a defined relationship between subcutaneous body fat and total body fat [317].

Equations have been developed for the purpose of predicting percent body fat from skinfold measurements in the pediatric population [318]. However, it should be noted that differences between the population being studied and those from which the equations were derived compromise its cross-validity [319]. When DEXA was used as a criterion, use of the Slaughter equation was unable to be validated in a heterogeneous group of African-American and Caucasian children [320]. Brook developed a method for estimating FM in children with the following logarithmic equation:

Body fat % of weight = [(4.95/body density) – 4.5] * 100 [321]

The Brook equation was found to predict FM with negligible bias in prepubertal boys compared to hydrodensitometry, which was used as the reference method [322].

2.7.4.3. Bioelectrical Impedance Analysis

Bioelectric impedance analysis (BIA) is a relatively simple and inexpensive method used to predict body composition in children and adults by measuring the impedance or resistance of the body to a small electrical current. This technique is based on the principle that there is a difference in electrical conductive properties of various tissues where conductivity of an electrical impulse is greater through fat-free tissue than fat, which contain a lower percentage of

fluid and electrolytes. Thus, a greater proportion of FFM and total body water will result in less resistance to the electrical flow.

Evidence suggests that BIA is a less precise method for measuring FM in young children compared to skinfold measurements [79]. In eleven, 9-14 year old boys, BIA underestimated FM by approximately 4 kg compared with skinfold measurements [307]. The generic theoretical model views the body as single cylinder and measurements are made between electrodes placed on the wrist and ankle [313]. This adjustment for height allows total body water to be estimated using regression equations, which in turn calculate FFM. There are several challenges associated with the accuracy of this method as the association between BIA values and total body water is influenced by the characteristics of the population being assessed [313]. Considering that body build is fairly consistent over short time periods in growing children, BIA may be used to indicate the direction of change in FFM but it is unlikely to accurately quantify the magnitude of these changes [313]. Consequently, BIA may not be a reliable method for estimating body composition in children.

2.8. Summary

The popularity of VGP is a recent environmental change that has independently been associated with obesity. This activity is hypothesized to promote weight gain by both displacing time that is dedicated to physical activity and increasing energy intake. Boys spend a substantial amount of time VGP, and this typically occurs outside of mealtime, but it is unknown if FI at mealtime is affected by prior use of this activity or if physiological signals in the regulation of FI are compromised. A greater understanding of the interaction between VGP and the physiological mechanisms that affect eating behaviour is needed to inform guidelines on the optimal use of video games. Therefore, the objective of this research was to compare the effect of VGP for 30 min to sitting on short-term FI, subjective appetite, satiation and satiety signals from a glucose preload in 9-14 year old NW boys.

Chapter 3. Hypothesis & Objective

3.1. Hypothesis

Video game playing for 30 min increases subjective appetite, reduces satiation, and diminishes satiety signals from a glucose preload at a test meal consumed immediately after in 9-14 year old NW boys.

3.2. Objective

To determine the effect of video game playing for 30 min before a mixed meal on subjective appetite, satiation and satiety signals from a glucose preload in NW boys.

Chapter 4. Methods

4.1. Experimental Design

A within-subject 2x2 repeated measures factorial design was used to examine the effect of video game playing (VGP) vs. no VGP and preload treatment on FI (glucose vs. control). The subjects were randomly assigned to a particular treatment order, which was balanced. On four separate mornings, in random order, 7 d apart, subjects arrived at Mount Saint Vincent University (MSVU), 2 h after a standard breakfast of milk, cereal, and orange juice. The subjects consumed equally sweetened drinks of a SPLENDA® Sucralose control or 50 g of glucose made up to 250 mL of water (within 5 min) prior to VGP (Sony® PlayStation 3) or sitting quietly for 30 min. Immediately following each treatment condition, participants were escorted to the feeding room, where they were seated in individual cubicles and were provided with an *ad libitum* pizza meal. Subjective appetite was measured at regular intervals during the study.

4.2. Subjects

Twenty-three NW 9- 14 y old boys who were born at full term (37-42 weeks gestational age) and normal birth weight (2500-4499 g [323]) participated in the study, 19 of which were included in the final analysis. Of the 23 boys who participated, two dropped out voluntarily, and one was excluded from the analysis based on outlier criteria. In addition, a set of identical twins participated in the study, and one brother was excluded at random from the analysis because they were expected to respond similarly to the caloric and VGP treatments. Children were primarily recruited through word-of-mouth, as well as advertisements in the Metro newspaper, online classifieds (i.e. Kijiji) and community locations in the Halifax Regional Municipality **(Appendices 9.1. and 9.2.)**. In addition, participants from past studies conducted in the lab were contacted.

Children were not included in the study if they had food allergies to wheat, milk, or nuts, were dieting, taking medication or had any significant learning, behavioral or emotional difficulties. It was also mandatory that the included boys approved of the standardized breakfast and lunch and were willing to consume them. Information pertaining to the inclusion criteria was obtained through a two-step screening process that was completed with each child and his parent to confirm the participant's qualification for the study.

Initially, parents who volunteered their children completed, by telephone, a semi-structured interview **(Appendix 9.5.)** that was designed to assess whether the child met the inclusion criteria. If a child met the study requirements, an in person screening session was arranged for

the parent and child at the Department of Applied Human Nutrition. During the screening session, a research assistant explained the details of the study to the child and parent. Additionally, anthropometric measures were obtained to determine BMI and estimate FM. These measurements included height (m), weight (kg), and sum of 4 skinfolds (mm). Height and weight were measured using a calibrated, standard medical scale (Detecto, Webb City, Missouri, U.S.A) and the measurements were subsequently used to calculate BMI [weight (kg)/height^2 (m^2)]. Because there are no Canadian BMI references for children, CDC BMI charts are recommended for monitoring individual Canadian children [324]. Using CDC growth charts, NW was classified as having a BMI between the 5th and 85th percentile. Skinfold thickness was determined using the sum of 4 skinfolds measured with the Harpenden skinfold caliper at the triceps, biceps, supra-ilial and subscapular locations. For greatest accuracy, each skinfold site was measured three times, and the average skinfold was calculated to the nearest 0.1 mm. The sum of 4 skinfold measurements were used to estimate percent FM from a sex specific regression equation.

The screening session served as an opportunity for the boys to become familiar with the test instruments used during the four study sessions. They were also asked to complete the Dutch Eating Behaviour Questionnaire (DEBQ) **(Appendix 9.8.)**, and rank their preference for the two types of pizza (pepperoni and three-cheese) that were served at the test meal during the sessions. If the subject met all the inclusion criteria, both informed consent, which was signed by the parent **(Appendix 9.3.)** and assent, which was signed by the child **(Appendix 9.4.)** were obtained.

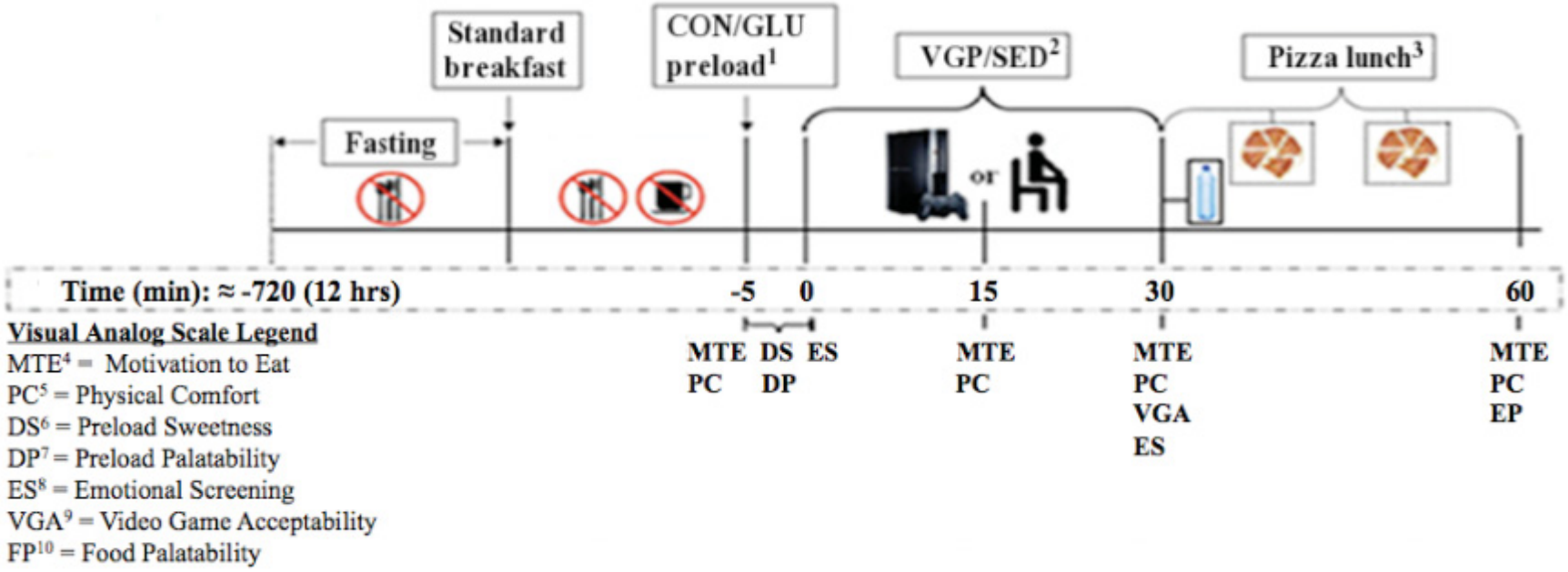
4.3. Experimental Procedure

Boys were instructed to adhere to an overnight fast, initiated 12 h prior to their participation in each of the study sessions, which were scheduled on four separate weekend mornings, 7 d apart. The morning after the fasting period (starting at 0800 or 0900 h), boys consumed the entire contents of a standardized breakfast, which consisted of Parmalat fat-free skim milk, (250mL, 91 Kcal), Honey Nut Cheerios, (26g, 103 Kcal), and Tropicana orange juice (236mL, 110 Kcal) at home. During the 2 h period after breakfast until their arrival, they abstained from eating, and participating in screen activities. Water was allowed up to 1 h before arriving for the study. Subjects arrived at the Department of Applied Human Nutrition, Mount Saint Vincent University (1000 or 1100 h) at the start of each study session.

For each session, the child arrived at a consistent time (i.e. always 1000 h). Upon arrival, the boys were asked if they consumed the entire breakfasts, if any other foods were consumed 13 hours prior to arrival and if they were taking any medication **(Appendix 9.7.).** If they reported significant deviations from their usual patterns, they were asked to reschedule. Participants were required to fill out a baseline VAS assessing motivation to eat **(Appendix 9.6a.)** and physical comfort (**Appendix 9.6e.**). After completion of baseline VAS, boys were given either a non-caloric control drink or a glucose preload drink, which had to be consumed within a 5 min period. Following consumption of the beverage, the boys completed two separate VAS to assess sweetness **(Appendix 9.6d.)** and palatability of the preload **(Appendix 9.6b.)**.

Immediately following completion of the VAS, the boys completed one of the test sessions. The video game condition consisted of playing the mini-game *Angry Birds* (Rovio Entertainment Inc) on Sony PlayStation®3, while the sedentary condition involved sitting quietly. Two additional questionnaires were administered at 30 min to assess subjective enjoyment of the video game **(Appendix 9.6f.)** and emotional response to the video game **(Appendix 9.6g.)**. Upon completion of the VAS, the boys were escorted to a feeding room where they were served an *ad libitum* pizza lunch for a minimum period of 30 min. A freshly baked tray of pizza was provided to the subjects starting at 30 min and every 8-10 minutes thereafter. The children were instructed to eat until they were "comfortably full". After a fresh tray was provided, the previous tray was removed. Upon completion of the test meal, the boys completed three final VAS to assess appetite, physical comfort and palatability of the test meal.

Figure 4.1. Test Day Procedure



[1] 250 mL of a control (non-caloric sweetener) or glucose (50 g) preload was given in an opaque covered mug with a straw and consumed within 5 min followed by 100 mL of water

[2] Video game playing or sedentary condition was assigned in random order

[3]A 500 mL bottle of spring water was provided with the test meal and replaced with a new full bottle once emptied

[4]Motivation-to-Eat VAS were administered to determine subjective appetite and thirst

[5]Physical Comfort VAS were administered to determine subjective physical well-being

[6]Preload Sweetness VAS were administered to determine subjective sweetness of the preload

[7]Preload Palatability VAS were administered to determine subjective palatability of the preload

[8] Emotional Screening VAS were administered to determine subjective emotional response to the video game and sitting

[9] Video Game Acceptability VAS were administered to determine subjective enjoyment of the video game

[10] Food Palatability VAS was administered to determine subjective palatability of the test meal

4.3.1. Video Game Playing Protocol

During the video game condition, the boys engaged in playing *Angry Birds* on a video game console (Sony PlayStation 3®) that was connected to a television screen for 30 min. A research assistant was present during the activity to ensure the game was played for the entire duration, and game play was only interrupted to ensure the VAS was completed halfway through the interval and at the end.

