Mount Saint Vincent University

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Does partial reduction of added sugar in chocolate milk and yogurt benefit glycaemic response?

by

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Abstract

Commercially available dairy products such as chocolate milk, or yogurt have added sugar (5.5% or higher of total calories) that contributes to a higher energy density of these products. The objective of this study was to investigate whether chocolate milk and yogurt with reduced sugar content have any benefits on blood glucose (BG) control in humans. We hypothesized that chocolate milk and yogurt formulated with the reduced level of added sugar will benefit mean glycaemic response of these products. Methods: Ten male and ten female aged 19-35 completed a cross-over, single-blinded, randomized study attending five sessions with one week washout between the sessions. The treatments were randomly assigned and included a serving of chocolate milk (250ml) with 3.3% (C3.3%) and 5.5% added sugar (C5.5%), a serving of yogurt (175g) with 3.3% (Y3.3%) and 5.5% added sugar (Y5.5%), and water control (250ml). Blood samples were collected and the subjective appetite rating and feeling of physical comfort were recorded at 0, 15, 30, 45, 60, 90, and 120 min. *Ad libitum* food intake (FI) was measured with a pizza meal at 120 minutes. The changes in blood glucose, appetite and physical comfort parameters over time were analyzed with two-way ANOVA, and food intake, blood glucose and appetite AUCs, and the palatability of the treatments and pizza meal were analyzed with one-way ANOVA. The Tukey-Kramer post-hoc test was used for pairwise comparisons. Results: There was an effect of a treatment on blood glucose $iAUC_{0\text{-}120min}$ ($P=0.0025$). The treatments with C3.3%, C5.5% and Y5.5% resulted in a higher BG $iAUC_{0\text{-}120min}$ compared to water control ($P<0.05$), while the treatment with Y3.3% led to a similar BG $iAUC_{0\text{-}120min}$ as the water control ($P>0.05$). Additionally, all the dairy treatments resulted in a similar BG $iAUC_{0\text{-}120min}$ ($P>0.05$). All caloric treatments resulted in reduced subjective appetite compared to water control over two hours ($P<0.05$). There was no effect of a treatment on cumulative FI over two hours; however, there was an effect of a

treatment (P=0.02) on *ad libitum* FI with pizza meal at 120 min. The treatments with C3.3% led to reduced FI compared to water control (P=0.02) and a similar trend was observed for the treatment with Y3.3% (P=0.05). All treatments resulted in a similar subjective rating of energy, fatigue, thirst physical and comfort parameters (P>0.05). Conclusion: The treatments with chocolate milk and yogurts with full and reduced added sugar content result in a similar glycaemic response over two hours. However, the reduction of added sugar in yogurt results in BG response similar to water control and tended to lower *ad libitum* FI compared to water control, while chocolate milk with reduced added sugar content results in a lower *ad libitum* FI compared to water control. Both chocolate milk and yogurt with reduced sugar content similarly suppress subjective appetite over two hours as their full sugar counterparts, and do not cause any physical discomfort. The reduction of added sugar does not negatively impact the sensory properties of chocolate milk and yogurt. Although the reduction of added sugar in chocolate milk and yogurt does not cardinally impact postprandial glycaemic response, both chocolate milk and yogurt with reduced added sugar content possess with unique metabolism that may position them as potential functional products for metabolic control.

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A	
ATP	Adenosine Triphosphate
α-La	Alpha-lactalbumin
ANOVA	Analysis of Variance
APP	Appetite
AA	Average Appetite
A1C	Glycated Hemoglobin
B	
β-Lg	Beta-lactoglobulin
BMI	Body Mass Index
C	
CVD	Cardiovascular Disease
C5.5%	Chocolate milk with 5.5% added sugar
C3.3%	Chocolate milk with 3.3% added sugar
D	
DTE	Desire to Eat
DM	Diabetes Mellitus
DHA	Docosahexaenoic Acid
E	
EPIC	European Prospective Investigation into Cancer
F	
FPG	Fasting Plasma Glucose
FFA	Free-fatty Acid
G	
GLP-1	Glucagon-like peptide-1
GLUT	Glucose Transporter
GI	Glycemic Index
GL	Glycemic Load
H	
HbA1C	Hemoglobin Glycated Hemoglobin
HGP	Hepatic Glucose Production
HDL	High-density Lipoprotein
HDPE	High Density Polyethylene Plastic
I	
iAUC	Incremental Area Under Curve
Ig	Immune Globulin
IFG	Impaired Fasting Blood Glucose
IGT	Impaired Fasting Glycemia
IHD	Ischemic Heart Disease
M	
M.F.	Milk Fat
MUFA	Monounsaturated Fatty Acids
N	
niAUC	Net Incremental Area Under Curve

O	
OGTT	Oral Glucose Tolerance Test
P	
PG	Plasma Glucose
Psi	Poung-force per square
S	
SQ	Satiety Quotient
SST	Serum Separating Tube
SD	Standard Deviation
SSB	Sugar-sweetened Beverages
T	
trt	Treatment
tAUC	Total Area Under Curve
T1DM	Type 1 Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
U	
UHT	Ultra Heat Treatment
V	
VAS	Visual Analog Scale
W	
WC	Waist Circumference
W	Water
WHO	World Health Organization
Y	
Y5.5%	Yogurt with 5.5% added sugar
Y3.3%	Yogurt with 3.3% added sugar

1 Introduction

In Canada, 73% of adults aged 65 years or older have at least one common chronic disease, such as obesity, diabetes, cardiovascular disease, cancer, hypertension, asthma, anxiety, or osteoarthritis (1). Of these common chronic diseases, obesity, diabetes, cardiovascular disease, and cancer can be diet-related (1). In 2018, 26.8% and 36.3% of Canadian adults were obese and overweight, respectively; moreover, one in three adults are diabetic or pre-diabetic (2,3). Cancer and cardiovascular disease (CVD) are the leading causes of death globally (4). In 2015, World Health Organization reported that 61% of deaths and 49% of the global disease burden were related to chronic diseases (5). By 2030, the expected number of deaths due to chronic diseases will increase to over 70% (6).

Chronic diseases continue to be an important global public health issue; because of the lifestyle, chronic diseases have a significant economic and social burden (7,8). For example, over 10 years, the total cost of diabetes in Canada was $15.36 billion (9). Additionally, the Canadian federal government recently provided funding of $300 million to fight chronic disease, emphasizing improving diet and physical activity level (10). The new Canada's Food Guide identifies the importance of protein-rich foods by recommending unsweetened lower-fat yogurt and milk (11). Research has also underlined the high consumption of sugar and impact on metabolic health (12). Higher consumption of sugar-sweetened beverages is related to weight gain and an increased risk for developing chronic diseases (13). Naturally occurring sugars and added sugars are two types of sugars in the diets. Natural sugars come from plant food sources (fructose, dextrose and sucrose) and milk (lactose), while added sugars are all sugars added to foods and beverages to improve taste or for preservation. It is known that type-2 diabetes is associated with high added sugar intake (14).

In Canada, the recommended amount of free sugar intake per day should be less than 10% of a person's total daily calorie intake (14). One possible way to decrease added sugar consumption among Canadians is to reduce the added sugar content in sugar-sweetened beverages (14). In addition, a reduction of free sugar consumption to less than 5% of total daily calorie intake might provide additional health benefits to reduce the risk of diabetes (14). This could possibly boost Canadian economy through decreasing health care costs related to chronic diseases (15).

The primary objective of the study is to investigate the effect of a partial reduction of added sugar in chocolate milk and yogurt on postprandial blood glucose. The secondary objective is to evaluate whether the reduction of added sugar in sweetened dairy products affect subjective appetite and short-term food intake as well as other parameters related to food sensory perception and subjective feeling of physical comfort.

2 Literature Review

2.1 Non-communicable/Chronic Diseases in Canada

Chronic diseases are characterized as non-communicable diseases due to the highest cause of death in the world (16). World Health Organization (WHO) also specifies non-communicable diseases as long-duration diseases that are resulted by a combination of genetic, physiological, environmental and behavioural factors (17). According to Statistics of Canada, obesity (26.8%), diabetes (11%), cancer (8%), hypertension (25%), and ischemic heart disease (8%) are the most common non-communicable diseases in Canada (1). In addition, obesity, diabetes, cancer, and cardiovascular disease can be related to an individuals' diet and lifestyle (1).

2.1.1 Overweight/Obesity

Obesity is a chronic disease defined as an excess proportion of adipose or fat tissue (18). Body Mass Index (BMI) is used to identify obesity; BMI provides a good estimation of the amount of body fat (19). The calculation of BMI is completed by dividing a person's weight (kg) by their height squared (m^2) (19). BMI helps to classify individuals into categories; according to WHO, a BMI below 18.5 kg/m^2 is underweight, a BMI between 18.5–24.9 kg/m^2 is normal weight, 25.0–29.9 kg/m^2 is pre-obesity/overweight, 30.0–34.9 kg/m^2 is obesity class I, 35.0–39.9 kg/m^2 is obesity class II, and above 40 kg/m^2 is obesity class III (20).

In Canada, there was an increase in the prevalence of obesity from 2015 to 2018 (2). In 2018, the prevalence of overweight and obesity was 36.3% and 26.8% among Canadians 18 years and older, respectively (2). Chronic diseases such as cardiovascular disease, type-2 diabetes, obesity related cancers, osteoarthritis, non-alcoholic fatty liver disease and psychological disturbance are associated with increasing levels of obesity (21). For example, in 2018, 29.5% and 6.0% of adults with obesity were diagnosed with high blood pressure and heart disease, respectively. In contrast,

9.5% and 2.7% of adults with a normal BMI were diagnosed with high blood pressure and heart disease, respectively (22). The prevalence of type-2 diabetes is lower among Canadians with a normal BMI (2.9%) compared to Canadians with obesity (13.4%) (2). Research has shown that obese children are at a higher risk of becoming obese during adulthood, especially if their parents are obese (2). Genetics, inadequate physical activity, unhealthy diet, socio-economic status, behavioral, cultural, and physical environmental risk factors are the well-known risk factors related to obesity (2).

2.1.2 Cardiovascular Diseases

Heart disease is the most common type of heart condition in Canada and is the second leading cause of death resulting in 20% of Canadians reported death in 2012 (2). In 2015, 45% (8.9 million) of all non-communicable disease deaths were reported as a cause of heart disease (2). Since 2000, the prevalence of ischemic heart disease (IHD) in Canada, has increased from 1.5 to 2.4 million (2). In 2012, Canadian adults with IHD had three times higher mortality risk than individuals without IHD (2). The mortality risk for those with IHD was 18 and 11 times higher for younger women and men aged 20 to 39, respectively, than individuals of the same age without IHD (2). IHD or coronary heart disease is known as a heart disease in which the heart muscle is damaged and does not function properly (2). Lifestyle behaviours such as smoking, poor diet, drugs, physical inactivity, and excessive alcohol use are risk factors for IHD (24). Supporting this statement, research with younger adults showed that there was a positive association with CVD mortality and a sugar-sweetened beverage intake of ≥2 servings/day (25). Medical conditions such as high blood pressure, diabetes and high cholesterol also can increase the risk of heart disease (24).

2.1.3 Diabetes

Diabetes is a chronic disease characterized by high levels of blood glucose due to the inability to be metabolized in cells (26). The pancreas loses its ability to produce or use insulin properly (26). The three main types of diabetes are type I, type II, and gestational diabetes (26). Type I diabetes is an autoimmune disease in which the pancreas does not produce insulin (26). Type II diabetes is a metabolic disorder where body cells are resistant to insulin production and therefore gradually decreasing production of insulin (26). Gestational diabetes is a maternal glucose intolerance which presents during pregnancy and is associated with issues during pregnancy and birth. Women with gestational diabetes are at a higher risk for type II diabetes after birth (26). There are two other categories of glucose intolerance, impaired fasting glucose (IFG) and impaired fasting glycemia (IGT), they present as fasting blood glucose levels between the ranges of normal (between 3.6 mmol/l and 6 mmol/l and diabetic (6.1 mmol/l and 6.9 mmol/l) (27). People with IFG and IGT are at higher risk of cardiovascular disease compared to people with normal blood glucose levels (28,29). In 2017, the prevalence of type II diabetes was the highest among Canadian adults (26). The prevalence of diabetes increased by approximately 3.3% per year from 2000 to 2018, and 2.16 million new cases are projected by 2022 (4,35). Diabetes causes an economic burden for many Canadians; the out-of-pocket costs are approximately $1,100-$2,600 per year for daily insulin injection, $1,400-$4,900 per year for insulin pump therapy, and $1,200-$1,900 per year for oral medication (26). Health planners and policymakers who focus on diabetes prevention should consider using different intervention strategies against diabetes cases for the future (31).

2.2 Diabetes: Classification, Pathogenesis, and Diagnosis

Diabetes Canada described Diabetes mellitus (DM) as a metabolic disorder characterized by chronic hyperglycemia and disturbances of carbohydrate, fat and protein metabolism resulting from defects of insulin secretion or/and insulin action (29). Chronic hyperglycemia is commonly

related to subclinical inflammation, and long-term microvascular damage affecting the eyes, kidneys, and nerves and increasing the risk of cardiovascular disease (32).

2.2.1 Classification

Diabetes mellitus is classified into three main types that are type I diabetes mellitus (T1DM), type II diabetes mellitus (T2DM), gestational diabetes mellitus (GDM) (29). In addition, prediabetes is another diagnosis that is related to developing diabetes (29).

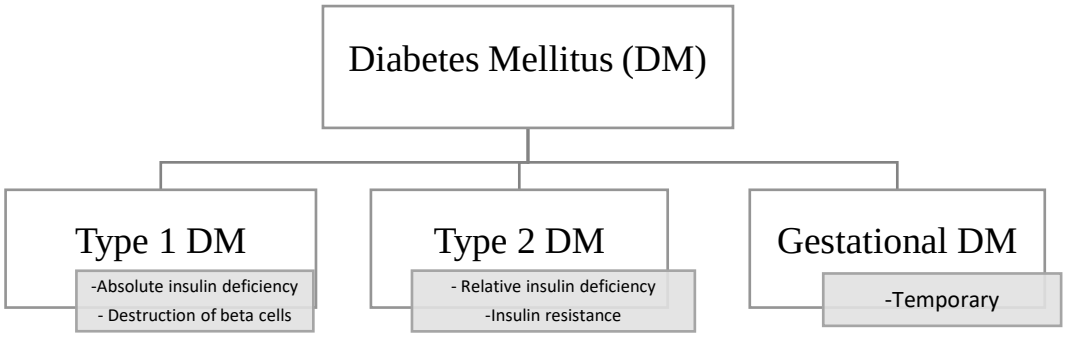


Figure 2. 1 The Classification of Diabetes (29)

T1DM (10% of cases) is an autoimmune disease known as insulin-dependent diabetes (29). The body would has and issue utilizing the insulin produced, or the body would not produce enough insulin (29). Also, T1DM usually develops in childhood, although it can also occur in adulthood (29). T2DM (90% of cases) is known as insulin-independent diabetes. T2DM is commonly developed in adulthood; however, it can also occur in childhood (29). GDM (3%-20% of pregnant women) is a temporary form of diabetes that occurs during pregnancy. GDM increases the risk of developing T2DM later in life (29).

2.2.2 Pathogenesis of Diabetes

Genetic and environmental factors affect the development of T1DM. Genetic factors affect the predisposition for triggering islet autoimmune responses and help to accelerate the failure of β -

cell secretion in response to environmental factors, such as obesity and diet. The environmental risk factors for T1DM are maternal factors, virus infections, dietary factors, psychological stress, and toxic substances (32).

T1DM results from an almost complete loss of insulin, while in T2DM, the peripheral tissues resist the effects of insulin (33). In T1DM, the pancreas does not secrete insulin, which predisposes people to ketoacidosis due to the destruction of the pancreatic β-cells (32).

T1DM includes two distinct stages in genetically susceptible individuals. Anti-islet and autoantibodies usually develop by 9 months and 9 months to 3 years, respectively (34). Firstly, there are triggers of autoimmunity resulting in one or multiple islet cell autoantibodies associated with the gradual destruction of β-cell (32). Secondly, the loss of β-cell secretory function results in the loss of first-phase insulin release, reduced C-peptide levels, glucose intolerance, and finally hyperglycemia (32). The destroyed β-cells are explained as the infiltration of pancreatic islets by CD4 + and CD8 + T lymphocytes and macrophages leading to insulitis (32).