4.3.2. Sedentary Protocol

In the sedentary condition, the boys were required to sit quietly for 30 min. They were not allowed to participate in any form of entertainment, including reading, games or cellphone use because the cognitive demand associated with these activities have the potential to effect appetite and FI [232]. Similar to the VGP condition, the sedentary condition was fully supervised to ensure protocol was being followed properly. The research assistant encouraged conversation but deterred the boys from talking about anything related to food or eating.

4.3.3. Food Intake Protocol

Immediately following completion of either the video game or sedentary condition, and 30 min after consumption of the control or glucose preload, the subjects filled out two VAS which measured appetite and physical comfort, and two VAS which measured enjoyment of the video game and emotional response to the video game during the two experimental sessions where the video game was played. After this, each boy was escorted to a cubicle free of external cues in the feeding room. While sitting in the cubicle, the boys were served an *ad libitum* pizza meal (Deep 'N Delicious, McCain Canada Ltd., Florenceville, Canada) of the variety selected by the subject at the screening accompanied by a 500 mL bottle of spring water (Danone Crystal Springs). Subjects were instructed to eat until they were "comfortably full" and were given a fresh hot tray of three pizzas at the start of the meal and again at 8-10 min intervals after initiation of the meal. When a new tray was given, the old tray was removed. All the boys were given a minimum of 3 trays of pizza. The end time of the meal may have varied, depending on the child because if the subject was still eating at the end of this period, one additional tray was served in order to detect a difference in FI between sessions. Thus, if the subject appeared to be still eating after 30 min, or expressed a desire for an additional tray, a fourth tray was served and if the subject expressed no more interest in eating, the meal session ended. Immediately after the

meal terminated, the boys were moved to another room where they filled out VAS that assessed appetite, physical comfort and pleasantness of the pizza meal **(Appendix 9.6c.)**.

4.4. Test Measurements

4.4.1. Preload treatment

Each participant received a control or glucose solution with or without VGP over the course of four sessions, and the order of the treatments was randomly determined. The preloads were matched for sweetness and flavor; they only differed in the source of sweetener, which was added to 250 mL of spring water. The caloric preload contained glucose (50 g in the form of glucose monohydrate; Grain Process Enterprises, Toronto, Ontario, Canada) while the non-caloric preload contained 150 mg of SPLENDA® Sucralose because it is not metabolized in the body and does not alter BG or insulin secretion [325]. In a previous study, the same preloads were pretested in a group of NW boys of the same age, and they were not found to be significantly different in sweetness [5]. In addition, 1.1 g of aspartame-sweetened, orange-flavoured crystals (Sugar Free Kool-Aid, Kraft Canada Inc., Don Mills, Ontario, Canada) was added to each preload treatment to standardize the flavour. Test preloads were prepared the day before they were consumed in covered, opaque cups and refrigerated. Subjects consumed the chilled drinks, followed by 100 mL of water to minimize aftertaste, in less than 5 min.

4.4.2. Food Intake

Two varieties of McCain Foods Deep 'N Delicious 5" diameter pizza were used as the test meal: pepperoni and three-cheese. Each tray of pizza contained two pizzas of the participant's first choice and one of their second choice. The pizzas were cooked in the Foods Laboratory at Mount Saint Vincent University, weighed on a digital food scale and cut into 4 equal pieces prior to serving. Children were instructed that additional hot tray replacements would be presented in 10 min intervals. An advantage of using these pizzas is the lack of crust, which results in a pizza with a more uniform energy content and the elimination of the possibility of subjects eating the denser filling and leaving the crust of the pizza. The two varieties of pizza were comparable in caloric and macronutrient content. Each pepperoni pizza (87 g) contained 9 g of protein, 6 g of fat, and 23 g of carbohydrates for a total energy content of 180 kcal. Similarly, each three-cheese pizza (81 g) contained 9 g of protein, 6 g of fat and 22 g of carbohydrate for a total energy content of 180 kcal. The amount left after the meal was

subtracted from the initial weight to provide a measure of FI. Each variety of pizza was weighed separately and the energy consumed (in kcal) was calculated by converting the net weight consumed to kcal consumed by use of information provided by the manufacturer (McCain Food Ltd.).

4.4.3. Water Intake

Participants were provided with a 500 mL bottle of spring water (Danone Crystal Springs) at the start of the test meal. If the entire volume of water was consumed during the test meal, it was replaced with an additional bottle. The amount of water ingested during the test meal period was calculated by weighing the water before and after the test meal.

4.4.4. Subjective Sweetness

The sweetness VAS were used to assess the subject's enjoyment of the preload by asking the question 'How sweet have you found the beverage?' with a corresponding range of 'Extremely sweet' to 'Not sweet at all' [5, 278, 304, 305, 307, 326]. The VAS used to measure participants' subjective sensations, feelings, and opinions consisted of a question that preceded a corresponding answer that consisted of two opposing statements at either end of a 100 mm line. Subjects responded to each question by marking an "X" on the location of the line that most closely represented their feelings at that moment. Scores were determined by measuring the distance (mm) from the left starting point to the intersection of the "X".

4.4.5. Subjective Pleasantness

The palatability VAS were used to assess each subject's enjoyment of the preload and meal by asking the questions 'How pleasant have you found the preload?' and 'How pleasant have you found the food?' with both questions corresponding to a range of 'Very pleasant' to 'Not at all pleasant' [5, 278, 304, 305, 307, 326, 327]. The results were analyzed to determine if preload treatment or VGP were factors that affected subjective palatability, and were reported as means (mm) ± SEM.

4.4.1. Subjective Physical Comfort

The physical comfort VAS were used to assess each subject's well being by asking the subject 'How well do you feel?' with a corresponding range of 'Not well at all' to 'Very well' [5, 278, 304, 305, 307, 326, 327]. Information on physical comfort was collected at baseline because it was imperative that the boys felt well during the sessions in order to gain meaningful results. A low physical comfort score would have indicated they should not participate in the session. Physical comfort scores were analyzed to determine if preload treatment or VGP were factors that affected physical comfort, which were reported as the change from baseline (physical comfort at 30 min - physical comfort at 0 min).

4.4.1. Video Game Acceptability

The video game acceptability was used to assess the subject's enjoyment of the video game by asking the question 'How much did you enjoy the video game' with a corresponding range of 'Not well at all' to 'Very well.' Pearson correlation coefficient was calculated to correlate this VAS with dependent measures.

4.4.2. Subjective Appetite

Subjective appetite and thirst were assessed using a motivation to eat VAS, which consisted of five questions:

How strong is your desire to eat? ('Very weak' to 'Very strong')
How hungry do you feel? ('Not hungry at all' to 'As hungry as I've ever felt')
How full do you feel? ('Not full at all' to 'Very full')
How much food do you think you could eat? ('Nothing at all' to 'A large amount')
How thirsty do you feel? ('Not thirsty at all' to 'As thirsty as I have ever felt')

To determine an average appetite (AA) score, DTE, hunger and PFC (ie. How much food do you think you can eat) as well as 100 minus fullness were added and divided by four [AA (mm) = [DTE + hunger + (100 – fullness) + PFC)/4]. This VAS, without the question regarding thirst, as well as the calculation for AA has been used previously [5, 304, 305, 307, 326].

This VAS provided important additional information concerning the impact of the caloric

preload used in the study on satiety. Subjective appetite scores were analyzed to determine if they were predictors of FI.

4.4.3. Subjective Emotions

The emotion screening VAS was used to assess the immediate impact of video game playing on each subject's current emotions and it consisted of seven questions:

How aggressive do you feel? ('Not aggressive at all" to 'Very aggressive')
How angry do you feel? ('Not angry at all' to 'Very angry')
How excited do you feel? ('Not excited at all' to 'Very excited')
How disappointed do you feel? ('Not disappointed at all' to 'Very disappointed')
How happy do you feel? ('Not happy at all' to 'Very happy')
How upset do you feel? ('Not upset at all' to 'Very upset')
How frustrated do you feel? ('Not frustrated at all' to 'Very frustrated')

This VAS was administered because emotions have the ability to impact FI. Scores were analyzed to determine if emotions decreased or increased from the beginning of each treatment to the end. They were also correlated with subjective appetite and FI.

4.4.4. Estimation of Fat Mass

A Harpenden skinfold caliper was used to measure skinfolds at four sites (triceps, biceps, supra-ilial, and subscapular) and recorded to the nearest 0.1 mm [328] The mean skinfold measurements at each site was used for estimation of percent body fat. Body density was first calculated from sex specific regression equations (Equation 1) [329] and then applied to another calculation (Equation 2) to determine percent body fat [321], as previously used by us and others in children [5, 6, 304, 305, 307, 326].

Equation 1: Density = 1.1533 – 0.0643 * log sum of skinfold thicknesses at 4 sites
Equation 2: Body fat % of weight = [(4.95/body density) – 4.5] * 100

4.4.5. Dutch Eating Behaviour Questionnaire

The DEBQ [330] was first developed by Van Strien and colleagues in 1986 [330], and it consists of 33 items that assess three factors: (1) restrained eating (2) emotional disinhibition, and (3) external disinhibition. It has previously been used and reported in children of the same age [7, 322, 323, 338, 349]. Emotional disinhibition is divided into two additional factors: (a) loss of cognitive control of eating due to specific emotions (i.e. an individual may be upset because of something bad that recently happened) or (b) loss of cognitive control of eating due to diffused emotions (i.e. eating due to boredom). Dietary disinhibition is defined by the combination of emotional and external disinhibition. Externality theory, Psychosomatic theory, and Restraint Eating theory postulate that these three factors may have an effect on FI, which is a main dependent variable in the study [331-333]. In a series of studies used to develop and assess use of the DEBQ, Van Strien and colleagues [330] reported that the three scales have a high internal consistency as well as a high factorial validity. Cronbach's alpha coefficients reflected adequate internal consistency for obese and non-obese men and women (range: 0.80 – 0.95). Furthermore, correlation coefficients for items within each scale were high [330].

4.4.6. Ethical Considerations

Ethics approval for this study was obtained from the Mount Saint Vincent University Research Ethics Board (UREB File # 2011- 014). Parental consent and child's assent for participation was obtained at the initial screening session before beginning experimental sessions. Use of a code and a number to identify all subjects in documents, records and files ensured confidentiality was maintained. All data pertaining to the study were entered into Microsoft Excel files and were available to the primary investigators. All records relating to subjects were kept confidential in a locked cabinet in the Department of Applied Human Nutrition (Evaristus 365) at Mount Saint Vincent University. No disclosure of personal information of subjects or parents/guardians took place except if required by law. All documents pertaining to the study will be kept for a minimum of five years following completion of the study and will then be securely destroyed.

4.5. Statistical Analysis

Statistical analyses were performed using SAS, version 9.2 (SAS Institute Inc, Cary, NC, USA). All data are reported as mean ± SEM (standard error of the mean) and results were considered significant at $p \leq 0.05$.

Treatment and condition effects on food and water intake, cumulative food intake, sweetness and pleasantness were analyzed using a 2-factor MIXED MODEL in SAS with preload treatment, and VGP as main factors. The 3-factor MIXED MODEL procedure was used to assess the effect of preload treatment, VGP and time on subjective appetite scores (AA, DTE, hunger, fullness, PFC), subjective emotion scores (anger, aggression, excitement, disappointment, happiness, frustration, upset) as well as physical comfort and thirst. Subjective appetite scores, subjective emotion scores, physical comfort and thirst are reported as absolute values. Subjective appetite, subjective emotion and physical comfort scores are also expressed as change from baseline, which was done to help control for the variation in subjective appetite upon arrival to test sessions. Change from baseline scores were calculated by subtracting baseline scores from pre-meal scores at 15, and 30 minutes. Student's paired t-tests were used to analyze caloric compensation, and enjoyment of the video game. When statistically significant differences in main effects or interactions were found, post-hoc analysis using Tukey-Kramer's test, adjusted for multiple comparisons was performed.

Pearson correlation coefficients were reported to assess associations between dependent measures and FI including: absolute subjective appetite scores (AA, DTE, hunger, fullness, PFC, thirst), subjective emotion scores (anger, aggression, excitement, disappointment, happiness, frustration, upset), preload sweetness, preload pleasantness, body composition (BW, FM, FFM), dietary restraint, disinhibition and emotional eating.

Caloric compensation, a measure of how well subjects reduced their FI at the test meal after the glucose solution relative to the control, was calculated using the following formula as reported previously [5, 304, 305, 307, 326].

Caloric compensation (%) = [Control intake (kcal) – Treatment intake (kcal) / kcal in Treatment preload] x100

Chapter 5. Results

5.1. Subject Characteristics

Nineteen NW boys (mean ± SEM; 12.0 ± 0.5 y) with a mean BMI percentile of 59.5 ± 5.3 were included in this study. Baseline characteristics, including fat mass (9.6 ± 0.9 kg), fat-free mass (34.6 ± 2.2 kg), and DEBQ average score (2.1 ± 0.1) are reported in **Table 5.1.**

Table 5.1. Baseline Characteristics for Test Participants

Subject Characteristics	Subjects
Age (y)	12.0 ± 0.5
Body Weight (kg)	44.1 ± 2.9
Height (m)	151.9 ± 3.6
BMI (kg/m^2)	18.7 ± 0.5
BMI Percentile	59.5 ± 5.3
Fat Mass (kg)[1]	9.6 ± 0.9
Fat Mass (%)[1]	21.3 ± 1.2
Fat-Free Mass (kg) [1]	34.6 ± 2.2
Fat-Free Mass (%)[1]	78.7 ± 1.2
DEBQ Average Score[2]	2.1 ± 0.1
Restraint[2]	1.8 ± 0.1
Overall Disinhibition[2]	2.2 ± 0.1
Diffuse Emotional Disinhibition[2]	1.8 ± 0.2
Specific Emotional Disinhibition[2]	1.7 ± 0.2
External Disinhibition[2]	2.9 ± 0.1

Data are presented as mean ± SEM, n = 19. Abbreviations: BMI, body mass index; DEBQ, Dutch Eating Behavior Questionnaire. [1]FM and FFM were determined from the sum of skinfold measurements at four points. [2]Dietary restraint and disinhibition were assessed using the DEBQ.

5.1.1. Food Intake

Preload treatment ($p < 0.0001$) and VGP ($p = 0.05$) affected FI but there was no preload x VGP interaction ($p = 0.24$). FI was reduced by the glucose preload compared to the control treatment ($p < 0.0001$), and VGP resulted in lower FI compared with the sitting control condition ($p = 0.05$). The lower FI during the VGP condition was due to an overall combined decrease in FI after the control ($p = 0.12$) and glucose ($p = 0.92$). Cumulative FI (preload + meal) was affected by VGP ($p = 0.05$) but not preload treatment ($p = 0.38$) and there was no preload treatment x VGP interaction ($p = 0.24$). **(Table 5.2).**

5.1.2. Water Intake

Water Intake (WI) at the test meal was not affected by preload treatment ($p = 0.22$) or VGP ($p = 0.67$), and there was no preload treatment x VGP interaction ($p = 0.08$) (**Table 5.2**).

5.1.3. Subjective Sweetness

Subjective sweetness of the test beverages was affected by preload treatment ($p = 0.04$) but not VGP ($p = 0.89$) and there was no preload treatment x VGP interaction ($p = 0.77$) **(Table 5.2).**

5.1.4. Preload Pleasantness

Subjective pleasantness of the test beverages was affected by preload treatment ($p < 0.0001$) but not VGP ($p = 0.40$), and there was no preload treatment x VGP interaction ($p = 0.14$). In the sitting control condition, the control beverage was perceived to be no different than the glucose beverage ($p = 0.07$), but less pleasant than the glucose beverage in the VGP condition ($p = 0.03$). In the VGP condition, the control beverage was perceived to be less pleasant than the glucose beverage ($p < 0.001$), and less pleasant than the glucose beverage in the sitting control condition ($p < 0.01$) **(Table 5.2).**

5.1.5. Subjective Physical Comfort

Physical comfort (PC) scores were not affected by preload treatment ($p = 0.77$), VGP ($p = 0.60$), or time ($p = 0.33$). There was no preload treatment x VGP interaction ($p = 0.89$), preload treatment x time interaction ($p = 0.73$), VGP x time ($p = 0.79$), or preload treatment x VGP x time ($p = 0.38$). **(Figure 5.1).**

Change from baseline PC scores were not affected by preload treatment ($p = 0.20$), VGP ($p = 0.09$) or time ($p = 0.55$). There were no interactions from preload treatment x VGP ($p =0.43$), preload treatment x time ($p = 0.67$), VGP x time ($p = 0.89$), or preload treatment x VGP x time ($p = 0.25$). **(Figure 5.2).**

5.1.6. Video Game Acceptability

The video game was rated as acceptable during the control treatment (80 ± 5) and the glucose treatment (81 ± 5) and video game acceptability did not differ between the test conditions ($p = 0.51$) **(Table 5.2).**

5.1.7. Caloric Compensation

Caloric compensation did not differ due to VGP ($p = 0.25$) **(Table 5.2).**

5.1.8. Test Meal Acceptability

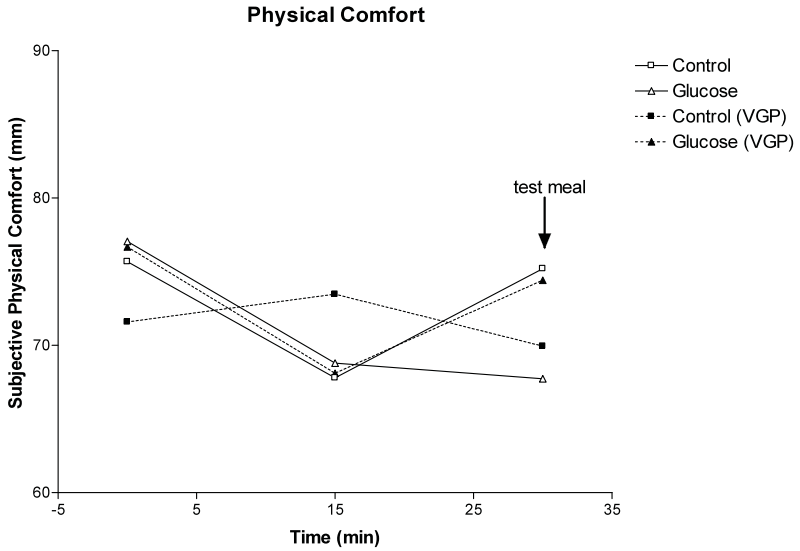
Test meal acceptability scores were not affected by preload treatment ($p = 0.46$), or VGP ($p = 0.48$), and there was no preload treatment x VGP interaction ($p = 0.73$). **(Tables 5.2).**

Table 5.2. Effect of Preload Treatments and Video Game Playing on Test Meal Intake, Caloric Compensation, and Subjective Measures Before and During Test Meal

	Control	Glucose	Control (VGP)	Glucose (VGP)
FI[1] (kcal)	977 ± 54[a]	769 ± 69[c]	898 ± 66[ab]	749 ± 65[c]
FI+Preload[2] (kcal)	977 ± 54	969 ± 69	898 ± 66	949 ± 65
Water Intake (g)	203 ± 35	215 ± 39	233 ± 41	166 ± 32
Sweetness (mm)	81 ± 4	76 ± 3	82 ± 4	76 ± 5
Preload Pleasantness (mm)	39 ± 6[ab]	55 ± 6[c]	28 ± 6[a]	58 ± 6[c]
Physical Comfort (mm)	72 ± 3	72 ± 2	73 ± 3	73 ± 3
Videogame Acceptability (mm)	—	—	80 ± 5	81 ± 5
Caloric Compensation[3] (%)	—	104 ± 17	—	75 ± 19
Test Meal Acceptability	78 ± 5	74 ± 5	74 ± 5	73 ± 5

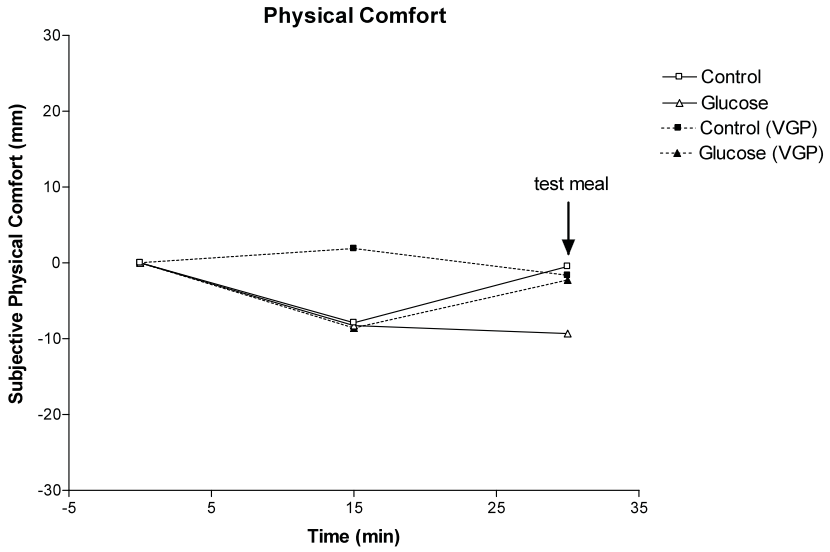
Treatment effects were analyzed using the PROC MIXED procedure with treatment and video game playing as main factors. Data are presented as means ± SEM; n = 19. Means in a row with different letters differ at $p < 0.05$ (Tukey's Test). Abbreviations: VGP, video game playing.
[1]Pizza intake at test meal. [2]Cumulative energy intake from test meal and preload (kcal).
[3] CC (%) = [control intake (kcal) – treatment intake (kcal)/ kcal in treatment preload] x 100

Figure 5.1. Absolute Physical Comfort After Preload Treatments and Video Game Playing to 30 Minutes



Subjective physical comfort at 0, 15, and at 30 min. Values are means, n = 19 per group.

Figure 5.2. Change from Baseline Physical Comfort After Preload Treatments and Video Game Playing to 30 Minutes



Change from baseline physical comfort at 0, 15, and at 30 min. Values are means, n = 19 per group.

5.2. Subjective Appetite Scores

5.2.1. Average Appetite

Absolute average appetite (AA) scores were affected by time ($p = 0.003$), but not preload treatment ($p = 0.16$) or VGP ($p = 0.27$), and there were no significant interactions. (**Figure 5.3a**).

Change from baseline AA scores were affected by preload treatment ($p = 0.01$), and time ($p = 0.003$) but not VGP ($p = 0.64$), and there was no significant interactions (**Figure 5.4a**).

5.2.2. Desire to Eat

Absolute desire to eat (DTE) scores were affected by VGP ($p = 0.05$), and time ($p = 0.05$) but not preload treatment ($p = 0.11$), and there were no significant interactions. (**Figure 5.3b**).

Change from baseline DTE scores were affected by time ($p = 0.01$) but not preload treatment ($p = 0.42$) or VGP ($p = 0.69$), and there was no significant interactions (**Figure 5.4b**).

5.2.3. Hunger

Absolute hunger scores were affected by time ($p = 0.02$), but there was no effect of preload treatment ($p = 0.18$) or VGP ($p = 0.33$), and there were no significant interactions. (**Figure 5.3c**).

Change from baseline hunger scores were affected by time ($p = 0.02$), and preload treatment ($p = 0.01$), but not VGP ($p = 0.83$), and there was no significant interactions (**Figure 5.4c**).

5.2.4. Fullness

Absolute fullness scores were affected by time ($p = 0.009$), but there was no effect of preload treatment ($p = 0.23$) or VGP ($p = 0.42$), and there were no significant interactions. (**Figure 5.3d**).

Change from baseline fullness scores were affected by time ($p = 0.008$), and preload treatment ($p = 0.01$) but not VGP ($p = 0.17$), and there was no significant interactions (**Figure 5.4d**).

5.2.5. Prospective Food Consumption

Absolute prospective food consumption (PFC) scores were affected by time ($p = 0.02$), but there was no effect of preload treatment ($p = 0.26$) or VGP ($p = 0.18$), and there were no significant interactions (**Figure 5.3e**).

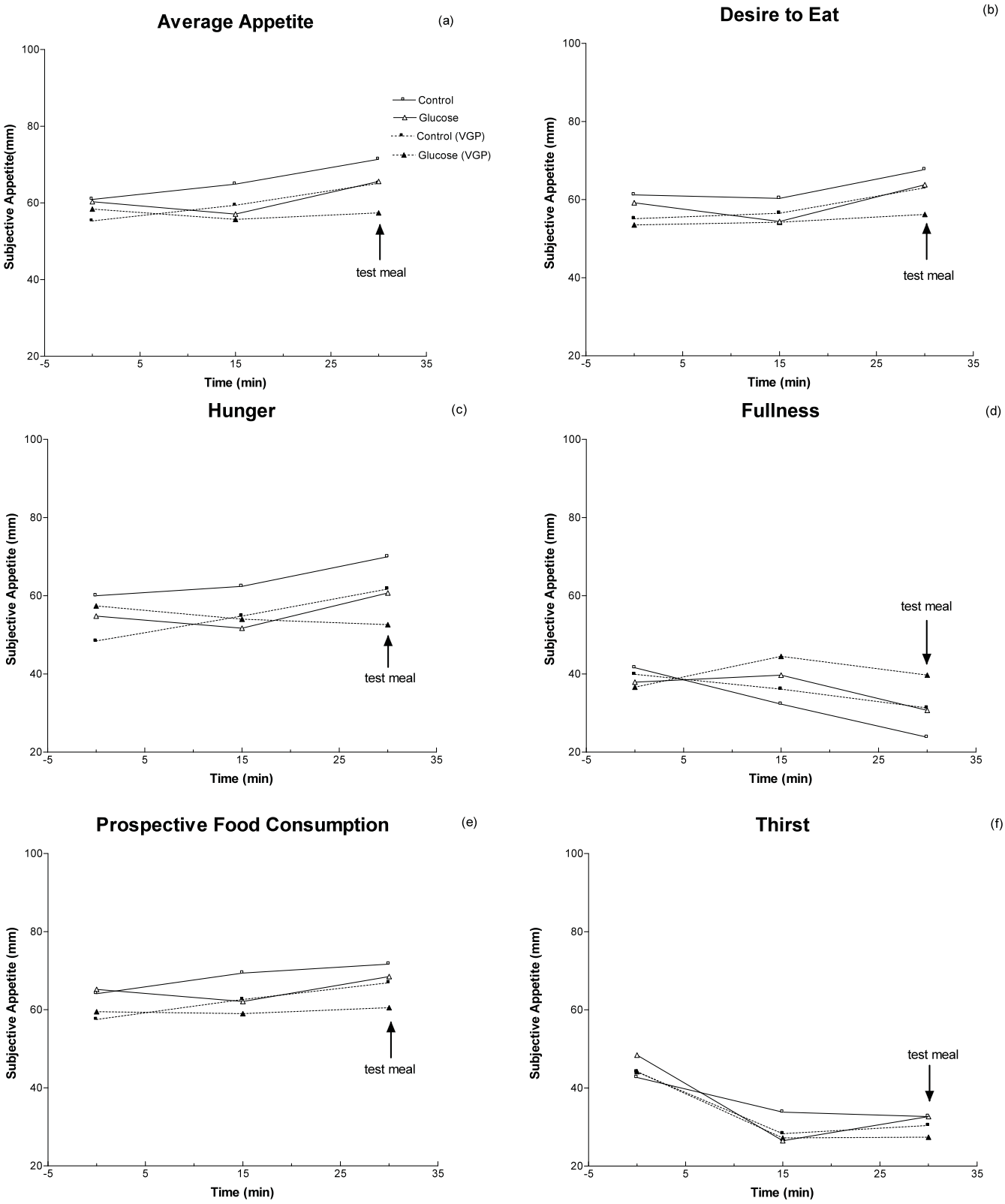
Change from baseline PFC scores were affected by time ($p = 0.04$), and preload treatment ($p = 0.02$) but not VGP ($p = 0.88$), and there were no significant interactions. (**Figure 5.4e**).