T2DM is caused by a decrease in insulin secretion by pancreatic β-cells and the inability of insulin-sensitive tissues to respond to insulin appropriately; this can be due to a combination of genetic, metabolic, and environmental risk factors (35). Obesity may cause insulin resistance, resulting in β-cell's inability to regulate normal glucose homeostasis (32). Thus, individuals' insulin sensitivity must be considered to assess β-cell function, the main stimulus for insulin secretion (32). β-cells are responsible for the regulation of blood glucose (36,37). Glucose transporter (GLUT2), located on the β-cells, transports glucose inside the β-cells (36). After the transportation of glucose into the β-cells, glucose undergoes glycolysis, and adenosine triphosphate (ATP) is generated (36). Generated ATP results in an increased ATP/ADP ratio, which leads to the closure of ATP-sensitive K+-channels (KATP-channels) (36). The K+-channels depolarize the cell membrane and causes

the opening of voltage-dependent Ca+-channels (36). The increase in intracellular calcium concentrations helps fuse insulin-containing granules with the membrane, and insulin secretion begins (36).

The mass, compensation, and function are important factors in β-cell regulation and maintenance (38). In T2DM, the survival of β-cells is essential because T2DM is characterized by a progressive loss of these cells (39). The regulation of β-cell mass is crucial for islets to maintain normal glucose concentrations against increasing glycemia and decreasing insulin sensitivity; otherwise, β-cells cannot meet insulin demands (35,40).

In summary, inadequate glucose concentrations decrease insulin secretion and causes β-cell dysfunction; when glucose concentrations increase it manifests as hyperglycemia (38). A well-balanced, healthy diet with limited saturated fat content and recommended daily intake of nutrients protects β-cells from destruction, and also helps to enhance longevity in healthy individuals (41). Furthermore, reduced energy intake combined with exercise is essential for improving insulin sensitivity (41). A Mediterranean diet reduces insulin resistance due to decreased dietary fat intake, saturated fatty acids, and trans-fatty acids; insulin resistance, the strongest predictor for T2DM, is mainly caused by increased adiposity tissue due to chronic high energy intake (41). For example, a study showed that a 12-weeks hypocaloric diet in T2DM patients helped decrease liver fat content (85%), explicitly associated with normalization in hepatic insulin sensitivity and reduced fasting hyperglycemia (42).

2.2.3 Diagnosis of Diabetes

Diagnosing diabetes is standardized with quality assured laboratory methods based upon venous blood samples (43). There are four methods to diagnose diabetes: (1) fasting plasma glucose (FPG) ≥ 7.0 mmol/L after at least eight hours fasting, (2) glycated hemoglobin value (HbA1C) ≥ 6.5%,

(3) two-hour plasma glucose (2hPG) value with including an oral glucose tolerance test (OGTT) of 75 g of glucose ≥ 11.1 mmol/L, and (4) random plasma glucose (PG) at any time of the day without regard to food intake ≥ 11.1 mmol/L (43). American Diabetes Association (2013) mentioned that the use of HbA1C is not recommended as a diagnostic test for diabetes because HbA1C testing shows an estimate of mean plasma glucose levels for the last 8 to 12 weeks and may not show the correct glycemic status when the red blood cell turnover short or extend (44,45). In recent years, A1C has been recommended for diagnosing T2DM and not for diagnosing diabetes in children and adolescents, screening for gestational diabetes, or T1DM (46).

Table 2. 1 Normal, Pre-diabetes, and T2DM Blood Test Parameters (46)

Test Name	Normal	Pre-Diabetes	T2DM
FPG	< 6.1mmol/L	6.1 - 6.9 mmol/L	≥ 7.0 mmol/L
HBA1C	< 6.0%	6.0% - 6.4%	≥ 6.5%
OGTT	< 7.8 mmol/L	7.8 - 11 mmol/L	≥ 11.1 mmol/L

2.3 Blood Glucose Regulation

2.3.1 Blood Glucose Concentration

The level of blood glucose is homeostatically regulated with insulin and glucagon to restore blood glucose to normal levels (36,47). β-cells in the pancreas are responsible for insulin secretion when blood glucose concentrations are high, and insulin stimulates body cells to increase the level of glucose uptake from the blood (36). As a result of this insulin secretion, blood glucose concentrations decrease (36). Additionally, insulin stimulates the formation of glycogen from glucose, which is stored in liver and muscle cells (44,55). Thus, blood glucose concentrations

decrease because glucose in the blood is used to create glycogen (36). In addition, α-cells in the pancreas are responsible for glucagon secretion when the blood glucose concentrations are low (36,48). Glucagon helps to break down glycogen to glucose in liver and muscle cells and increase blood glucose concentrations (36).

2.3.2 Glucose Homeostasis

Glucose is a primary carbohydrate required for energy production in the brain, and it is essential for the metabolic substrates in all cells of the body (49). Blood glucose concentrations is regulated in the body to avoid both hyperglycemia and hypoglycemia that can lead to serious clinical outcomes (49). For example, low blood glucose concentrations (hypoglycemia) can cause the loss of consciousness, seizures, and death (49). On the other hand, long-lasting elevation of blood glucose (hyperglycemia) may cause renal failure, neuropathy, blindness, and vascular disease (49).

Hormones and neuropeptides released from the pancreas, liver, intestines, brain, muscle, and adipose tissue affect the complex regulation of glucose metabolism (50). The net effect of the influx and efflux mechanisms of glucose from the arterial blood glucose level is between 3.5 mmol/L (after exercise) to 9 mmol/L (following a meal), whereas fasting levels are limited within a narrow range of 4-5.5 mmol/L (50).

Glucose regulation was traditionally explained through the islet-centered hormones, especially insulin and glucagon, and are important for glucose homeostasis (51,52). According to normal conditions in the islet-centred model, glucose homeostasis is controlled primarily by the effect of rising blood glucose levels, stimulating insulin secretion; insulin affects the peripheral tissues such as liver to reduce the hepatic glucose production (HGP) (52,53). At the same time, adipose tissue reduces lipolysis and muscle stimulates glucose uptake in response to insulin action (52). Some individuals' islet functions create insulin-resistance due to dietary and genetic factors (54). The

capacity of the islet to increase insulin secretion preserves glucose homeostasis in a compensatory manner (38,52). Moreover, glucose intolerance results if the islet dysfunction precludes the increase of insulin secretion needed to overcome insulin resistance (38,51,52). Increased HGP with reduced tissue glucose uptake can progress islet dysfunction, eventually causing overt hyperglycemia and diabetes (52).

Traditionally, glucose regulation was explained through the pancreatic islet-centered glucose control system. Now, a new model explains glucose regulation through the contributions of defective brain- and islet-centred glucoregulatory systems (52). In the new glucose regulation model, the brain and pancreas are the primary control organs for glucose homeostasis. However, glucose regulation is also affected by liver, gut, adipose tissue and muscle through hormones, neurotransmitters, and cytokines (52).

Islet-centered glucose homeostasis is determined by blood glucose levels (50). After a meal, increased blood glucose levels activate the β-cells of the pancreatic islets of Langerhans to secrete insulin (50). Opposite effects occur during the fasting state or inter-prandial state. After fasting, lower blood glucose levels stimulate the glucagon secretion from α-cells of the pancreatic islets (55). The brain controls nutrients and hormones through sensing glucose levels after ingesting the ingestion of a meal (51). In summary, the brain-centred glucose regulatory system can regulate tissue glucose metabolism and plasma glucose levels whether insulin-dependent or insulin-independent (52). Both islet and brain-centred pathways have critical roles to regulate blood glucose levels and tissue insulin sensitivity (52).

2.3.3 Glucose Transportation

The transport of glucose into insulin-sensitive cells is catalyzed by transport proteins (GLUT) (56). GLUT4 is insulin-dependent, while GLUT1, GLUT3, and GLUT2 are insulin-independent (56).

GLUT2 is the major glucose transporter responsible for inactivating, suppressing glucose uptake, and increasing insulin secretion (57). GLUT2 is stated in liver, intestine, kidney and pancreatic islet β-cells (57). GLUT4 is responsible for metabolizing or storing the glucose in muscle and adipose tissues when insulin is stimulated, for example, after a meal. However, GLUT4 as an insulin-dependent glucose transporter mediates the insulin-stimulated glucose removal in muscle tissue (56,57). GLUT3 controls the uptake of glucose into the neuron while GLUT1 transports the glucose across the blood-brain barrier (58). There are two functional components of glucose 6-phosphate formation within the cell to help understand glucose's entrance way, whether GLUT1 or GLUT4 (56). If glucose is entering through GLUT4, glucose phosphorylated by hexokinase II is directed to glycogen synthesis and glycolysis (56,57). However, if glucose is entering through GLUT1, glucose is phosphorylated by hexokinase I and, the produced glucose 6-phosphate is available for all metabolic pathways, especially hexosamine pathway (56,57). Hexosamine pathway negatively affects GLUT4 because it decreases insulin-dependent glucose transport (56). As GLUT1 and GLUT4 are important for glucose homeostasis, abundant GLUT1 can decrease the insulin-mediated glucose transport through GLUT4 leading to insulin resistance (56,57).

2.3.4 Insulin Secretion

Insulin is an important regulator of metabolism, produced in β-cells (59). In the beginning, insulin is synthesized as preproinsulin and then forms to the proinsulin (59). Proinsulin is transformed to insulin and C-peptide before storing in secretary granules to release on demand (59). Insulin secretion has two main phases. In the first phase of insulin response, after eating a meal, insulin immediately releases from insulin secretor vesicles at the β-cell membrane and peaks by five minutes lasting no longer than ten minutes (60). The second phase results from recruitment of insulin secretory vesicles to the β-cell membrane controlled by intracellular calcium levels (60).

The second phase is also known as a long-term release of insulin and happens if blood glucose remains high (61). First phase insulin response is crucial in diabetes research because the release of insulin determines the efficiency of subsequent meal glucose disposal (51,60). Insulin regulates postprandial blood glucose rise by inhibiting gluconeogenesis and glycogenolysis within 30-60 minutes after a meal intake (47).

2.3.5 Postprandial Glycemia

Postprandial glycemia is described as the plasma blood glucose concentrations after a meal (62). Carbohydrate absorption, insulin and glucagon secretion, and their effects on glucose metabolism in the liver and peripheral tissues determine the postprandial glucose (62). Fasting plasma glucose concentrations range between 70 and 110 mg/dL (3.9-6.1 mmol/L) in nondiabetic individuals (62). Ten minutes after starting a meal, glucose concentrations begin to increase due to the absorption of dietary carbohydrates (62). Plasma glucose concentration peak 30-60 minutes after the meal and can reach a maximum of 140 mg/dl, and returns to pre-prandial levels within 2-3 hours depending on the composition and quantity of the meal, as well as processing and cooking methods (62). The absorption of ingested carbohydrate continues for a minimum of 5-6 hours (62).

High plasma glucose concentrations are toxic to the body, therefore, homeostatic mechanisms maintaining glucose tolerance regulate the plasma glucose concentrations (63). Postprandial glycemia plays an essential role in body weight control by affecting appetite, or increased insulin and glucose on nutrient partitioning (64). Furthermore, postprandial glucose is also related to hyperinsulinemia and lipidaemia, implicated in chronic metabolic diseases such as obesity, T2DM, and CVD (64).

The amount and type of carbohydrates is important for individuals to regulate blood glucose because different carbohydrates have different glycemic effects. Foods with low glycaemic index

(GI) are slowly digested and release glucose gradually compared to foods with high GI (65). Studies have shown that low GI foods help control blood glucose excursions result from a meal (66).

2.4 Effect of Macronutrients on Postprandial Blood Glucose Levels

Many systems in human body aid in maintaining stable blood glucose levels, it means hormones generated by the diet (67). Insulin is the hormone that is involved in the metabolic communication system; however, increased inflammation affects these communication systems and causes metabolic defects such as diabetes, obesity, and metabolic syndrome (68). Thus, keeping insulin within a therapeutic zone is essential for humans' survival because insulin is the primary regulator of macronutrients, including carbohydrates, fat, and protein (68). Long-term insulin dysfunction causes insulin resistance, a metabolic disorder related to many pathways such as lipid metabolism, energy expenditure, and inflammation (69,70). Insulin dysregulation, or insulin resistance, is the main reason for T2DM (71). The amount and type of foods and macronutrients are important for managing post-prandial blood glucose (72).

2.4.1 Dietary Protein

There is insufficient evidence that protein intake should be modified in diabetic patients with normal renal function (73). It is documented that diabetes may cause protein deficiency; however, whether there is an effect of long-term high protein intake above 20% of total energy on renal function is unclear (73–75). In addition, although short-term high protein intake may improve glycemia and weight control, it has not been determined whether these benefits are the same for long-term high protein intake (76,77). Some studies mention that very high protein diets, long-term, especially animal protein, might increase the risk of T2DM (78). Dietary proteins and amino acids strongly facilitate glucose metabolism and affect satiety and energy intake. However, well-

controlled long-term studies are needed to know the optimal macronutrient composition for the treatment and prevention of obesity and T2DM (79).

2.4.2 Dietary Fat

Insulin resistance is related to damaged communication between insulin and the interior of a target cell due to inflammation, affected by the fatty acid composition of the diet (67). Thus, fat content in a diet, and the composition of fatty acids, might affect the modulation of insulin resistance (67). However, the fat types play an important role in whether inflammation in insulin resistance is beneficial or harmful (67). Some epidemiologic studies have found that saturated fat or meat intake harm insulin sensitivity, while polyunsaturated fat (or vegetable fat) intake improves insulin sensitivity and glucose tolerance (80–83). Controlled clinical trials also have found that high saturated fat (SAFA) diets worsen insulin sensitivity and glucose tolerance relative to high monounsaturated fatty acid (MUFA) diets (84). Furthermore, a multicenter study reported that the beneficial effect of MUFA diet disappears when total fat intake exceeds 38% of total energy (85). Hence, researchers suggested that total fat intake may have a more substantial impact on insulin sensitivity than fatty acid composition (85). In relation, another study demonstrated that insulin sensitivity measured by intravenous glucose tolerance test was 40% higher in a moderate-fat diet (34% total fat, 16% MUFA) than a high-MUFA diet (39% total fat, 21% MUFA) (86). In addition, a high-MUFA diet relative to a high carbohydrate diet reduced postprandial glucose and insulin levels in T2DM patients; however, a high-MUFA diet did not improve insulin sensitivity in patients with impaired glucose tolerance (87,88).

Omega-6 and omega-3 are the two categories of polyunsaturated fatty acids. In particular, omega-6 fatty acids are considered pro-inflammatory molecules, whereas omega-3 fatty acids are considered anti-inflammatory (67). Long-chain omega-3 fatty acids found in fish oil appear to

have a positive effect on insulin action in humans (89). Several studies have found that fish oil improved insulin sensitivity and intravenous glucose tolerance in healthy individuals (90). In addition, some studies demonstrated the importance of omega-3 supplementation (91). For example, studies discovered insulin sensitivity measured by frequently sampled intravenous glucose tolerance was improved by 17% in subjects supplemented with docosahexaenoic acid (DHA), one of the major omega-3 fatty acids (91).

In conclusion, high total fat in the diet appears to have an adverse effect on insulin sensitivity (89). Collectively, the data support the idea that a daily dietary fat intake of less than 30% of total energy, with a low intake of saturated fat, is appropriate to improve insulin sensitivity and prevent T2DM (89).

2.4.3 Dietary Carbohydrate

The amount of carbohydrates in a diet is an important factor for postprandial glucose in people with T1DM and T2DM (92). Besides the amount of carbohydrates, the quality of carbohydrates is vital for individuals to regulate blood glucose because different carbohydrates have different glycemic effects (32). Starch for example, influences glycemic response differently due to the amount of dietary fibre and the type of sugar (32). Foods that release their carbohydrates at a slow rate should be preferred in the presence of dysfunctional glucose regulation due to their effects on reducing postprandial glycemic fluctuation through reduced insulin demands (93). This interaction between types of carbohydrates and glycemic response is described as the glycemic index (GI) (93). Furthermore, in a comparison model for the glycemic response, GI compares a reference food with the same amount of carbohydrate, usually containing 50 g glucose as a drink or 50 g of available carbohydrate in a portion of white bread to test the food (32). GI is a scale that shows how much a carbohydrate-containing food or drink raises blood glucose and post-prandial blood

glucose response over 2 hours after it is consumed (32). In Canada and Australia, foods are differentiated into high (GI: 70 – 100), average (GI: 55 – 70) or low (GI: < 55) GI foods (32). Legumes, pasta, parboiled rice, wholegrain bread, oats, and certain raw fruits are low GI; while mashed potatoes, white rice, white bread, cookies, and sugary drinks are high GI (32). Foods with a low GI increase blood glucose less and slower than foods with a high GI, so it may affect HbA1c and insulin sensitivity and lower the incidence of hypoglycemic episodes (65,93).

On the other hand, glycemic load (GL) describes the quality (GI) and quantity of carbohydrates in a food serving, meal, or diet, while the GI compares the potential of foods containing the same amount of carbohydrate to raise blood glucose (94). Studies have found that low GI and GL diets may improve glycemic control and insulin sensitivity and reduce the risk of cardiovascular disease and T2DM (93,95).

2.5 The Link Between Sugar Consumption, Diabetes, and Other Metabolic Diseases

2.5.1 Sugar and Obesity

Simple sugar is known as a high GI food (94). Excessive sugar intake can lead to excessive energy intake, leading to overweight and obesity (96). Globally, over 17 million people die each year because of adiposity-related chronic diseases related to sugar-sweetened beverages. Sugar-sweetened beverages increase the risk of weight gain due to the high energy content (15). In addition, sugar-sweetened beverages are the biggest contributor in consuming added sugar in the United States (97). In the Western diet, 7.1% of total energy intake comes from sugar-sweetened soft drinks, the primary source of calories, therefore, childhood obesity increased parallel to sugar-sweetened beverages intake (98).

2.5.2 Sugar and Other Metabolic Disorders

There are two pathways in which sugar increases metabolic risk directly or indirectly. First, the added sugar intake may cause dysregulation of lipid and carbohydrate metabolism, increasing the risk of metabolic disease (99). Secondly, added sugar intake may promote weight gain, which causes dysregulation of lipid and carbohydrate metabolism (99). High carbohydrate intake, mainly sucrose and fructose, may increase serum triacylglycerol concentrations, dyslipidemia, and insulin resistance and decrease serum HDL cholesterol, therefore increasing CVD risk (100,101)**.**

2.5.3 Sugar and Type-2 Diabetes

According to Diabetes Canada, over 20 Canadians die every 24 hours because of diabetes-related complications (14). Excessive sucrose (sugar) intake was inversely related to the risk of T2DM whereas fructose and glucose intake have not been proven (102). A meta-analysis of a prospective cohort study determined that fructose intake was not related to T2DM, whereas sucrose intake was related to T2DM (103). However, even if the high consumption of sugar increases the risk of T2DM, sugar intake is not the only reason for the development of diabetes (104). The connection between sugar and T2DM illustrates high energy intake; high sugar-containing foods and drinks are calorie dense, therefore, the increased consumption of added sugar is correlated to gain weight (104). Sugar-sweetened beverage intake affects body adiposity and metabolism, so a growing body of evidence shows that increased added sugar intake is associated with the increased risk of T2DM (105). For example, 17 million individuals in the United States were affected by T2DM, in parallel with the obesity epidemic during the last decades; sugar-sweetened beverage intake increased by 61% within the past 20 years (13).

2.6 Appetite Control

Obesity is a critical global public health problem, increasing the risk of other metabolic diseases (106). A primary weight loss strategy is energy restriction; however, most regimens that reduce

energy intake for a long period have been shown to fail by participants, and an alternative solution may be appetite management as a method of weight control, consuming dietary macronutrients to intensify satiety effects on a meal and reduce food intake (107). Many factors that lead to hunger and eating play an essential role in food consumption, including various aspects that dictate the feelings, emotions, and actions towards the consumption of foods (108). The long-lasting feeling of fullness after a meal can be explained as satiety that is gradually generated during food consumption and reduces other meals. The feeling of hunger and satiety process cyclically and continues during the day (109).

The holistic point of view explains that sensory perception, physiologic and metabolic processes, psychological responses, and attitudes create the understanding of satiety (109). Sensory perceptions consist of the combination of visual stimulation, odor, and previous experiences, along with initial physiological responses that lead to the first responses to food ingestion (110). Also, the food texture is another relevant factor related to satiety because it affects metabolic processes due to state (liquid vs. solid), particle size, or viscosity (111).

From a physiological point of view, hormones in circulation have different responsibilities; for example, some act acutely to initiate or terminate a meal, where others reflect body adiposity and energy balance (112). In the epithelium of the gastrointestinal system, specialized enteroendocrine cells secrete hormones that are responsible for appetite control, such as cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), leptin, and peptide YY (PYY) into the blood stream (113). These hormones suppress hunger and food ingestion, delay gastric emptying, inhibit gastric acid production, induce pancreatic insulin secretion, contract the gall bladder, and slow gastrointestinal motility (113). In addition, one of the hormones, ghrelin, suppresses insulin secretion, promotes hunger, and initiates food ingestion (113). Furthermore, peripheral nerves and brain centers, such

as the hypothalamus and brain stem, integrate the signals to regulate central neuropeptides, which modulate feeding and energy expenditure (112).

Adiposity and body weight are mostly steady even though there are big variations between daily food intake and energy expended in most adults (112). This is due to the fact that there is a powerful and complex physiological system that balance energy intake and expenditure in human body (112). This physiological system comprises numerous pathways to maintain the desire to eat (112). As a result, the energy homeostasis regulates body weight in most cases; however, there is an argument that evolutionary pressure causes unlimited desire to eat when food is ready (112). The inequality between the hormonal system in human's body and the current availability of food may increase the prevalence of obesity contributing to over-eating (112).

2.6.1 Measuring Food Intake, Hunger, Satiety, and Satiation

The two processes that affect eating behaviors are satiation and satiety (114). When assessing these processes, objective measures such as energy intake, and subjective measures, such as the rating of appetite-related sensations, are used (114). Satiation is an experimental measurement of ad libitum food intake under investigation during an eating occasion for a study participant (114). Controlled conditions are essential for satiation measurement to minimize the impact of environment-related and cognitive confounding factors (114). On the other hand, satiety is a measurement indicating the importance of measuring the magnitude or duration of changes in subjective ratings of appetite-related sensations with or without energy intake at a subsequent meal (114). A self-report scale, such as a Visual Analogue Scales (VAS), is a method for the measurement of appetite sensations which consists of questions, in a controlled setting, in response to an eating occasion right before and after consuming a preload or a test meal and then at regular time intervals. The standard VAS is a 100-150 mm horizontal line. Furthermore, it is important to know the differences between satiation and satiety. For satiation, food form (liquid, solid), energy

density (kcal/g), and macronutrient composition (percentage of energy from fat, carbohydrate, protein), as well as the predominant sensory properties and intensities, including the palatability and variety of foods served are the described factors at the end of the meal (110). However, satiety describes the factors influenced by food composition and total calories consumed (110). Energy (caloric) intake measurement assesses satiation/satiety effects by accurately monitoring the food consumed ad libitum (114). Satiation is usually measured in terms of test food by itself or as a part of the measured meal (115). In contrast, satiety is based on the energy intake measurement from the test meal within a precisely determined time after the preload (114).

2.6.2 Physiological Implication of Sugars on Satiety

Between 45% and 60% of total energy intake comes from carbohydrates as the primary macronutrient in most diets (116). Besides providing energy, carbohydrate ingestion promotes the regulation of food intake affecting many aspects of brain function (117). According to the glucostatic theory of food intake regulation, glucose is central to regulating satiety and food intake (118). Furthermore, this theory proposes that if glucose is inadequately used by various organs, the food is ingested while blood glucose concentration is monitored (118). However, satiety and the termination of food intake arise after the blood glucose concentration increases (118). The complete physiological mechanism of sugar on satiety is still unknown, however research has suggested that sugars have considerable effects on appetite and food intake include sucrose, glucose and fructose (119,120).

In the hypothalamus, glucose-sensing cells determine blood glucose control, which is vital to regulating the energy status of an organism (119). However, there are two converse theories based on scientific evidence to explain the satiating effect of sugar (121). The first one suggests that consuming sugar or carbohydrates can increase satiety, regulating self-control mechanisms to

reduce the intake of other caloric foods (122). In contrast, the second theory suggests that consuming sugar or carbohydrates might increase the intake of other foods. In addition, some research indicated that sugar-rich foods activate areas of the brain responsible for appetite control (123). For example, a study reported that the pleasure of eating and appetite can return when a dessert is served even after a large meal (124).

It has been assumed that the sweet taste encourages food intake and causes excessive consumption (125). Conversely, some studies mentioned that sweetness does not stimulate food intake and may have a weak impact on satiety (126,127). For example, some studies related to the effect of sucrose drinks on satiety and food intake showed that the level of sweetness in a control treatment may influence the interpretation of the data (125). On the other hand, some studies support that the sweet taste alone has been contributing to reducing hunger and increasing feelings of fullness (128). For instance, in a study with young males, drinks containing 25 g (418 kJ) and 50 g (836 kJ) of sucrose did not differ from the water control; however, they were different from the noncaloric sweetened control on food intake (129).

Sucrose and reduced food intake satiated both adults and children when the time intervals between the preload and the test meal were sufficient for the dose consumed (125). It was demonstrated that consuming drinks containing ≥ 50 g sucrose presented 20–60 min before a meal reduced mealtime food intake, showing that appetite regulatory centers respond to sugar's energy content (130). In another study, the consumption of a beverage containing a more substantial amount of sucrose (135 g) increased the feeling of fullness and reduced hunger compared to the water control for 2-3 hours (131). Moreover, studies have demonstrated that even a small amount of sucrose can result in decreased food intake. For example, when young male participants consumed a 300 mL

beverage containing 25 g, 50 g, or 75 g sucrose, the lowest dose, 25 g (418 kJ), increased subjective satiety and suppressed food intake from a pizza meal 1 h later compared to the water control (129).

Another study showed that two different sugar-sweetened beverages consisting of 20 g (83 kcal) and 40 g (166 kcal) sucrose did not show a statistical difference in food intake and appetite ratings (127). Similarly, most of the literature suggests that caloric preloads do not consistently decrease food intake if they include less than 200 kcal (130). As a result, further studies need to be conducted to fully understand sugar and its effects on satiety, by comparing other carbohydrate sources (120).

2.6.3 Dairy Products, Satiety and Food Intake

Sensory attributes are important for eating behaviours, encouraging food intake (132). Food choice and portion size are usually decided before food consumption depending on food liking, flavour, odour, and texture (133,134). As texture and flavour are essential dimensions of sensory attributes, the type of food products might have different effects on satiety (135). For example, liquid products generally have a lower satiating capacity than iso-caloric semi-solid and solid products (136). In addition, shorter duration or/and lower intensity of the sensory signal may also reduce satiation (137). For instance, semi-solid or solid food intake compared with liquid food intake may attribute to a higher sensory signal in satiating capacity (138). A study explored the expected satiation on six different commercially available dairy foods; results indicated that increased thickness of dairy products presented an increase in expected satiation, whereas flavour did not significantly affect the expected satiety (135).

Another study demonstrated the role of oral sensory in learned satiation by including different treatments such as liquid yogurt with a straw, liquid and semi-solid yogurt with a spoon, and pudding with a spoon; all the treatment groups were repeated with low and high-energy densities using noncaloric sweeteners (139). The liquid and semi-solid yogurts consumed with a spoon did

not affect the eating rate (139). Conversely, liquid yogurt consumed with a straw showed a 20% higher intake than liquid yogurt consumed with a spoon; however, hunger and fullness after the ad libitum breakfast did not show any difference between intervention groups (139). Besides sensory attributes, metabolic consequences after a meal consumption are also connected to food intake (140). Similar to this suggestion, a research study compared the effect of water (0 kcal), soy beverage (200 kcal), 2% milk (260 kcal), 1% chocolate milk (340 kcal), orange juice (229 kcal) and cow's milk-based infant formula (368 kcal) on their food intake, subjective appetite, and blood glucose (141). The results showed that the two beverages with the highest energy content, chocolate milk and infant formula, suppressed food intake more at 30 min than the control group (water) (141).

2.7 Sugar Intake and Nutritional Policies

2.7.1 Sugar intake in Canada

There are two types of sugars in North American diets which are naturally occurring sugars and added sugars. Natural sugars are a type of carbohydrate that come from fruits (fructose) and milk (lactose). Added sugars are all sugars added to foods and beverages to improve taste or for preservation such as white sugar, brown sugar and honey, as well as other caloric sweeteners that are chemically manufactured (142). Refined (processed) sugar is produced by sugar cane and sugar beets, the extracted sugar is typically found as sucrose (the combination of glucose and fructose) (142). The process of extracting sugars from sugar cane and beets helps to produce a large variety of sugars, for example, granulated sugar, coarse sugar, superfine sugar, pearl sugar, liquid sugar, and liquid invert sugar are grouped as regular sugars (143). Furthermore, brown sugar, icing sugar, demerara-style sugar, muscovado sugar, turbinado-style sugar, organic sugar, golden syrup, and molasses described as specialty sugars (143). The last category of sugars are named as other sugars and include raw sugar and evaporated cane juice (143). The purification process helps to produce

different crystal sizes and liquid sugars (143). These different sugar characteristics creates a variety of functions in food products (143). There are six main roles that sugar plays in food including flavour balance (sugar balances acidic and bitter flavours with its sweetness), preservation (sugar delays bacteria growing and spoilage), texture and mouthfeel (sugar gives the soft structure in baked goods and the smoothness in frozen dairy products), volume (sugar allows to different products to be tall, fluffy, or soft), colour (sugar caramelizes with heat to create a golden brown colour) and taste (sugar provides better taste for high-fibre foods) (144). As a result, sugar replacement and reformulation are challenging in some recipes, because there is no sugar replacement that can be used in every application (144).

In 2004, the average total sugar intake for Canadians was 110.0 grams, equivalent to 26 teaspoons per day, and this constituted approximately 20% of their total daily energy intake (145). According to Statistics Canada, in 2015, the data from more than 45,000 Canadians living in 10 provinces showed that the average daily total sugars intake from food and beverages was 101 grams (compared with 104 grams in 2004) for children aged 2 to 8, 115 grams (compared with 128 grams in 2004) for children aged 9 to 18, and 85 grams (compared with 93 grams in 2004) for adults aged 19 years and older (146). In addition, over one-third of the total sugar intake, for children aged 2 to 18, came from beverages (146). The statistics show that the consumption of daily total sugars decreased for all age groups between 2004 and 2015 (146). These results might be related to increased health consciousness (147).

Added sugar in children's (ages 1-8 years), adolescents' (9-18 years), and adults' (19+ years) diets accounted for 39%, 57% and 50% of their total sugar intake, respectively. Children (1-8 years) consumed a higher percentage of sugars from natural sources such as milk, fruit, fruit juice,

however; adolescents (ages 9-18 years) consumed a higher percentage of sugars from added sugars such as soft drinks, fruit drinks, cereals, molasses, honey, grains, and pasta (148).

2.7.2 Nutritional Policies on Sugar Intake

Free sugars are described as added monosaccharides (e.g., glucose, fructose) and disaccharides (e.g., sucrose, lactose, maltose) to foods and beverages (149). In Canada, the recommended amount of free sugar should include less than 10% of total daily energy intake (14). The World Health Organization (2015) also recommends that the consumption of free sugars should not exceed 10% of total daily energy for both adults and children (14). In addition, a reduction of free sugar consumption less than 5% of total daily energy intake might provide additional health benefits (149).

2.8 Dairy Products and Composition

2.8.1 Milk

Milk composition is nutritionally crucial to consumers and economically crucial to milk producers (150). Milk provides an all-inclusive source of nutrition for mammalian neonates (151). In addition, mature humans and children of sufficient renal development can consume the milk of other species, even if milk composition has differences between species because it provides essential macro- and micro-nutrients than most other food sources (152,153). There are different types of milk for human consumption, such as goat milk, buffalo milk, sheep milk, yak milk, equine milk, and camel milk (154). Thus, milk has been produced from livestock for human consumption for a long time; bovine milk is the most commercially available for large-scale production (155). Milk can provide a range of daily human dietary requirements due to the availability of fat, protein, lactose, soluble salts, vitamin, and minerals (156).

Triacylglycerol accounts for about 95% of the lipid fraction in milk, which contains fatty acids of short- (C4-C10), intermediate- (C12-C16), or long-chain (C18) length (150,156). Furthermore, other milk lipids are diacylglycerol (about 2% of the lipid fraction), cholesterol (less than 0.5 %), phospholipids (about 1%), and free fatty acids (FFA) (less than 0.5%) (157). In addition, the level of FFA affects the flavour in milk and dairy products; for example, increased levels of FFA in milk may result in off-flavour (156).