5.2.6. Thirst

Absolute thirst scores were affected by time ($p < 0.0001$) but not preload treatment ($p = 0.70$), or VGP ($p = 0.31$), and there were no significant interactions (**Figure 5.3f**).

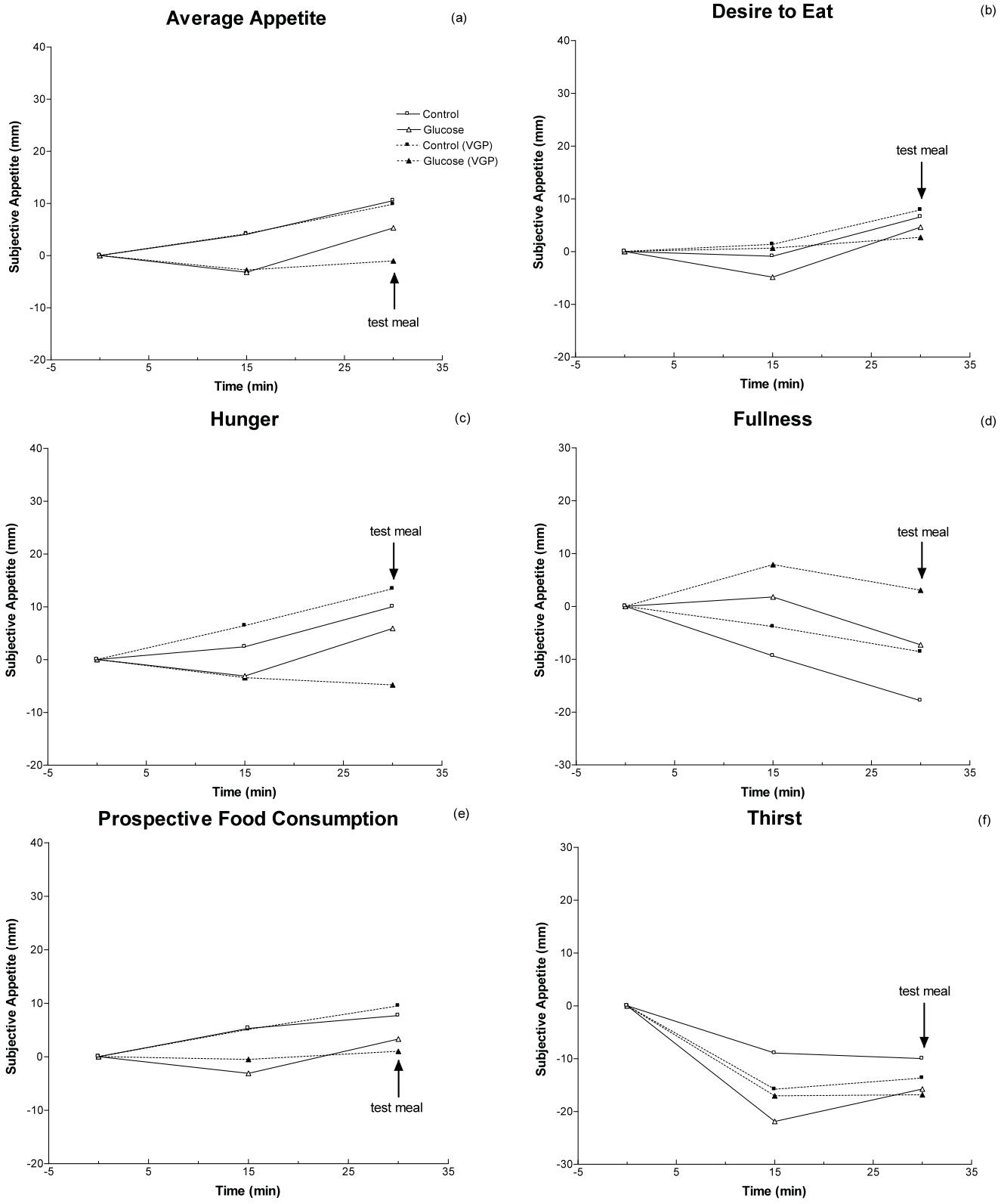
Change from baseline thirst scores were not affected by preload treatment ($p = 0.13$), VGP ($p = 0.76$), or time ($p = 0.36$), and there were no significant interactions (**Figure 5.4f**).

Figure 5.3. Absolute Subjective Average Appetite After Preload Treatments and Video Game Playing to 30 Minutes



Appetite ratings for (a) AA, (b) DTE, (c) hunger, (d) fullness (e) PFC and (f) thirst at 0, 15, and 30 min. Values are means, n = 19 per group. Average and individual appetite scores changed over time ($p < 0.001$).

Figure 5.4. Change from Baseline Subjective Average Appetite After Preload Treatments and Video Game Playing to 30 Minutes



Change from baseline appetite ratings for (a) AA, (b) DTE, (c) hunger, (d) fullness, (e) PFC and (f) thirst at 0, 15, and 30 min. Values are means, n = 19 per group. Average and individual appetite scores changed over time ($p < 0.001$)

5.3. Subjective Emotions Scores

5.3.1. Aggression

Absolute aggression scores were not affected by preload treatment ($p = 0.30$), VGP ($p = 0.66$), or time ($p = 0.91$), and there were no treatment x VGP interaction (**Figure 5.5a**).

Change from baseline aggression scores were affected by VGP ($p = 0.048$), but not preload treatment ($p = 0.88$), and there was no preload treatment x VGP interaction ($p = 0.69$) (**Figure 5.6a**).

5.3.2. Anger

Absolute anger scores were not affected by preload treatment ($p = 0.69$), VGP ($p = 0.13$), or time ($p = 0.29$), and there were no significant interactions ($p = 0.29$) (**Figure 5.5b**).

Change from baseline anger scores were not affected by preload treatment ($p = 0.84$) or VGP ($p = 0.87$), and there was no preload treatment x VGP interaction ($p = 0.26$) (**Figure 5.6b**).

5.3.3. Disappointment

Absolute disappointment scores were not affected by preload treatment ($p = 0.96$), VGP ($p = 0.90$) or time ($p = 0.31$). There was a preload treatment x VGP interaction ($p < 0.05$) (**Figure 5.5c**).

Change from baseline disappointment scores were not affected by preload treatment ($p = 0.99$) or VGP ($p = 0.21$), and there was no preload treatment x VGP interaction ($p = 0.78$) (**Figure 5.6c**)

5.3.4. Excitement

Absolute excitement scores were not affected by preload treatment ($p = 0.66$), VGP ($p = 0.64$), or time ($p = 0.60$), and there were no significant interactions (**Figure 5.5d**).

Change from baseline excitement scores were not affected by preload treatment ($p = 0.39$) or VGP ($p = 0.65$), and there was no preload treatment x VGP interaction ($p = 0.87$) (**Figure 5.6d**).

5.3.5. Frustration

Absolute frustration scores were affected by VGP ($p < 0.05$), but not preload treatment ($p = 0.32$) or time ($p = 0.05$) and there were no significant interactions **(Figure 5.5e).**

Change from baseline frustration scores were affected by VGP ($p < 0.05$), but not preload treatment ($p = 0.71$), and there was no preload treatment x VGP interaction ($p = 0.33$) **(Figure 5.6e).**

5.3.6. Happiness

Absolute happiness scores were not affected by preload treatment ($p = 0.34$), VGP ($p = 0.93$), or time ($p = 0.26$), and there were no significant interactions (**Figure 5.5f**).

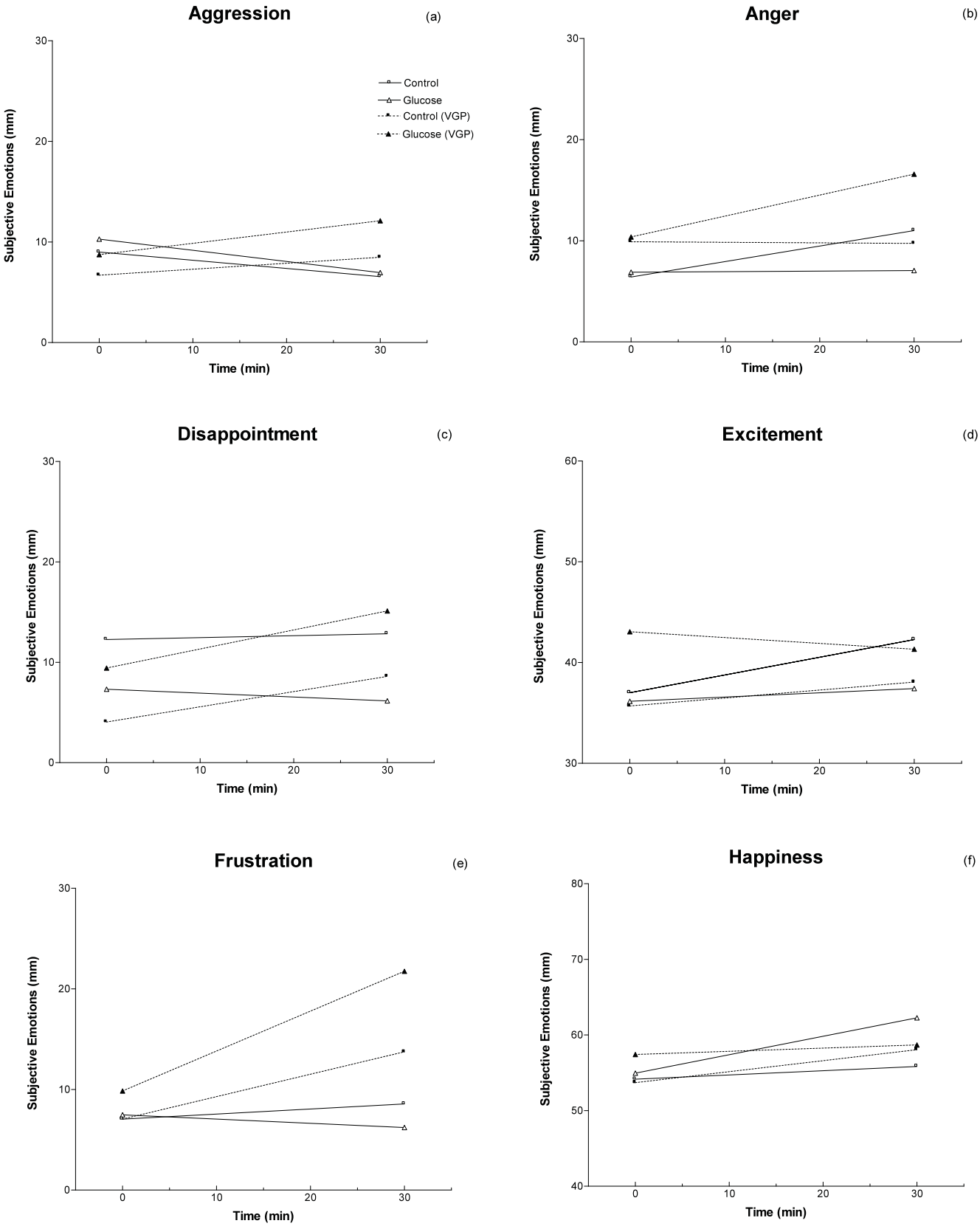
Change from baseline happiness scores were not affected by preload treatment ($p = 0.69$) or VGP ($p = 0.76$), and there was no preload treatment x VGP interaction ($p = 0.27$) (**Figure 5.6f**).

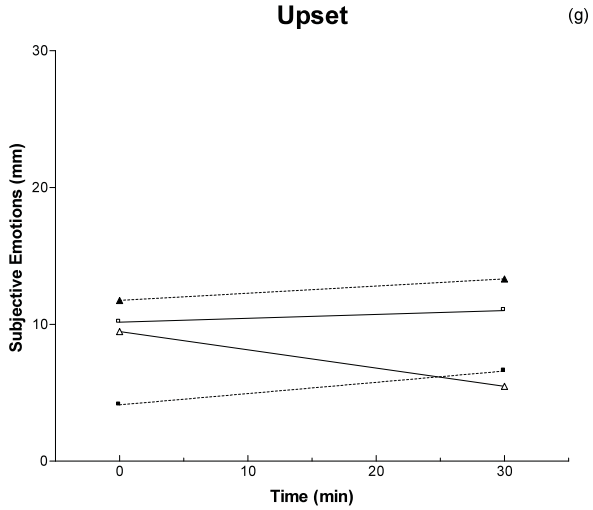
5.3.7. Upset

Absolute upset scores were not affected by preload treatment ($p = 0.33$), VGP ($p = 0.99$) or time ($p = 0.94$). There was a preload treatment x VGP interaction ($p < 0.05$) (**Figure 5.5g**).

Change from baseline upset scores were not affected by preload treatment ($p = 0.40$) or VGP ($p = 0.21$), and there was no preload treatment x VGP interaction ($p = 0.58$) (**Figure 5.6g**).

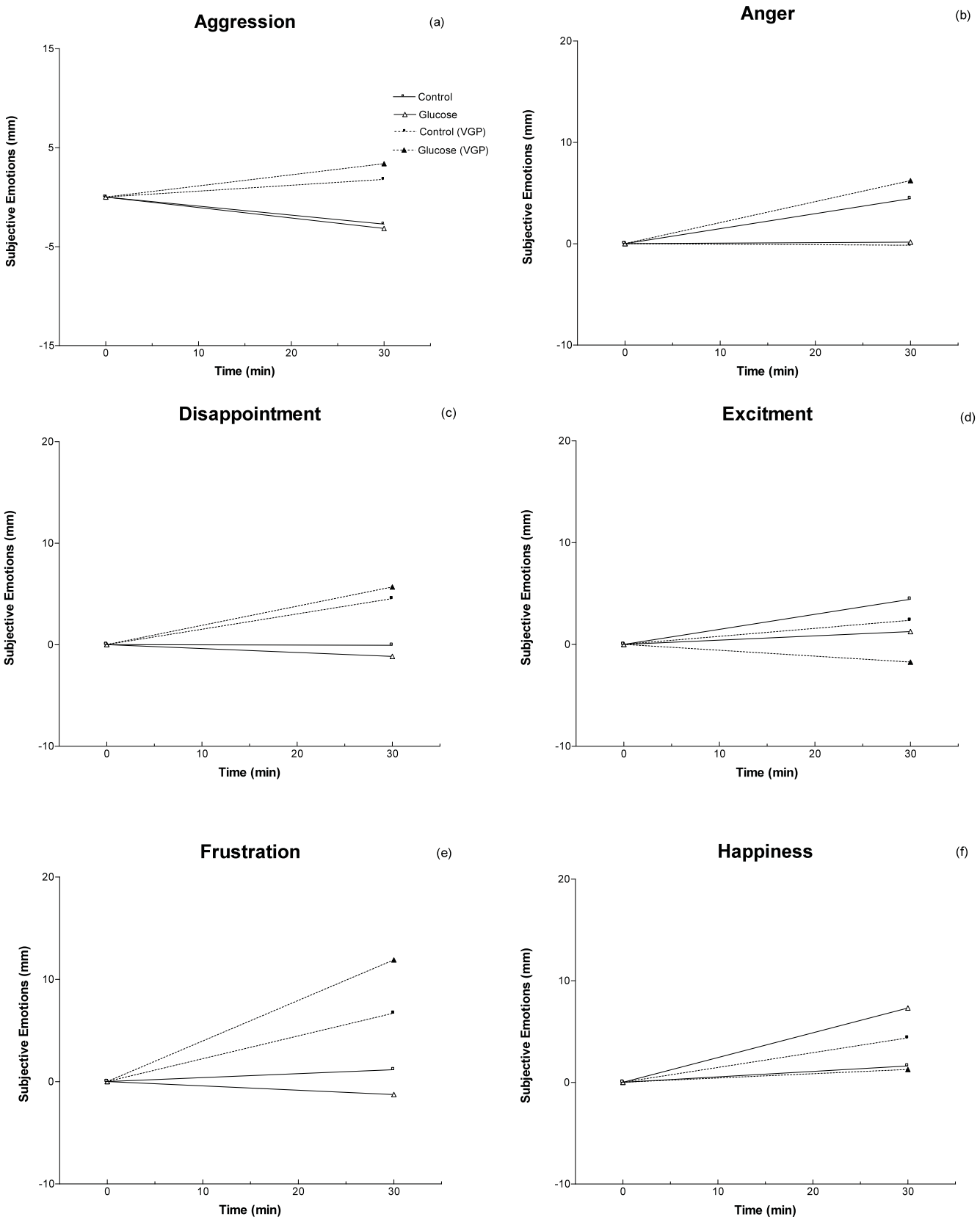
Figure 5.5. Absolute Subjective Emotions After Preload Treatments and Videogame Playing to 30 Minutes

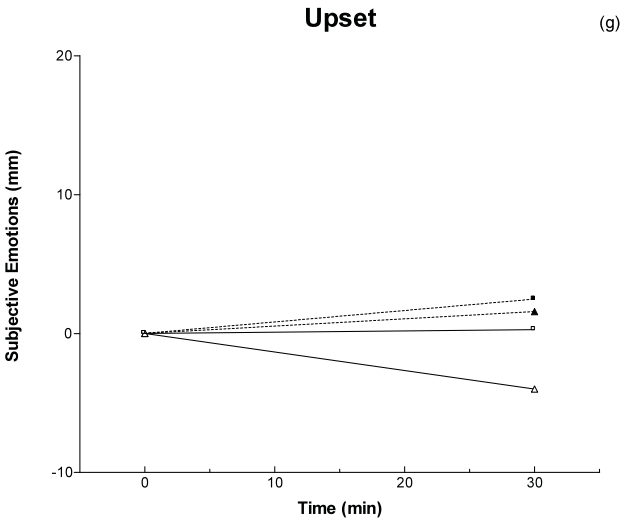




Subjective ratings for a) aggression, b) anger, c) disappointment, d) excitement, e) frustration, f) happiness and g) upset at 0 and 30 min. Values are means, n =19.

Figure 5.6. Change from Baseline Subjective Emotions After Preload Treatments and Video Game Playing to 30 Minutes





Change from baseline ratings for a) aggression, b) anger, c) disappointment, d) excitement, e) frustration, f) happiness and g) upset at 0 and 30 min. Values are means, n =19.

5.4. Association of Subjective Measures of Motivation to Eat, Thirst, and Subjective Emotions on Food Intake

5.4.1. Average Appetite

FI after all treatments did not correlate with AA scores after any of the preload treatments at any of the measurement time points **(Table 5.3.)**

Table 5.3. Associations between average appetite scores and food intake

Time	0	15	30	60
Control	-0.24	-0.04	-0.22	0.16
Glucose	-0.23	0.05	0.01	-0.18
Control (VGP)	-0.02	0.05	-0.04	0.17
Glucose (VGP)	-0.41	-0.06	-0.05	0.28

Pearson correlation coefficients; n =19. Abbreviations: VGP, video game playing

5.4.2. Desire to Eat

FI after the glucose preload and subsequent VGP was inversely correlated with DTE scores at 0 min ($r = -0.57$, $p = 0.01$) **(Table 5.4.)**

5.4.3. Hunger

FI after the glucose preload and subsequent VGP was inversely correlated with hunger scores at time 0 ($r = -0.52$, $p = 0.02$) **(Table 5.5.)**.

5.4.4. Fullness

FI after all treatments did not correlate with fullness scores at any time point after any of the preload treatments with or without VGP **(Table 5.6.)**

5.4.5. Prospective Food Consumption

FI after all treatments did not correlate with PFC scores at any time point after any preload treatment with or without VGP **(Table 5.7.).**

Table 5.4. Associations between desire to eat scores and food intake

Time	0	15	30	60
Control	-0.26	0.03	-0.14	0.17
Glucose	-0.17	0.08	-0.07	-0.35
Control (VGP)	-0.15	0.10	0.15	-0.03
Glucose (VGP)	-0.57*	-0.08	-0.22	0.05

Pearson correlation coefficients; n = 19. Abbreviations: VGP, video game playing. *$p < 0.05$

Table 5.5. Associations between hunger scores and food intake

Time	0	15	30	60
Control	-0.26	-0.17	-0.20	0.07
Glucose	-0.22	0.01	-0.17	-0.21
Control (VGP)	-0.09	-0.05	-0.12	-0.02
Glucose (VGP)	-0.52*	-0.25	-0.34	0.23

Pearson correlation coefficients; n = 19. Abbreviations: VGP, video game playing. *$p < 0.05$

Table 5.6. Associations between fullness scores and food intake

Time	0	15	30	60
Control	0.37	0.04	0.18	-0.15
Glucose	0.25	-0.12	-0.18	-0.15
Control (VGP)	0.05	-0.07	0.21	-0.36
Glucose (VGP)	0.37	0.01	-0.15	-0.25

Pearson correlation coefficients; n = 19. Abbreviations: VGP, video game playing

Table 5.7. Associations between prospective food consumption scores and food intake

Time	0	15	30	60
Control	0.13	0.07	-0.20	0.25
Glucose	-0.20	-0.05	0.18	-0.12
Control (VGP)	0.22	0.06	0.05	0.18
Glucose (VGP)	-0.20	0.12	0.25	0.39

Pearson correlation coefficients; n = 19. Abbreviations: VGP, video game playing

5.4.6. Aggression

FI did not correlate with absolute or change from baseline aggression scores at any time point after any preload treatment with or without VGP.

5.4.7. Anger

FI did not correlate with absolute anger scores at any measurement time point after any of the preload treatments. Change from baseline anger scores were positively correlated with FI after the glucose treatment and sitting ($r = 0.52$, $p = 0.02$).

5.4.8. Disappointment

FI did not correlate with absolute or change from baseline disappointment scores at any time point after any preload treatment with or without VGP.

5.4.9. Excitement

Absolute excitement scores were inversely correlated with FI after the glucose preload/sitting at 0 min ($r = -0.64$, $p = 0.003$) and 30 min ($r = -0.62$, $p = 0.005$), immediately before the test meal. There was also an inverse correlation between absolute excitement score and FI after the control preload/VGP at 0 min ($r = -0.54$, $p = 0.02$) and 30 min ($r = -0.50$, $p = 0.03$), immediately before the test meal. FI did not correlate with change from baseline excitement scores after any preload treatment with or without VGP.

5.4.10. Frustration

FI did not correlate with absolute or change from baseline frustration scores at any time point after any preload treatment with or without VGP.

5.4.11. Happiness

FI after the control preload/sitting was inversely correlated with absolute happiness scores at 0 min ($r = -0.59$, $p = 0.007$). FI after the glucose preload/sitting was inversely correlated with absolute happiness scores at 0 min ($r = -0.62$, $p = 0.005$) and 30 min ($r = -0.47$, $p = 0.04$), immediately before the test meal. FI after the control preload/VGP was inversely correlated with absolute happiness scores at 0 min ($r = -0.47$, $p = 0.04$) and 30 min ($r = -0.54$, $p = 0.02$), immediately before the test meal. FI did not correlate with change from baseline happiness scores after any of the preload treatments.

5.4.12. Upset

FI did not correlate with absolute or change from baseline upset scores at any time point after any preload treatment with or without VGP.

5.4.13. Body Composition

BMI was positively associated with FI after the control ($r = 0.58$, $p = 0.01$), glucose ($r = 0.61$, $p = 0.006$), the control & VGP ($r = 0.57$, $p = 0.01$), and glucose & VGP ($r = 0.67$, $p = 0.002$). FM was positively associated with FI after the control ($r = 0.60$, $p = 0.007$), glucose ($r = 0.60$, $p = 0.006$), and glucose & VGP ($r = 0.54$, $p = 0.02$). FFM, weight, and height were positively associated with FI after all treatment conditions **(Table 5.8.).**

Table 5.8. Associations Between Food Intake and Caloric Compensation and Weight, Height, BMI, Percent Fat-Mass, Total Fat-Mass, Percent Fat-Free Mass and Total Fat-Free Mass after Preload Treatments and Video Game Playing

	Weight(kg)	Height(m)	BMI	FM(%)[1]	FM[1] (kg)	FFM(%)[1]	FFM[1] (kg)
FI Control	0.78***	0.78***	0.58**	0.11	0.60**	-0.11	0.79***
FI Glucose	0.81***	0.80***	0.61**	0.08	0.60**	-0.08	0.82***
FI Control (VGP)	0.69***	0.67**	0.57*	-0.03	0.44	0.03	0.73***
FI Glucose (VGP)	0.80***	0.76***	0.67**	0.03	0.54*	-0.03	0.83***
CC[2] Glucose	-0.41	-0.37	-0.32	0.01	-0.27	-0.01	-0.42
CC[2] Glucose (VGP)	-0.17	-0.14	-0.16	-0.11	-0.17	0.11	-0.15

Pearson correlation coefficients; n = 19. Abbreviations: BW (kg), body weight; CC, caloric compensation; FFM, fat-free mass; FI, food intake (kcal); FM, fat-mass; VGP, video game playing. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. [1] FM and FFM estimated from the sum of skinfold measurements at four points (159). [2] CC (%) = [control intake (kcal) – treatment intake (kcal)/ kcal in preload treatment] x 100.