Bovine milk contains about 32 g protein/L (156). Milk proteins fall into two major groups: casein, which accounts for 80% of the total protein in bovine milk, and whey proteins, which account for 20% of total milk protein (156,158). Casein consists of four fractions, which are α-s1, α-s2-, β-casein, and k-casein at 40%, 10%, 45% and 15%, respectively (159). Whey protein consists of β-lactoglobulin (β-Lg), α-lactalbumin (α-La), serum albumin (BSA), immunoglobulins (Ig), and others (159). Casein is responsible for carrying calcium and phosphate and forming a clot in the stomach for efficient digestion (156,158). On the other hand, whey protein is rapidly digested (156).

Lactose is the main carbohydrate in milk and consists of one molecule of glucose and one molecule of galactose connected with a 1-4 carbon linkage as beta-galactoside (150). Lactose regulates water and osmotic content in milk, and because of this principal biological function, lactose becomes the most constant constituent in milk, averaging 4.6% (150). Milk also contains other minor amounts of carbohydrates such as monosaccharides, sugar phosphates, nucleotide sugars, and oligosaccharides (150).

Table 2. 2 Bovine Milk Composition (150)

Energy (kcal/100 g) = 66	Composition (g/100 g)
Water	87.3
Fat	3.9
Casein	2.6
Whey protein	0.6
Lactose	4.6
Ash	0.7

2.8.2 Yogurt

Yogurt is generally standardized from whole, partially defatted milk, condensed skim milk, cream, or non-fat dry milk (160). Nutritional significance in various milk constituents are protein, fat, lactose, and minerals (161). Also, live and active yogurt possesses provide additional health benefits (160). According to health claims by U.S. Food and Drug Administration, yogurt can claim to be an "Excellent Source" for calcium, riboflavin, and phosphorus and a "Good Source" for potassium, protein, and zinc. In addition, fat-free yogurt can claim to be free of saturated fats and can claim to be low in cholesterol (162).

Additionally, manufactured flavoured yogurt includes an extra sweetening agent added to the yogurt base (160). The level of sweetness in the yogurt mix depends on the fruit and flavouring ingredients (160). For example, most fruit-flavoured yogurts contain approximately 10%–13% sugar equivalent, while other flavours (vanilla, lemon, coffee, etc.) contain 8%-10% sucrose (160). The total sugar solids in the yogurt mix should not exceed 10%–11% before fermentation because

sugar might inhibit the yogurt culture. Therefore, the sugar is usually added before pasteurization, and non-nutritive high-intensity sweeteners are used to produce low-calorie yogurts (160).

2.9 Per Capita Intake of Flavoured Fluid Milk, Regular Milk and Yogurt

In 2018 in Canada, an annual per capita intake of total fluid milk was 65.2L (179 mL per day), and 9.87L (27 mL per day) for yogurt (163). Also, 2% milk represents the largest intake of total fluid milk consumption with 32.8L per capita, 1% milk follows as a second largest intake of total fluid milk consumption with 11.9L, whole (3.25% m.f.) milk continues as the third with 10.41L, and chocolate and other flavoured milk consumption takes the fourth largest intake of total fluid milk consumption with 5.53L (163). Skim milk has the least intake of the total fluid consumption with 4.57L per capita in Canada (163). In summary, the consumption of dairy products per capita in Canada has decreased approximately by 20 liters per year from 2004 to 2019 (163).

2.10 The Role of Dairy Intake in Diabetes

Diabetes is characterized by insulin resistance, dyslipidemias, increased oxidative stress, low-grade inflammation, and increased body weight (164). Clinical trials provided important information about lifestyle interventions for primary prevention of type 2 diabetes; however, there is still uncertainty between dietary factors and diabetes prevention (165). Some evidence supports that one of the specific dietary priorities, consumption of dairy products, may contribute to healthy lifestyles, impacting the risk of chronic diseases (166). To further support the role of dairy products in health, a public health and health economic analysis reported that if Americans consumed at least 3-4 servings/day of dairy products, the 5-year cumulative health care savings would be over $200 billion (167). However, there is still contrasting evidence on the impacts dairy products play on the risk of diabetes and other metabolic disease development (168).

A prospective study with healthy male participants demonstrated that each additional daily serving of total dairy intake decreased risk for T2DM by 9% (169). Similarly, a clinical trial that compares dairy and non-dairy snacks with the same amount of carbohydrates (25 g), found blood glucose response was lower ($p<0.01$), and insulin levels were higher ($p<0.0001$) in children who consumed dairy snacks (170). Furthermore, a meta-analysis compared the differences between the highest and lowest dairy consumption; the higher dairy consumption significantly reduced the risk of T2DM by 14% compared to the lowest dairy consumption (171). Related to previous studies, a systematic review and meta-analysis of observational studies, including ten cohorts and seven cross-sectional studies, demonstrated the relationship between different types and levels of dairy consumption and T2DM risk (172). For example, seven cohort studies showed that each additional 200g/d of total dairy product intake was related to a lower risk of metabolic syndrome components and a 12% lower risk of obesity (173). In addition, each additional 100g/d of yogurt intake showed a 16% lower risk of hyperglycemia (173).

An epidemiological study by Pei et al. mentioned that yogurt has postulated role in gut health and may help to protect the intestinal barrier and reduce inflammation in diabetic patients (165). According to European Prospective Investigation into Cancer (EPIC), yogurt may be more convenient than some types of dairy products for the protection against diabetes (174). Similarly, a study which involved eight European cities with 511 adolescent participants determined higher consumption of yogurt and milk was associated with lower body fat, lower risk for cardiovascular disease, and better fitness (175). These epidemiological studies clearly showed that yogurt has a protective effect on diabetes by lowering insulin resistance, glycosylated hemoglobin, and plasma glucose; on obesity by reducing central fat and body weight; and on metabolic syndrome by

decreasing waist circumference (WC), blood pressure, plasma triglycerides, and fasting glucose (165,176–182).

2.11 The Manufacture of Chocolate Milk and Yogurt

2.11.1 Processing of Chocolate Milk

Flavored milks are consisting of unfermented milk of different fat contents, mixed with sugar, cocoa powder, and/or other ingredients and additives depends on the type of flavored milk (161). Fat content (full fat: 3.5%, semi-skimmed: 1.5-1.8%, or skimmed <0.5%) of the flavored milk affects the creaminess, texture, and the color of the product (161). Also, milk fat masks the strong cocoa flavor; for example, full fat chocolate milk tastes less chocolaty than skimmed chocolate milk (161). Also, pasteurized, ultra-pasteurized, or ultra-high temperature-treated (UHT) milk are used for processing flavored milks (161). There are additional ingredients such as carrageenan (European additive number E407), pectin (E440), sodium alginate (E401), and carboxymethylcellulose (E 466) as thickeners (161). Additionally, sodium phosphate (E339) or diphosphate (E450) and sodium hydroxide (E524) may be used as stabilizing agents for the products applied for intense heat treatment (161).

First, the cocoa particles produced from defatted cocoa are mixed into milk at a ratio of 1:2 and left for 2-3 hours or is heated up to 80-90 ˚C for 30 minutes (161). Carrageenan and sugar [sucrose (5–8%)] are mixed before being added to the milk (161). Then, the cocoa particles and the premixed carrageenan with the other ingredients, are added into the main part of milk at 40˚C (161). Homogenization with 180-200 bar pressure is applied before pasteurization, ultra-pasteurization) or after the heat treatment (UHT) for full-fat and semi-skimmed flavored milks (161). The recommended pressure reduces the size of the fat globules without affecting the size of the cocoa particles (161). The heat must be above 60˚C to dissolve carrageenan molecules properly

and interact with the casein micelles (161). Lastly, UHT products are cooled to 20˚C while pasteurized and ultra-pasteurized products are cooled to 4˚C before filling aseptically into the beverage cartons or HDPE plastic bottles; the cooling process increases the strength of the gel (161). The filling temperature should not be higher than recommended temperature (20˚C and 4˚C), otherwise cocoa sedimentation may occur (161). Occasionally, guar gum is added into carrageenan as an option to prevent sedimentation of cocoa powder during the cooling process (161). Homogeneous appearance is very important for chocolate milk, avoiding strong gel and sediment (161).

2.11.2 Processing of Yogurt

The manufacturing of yogurt can fall under two categories depending on the variety of bacteria cultures used to produce the yogurt (183). Standard yogurt is made with added starter culture strains *Lactobacillus delbrueckii* subsp. *bulgaricus* and *Streptococcus thermophilus*, while probiotic (bio-yogurt) is enhanced with live probiotic microorganisms such as *Bifidobacterium sp.* and *Lactobacillus acidophilus* (183).

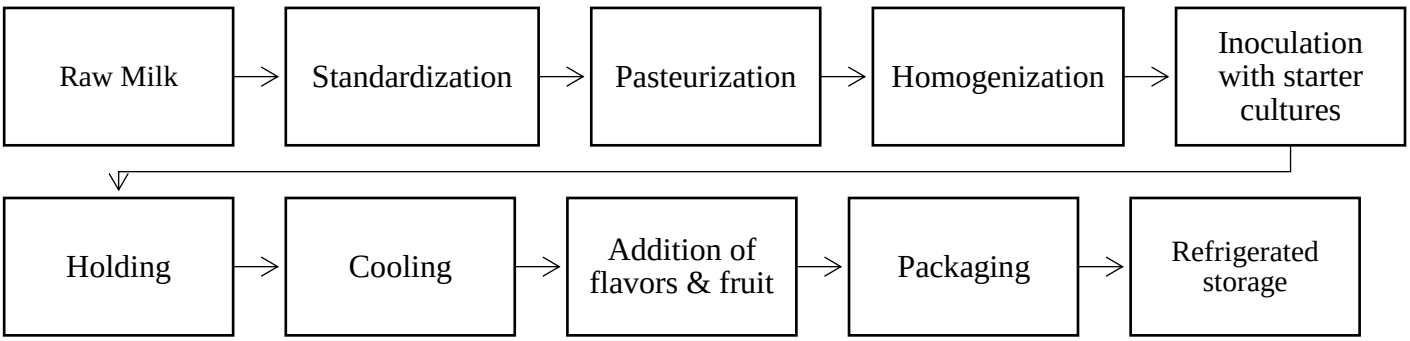


Figure 2. 2 A General Summary of the Steps Required for Making Flavored Yogurt (184)

Milk, skim milk, non-fat milk and dry milk is typically used in yogurt production by manufacturers (184). Standardization of milk composition may be important to reach required fat and solids

content (184). Ingredients such as stabilizers may be added at this stage (184). Pasteurization is the next step with heat treatment at 85°C for 30 minutes or 95–99°C for 7–10 minutes and is an important step in yogurt manufacturing (184). The intense heat treatment during yogurt pasteurization destroys all the pathogenic microbiota and most vegetative cells of all microorganisms contained in the milk (184). In addition, high heat treatment allows the whey (serum) proteins to denature, and the proteins also become to form a more stable gel, which avoids the separation of the water during storage (184). The high heat treatment provides a better environment for the starter cultures to grow, reducing the number of damaged organisms; if the yogurt is fermented before pasteurization, the cultures will be inactivated (184). Therefore, pasteurization is crucial before adding the starter cultures to ensure that the cultures stay active in the yogurt (184). Then, a high-pressure pump forces the mix through extremely small orifices with average of 17 MPa (1500 psi) at 60°C for homogenization (184). Homogenization of the mixture helps mix all ingredients entirely and improves the consistency of yogurt (184). After homogenization, starter cultures are added to the mixture for fermentation (184). The milk is held at 108°F (42°C) for several hours until a pH of 4.5 is reached (184). The holding process provides the fermentation to progress to form a soft gel and the characteristic flavor of yogurt (184). Next, the yogurt is cooled to 15–20°C in 1–1.5 hours (184). There are different steps for adding fruits and flavour depending on the type of yogurt (184). For example, if the fruit is in the bottom of the cup and then the inoculated yogurt is on top, the yogurt is fermented in the cup (161). Lastly, the yogurt is taken from the fermentation container and packaged as needed and stored in a refrigerator at ≤ 10°C to stop the fermentation process (161,184).

2.11.2.1 Fermentation/Starter Cultures

The production of yogurt should include the bacteria that are *Streptococcus thermophilus* and *Lactobacillus delbrueckii* subsp. *bulgaricus* (161). However, yogurt starter cultures may include

other microorganisms such as *Lactobacillus acidophilus*, *Lactobacillus casei*, *Lactobacillus lactis*, *Lactobacillus jugurti*, *Lactobacillus helveticus*, *Bifidobacterium longum*, *Bifidobacterium bifidus* and *Bifidobacterium infantis* (184). *Streptococcus thermophilus* subsp. *thermophilus (ST)* is the only species in the *streptococcus genus* as dairy starter cultures (184). In some countries, *Lb. delbrueckii* subsp. *bulgaricus* is a requirement as the starter culture for any dairy product named as 'yogurt' (161). However, in other countries, the starter culture might be different from normal yogurt as a product is described 'yogurt-like', 'mild-yogurt' or 'bioyogurt' (161).

The starter cultures are very important because there are numerous strains categorized as *Sc. thermophilus* and *Lb. delbrueckii* subsp. *bulgaricus* in commercial use (161). Yogurt manufacturers and culture suppliers isolated and retained *Sc. thermophilus* and *Lb. delbrueckii subsp. bulgaricus* strains because each culture has unique characteristics to provide the yogurt (161). For example, the natural yogurts containing 10 g lactic acid kg^{-1} is acidic enough for the average consumers; however, some strains of *Lb. delbrueckii* subsp. *bulgaricus* might generate levels of lactic acid >18 g kg^{-1} of yogurt (161). In addition, the metabolism and growth of the starter cultures *Sc. thermophilus* and *Lb. delbrueckii* subsp. *bulgaricus* are necessary to produce a good yogurt, so the damaged starter culture will produce a below standard product (185). To summarize, the selection of a strain of *Lb. delbrueckii* subsp. *bulgaricus* is beneficial for yogurt manufacturers to avoid the over acidification of the product during transport and storage because the selection of a strain stops to release lactic acid at 10 g kg^{-1} (161).

*2.11.2.2 **Optional Probiotics***

Lactic cultures are used to develop probiotic products that help to increase health benefits (161). Yogurt manufacturers produce probiotic yogurt products with live strains of *L. acidophilus*, and species of *Bifidobacterium* added to the conventional yogurt organisms, *S. thermophilus* and *L.*

bulgaricus (161). The required number of probiotic bacteria to produce a beneficial effect has not been determined. However, Kurmann and Rasic (1997) recommended a minimum of $>10^6$ cfu/mL probiotic organisms in a probiotic product to reach ideal therapeutic effects (186). However, other studies mention that the numbers may change according to the type of species, and strains within a species; for example, $>10^7$ and 10^8 cfu/mL are the suggested number of probiotic bacteria to produce bio-yogurt (186). One study recommended that the consumption of live probiotic cells per day should be 10^8 cfu/mL. Furthermore, the frequent consumption of 400-500 g/week of probiotic yogurt will provide $>10^6$ cfu/mL as a recommended number of probiotic bacteria (187). Also, National Yogurt Association of the United States requires at least 10^8 cfu/g of lactic acid producing bacteria to qualify "Live and Active Culture" logo on containers of products (186). In contrast, the Fermented Milks and Lactic Acid Bacteria Beverages Association of Japan has standards that require a minimum of 10^7 cfu/mL to be named as a fresh dairy product (188).

2.11.2.3 ***Pasteurization Stages***

The main purpose of pasteurization was to destroy *Mycobacterium tuberculosis*, which is one of heat-resistant pathogens that might be present in milk (161). However, today's requirements are to destroy all heat-sensitive bacteria and extend the shelf-life of raw milk (161). There are various processes with different temperatures and holding time in pasteurization stage depending on products (162).

Table 2. 3 Pasteurization Temperature and Holding Time for Various Processes (183)

Pasteurization Temperature and Holding Time for Various Processes		
Temperature		**Time**
°C	**°F**	
63	145	30 min
72	161	15 s
89	191	1 s
90	194	0.5 s
94	201	0.1 s
96	204	0.05 s
100	212	0.01 s

2.11.3 Commercially Available Chocolate Milk and Yogurt Products

The dairy beverage market is growing and there is a huge competition between different brands, so consumer preferences play an important role in this sector (161). Thus, dairy processors have created different kinds of products, including chocolate and other flavored milks which vary in sensory characteristics such as flavour, colour, viscosity, sweetness, and creaminess (193). For example, partly skimmed 2% plain milk usually contains 0.03 g fructose, 0.003 g glucose, 0.03 g sucrose, and 12.92 g lactose per serving (250 mL) (190). Compared to 2% plain milk, chocolate milk commonly contains 1.08 g fructose, 1.4 g glucose, 12.65 g sucrose, and 10.12 g lactose per serving (191). This shows that the regular formulations of chocolate milk in Canada contains between 13-14 g of added sugar for each serving of 250 mL, or 264 g (191).