5.4.14. Sweetness of Preload

Preload sweetness was not associated with FI during any of the test conditions (**Table 5.9**).

Table 5.9. Associations Between Food Intake and Sweetness of Preloads After Preload Treatments and Video Game Playing

	Sweetness
FI Control	-0.13
FI Glucose	-0.32
FI Control (VGP)	-0.26
FI Glucose (VGP)	-0.23

Pearson correlation coefficients; n = 19. Abbreviations: FI, food intake; VGP, video game playing.

5.4.15. Pleasantness of Preload

Preload pleasantness was not associated with FI during any of the test conditions (**Table 5.10**).

Table 5.10. Associations Between Food Intake and Pleasantness of Preloads After Preload Treatments and Video Game Playing

	Pleasantness
FI Control	0.25
FI Glucose	-0.18
FI Control (VGP)	0.32
FI Glucose (VGP)	0.32

Pearson correlation coefficients; n = 19. Abbreviations: FI, food intake; VGP, video game playing.

Chapter 6. General Discussion

6.1. Discussion

The results of this study do not support the hypothesis that video game playing before a mixed meal increases subjective appetite, reduces satiation, and diminishes satiety signals after a glucose preload in NW 9-14 year old boys. Glucose suppressed FI compared with the sweetened control, and in contrast to the hypothesis, VGP decreased FI by approximately 50 kcal. Subjective average appetite changed over time, and was affected by the preload treatment, but not VGP.

Lower FI after VGP was unexpected, and is inconsistent with the only other study published in older adolescents that explored the effect of VGP on next meal FI [239]. In older adolescents and young adults, playing a soccer video game for one hour increased caloric intake by approximately 80 kcal at an *ad libitum* meal compared to sitting [239]. However, there are several methodological differences that impact these differing observations. The energy density of the pizzas used in the present study was more uniform than the mixed meal in the previous study (spaghetti bolognese), and therefore, it was less likely that the more energy dense ingredients were consumed in isolation or disproportionately. Another factor that may explain these differences is the interval between consumption of breakfast and initiation of the test meal. Boys in the current study refrained from consuming any energy after the standardized breakfast for a shorter duration (2.5 h) when they were given the non-caloric preload than boys in the previous study (3.5 h). The higher FI in older adolescents may be a result of the longer pre-meal interval, and lack of a snack, which minimized the potential for satiety signals from the breakfast to reduce FI at lunch. Furthermore, the average age of the participants was younger in the present study (12.0 compared to 16.7 years in the previous study). It has been reported that the ability to self-regulate FI declines with increasing age, but the influence of pubertal development on this decline is poorly understood [334].

A significant decrease in FI after a glucose preload is consistent with results of previous studies of NW adult men [306] and our studies in children [5, 266, 269, 335]. In our recent study, a 50 g (200 kcal) glucose preload reduced FI at a test meal provided 30 min later by 235 kcal in NW 9-14 y old boys [5]. Similarly, boys reduced FI by approximately 208 kcal after the glucose preload compared to the control. The ability of a glucose preload to suppress FI is attributable to several mechanisms. Glucose is quickly absorbed in the bloodstream and, when administered orally, it produces rapid postprandial plasma glucose and insulin responses [336], which reduce FI and increase satiety in the short term [279]. The inverse relationship between glycemic response and FI is consistent with the glucostatic hypothesis, which states that a rise in BG concentrations signals satiety and the termination of feeding, and conversely, a drop in BG concentrations has the opposite effect [252]. In addition, glucose may contribute to satiety though the release of GLP-1 in the small intestine [337], and the slowing of gastric emptying [281]. Full compensation for the energy content of the glucose without VGP (104%) is consistent with previous reports showing that children are able to adjust their caloric intake from carbohydrates [5, 6], suggesting that sugars in liquid form do not bypass FI regulatory mechanisms [338].

Decreased caloric compensation in boys, from 104% to 75%, for the calories of the glucose drink in response to VGP for 30 min was consistent with our hypothesis. Video game playing was hypothesized to diminish satiety signals from the glucose preload because comparable studies on distraction during mealtime demonstrated that TVV impairs the ability for a glucose drink consumed 30 min earlier to suppress FI in 9-14 y old boys [5] and peripubertal girls [6]. Although not statistically significant compared to the no VGP condition, caloric compensation for previously ingested glucose was incomplete (75%) after VGP in boys, similar

to previous findings that children have an impaired ability to compensate for calories consumed prior to TVV [5, 6]. However, the present findings of lower compensation scores in the presence of VGP is not an indication of lower response to calories from the glucose drink. Rather it is an artifact of the slightly lower FI after VGP compared to no VGP after the control drink. Food intake was approximately 80 kcal (8%) lower after the control drink and VGP compared to no VGP ($p = 0.12$) but there was only a modest (3%) decrease in FI after the glucose drink and VGP compared to no VGP ($p = 0.92$). Thus, the apparent decreased compensation after the glucose drink in boys with exposure to VGP may be attributed to a decrease in energy intake in the control condition combined with a less pronounced decrease in energy intake in the glucose condition.

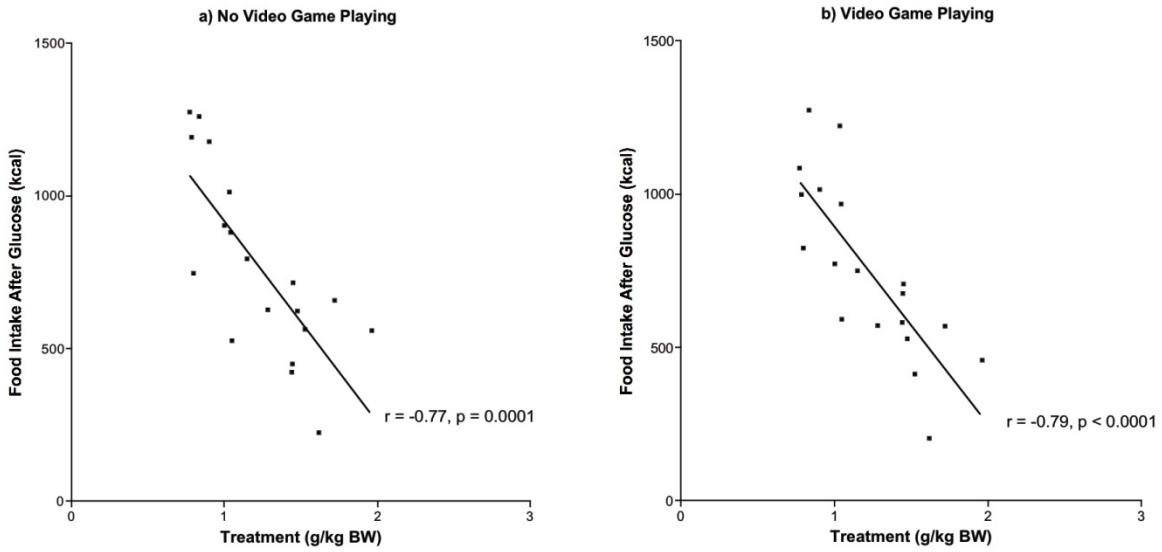
In contrast to our previous studies [5, 6, 304, 307], subjective appetite was lower after glucose. In accordance with previous studies in adults [278, 339-341], glucose decreased AA, hunger, and PFC and increased fullness compared to the control. With the exception of fullness and DTE, average and individual appetite scores for glucose were attenuated compared to the control, and therefore, it challenges the notion that children do not understand the scales, and provides validation that VAS scores can meaningfully represent children's subjective feelings of hunger. Therefore, the notion that sugars in solution bypass appetite control mechanisms is not supported by the current study. Furthermore, it is evident that larger solid caloric loads in the form of *ad libitum* test meals decrease appetite in children [5, 6, 304, 306], and similarly, boys in the current study reported a decrease in AA, DTE, PFC, and hunger, as well as an increase in fullness after consumption of the pizza meal.

Ingestion of calories from glucose and the test meal resulted in a subsequent decrease in subjective appetite sensations. A decrease in subjective appetite following glucose was predictive

of FI at the test meal because it was paralleled by a reduction in food quantity (g) and caloric intake (kcal) at the test meal. Average and individual appetite scores before and after the test meal were not correlated with FI, which is inconsistent with previous findings of a significant correlation between subjective appetite immediately before the test meal and FI in boys [5] and post-pubertal girls [6]. The absence of a correlation in the present study could be the result of a false negative due to the small sample size. Insignificant correlations were consistently observed in every treatment condition, which suggests that the relationship between AA and later FI is not dependent on prior VGP. Thus, it is likely that VGP does not favor a disconnect between subjective appetite sensations and measured energy intake.

In the present study, height, weight, BMI, FM, and FFM were all positively associated with FI but not with caloric compensation after the glucose preload in each treatment condition. The use of a fixed (50 g) dose of glucose may explain why boys with a larger body size ate more in the study because FI was inversely correlated with the glucose content of the treatment preload expressed as g/kg BW **(Figure 5.7)**. The boys at the higher end of the normal range received a lower dose of glucose relative to their BW, which may have contributed to higher FI than boys who received a higher dose. However, body size does not explain the lower FI after VGP, because body size was not associated with suppression of energy intake. In addition, the Pearson correlation coefficients for the association between glucose dose (g/kg BW) and FI were similar for VGP ($r = -0.77$) and no VGP ($r = -0.79$). Therefore, the use of a fixed dose did not override the effect of VGP on FI. Glucose dose (g/kg BW) was not associated with caloric compensation scores. Therefore, glucose effectively suppressed FI in boys, regardless of body size. This suggests that the use of a fixed dose was not a confounding variable that contributed to variance in caloric compensation scores, which strengthens the internal validity of this study.

Figure 5.7. Associations Between Preload Gm per Kilogram Body Weight and Food Intake



Preload dose (g/kg BW) inversely correlated with food intake following the caloric preloads ($p < 0.001$); n = 19. Abbreviations: Kg, kilogram; BW, body weight.

Sweetness is an important property of food that may have an impact on FI. Oral ingestion of food is more effective at suppressing appetite than meals directly infused in the stomach or duodenum, which suggests that oral factors have a role in the development of satiety [342]. Sweet taste alone is associated with neurochemical changes in brain regions involved in reward [343]. Furthermore, sweet taste receptors located on the tongue have also been identified in the endocrine cells of the gastrointestinal tract, and the number of sweet receptors is hypothesized to affect the type and quantity of food consumed [344]. Pleasantness of the preloads was also measured because it has been suggested that palatability of foods can affect subjective ratings of hunger and satiety [345]. Participants rated the control beverage as significantly sweeter and less pleasant than glucose. Neither mean sweetness nor pleasantness ratings were associated with mean FI for the control or glucose treatments, which suggests that sweetness and pleasantness of the beverages were not major determinants of FI.

Video game playing can stimulate both positive and negative emotions [346], which are known to impact eating behaviour [347], and therefore, it was important to determine if susceptibility to emotional eating may have impacted the results of this study. This was assessed with the DEBQ, which measured the tendency to eat in response to diffuse emotions (i.e. eating when feeling lonely or bored) and the tendency to eat in response to specific emotions (i.e. eating in response to anger or irritation) [330]. Diffuse and emotional eating scores were low, and they did not differ among individuals in the study or correlate with FI. Therefore, susceptibility to emotional eating was unlikely responsible for inter-individual differences in FI.

In a study on young adult women, negative emotions (frustration, sadness, anxiety, anger and disgust), measured on a Likert scale, increased significantly after watching a four minute video, but intake of snack food after the video was not related to the change in emotions [348]. In the present study, frustration and aggression scores both increased from baseline during the VGP condition. However, frustration and aggression scores did not correlate with FI after any of the treatment conditions. This result was expected based on the DEBQ indication that the boys were unlikely to eat more in response to negative emotions. However, FI was inversely correlated with excitement and happiness scores at 0 and 30 min after the control preload/VGP and after the glucose preload/sitting, as well as happiness scores at time 0 after the control preload/sitting. There is limited research on the effect of positive emotions on FI in humans. Positive emotions increased FI in adults who were restrained eaters because they impair the cognitive control over eating [349], and higher Likert scores for excitement and elation before eating under natural conditions were significantly correlated with increased FI in adolescents [350]. In the present study, there was a trend for lower FI after the control and VGP ($p = 0.12$), and this may have been moderated by higher subjective arousal (happiness and excitement). Higher states of

subjective arousal were unrelated to exposure to VGP and preload treatment, and therefore, it is unclear why this association occurred after certain conditions, but not others. Collectively, these results suggest that subjective arousal evoked by VGP may be an important factor that affects caloric intake in children but further research is required to determine if there are unique physiologic mechanisms associated with VGP that affect FI.

In conclusion, video game playing for 30 min before a meal increased satiation through a decrease in energy intake at mealtime, but did not diminish satiety signals from the glucose preload in 9- to 14- year-old normal weight boys. Although VGP is less likely to promote overeating at a subsequent meal than an equal duration of TVV during mealtime in children, it should not be assumed that playing video games produces a net benefit on weight. Excessive VGP may directly and indirectly influence weight status, and health through mechanisms unrelated to FI, including decreased sleep duration, and physical activity. In addition, the potential for VGP to reduce FI at a subsequent meal may diminish with increased duration of use. Therefore, participation in moderate sessions (30 min) of VGP would be an optimal recommendation for children to adhere to. There is a need for further research on the mechanisms through which VGP affects FI, and the influence of VGP on energy balance in the long-term.

6.2. Limitations

A limitation of the study is that boys were assigned to play a specific type of video game for a predetermined, fixed period of time, which may not reflect their typical gameplay patterns at home. *Angry Birds* is a strategic game, which may be more mentally demanding and stressful than other available game genres. Prior research has shown that computerized activities are mentally stressful, and contribute to increased FI at a later meal in young women [233]. It is unclear from this study how VGP affected the physiological stress response because biomarkers such as cortisol were not measured. However, a possible unique effect of stressful video games on appetite control limits generalizability of the study's findings to other game genres. Research on various types of video games is needed to make conclusive generalizations about the effect of VGP on FI.

Chapter 7. Future Directions

7.1. Duration of Video game playing and its Effect on Short Term Food Intake

Although the present study found that 30 min of VGP was a sufficient amount of time to decrease FI compared to sitting, the results did not confirm that satiety signals from the glucose preload were attenuated by VGP. It has been demonstrated that the ability for sugars to suppress FI is dependent on the time interval between the preload and the test meal. A previous study found that in 9-14 y old boys, caloric compensation for a glucose preload was higher at a test meal consumed 30 min after the preload (106%) than 60 min (67%), indicating the ability for glucose to suppress FI decreased with time [304]. The duration of 30 min may not have been long enough to interfere with physiological signals from the glucose prelaod in the present study, but extending exposure to VGP to 60 min may be too long. Therefore, it might be appropriate to examine the effect of VGP for 45 min after a glucose preload on satiety and satiation. This is also similar to the average amount of time boys spend with video game consoles a day (48 min) [122], which would more closely replicate the real life environment.

7.2. Exposure to Food Cues During Video Game Playing and Food Intake

The purpose of the present study was not to examine the effect of exposure to food cues, which are known to influence FI, and this is why a game was selected that did not contain any content associated with food or eating. Playing advergames that promote food brands influences brand preference, and the nutritional value of food selected [193-195] but it is unknown if exposure to food cues during VGP have an effect on mechanisms regulating FI. Video games may be used as an avenue for promoting brands, and therefore, it might be relevant to compare the effect of video games containing food content, compared to a neutral video game that doesn't contain this type of content.

7.3. The Effect of Active Video Game Playing Before a Mixed Meal on Subjective Appetite, and Satiety Signals from a Glucose Preload in Normal Weight and Overweight/Obese Children

Children who play active video games expend a significantly greater amount of energy compared to when they play non-energy expending video games [182], but it is unknown if they compensate for this energy by increasing their FI at a later meal. In a previous study, walking on a treadmill for 45 min at a moderate intensity resulted in higher energy intake at a later meal in normal weight girls, but not boys [326]. It is of interest to investigate the net effect of playing active video games prior to a meal on energy balance, and satiety signals from a previously consumed glucose preload, and if the relationship is dependent on sex, and body fatness.

7.4. Effect of Multi-player Video Games on Food Intake

The majority of children and teenagers report playing video games with other people, in addition to playing them alone [351]. Adding a social and competitive aspect to the gaming experience may have a different effect on FI than a single-player video game, which is played in isolation. Future research is needed to determine if the amount of players in the video game playing environment is an important factor that affects children's response to video games.

7.5. Effect of Video Game Genre on Food Intake

It is unknown if all video games affect children's FI in a generalized fashion, or whether the relationship is dependent on video game genre. This is a relevant question because children and adolescents typically play a variety of different video game genres (i.e. racing, puzzle, sports), as opposed to limiting themselves to one genre [351]. Therefore, future studies should investigate if manipulating the genre of video game produces similar or specific effects on FI and appetite. This type of research would inform more specific recommendations regarding the optimal type of VGP for preventing overeating.

7.6. The Effect of Sedentary Videogame Playing on Subjective Appetite and Short-term Food Intake After a Glucose Preload in Overweight and Obese Boys

There is evidence that the ability to self regulate FI by responding to short-term satiety signals is impaired in obese children [304], which suggests that the physiological mechanisms controlling FI is dependent on weight status. It is currently unknown if exposure to screen-time activities, such as VGP interferes with the response to previously consumed calories in children who are overweight and obese, and how VGP prior to mealtime affects FI in these groups. Therefore, future studies should explore if overweight and obese children compensate for energy intake from a glucose preload at a subsequent meal when VGP is present in the pre-meal environment. This research will provide preliminary information regarding the necessity for research on the mechanism of action by which BW status affects the interaction between VGP and the regulation of FI in children.

7.7. Longitudinal Research on the Relationship Between Video Game Playing and Weight Status

Longitudinal studies are needed to investigate the link between VGP practices and weight status in the long term. It may be beneficial for children to limit the amount of time that they play video games but there is limited research to inform the development of guidelines regarding video game use. Information is needed on how body composition is related to when, and how video games are played, and if changes in these variables produces different outcomes.

Chapter 8. References

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Chapter 9. Appendices

Appendix 9.1. Recruitment Poster

Mount Saint Vincent University

ATTN: PARENTS OF BOYS AGED 9-14 YEARS

We are conducting a nutrition and video game study
REQUIREMENTS: Healthy, 9-14 year old boys

INVOLVES: 1 screening + 4 weekend sessions (breakfast and lunch included)

As a reward for taking part, at each session the child will receive a theatre movie pass.
Parents will receive $5 travel reimbursement after each session.

For more information, please contact:
Ms. Alyson Branton at (902) 457-6378
OR
Mr. Brandon Gheller at (902) 457-6378

Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378	Alyson or Brandon 457-6378

Appendix 9.2. Recruitment Letter for Parents

Excellence • Innovation • Discovery

Effect of sedentary video game playing prior to meal time on food intake after a glucose preload in boys

Dear Parent

Mount Saint Vincent University is leading a team of researchers investigating the physiological and environmental determinants of energy intake regulation on the health of children and young adolescents. In our current work we are conducting studies aimed at understanding the controls of food intake in children, with the ultimate goal of finding ways to address the problems of overeating and obesity that are becoming a concern among those people involved in improving the long term health of Canadians.

We are asking the parents of boys aged 9 to 14 years old to allow their child to take part in a research study. Their participation is quite straightforward: on four separate weekend mornings, following a 12 hour fast, your child will consume a standard breakfast at home, and then consume a sweet beverage. They will then be asked to play a video game for 30 minutes, on two of the four visits, followed by a pizza lunch in the Department of Applied Human Nutrition, Mount Saint Vincent University. The study will take place on four weekend mornings at the Evaristus Building (Room 365), Department of Applied Human Nutrition.

There are criteria for participation that you need to be aware of, the child must:

- be between 9 and 14 years of age, and
- be healthy, and have been born at term, and
- not be taking medications.
- not have allergies to milk, wheat or nuts

As a reward for taking part, at the end of each session the child will be given a gift certificate to Empire Theatres ($10 gift certificate).

If you would like your son to participate, or to get further information beyond that provided in this letter, please contact Ms. Alyson Branton, Project Coordinator at (902) 457-6378, or Dr. Nick Bellissimo, Principal Investigator [redacted] at Mount Saint Vincent University (Department of Applied Human Nutrition).

If you have questions about how this study is being conducted and wish to speak with someone who is not directly involved in the study, you may contact the Chair of the University Research Ethics Board (UREB) c/o MSVU Research and International Office, at 457-6350 or via e-mail at research@msvu.ca

Thank you for your support in this important research.
Sincerely,
Dr. Nick Bellissimo, Department of Applied Human Nutrition, Mount Saint Vincent University.
Ms. Alyson Branton, Department of Applied Human Nutrition, Mount Saint Vincent University.

Appendix 9.3. Study Information Sheet and Parent's Consent Form

Excellence • Innovation • Discovery

Effect of sedentary video game playing prior to meal time on food intake after a glucose preload in boys
Study Information Sheet and Parent's Consent Form

Investigators:

Dr. Nick Bellissimo, PhD, BEd
Principal Investigator
Department of Applied Human Nutrition at Mount Saint Vincent University
Phone: [redacted]
Email: nick.bellissimo@msvu.ca

Ms. Alyson Branton, MSc.AHN (c)
Project Coordinator
Phone: (902) 457-6378
Email: alyson.branton@msvu.ca

Invitation:

Mount Saint Vincent University is leading a team of researchers investigating the physiological and environmental determinants of energy intake regulation on the health of children and young adolescents. In our current work we are conducting studies aimed at understanding the controls of food intake in children, with the ultimate goal of finding ways to address the problems of overeating and obesity that are becoming a concern among those people involved in improving the long term health of Canadians. We are asking the parents of 9-14 year old boys to allow their children to take part in a research study.

Purpose of Research:

The purpose of this study is to determine the effects of videogame playing on food intake regulation in normal weight and obese 9-14 year-old boys. This experiment is being conducted through the Department of Applied Human Nutrition at Mount Saint Vincent University by Dr. Nick Bellissimo. Your child will be required to attend four experimental sessions conducted over a 4-week period, for a total of 5 visits (4 food intake measurement sessions + 1 information/screening visit) to the Mount Saint Vincent University campus. Each visit will last approximately 60 minutes.

Procedure:

Appetite Assessment:

For those parents who express interest in having their child participate, some information about the child will be requested by telephone. If the child was born at term, is healthy and does not receive any medications, a screening session will be arranged.

During the information/screening session, the researcher will explain the full details of the study. Parents

that give consent to have their child participate will sign a consent form. The parent will receive copies of consent forms and of the study information sheet. If the child wishes to participate and signs a children's assent form, their weight, height, and body fat by skinfold caliper at 4-points (biceps, triceps, supra-ilial, and subscapular), will be measured.

The children will then be asked to rank their preference for pizza that will be served as the lunch meal at each session.

The children who participate in this study will be requested to go to the Evaristus Building (Rm. 365), Department of Applied Human Nutrition, Mount Saint Vincent University, for four individual weekend morning sessions over a four week period.

On each of the four test days, the children will have a standardized breakfast of cereal, milk and orange juice at home, either at 8:00 am, 9:00 am or 10:00 am (the time will be consistent for each child). The children will arrive at the Evaristus Building, either at 10:00 am, 11:00 am or 12:00 pm (but consistent throughout for each child).

Children will fast for 12 hours before breakfast and after breakfast until their arrival, except for water (which will be allowed up to one hour before their arrival).

Each child will receive one glass of sweetened Kool-aid containing sugar or an artificially sweetened beverage. The sugar drink will contain similar amounts of sugar as common soft drinks or fruit juice.

The children will be fully supervised during the study sessions. They will be engaged in video game playing (e.g. Angry Birds, Sonic Unleashed, Lego Star Wars) on two of their four visits, before the pizza lunch, for 30 minutes.

McCain pizza and spring water (purchased at Sobey's or Atlantic Superstore) will be served 30 minutes after the children have consumed their beverage and started playing video games. Children will be told that they may eat as little or as much as they like. The amount of food eaten by each child will be measured.

The children will also be requested to complete scales on which they will place a pencil mark to describe their desire to eat ("Very weak" to "Very strong"), hunger ("Not hungry at all" to "As hungry as I've ever felt"), fullness ("Not full at all " to "Very full"), how much food they could eat ("A large amount" to "Nothing at all"), thirst ("Not thirsty at all" to "As thirsty as I ever felt") sweetness of the drinks ("Not sweet at all" to "Extremely sweet"), pleasantness of the preload and pizza ("Not at all Pleasant" to "Very Pleasant", and physical comfort ("Not well at all" to "Very well"). They will complete these scales during the information/screening session, in order to become familiar with the test instruments. They will also be asked to describe their feelings after playing the video game, aggression (I feel "very aggressive" to "no aggression), excitement ("very excited" to "not excited at all"), anger ("very angry" to "not angry at all"), violent ("very violent" to "not violent at all"), disappointed ("very disappointed" to "not disappointed at all") happy ("very happy" to "not happy at all"), upset ("very upset" to "not upset at all") and satisfied ("very satisfied" to "not satisfied at all").