Commercially prepared chocolate milk products are widely available in the Canadian market. The Brand Natrel has a variety of chocolate beverage products in Canada. For instance, fine-filtered

2% chocolate milk which has 23 g sugars and 8 g proteins per serving (250 mL), Natrel Plus chocolate 2% chocolate milk which contains 12 g sugars (sucralose and lactose-free) and 18 g proteins per serving (250 mL), organic fine-filtered 2% chocolate milk which has 25 g sugars and 9 g proteins per serving (250 mL), Natrel milk 1% chocolate milk which has 29 g sugars and 9 g proteins per serving (250 mL), Natrel Dark 1% chocolate milk which contains 31 g sugars and 9 g proteins per serving (250 mL), Natrel 1% chocolate milk which contains 21 g sugars and 9 g proteins per serving (250 mL), and Natrel Lactose-free 1% chocolate milk which contains 22 g sugars and 9 g proteins per serving (250 mL) (192).

Sealtest brand has only one chocolate milk product in Canada which is the 1% partly skimmed chocolate milk that contains 25 g sugars, and 7 g proteins per serving (250 mL) (193). Another brand, Northumberland, has two kinds of chocolate milk. For example, Northumberland 2% chocolate partly skimmed milk contains 28 g sugars and 8 g proteins per serving (250 mL), and Northumberland 1% chocolate partly skimmed milk contains 26 g sugars and 8 g proteins per serving (250 mL) (194).

Health consciousness has increased among Canadian consumers, so the share of healthier milk products increased on the Canadian market, including the products with reduced sugar, ultra-filtered (high-protein), fat-free, or with lower fat (195,196). There are a few brands that produce reduced sugar chocolate-flavoured milk in Canada (166). For instance, Milk2Go brand produces a reduced sugar chocolate milk containing only 6 grams of added sugar per serving (250 mL) (197). Similarly, Neilson's brand has a reduced sugar chocolate milk product containing 7 grams of added sugar per 250 mL and is less sweet than the regular chocolate milk (196).

Nesquik products have a variety of chocolate beverage products such as chocolate milk powders, syrups, and ready-to-drink fluid chocolate milk (198). The Nesquik ready-to-drink fluid chocolate

milk products contain approximately 9 grams of added sugar per serving (250 mL) (198). Additionally, the Nesquik chocolate milk powders contain 11 grams of added sugar per serving, the Nesquik syrups contain 11 grams of added sugar per serving, and Nesquik less-sugar syrups contain 8 grams of added sugar per 250 mL (198).

There are three brands of ultra-filtered chocolate milk in Canada. For example, Joyya ultra-filtered 2% partly skimmed milk contains 9 grams of added sugar per serving (199). Joyya chocolate milk products also contain artificial sweeteners such as sucralose (9.73 mg/50 mL) and acesulfame potassium (8.58 mg/250 mL) (199). Another brand is Lactantia, ultra-filtered 1% skim milk, contains 7 grams of added sugar per serving (250 mL), and this product does not contain sucralose and artificial sweeteners; however, contains 50% more protein than regular chocolate milk (200). Similar to previous ultra-filtered chocolate milk products, the Fairlife chocolate 2% ultra-filtered lactose-free milk contains approximately 13 grams of added sugar per serving (250 mL) and sucralose (201).

In the USA, a2 Milk company has chocolate 2% reduced-fat milk which contains only 5.5 grams of added sugars per 250 mL, and stevia (202). Similarly, TruMoo 1% low fat chocolate milk contains 7 grams of added sugar (203). McDonald's in the USA had a fat free chocolate milk containing 22 grams of total sugar (10 g added sugars), and liquid sucrose and fructose were used for this product (204).

On the other hand, there are various yogurt products in Canada. For example, the IOGO brand usually uses skimmed milk, cane sugar, water, cream, modified corn starch, pectin, active bacterial culture, and natural flavours (205). IOGO produces mostly flavoured yogurt products; for instance, blueberry Canadian Harvest yogurt includes (3.2% M.F.) 14 grams of total sugar per serving (175 g) (205). Another well-known brand is Olympic organic yogurt. 0% M.F.

Olympic organic plain yogurt contains 7 grams of total sugar per serving (175 g) and 8 grams of protein (206). In comparison, 0% M.F. Olympic organic vanilla yogurt has 19 grams of total sugar per serving (175 g) and 8 grams of protein (206).

2.12 Consumption Patterns of Chocolate Milk and Yogurt

Chocolate milk contains various nutrients such as carbohydrates, protein, electrolytes (potassium), calcium, and vitamin D, therefore, it can be used as a sports recovery drink because it may help recover harmed muscles (207). Another study showed that the consumption of chocolate milk as a post-exercise drink in a four-day heavy soccer training resulted in lower serum creatine kinase levels and provided muscle recovery responses (208). Also, one study compared the effects of consumption of chocolate milk and CHO-only beverage after exercise, and they found that chocolate milk intake in the post-exercise was effective to maintain muscle glycogen during the recovery period (209).

On the other hand, many populations worldwide do not consume enough yogurt to meet some nutrient needs, especially calcium (210). For example, only about 6% of people in the USA consume yogurt daily (210). Also, the consumption patterns of yogurt vary depending on the region in the world (210).

2.13 Food Labelling in Canada

2.13.1 Front of Packing Label for Risk Ingredients

According to Health Canada, the front-of-package label is required with a nutrition symbol to explain saturated fat, sugars, and sodium levels on foods because high consumption of these nutrients may cause some chronic diseases, such as obesity, high blood pressure, heart disease, and T2DM (211). In Canada, the front-of-package label includes a footnote for these ingredients

at the bottom of the nutrition facts table (212). The sign on the footnote shows that 5% or less is a little, and 15% or more is a lot of a nutrient (213).

2.13.2 Food Labelling for Sugar

A list of ingredients and nutrition facts table for food can help people to make healthier food choices and help health professionals to educate consumers (214). In addition, the list of ingredients on packaged foods and beverages play an important role for people with food allergies and intolerances who cannot consume certain foods or beverages (215). Food Standards Agency also mentioned some labelling requirements for certain food and drink products to tell consumers if the food or beverage includes sweeteners or sugars, aspartame or colourings, liqourice, caffeine or polyols (215). Related to this, one study showed that 95% of 340 subjects read the food labels, and even basic information was enough for them to make healthier choices (147).

According to new format labeling in Canada, the new Nutrition Facts Table includes a percent daily value and grams for total sugars instead of only amount of the sugar in grams (96). In addition, the ingredient list includes the names of sugar types by opened parentheses on the same line to provide more information to consumers (96). The nutrition facts label put all types of sugar into the same category and a “% daily value” is included for total sugars, but not for added sugars in Canada (96). However, in the United States, naturally occurring sugars and added sugars are separated from each other on the label of food packages (216). For instance, there is a separate section for "Added sugars" which shows in grams and as percent daily value on the label (216).

2.13.3 Proposed Restriction of Food Advertisements to Children

In Canada, nearly 1 in 3 children are overweight or obese, and overweight that can lead to the development of health problems such as heart diseases, T2DM, and high blood pressure (217). A study found that Canadian children see the advertisements for foods on TV every hour per station;

there are approximately 54 million food ads and 90% of them are for highly processed foods (218). Also, food advertising impacts children's eating patterns, taste preferences and purchase requests, so the Child Health Protection Act decided to prohibit or limit the advertising of foods that include high sodium, saturated fat, or sugar for children under 13 years of age (219).

2.14 Chocolate Milk as a Sugar-Sweetened Beverage

The consumption of sugar-sweetened beverages is high in many parts of the world (98). Sugar-sweetened beverages have large amounts of added sugars such as sucrose or fructose which contribute to the overall energy density of diets and the lack of nutritional value may not provide a feeling of fullness as much as a solid food (220).

Flavored milk such as chocolate milk has the same nutrients as plain milk, but flavored milk usually contains added sugar and less fat (221).Chocolate milk in Canada contains approximately 5-5.3% added sugar (13-14 grams) for each 250 mL or 264.2 grams (191). Added sweeteners in chocolate-flavored milk other than sucralose may also include fructose, glucose, high-fructose corn syrup, maltose, and dextrose (96). According to Health Canada, the permitted sweeteners in chocolate-flavored milk may include acesulfame potassium, sucralose, and stevia (222).

2.14.1 Positions of Dietitians of Canada on Taxation of Sugar-sweetened Beverages

In Canada, Dietitians of Canada issued a position paper recommending that sugar-sweetened beverages sold in Canada should include at least 10% to 20% tax, and chocolate milk was categorized in this paper as a sugar-sweetened beverage (223,224). According to one study, beverage taxes could be important for public health; for example, a 24% reduction of sugar-sweetened beverage intake might reduce daily per capita consumption of sugar from sugar-sweetened beverages from 190-200 kcal to 145-150 kcal (225). In Canada, Newfoundland and

Labrador will be the first province that will impose 20 cents tax on each litre of SSB; however, there is a strong criticism on the idea of the taxation of SSB (226).

2.14.2 Sugar Reduction in Dairy Products

Health issues related to sugar overconsumption continues to increase worldwide (227). Also, dairy foods have a large market; however healthier products are leading to reduce sugar content in dairy products (227). Many consumers prefer the taste of artificial non-nutritive sweeteners even though natural ones are popular for label appeal (227). Texture of the food matrix and the presence of fat also affects the sweet taste perception (227). There are some sugar reduction techniques in dairy products which include hydrolysis of lactose, ultrafiltration, and direct reduction (227).

Replacing sugar with natural or artificial non-nutritive sweeteners is the most successful sugar reduction in dairy foods (228). Non-nutritive sweeteners are sweeter than regular sugar; however, might contribute some sensory characteristics such as mouthfeel and bitter taste (228).

Lactose hydrolysis also has been investigated as a sugar reduction method because hydrolysis of 70% of the lactose in milk increases the sweetness to the same degree as adding 2% sugar into the dairy products (229). The first method of lactose hydrolysis is adding β-galactosidase to pasteurized milk and holding the mixture at 35 to 45°C for a set amount of time (230). The lactase enzyme will be deactivated due to an additional heat process (230). The second method can be adding a sterile lactase to UHT milk before packaging (231). This method will help to break down lactose during the first few days of packaging (231).

Ultrafiltration is another well-known method for sugar reduction in dairy products (232). This method has a pressure-driven process to separate compounds in milk by molecular weight (232). Lower molecular weight compounds (lactose, water, minerals, and vitamins) pass through the membrane, while heavier molecular weight compounds (protein, fat) retain by ultrafiltration (232).

In addition, the ultrafiltration method is used for lactose removal, especially in reduced sugar milk, yogurt, and cheese production (233).

The last method is direct sugar reduction which is explained as a way for gradual reduction of sugar consumption (234). In this method, manufacturers reduce the sugar content in dairy products slowly and progressively, so consumers will gradually get used to the lower sugar concentrations without noticing a difference (234).

While dairy products such as chocolate milk and yogurt are nutrient-dense food that provide multiple important nutrients for human health, their consumption is impacted by a negative perception towards added sugar. While the complete reduction of added sugar results in products with a strong bitter (chocolate milk) or sour (yogurt) tastes, the partial reduction of added sugar can reduce the energy density of such products and make them more attractive for health-conscious consumers. The present work is aimed to investigate whether the partial reduction of added sugar in chocolate milk and yogurt provides any short-term physiological benefits.

3 Rationale, Objectives, and Hypothesis

3.1. Rationale

The consumption of fluid milk in Canada has decreased by approximately 22%; however, per capita intake of chocolate milk and yogurt products have increased by 58% and 179%, respectively, over the last two decades (235). Commercially available chocolate milk and yogurt products provide nutritional benefits through dairy proteins and calcium to consumers; however, these products also contain added sugars such as sucrose, fruit preparations, and honey (156,157,183). Flavored milk such as chocolate milk has the same nutrients as plain milk, but flavored milk usually contains added sugar and are perceived equal to other sugar-sweetened products such as juice and carbonated beverages due to their association with obesity (236). Some schools removed chocolate milk and sweetened dairy beverages from their menu due to their excessive sugar content (237). Lower-sugar formulations of sweetened dairy product may provide benefits of milk consumption (237). Previous studies have shown that the partial reduction of sugar in chocolate milk is not associated with any inferior sensory characteristics such as taste and pleasantness compared to their full-sugar counterparts (238). However, it is not known whether the reduction of added sugar in chocolate milk and yogurt results in benefits for blood glucose control. Therefore, this project will help with the better understanding of sweetened flavoured milk and yogurt through the formulation and evaluation of products with reduced sugar content and their examination on blood glucose control in human adults.

3.2 Objectives

The primary objective is to investigate the effect of partial reduction of added sugar in chocolate milk and yogurt on postprandial blood glucose response over two hours.

The secondary objective is to investigate how the ingestion of chocolate milk and yogurt with reduced added sugar content affect subjective appetite and physical comfort over two hours, and *ad libitum* food intake at 120 min.

3.3 Hypothesis

The treatments with chocolate milk and yogurt formulated with a reduced amount of added sugar (3.3% by weight) will reduce blood glucose response over two hours compared to the treatments with chocolate milk and yogurt with a full added sugar content (5.5% by weight).

This study also aimed to reveal any difference in glycaemic response between one serving of each product with full or reduced added sugar content.

4 Methodology

4.1 Ethical Statement

Ethical approval (file # 2019-202) was obtained from the Research Ethics Board of Mount Saint Vincent University.

4.2 Study Design

This study is a randomized single-blinded crossover consisting of 10 male and 10 female adult participants. Twenty normoglycemic healthy male and female adults with the age between 19 years and 35 years with the Body Mass Index (BMI) between 20kg/m^2 and 24.9kg/m^2 were recruited for the study. The exclusion criteria were having any metabolic disease, irregular menses in females, smoking cannabis or tobacco, taking medications that influence blood glucose, skipping breakfast, having emotional or learning problems, having known food allergies or lactose intolerance.

4.3 Study Participant Recruitment & Selection

The participants for this study were recruited by posting posters (Appendix 1a) around the University, keeping flyers at local libraries, advertising in social media platforms such as Facebook and Instagram, and by the word of mouth. The screening was done by using a telephone screening questionnaire (Appendix 2&2a) for interested participants. A brief information about the study also was given during the screening session. Potential participants were then scheduled for in-person screening and an information session. In the information session, first, information sheet and consent form (Appendix 4) were given to participants, and then their height, weight and body composition were measured via bioelectrical impedance analysis using a Tanita Body Composition Analyzer TBF-300A and Stadiometer HR-200 (Tanita Corporation of America, Inc, Arlington Heights, IL). Eligible male participants were scheduled for the experimental sessions one week apart. In contrast, eligible female participants were scheduled at the follicular phase of their menstrual cycle to avoid a potential effect of the menstrual phase on insulin sensitivity and food intake. An information email was sent to

the participants a day before the session. This email reminded participants that they should follow an usual diet and activity routine on the day before the study sessionand come to the study after an overnight fasting (10-12 hours) on the following day to the Appetite lab between 8 am and 10 am.

4.4 Study Protocol

Upon arrival, participants completed the physical activity questionnaires and restraint scale and three factor eating questionnaires only on the first session. Following this, participants completed health and activity questionnaires about their activity and intake from the previous day along with their perceived level of stress. Compliance with the fasting was assessed by taking a baseline blood glucose measurement from a discard tube and was measured with a handheld glucose meter (HemoCue® Glucose 201). Participants with baseline blood glucose under 4mmol/L or over 6mmol/L were rescheduled to another day. When the fasting blood glucose was in a normal range, participants completed the baseline level of subjective appetite and their physical comfort using 100 mm motivation to eat (MTE) visual analogue scale (VAS) questionnaire (114). Further to this, a registered nurse inserted an intravenous catheter into their arm for the blood collection. Blood collection was done by using a system manufactured by Becton, Dickinson and Company (Mississauga, ON) that included a shielded intravenous catheter (BD Insyte Autoguard™) inserted into the antecubital vein and connected with a Luer-Access split septum device (BD Q-Syte™) and Luer-Lok™ access device (BD Vacutainer®). The blood was collected into BD Vacutainer® Plus serum separation tubes.

An initial baseline sample was taken right after IV insertion at 0 min. Following this, one of the five treatments was given to the participant who had 10 minutes to consume the treatment. While participants were blinded to the treatment they were receiving, there was a noticeable difference in the visual appearance and taste of the treatments, such as yogurt treatments compared to

chocolate milk treatments. Similarly, the caloric free control, with only water, was obviously different than the other treatments. The palatability of the treatments was measured immediately after consumption with 100mm VAS (239). When the participant had the first sip or bite of the treatment, a timer was started to keep track of the time points. VAS and a 9-point hedonic scale were given to participants to assess the treatment palatability. The blood samples (5ml) were collected at 15, 30, 45, 60, 90 and 120 minutes for a total of 35 ml. VAS scales evaluating physical comfort, subjective appetite, and gastrointestinal wellness were completed by the participants at 15, 30, 45, 60, 90, and 120 minutes.

After the last blood collection (120 min), participants were given an *ad libitum* pizza meal with a glass of water to measure the food intake and water intake and instructed to eat until comfortably full. Food intake was measured using a weight difference, with the weight of pizza consumed used to determine caloric intake based on information provided on the nutrient facts table from the manufacturer (240). Water intake was also measured using a weight difference of the cup plus water before serving to the participant, minus the final weight of cup plus remaining water. Food and water intake values were marked in the food intake sheet. Participants were rescheduled for the next session after a washout period of at least one week.

4.5 Treatments

Treatments were prepared in the kitchen at the Shelia A. Brown Centre of Applied Research, Mount Saint Vincent University using fresh ingredients.

Five treatments for the study, chocolate milk with 3.3% added sugar (33 g of sucrose per 1L), chocolate milk with 5.5% added sugar (55 g of sucrose per 1L), yogurt with 3.3% added sugar (33 g of sucrose per 1L), yogurt with 5.5% added sugar (55 g of sucrose per 1L), and water were prepared before each session. The energy-free water control is used according to the according to

the recommendations set in *Health Canada Draft Guidance Document on Satiety Health Claims on Food* (114).

The list of ingredients is shown in Table 4.1.

Table 4.1 List of ingredients

Ingredients	**Universal product code (UPC)**
Skim milk	06799703300
Dry sugar	05889125222
Cream, 35% m.f.	06799775600
Cocoa base	(From Agropur)
Organic yogurt	05916170215
Water	N/A

Table 4. 2 Treatment formulations of chocolate milk

Ingredients	**3.3% added sugar chocolate milk**	**5.5% added sugar chocolate milk**
Milk, skim, ml	907	885
Dry added sugar, g	33	55
Cocoa base, g	26.5	26.5
Cream, 35%, m.f.	6.6	6.6
Water, ml	26.9	26.9

Table 4. 3 Treatment formulations of yogurt

Ingredients	3.3% added sugar yogurt	5.5% added sugar yogurt
Yogurt, organic, g	945	945
Dry added sugar, g	33	55
Water, ml	22	0

4.5.1 Preparing Directions

Each treatment was prepared for each participant by using fresh ingredients. The SOP for the preparation of chocolate milk was provided by Agropur. First, all dry ingredients (sugar and cocoa base) were measured individually and added into a bowl followed by mixing thoroughly using a spoon. The liquid ingredients (cream and water) were then measured individually and added into a separate bowl. The main ingredient (milk) was measured and added to a pot. Cream and water were then added to the pot and mixed with cold milk andheated until 65°C. When the temperature reached 65°C, the pot was taken to the cold area on the stove followed by addition of dry ingredients into the pot. All the ingredients in the pot were mixed with a whisk at 65°C for 20 min. A food thermometer was used to check the temperature and helped ensure that the chocolate milk stayed at 65°C. After 20 min, the pot was taken from the top of the stove and was allowed to cool down to 50°C. Then, the prepared chocolate milk was transferred into a pitcher and covered with a lid. Another big bowl was filled up with ice, and the pitcher was placed inside the iced bowl to speed up the cooling process.. The food thermometer stayed inside the pitcher until the chocolate milk cooled down to room temperature (24°C). When the chocolate milk reached room temperature, the food thermometer was taken out of the pitcher, and the pitcher was placed in the refrigerator at 4°C. When the participant

arrived at the lab the next day, 250 mL of chocolate milk was measured with a measuring cup and was served to the participant with a clean tissue.

The second treatment preparation was for yogurt. Yogurt treatment was prepared the same day before participants arrived at the lab. All the ingredients (sugar, water, yogurt) were measured individually and added to different bowls. First, the measured yogurt was added into a bowl. Next, water was added to the same bowl followed by the addition of sugar. All the ingredients were mixed for 3 min with a whisk until the sugar dissolved properly. Then, 175 g of sweetened yogurt mixture was measured and added into a smaller bowl and covered with a plastic food wrap. The treatment was served to participants with a spoon and a clean tissue. The nutrition composition of the treatments is shown in Table 4.4 and Table 4.5.

Table 4. 4 Nutrition Composition of The Chocolate Milk Treatments

Serving size: 250 mL	**3.3% added sugar chocolate milk**	**5.5% added sugar chocolate milk**
Protein, g	9	9
Total Fat, g	3	3
Total Carbohydrates, g	23	30
Added sugar, g	9	16
Energy, kcal	149	177

Table 4. 5 Nutrition composition of the yogurt treatments

Serving size: 175 g	3.3% added sugar yogurt	5.5% added sugar yogurt
Protein, g	7	7
Total Fat, g	3.5	3.5
Total Carbohydrate, g	12	16
Added sugar, g	6	10
Energy, kcal	123	139

4.6 Data Collection

4.6.1 Blood Glucose

Blood was collected from participants at 0, 15, 30, 60, 90, 120 min at each session. The registered nurse collected the blood into serum separation tubes (SST),and was immediately inverted 5 times, and left at room temperature for 30 minutes. Then, SST was centrifuged at 4°C at 1,300 *g* (RCF) for 10 minutes. The serum was immediately aliquoted into 0.5 mL microtubes and stored at -80˚C for glucose analysis. Blood serum was analysed for glucose using YSI 2900 Biochemistry Analyzer (YSI Inc., Yellow Springs, OH, USA).

4.6.2 Subjective Feelings of Appetite

Subjective appetite was measured using Visual Analogue Scale (VAS) questionnaires administered to participants at 0, 15, 30, 45, 60, 90, and 120 min using Compusense software (Compusense Inc., Guelph, ON). The average score for subjective appetite was calculated using the formula (241,242):

Appetite score = [desire to eat + hunger + (100 –fullness) + prospective consumption]/4.

The average score for average appetite satiety quotient calculated using the formula (243):

Satiety Quotient (mmol/kcal) = [(fasting appetite sensation- mean 60 min post meal AS)/ energy content of the test meal (kcal)]x100.

Further to this, several objective outcomes, including desire to eat, hunger, feelings of fullness, and subjective food intake speculation were measured using the following 4 questions (114): (1) how strong is your desire to eat? (2) how hungry do you feel? (3) How full do you feel? (4) How much food do you think you could eat?

4.6.3 Physical Comfort

The VAS for physical comfort was collected at the same time points at 0, 15, 30, 45, 60. 90-, and 120-min (244). VAS measurements on subjective comfort, thirst and gastrointestinal wellness were collected using the following 8 questions (242): (1) How thirsty do you feel? (2) How energetic do you feel right now? (3) How tired do you feel right now? (4) Do you feel nauseous? (5) Does your stomach hurt? (6) How well do you feel? (7) Do you feel like you have gas? (8) Do you feel like you have diarrhea?

4.6.4 Treatment Palatability

The palatability of the treatments was measured immediately after the treatment ingestion with 100mm VAS and 9-point hedonic scales (114,245). The questions were related to the pleasantness, sweetness, taste, flavour, creaminess, bitterness, aftertaste, and mouthfeel perception of the treatments (242).

4.6.5 Food intake

After the blood collection at 120 min, participants were served an ad libitum pizza meal and a glass of water. Participants were instructed to eat until they feel comfortably full. The food and water intake were calculated by the difference between the weight of the meal or water before and after serving.

4.7 Statistical Analysis

All statistical analyses were conducted using SAS version 9.4 (Statistical Analysis Systems, SAS Institute, Cary, NC, USA) and GraphPad Prism 9 (GraphPad Software, San Diego, CA, USA). Results were reported as the mean ± standard deviation, and the P values of ≤ 0.05 were considered statistically significant.

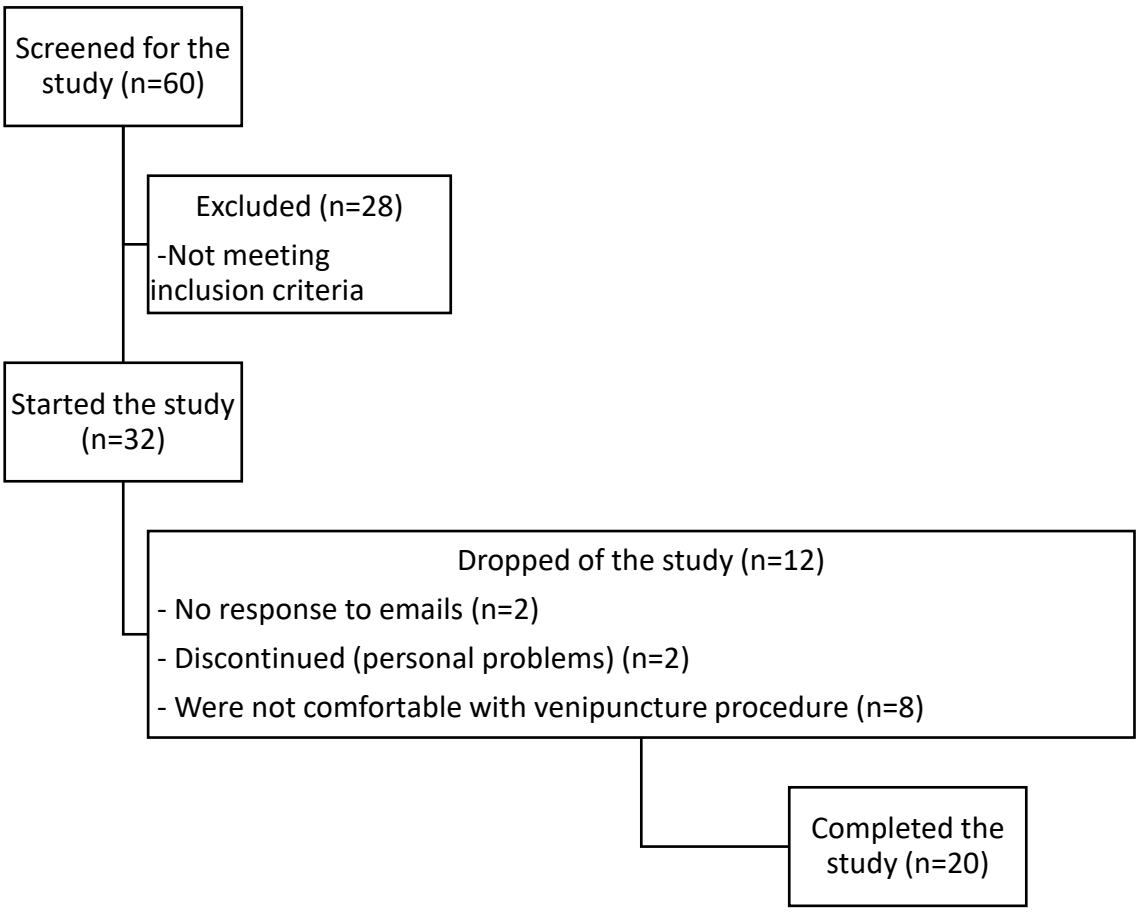
The analysis of serum concentrations of glucose was run in triplicate and the average value for each time point was calculated. The results for blood glucose were calculated as the mean value over 120 min and as the total (tAUC), incremental (iAUC) and net incremental (niAUC) areas under the curve (AUC) (246). Repeated measures (RM) Analysis of Variance (ANOVA) was used to calculate the effect of time, treatment, and their interaction. One-way RM ANOVA with Tukey's-Kramer post-hoc test was used to find the effect of a treatment on blood glucose AUCs, food intake, and treatment and pizza palatability. Two-way RM ANOVA was used to calculate the effect of time and treatment, and their interaction on blood glucose and parameters of subjective appetite and physical comfort over two hours. Pairwise comparisons were assessed with Tukey's-Kramer post-hoc test. The graphs were created by using GraphPad Prism 9.

5 Results

5.1 Participant Characteristics

A total of sixty potential participants were pre-screened into the study; however, twenty (ten females and ten males) normoglycemic healthy, non-smokers adults with the age between 19 years and 35 years and with the Body Mass Index (BMI) between 20kg/m^2 and 24.9kg/m^2 completed all five treatment sessions. The recruitment is explained in Figure 5.1.

Figure 5. 1 Recruitment CONSORT Diagram



The characteristics of the twenty participants are summarized in Table 5.1. The values for the last 24-hour food intake, last 24-hour physical activity, stress, and baseline blood glucose are shown below.

Table 5. 1 The Characteristics of Study Participants

Characteristics	Mean ± SD		
	Female	Male	*P value*
Age (y)	27.2±4.98	25.2±4.0	0.3
Weight (kg)	55.04±6.64[a]	69.3±5.4[b]	0.002
Height (m)	1.6±0.0[a]	1.7±0.1[b]	0.01
BMI (kg/m^2)	20.39±1.9[a]	23.2±1.3[b]	0.002
Fat Mass (kg)	13.8±5.2	11.8±2.5	0.3
Fat-free Mass (kg)	41.3±2.3[a]	57.6±4.1[b]	0.0
Restraint Scale	10.8±3.8	12.0±5.0	0.5
TFEQ Factor I (Behavioural Restraint)	6.8±4.1	6±3.2	0.7
TFEQ Factor II (Behaviour & weight)	10.7±3.5	10.3±2.2	0.8
TFEQ Factor III (Hunger & behaviour ramifications)	23.3±9.2	23.4±7.2	1.0
Total TFEQ	40.8±10.3	39.7±6.7	0.8
Last 24 hours physical activity (mm)	46.1±18.2	46.5±12.5	0.8
Last 24 hours food intake (mm)	48.4±12.4	48.2±11.2	1.0
Stress (mm)	49.1±22[a]	34.4±20.2[b]	0.02
Baseline Blood Glucose (mmol/L)	4.4 ± 0.4	4.5 ± 0.4	0.2

Means ± SD, Abbreviations: TFEQ= Three Factor Eating Questionnaires, n=20. The values for the last 24-hour food intake, physical activity, and stress are measured using 100 mm VAS and calculated with cumulative averages and standard deviations from each treatment session. Baseline blood glucose (mmol/L) reflected normal range of 10-12 hours fasting blood glucose levels (<6 mmol/L). Scoring of Three Factor Eating Questionnaires (TFEQ) are divided by three factors according to Stunkard, A. J. & Messick, S. (1985) scoring guidelines. Unpaired t- test followed for female and male comparison. The values with different letters are significantly different (P<0.05).

5.2 Blood Glucose

There was an effect of time (P<0.0001) and a treatment by time interaction (P<0.0001); however, there was no effect of treatment (P=0.2) on blood glucose (BG) over 120 min. Y3.3% resulted in

a lower blood glucose compared to the treatment with C3.3% at 30 min (P<0.05) (Table 5.2). Water control showed lower BG response compared to the treatments with C3.3%, C5.5% and Y5.5% on at 30 min (P<0.05). There was no difference between the treatment with Y3.3% and water control on BG at 30 min (P=1.0) (Table 5.2).