<u>Eating Behaviour Questionnaire:</u>

If you consent to your child's participation in this experiment, he/she will also be asked to fill out a short questionnaire about their eating habits during the information/screening visit or after one of the food intake sessions. A trained examiner will help your child fill out the questionnaire. The answers will be strictly confidential and will only serve to assist in the analysis of the data collected. Your child may skip

any questions on the questionnaire that makes them feel uncomfortable.

There will also be a "Screen Time and Feeding Behaviour Questionnaire" given to the child about their electronic game playing and television viewing habits, what they eat while they are engaged in screen time activities, and different meal time behaviours they engage in.

Confidentiality:

Records relating to participants will be kept confidential in a locked cabinet in the Department of Applied Human Nutrition and no disclosure of personal information of the children or parents will take place except where required by law. Participants will have a code and a number that will identify them in all documents, records and files to keep their name confidential. All data will be entered into Microsoft Excel files, available only to investigators. Each participant will have a file, also only available for investigators. All forms and printouts will be stored in the individual files – and clearly labelled. All documents will be kept for a minimum of five years following completion of the study and then securely destroyed.

Benefits:

As the causes of obesity remains undefined, the potential benefits from this study will be a better understanding of the regulation of food intake in children and might contribute to the prevention of obesity in children.

Questions and further information:

If you have any questions or would like further information concerning this research project, please do not hesitate to call: Dr. Nick Bellissimo [redacted] or Ms. Alyson Branton at (902) 457-6378.

Dissemination of findings:

A summary of results will be made available to you to pick up, or if requested will be sent by mail or e-mail, after the study is completed.

Consent:

I acknowledge that the research procedures described above and of which I have a copy, have been explained to me and that any questions that I have asked have been answered to my satisfaction. I know that I may ask additional questions now or in the future. I am aware that participation in the study will not involve any health risk to my child.

I understand that for purposes of the research project, if my child or I choose to withdraw from the study at any time, we may do so without prejudice.

Upon completion of each study session, my child will receive a $10 Empire Theatre gift certificate. I will also receive $5 to cover transportation costs following each study session. The final summary and results of the study will be available for me to pick up from the Department of Applied Human Nutrition, Mount Saint Vincent University. I am aware that the researchers may publish the study results in scientific journals, keeping confidential my son or daughter's identity.

If you have questions about how this study is being conducted and wish to speak with someone who is not directly involved in the study, you may contact the Chair of the University Research Ethics Board (UREB) c/o MSVU Research and International Office, at 457-6350 or via e-mail at research@msvu.ca

I hereby consent for my child, _____, to participate in this study.

_____ _____
(Name of parent or guardian) (Signature of parent or guardian)

_____ _____
(Name of witness) (Signature of witness)

Date: _____ (dd/mm/yy)

Appendix 9.4. Children's Assent Form

Effect of Sedentary Screen Time Activities before a Meal on Food Intake in Children
Children's Assent Form

Purpose of Research:
The purpose of this study is to determine the effect of television viewing, computer use, and video game playing on your appetite. My weight, height, and body fat will be measured during the information/screening visit. I will also be required to complete special scales to show if I am hungry or full during each session. I will fill-out a short questionnaire about my eating behaviours, and know that I am allowed to skip any questions that may make me feel uncomfortable. I will also be asked to fill out a short questionnaire about my video game playing, TV watching and computer use before and during meals. I will also be provided with a pizza lunch at the end of each study session (that I will eat in the Department of Applied Human Nutrition, Mount Saint Vincent University). All the experimental sessions will be on weekends or during summer vacation, so I don't need to be absent from school.

I know that my participation in the study will not involve any health risk to me.

Also, if at any time I decide to stop participating, that will be O.K. I understand that information related to me will be kept confidential. I know that I will receive a $10 Empire Theatre gift certificate after completion of each study session, as a "thank you" for my participation.

"I was present when _____read this form and gave his/her assent.

_____
Signature

Name of the person who obtained assent:

_____

Date: _____ (dd/mm/yy)

Appendix 9.5. Telephone Screening Questionnaire

Effect of Sedentary Video Game Playing Prior to Meal Time on Food Intake after a Glucose Preload in Children

Name: _____

Age: _____ years DOB (d/m/y) _____ Term baby? yes/no

Height: _____ cm. Weight: _____ kg. Normal birth weight? yes/no

Has your child gained or lost weight recently? yes/no (circle correct answer)

Does your child usually have breakfast? yes/no

Does your child like (foods that will be used in experiments 1, 2,3 & 4)

Fat-free milk	yes/no	**Honey-Nut Cheerios**	yes/no	**Pizza**	yes/no
Juice	yes/no	**Sports Drinks (E.g. Gatorade)**	yes/no		

Has your child consumed non-caloric beverages or foods containing either aspartame or Sucralose®? Ie: diet soft drinks or sugar-free snacks? yes/no

Is your child following a special diet? yes/no

Does your child have food allergies or sensitivities?
Milk, nuts, wheat yes/no

Health problems? yes/no
If yes, which problem? _____

Medication(s)? yes/no
If yes, which medication/s? _____

Education: Grade: _____ Special class? yes/no

Skipped or repeated grade? yes/no Learning difficulties/problems? yes/no

Behavioural or emotional problems yes/no
If yes, which problem? _____

Is your child willing to not spend time in front of a screen (i.e. television, computer, videogame, cell phone, etc) for12 hours prior to coming in to the school? yes/no

Include in study? yes/no
If not, why? _____

Appointment date: _____ _____ (d/m/y)

Investigator: _____ Date: _____ (d/m/y)

Appendix 9.6. Study Day Questionnaires

VAS – Motivation to eat
VAS – Pleasantness (preload beverage)
VAS – Pleasantness (pizza lunch)
VAS – Perceived Sweetness
VAS – Physical Comfort
VAS- Videogame Acceptability
VAS- Emotion Screening
Feeding Session Cover Sheet
Dutch Eating Behaviour Questionnaire

Appendix 9.6a. VAS Motivation to Eat

Time =

Visual Analogue Scale
Motivation to Eat

DATE: _____

ID: _____

These questions relate to your "motivation to eat" at this time. Please rate yourself by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

1. How strong is your desire to eat?

Very WEAK _____ Very STRONG

2. How hungry do you feel?

NOT Hungry at all _____ As hungry as I have ever felt

3. How full do you feel?

NOT Full at all _____ VERY Full

4. How much food do you think you could eat?

NOTHING at all _____ A LARGE amount

5. How thirsty do you feel?

NOT thirsty at all _____ As thirsty as I have ever felt

Appendix 9.6b. VAS Pleasantness of Preload

Time =

Visual Analogue Scale
Pleasantness of Preload

DATE: _____

ID: _____

This question relates to the palatability of the drink you just consumed. Please rate the pleasantness of the beverage by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

How pleasant have you found the preload?

NOT at all pleasant _____ Very pleasant

Appendix 9.6c. VAS Pleasantness of Test Meal

Time =

Visual Analogue Scale
Pleasantness of Test Meal

DATE: _____

ID: _____

This question relates to the palatability of the food you just consumed. Please rate the pleasantness of the food by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

How pleasant have you found the food?

NOT at all pleasant _____ Very pleasant

Appendix 9.6d. VAS Sweetness of Preload

Time =

Visual Analogue Scale
Sweetness

DATE: _____

ID: _____

Please rate the level of sweetness by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

How sweet have you found the beverage?

NOT sweet at all _____ Extremely sweet

Appendix 9.6e. VAS Physical Comfort

Time =

Visual Analogue Scale
Physical Comfort

DATE: _____

ID: _____

These questions relate to your "physical comfort" at this time. Please rate yourself by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

How well do you feel?

NOT well at all _____ VERY Well

Appendix 9.6f. VAS Videogame Acceptability

Time =

Visual Analogue Scale
Videogame acceptability

DATE: _____

ID: _____

This question relates to how well you enjoyed the video game. Please rate the program by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

How much did you enjoy the videogame?

Did not _____ Enjoyed
Enjoy Very Much
At all

Appendix 9.6g. VAS Emotion Screening

Time =

Visual Analogue Scale
Emotion Screening

DATE: _____

ID: _____

These questions relate to "your emotions" at this time. Please rate yourself by placing a small "**x**" across the horizontal line at the point which best reflects your present feelings.

1. How aggressive do you feel?

NOT aggressive _____ VERY aggressive
At all

2. How angry do you feel?

NOT angry _____ VERY angry
At all

3. How excited do you feel?

NOT excited _____ VERY excited
At all

4. How disappointed do you feel?

NOT
Disappointed _____ VERY disappointed
at all

5. How happy do you feel?

Not happy _____ VERY happy
at all

6. How upset do you feel?

Not upset _____ VERY upset
at all

7. How frustrated do you feel?
Not frustrated _____ VERY frustrated

Appendix 9.7. Feeding Session Cover Sheet

Feeding Session Cover Sheet
Department of Applied Human Nutrition, Mount Saint Vincent University

Food Intake Control in Children

Subject ID: _____ Session: _____

Date: _____

Baseline Questionnaire (to be asked by investigator)

1. Have you had the standardized breakfast this morning? YES/NO

2. At what time did you have the standardized breakfast? _____

3. Have you had anything to eat or drink for 10 - 12 hours before breakfast? YES/NO

If yes, please describe briefly _____

4. Have you had anything to eat or drink after breakfast before arriving here? YES/NO

If yes, please describe briefly _____

5. Are you taking any medication? YES/NO

If yes, please describe briefly _____

6. Have you watched television, used cell phones, played video games or been on the computer in the last 12 hours? YES/NO

7. When was the last time you played *Angry Birds*? _____

How long did you play it for? _____

8. What time did you go to bed? _____

9. What time did you wake up? _____

Comments/Notes

Appendix 9.8. Dutch Eating Behaviour Questionnaire

Dutch Eating Behaviour Questionnaire

Please read each question and circle the appropriate response.

1. If you have put on weight, do you eat less than you usually do?

Never Seldom Sometimes Often Very often

2. Do you try to eat less at meal times than you would like to eat?

Never Seldom Sometimes Often Very often

3. How often do you refuse food or drink offered because you are concerned about your weight?

Never Seldom Sometimes Often Very often

4. Do you watch exactly what you eat?

Never Seldom Sometimes Often Very often

5. Do you deliberately eat foods that are slimming?

Never Seldom Sometimes Often Very often

6. When you have eaten too much, do you eat less than usual the following day?

Never Seldom Sometimes Often Very often

7. Do you deliberately eat less in order not to become heavier?

Never Seldom Sometimes Often Very often

8. How often do you try not to eat between meals because you are watching your weight?

Never Seldom Sometimes Often Very often

9. How often in the evenings do you try not to eat because you are watching your weight?

Never Seldom Sometimes Often Very often

10. When you eat, do you take into account what you weigh?

Never Seldom Sometimes Often Very often

11. Do you have the desire to eat when you are irritated?

Never Seldom Sometimes Often Very often

12. Do you have the desire to eat when you have nothing to do?

Never Seldom Sometimes Often Very often

13. Do you have the desire to eat when you are depressed or discouraged?

Never Seldom Sometimes Often Very often

14. Do you have a desire to eat when you are feeling lonely?

Never Seldom Sometimes Often Very often

15. Do you have a desire to eat when somebody lets you down?

Never Seldom Sometimes Often Very often

16. Do you have a desire to eat when you are angry?

Never Seldom Sometimes Often Very often

17. Do you have a desire to eat when you are expecting something unpleasant to happen?

Never Seldom Sometimes Often Very often

18. Do you get the desire to eat when you are anxious, worried or tense?

Never Seldom Sometimes Often Very often

19. Do you have a desire to eat when things are going against you or when things have gone wrong?

Never Seldom Sometimes Often Very often

20. Do you have a desire to eat when you are frightened?

Never Seldom Sometimes Often Very often

21. Do you have the desire to eat when you are disappointed?

Never Seldom Sometimes Often Very often

22. Do you have a desire to eat when you are bored or restless?

Never Seldom Sometimes Often Very often

23. Do you have a desire to eat when you are emotionally upset?

Never Seldom Sometimes Often Very often

24. If food tastes good to you, do you eat more than usual?

Never Seldom Sometimes Often Very often

25. If food smells and looks good to you, do you eat more than usual?

Never Seldom Sometimes Often Very often

26. If you see or smell something delicious, do you have the desire to eat it?

Never Seldom Sometimes Often Very often

27. If you have something delicious to eat, do you eat it straight away?

Never Seldom Sometimes Often Very often

28. If you walk past the baker, do you have the desire to buy something delicious?

Never Seldom Sometimes Often Very often

29. If you walk past a snackbar or a cafe, do you have the desire to buy something delicious?

Never Seldom Sometimes Often Very often

30. If you see others eating, do you also have the desire to eat?

Never Seldom Sometimes Often Very often

31. Can you resist eating delicious foods?

Never Seldom Sometimes Often Very often

32. Do you eat more than usual when you see others eating?

Never Seldom Sometimes Often Very often

33. When your mother or father are preparing a meal, are you inclined to eat something?

Never Seldom Sometimes Often Very often