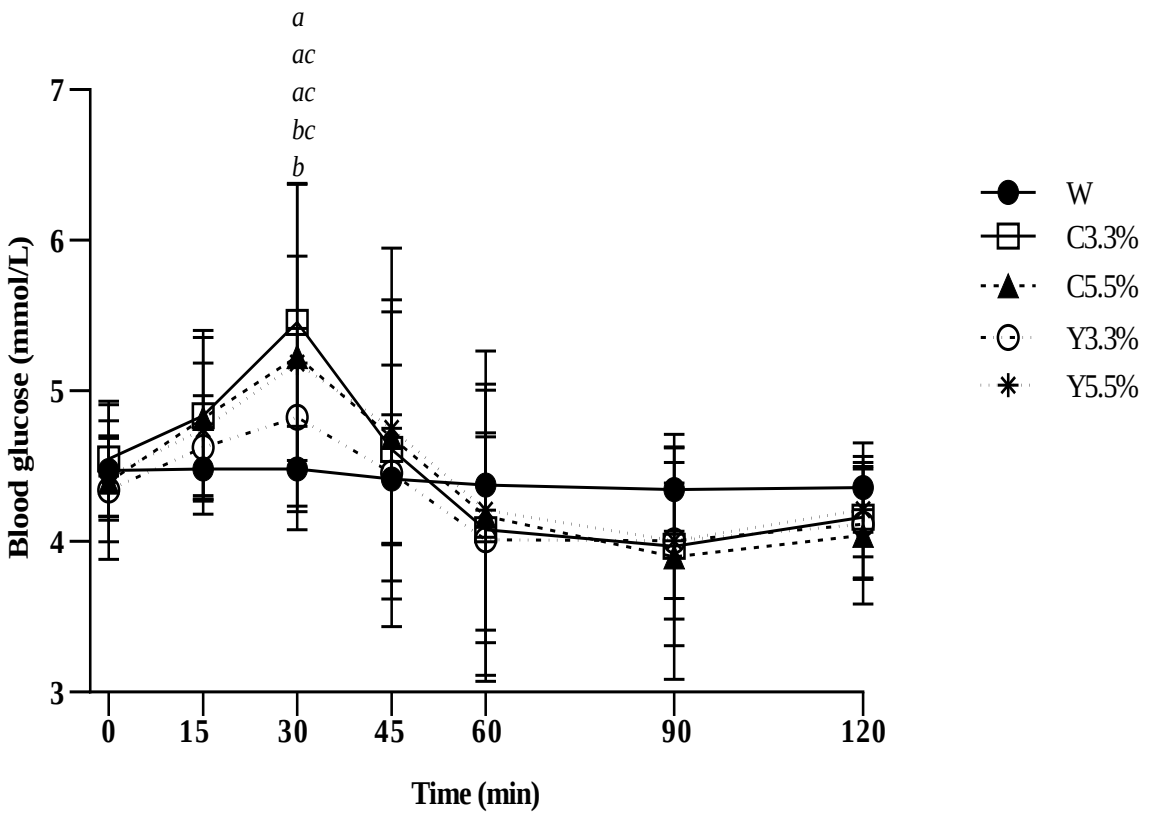
Table 5. 2 Mean Blood Glucose by Treatment over 120 min

Treatment	Mean Blood Glucose mmol/l ± SD						
	0 min	15 min	30 min	45 min	60 min	90 min	120 min
W	4.5±0.3	4.5±0.3	4.5±0.3[a]	4.4±0.4	4.4±0.3	4.3±0.3	4.4±0.3
C3.3%	4.5±0.4	4.8±0.6	5.5±0.9[b]	4.6±1.0	4.1±1.0	4.0±0.7	4.2±0.4
C5.5%	4.4±0.5	4.8±0.5	5.2±1.1[bc]	4.7±1.3	4.2±1.1	3.9±0.8	4.0±0.5
Y3.3%	4.3±0.3	4.6±0.3	4.8±0.6[ac]	4.5±0.7	4.0±0.7	4.0±0.4	4.1±0.4
Y5.5%	4.4±0.3	4.7±0.4	5.2±0.7[bc]	4.8±0.8	4.2±0.8	4.0±0.5	4.2±0.3

Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA followed by Tukey–Kramer post hoc test for mean blood glucose over 120 min. Effect of time (P<0.0001), treatment (P=0.2), and a time by treatment interaction (P<0.0001). Different lowercase letters represent significant differences between treatments (P<0.05).

No statistical differences were observed on mean blood glucose at 0, 15, 45, 60, 90, and 120 min. At 30 min, the mean blood glucose for the treatment with C3.3% was higher than the treatment with Y3.3% and water control (P<0.05). There was no difference between treatments with C3.3%, C5.5%, and Y5.5% (P>0.05). Y3.3% showed similar results compared to water (P>0.05).

Figure 5. 2 Mean Blood Glucose by Treatment over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA (treatment and time) with Tukey's-Kramer post-hoc test. Effect of time (P<0.0001), treatment (P=0.2), and a time by treatment interaction (P<0.0001). Values with different letters are significantly different (P<0.05).

5.2.1 Blood Glucose tAUC, iAUC and niAUC over 120 min

There was no effect of treatment on blood glucose $tAUC_{0\text{-}120min}$ (P=0.4) and $niAUC_{0\text{-}120min}$ (P=0.2); however, there was an effect of a treatment on blood glucose $iAUC_{0\text{-}120min}$ (P=0.003) (Table 5.3).

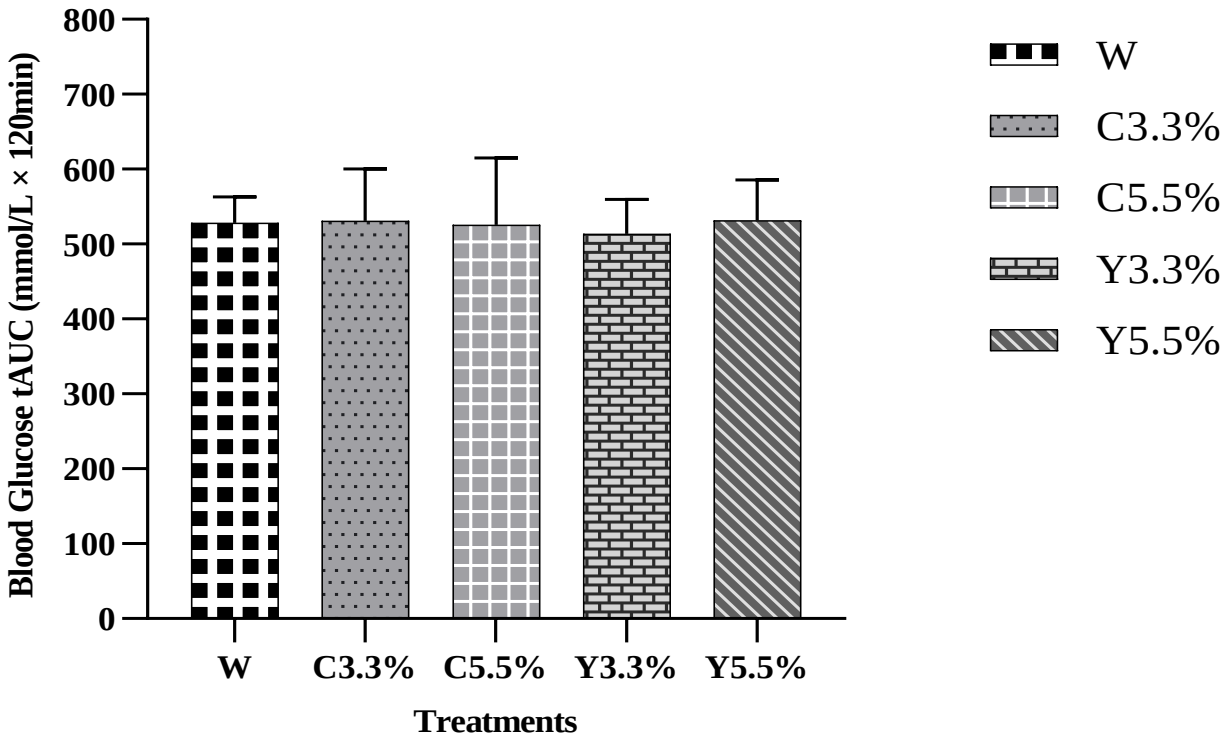
Table 5. 3 Mean Blood Glucose tAUC, iAUC and niAUC by Treatment over 120 min

	Mean ± SD					
	W	C3.3%	C5.5%	Y3.3%	Y5.5%	*P value*
Blood glucose tAUC(μIU×min)/L	528.4±34.7	530.8±69.3	525.5±89.3	513.4±46.2	531.4±54.0	P(trt)= 0.4
Blood glucose iAUC(μIU × min)/L	7.7±17.0[a]	28.5±24.8[b]	36.8±33.6[b]	20.2±14.9[ab]	31.5±36.5[b]	P(trt)= 0.003
Blood glucose niAUC(μIU × min)/L	-8.5±28.1	-15.0±54.7	-2.0±60.7	-7.4±36.6	-0.5±59.1	P(trt)= 0.2

Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, tAUC=total area under the curve, iAUC=incremental area under the curve, niAUC=net incremental area under the curve, n = 20. One-way ANOVA followed by Tukey–Kramer post hoc test for mean blood glucose tAUC, iAUC and niAUC over 120 min. Effect of treatment on blood glucose tAUC (P=0.4), iAUC (P=0.003) and niAUC (P=0.2). The values with different letters are significantly different (P<0.05).

No differences were observed between all treatments on blood glucose $tAUC_{0\text{-}120min}$ ($P>0.05$) (Figure 5.3).

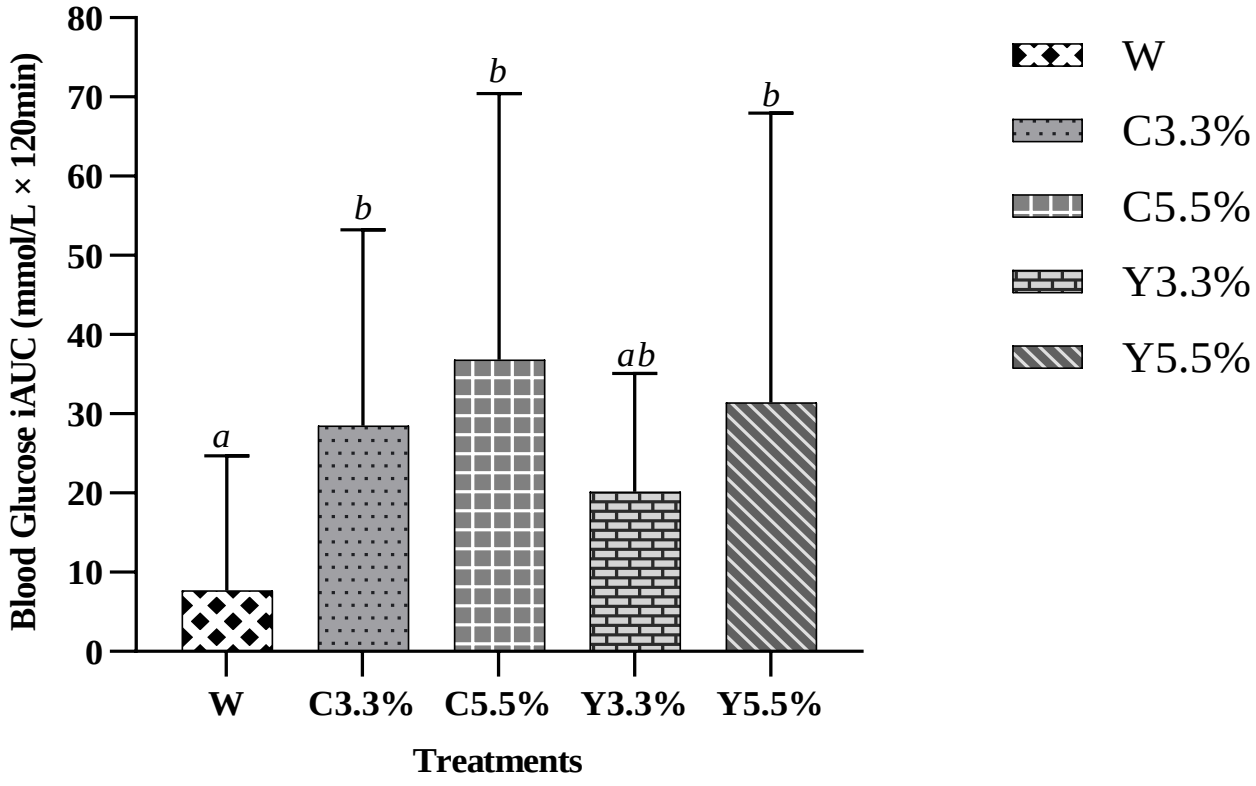
Figure 5. 3 Blood Glucose tAUC over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test.

Blood glucose $iAUC_{0\text{-}120min}$ was lower for water control compared to treatments with C3.3%, C5.5% and Y5.5% ($P<0.05$) (Figure 5.4). There was no significant difference between the treatment with Y3.3% and water control ($P>0.05$) (Figure 5.4). There was no significant difference between the treatments with C3.3%, C5.5%, Y3.3% and Y5.5% on blood glucose $iAUC_{0\text{-}120min}$ ($P>0.05$) (Figure 5.4).

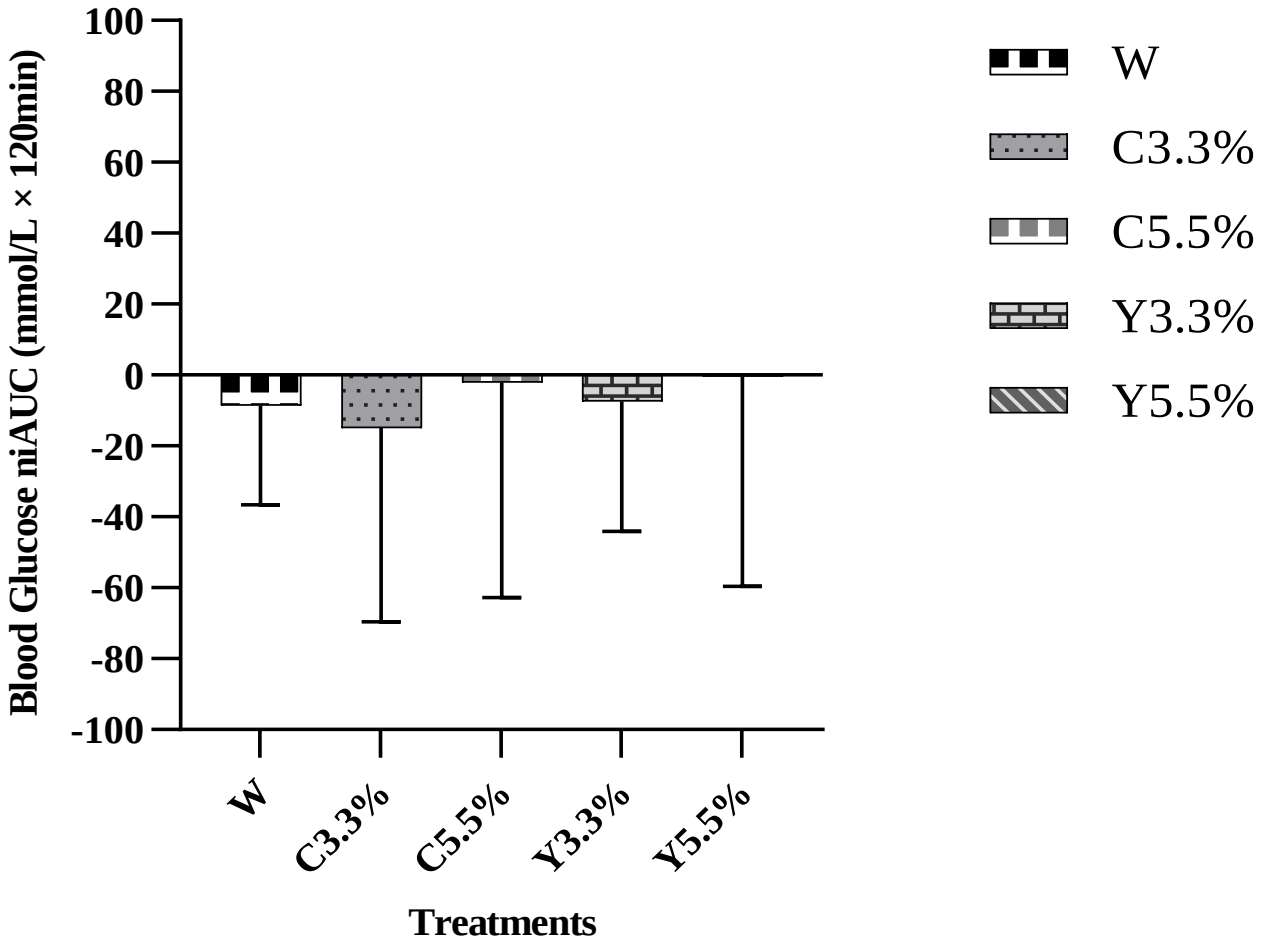
Figure 5. 4 Blood Glucose iAUC over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. The values with different letters are significantly different (P<0.05).

There was no significant difference between all the treatments on blood glucose $niAUC_{0\text{-}120min}$ (P>0.05) (Figure 5.5).

Figure 5. 5 Blood Glucose niAUC over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test.

5.3 Food and Water Intake

There was an effect of a treatment (P=0.02), but there was no session (P=0.7), and session by treatment interaction (P=0.7) on *ad libitum* FI at 120 min (Table 5.4).

There was no effect of a treatment (P=0.1), session (P=0.6), and a session by treatment interaction (P=0.7) on cumulative FI over 120 min (Table 5.4).

There was an effect of a treatment on water intake with pizza meal at 120 min (P=0.001). There was no effect of session (P=0.8), and a session by treatment interaction (P=0.6) on water intake with pizza meal at 120 min (Table 5.4).

There was no effect of a treatment (P=0.2), session (P=0.5), and a session by treatment interaction (P=0.9) on caloric compensation (%) (Table 5.4).

No differences were observed between all treatments (P>0.05) on pizza pleasantness (VAS). There was no effect of a treatment (P=0.6), and session (P=0.2); however, there was an effect a session by treatment interaction (P=0.047) on pizza pleasantness (VAS).

There was no difference between all treatments (P>0.05) on pizza pleasantness (Hedonic). There was no effect of a treatment (P=0.5), session (P=0.6), and a session by treatment interaction (P=0.2) on pizza pleasantness (Hedonic).

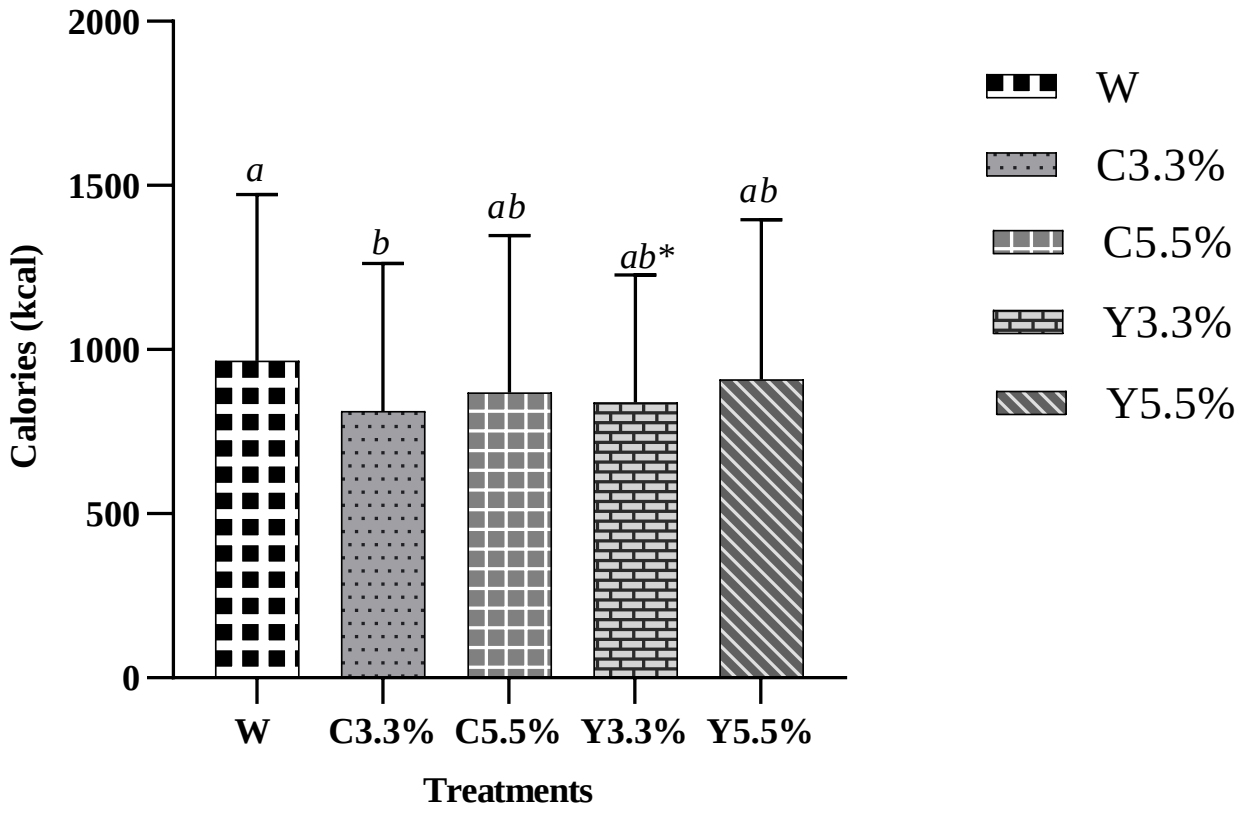
Table 5. 4 Mean Food Intake, Water Intake and Caloric Compensation by Treatment

	Mean ± SD					
	W	C3.3%	C5.5%	Y3.3%	Y5.5%	*P value*
Ad libitum food intake at 120 min (kcal)	966.1±506.4[a]	812.6±449.5[b]	869.8±477.0[ab]	839.8±387.4 [ab*]	909.7±486.8[ab]	P(trt)= 0.02 P(session)=0.7 P(trt x session)=0.7
Cumulative food intake over 120 min (kcal)	966.1±506.4	961.6±449.5	1040.8±477.0	962.8±387.4	1048.7±486.8	P(trt)= 0.1 P(session)=0.6 P(trt x session)=0.7
Water intake with pizza meal at 120 min (g)	171.1±96.2[a]	184.7±119.2[a]	321.6±111.3[bc]	257.6±100.7[ac]	287.4±143.5[bc]	P(trt)= 0.001 P(session)=0.8 P(trt x session)=0.6
Caloric compensation (%)	_	103.0±128.8	56.3±100.2	102.7±170.2	40.6±160.8	P(trt)= 0.2 P(session)=0.5 P(trt x session)=0.9

*Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA followed by Tukey–Kramer post hoc test for ad libitum food intake, cumulative food intake, water intake at 120 min and caloric compensation. P indicates P values for the effect of a treatment (P trt), session (P session), and a treatment by session interaction (P trt × session). Different lowercase letters represent significant differences between treatments (P<0.05). *P=0.05 compared to W*

The treatment with C3.3% reduced *ad libitum* FI at 120 min compared to water control (P=0.02) (Figure 5.6). The treatment with Y3.3% tended to reduce *ad libitum* FI compared to water control (P=0.05). No statistical differences were observed between treatments with C3.3%, C5.5%, Y3.3%, and Y5.5% on food intake at 120 min (P>0.05). There was no difference between treatments with C5.5%, Y5.5% compared to water control (P>0.05) on FI at 120 min.

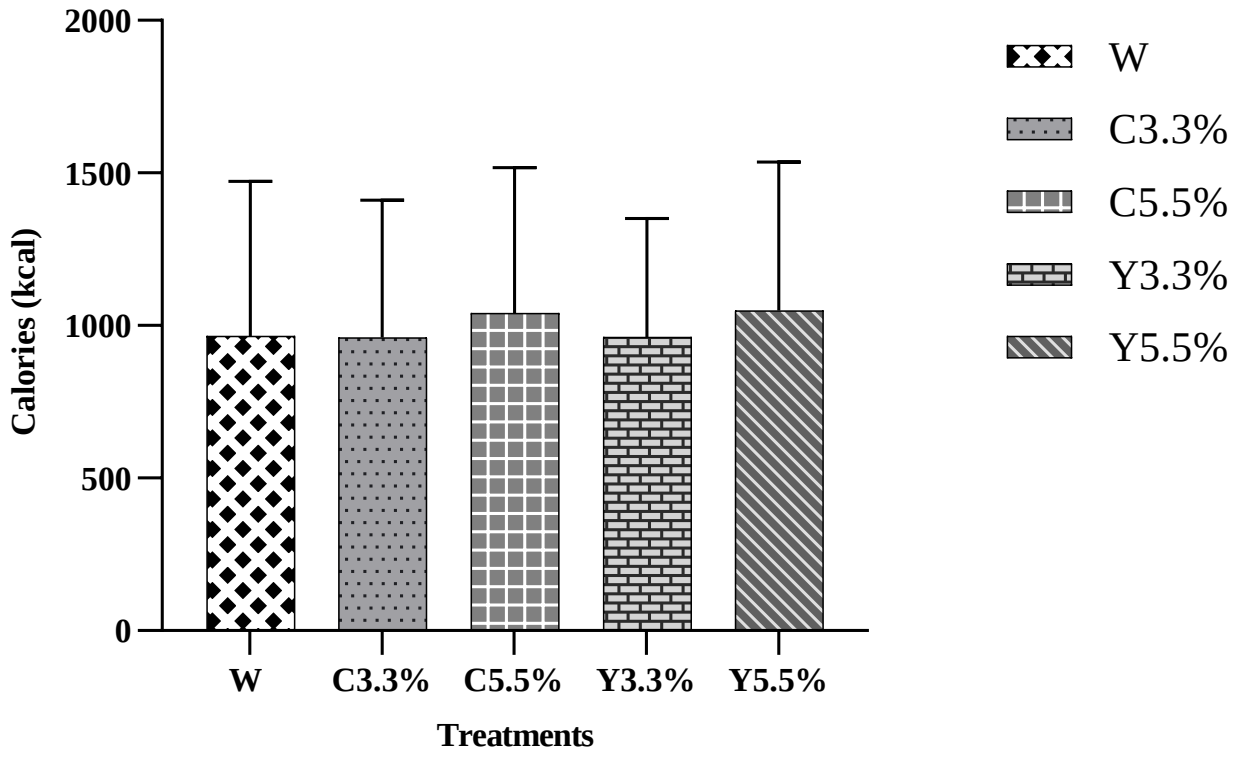
Figure 5. 6 *Ad Libitum* Food Intake at 120 min



*Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05). *P=0.05*

No differences were observed between all treatments on cumulative FI over 120 min (P>0.05) (Figure 5.7).

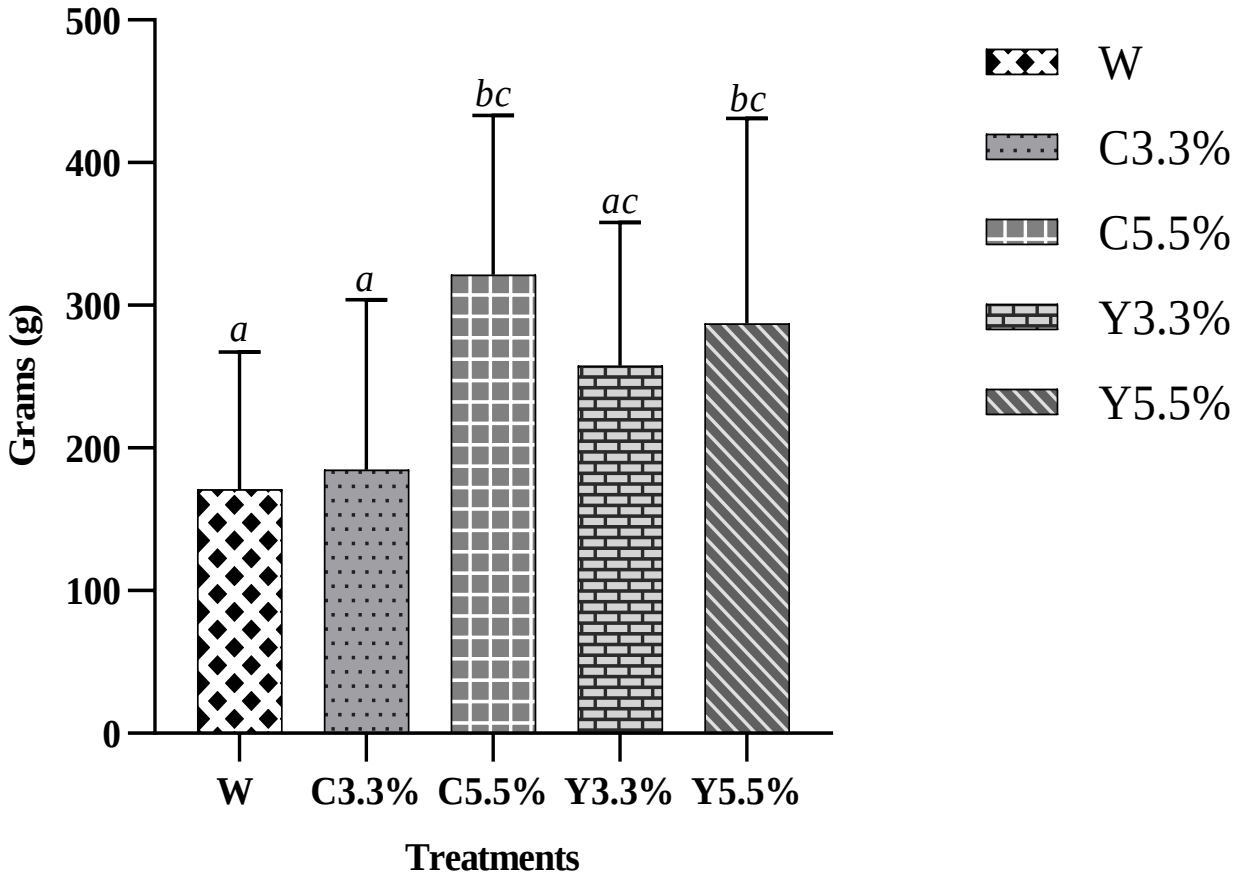
Figure 5. 7 Cumulative Food Intake over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test.

No differences were observed between the treatments with C3.3%, Y3.3% compared to water control (P>0.05) on water intake with pizza meal at 120 min (Figure 5.8). The treatments with C5.5% and Y5.5% resulted in a higher water intake compared to water control and C3.3% (P<0.05). No differences were observed between the treatments with C5.5%, Y3.3%, and Y5.5% on water intake with pizza meal at 120 min (P>0.05).

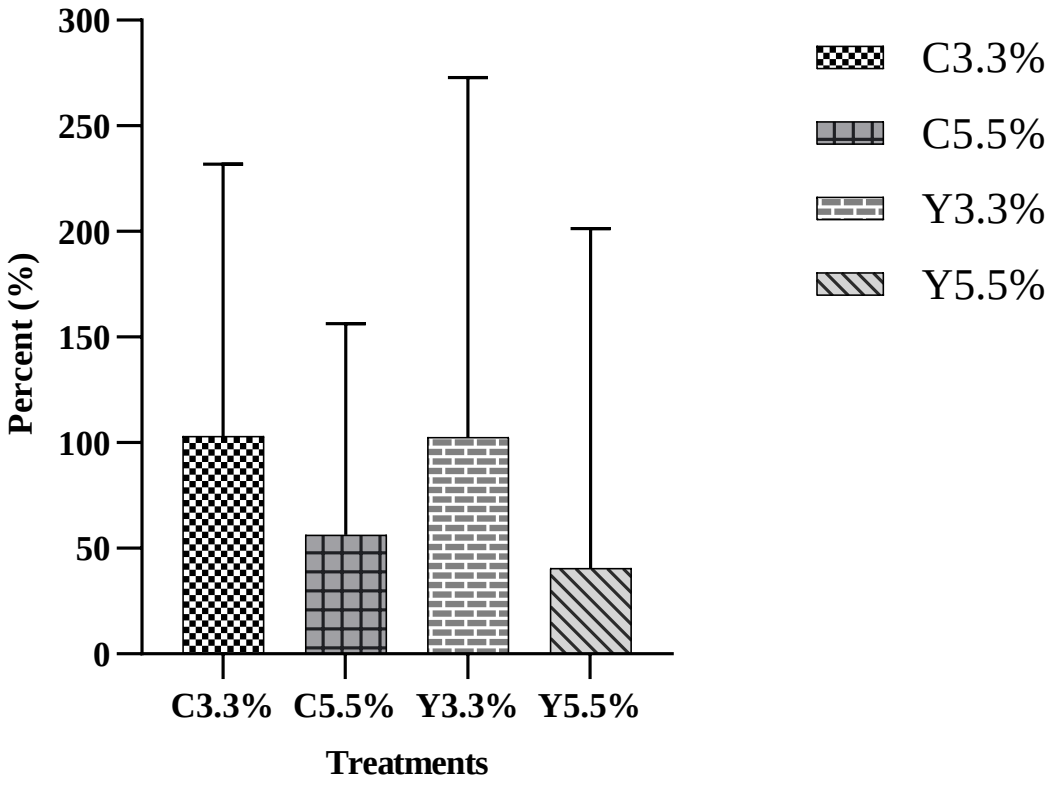
Figure 5. 8 Water Intake with Pizza Meal at 120 Min (g)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. The values with different letters are significantly different (P<0.05).

No differences were observed between treatments with C3.3%, C5.5%, Y3.3% and Y5.5% on caloric compensation (%) (P>0.05) (Figure 5.9).

Figure 5. 9 Caloric Compensation after the Treatments



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test.

The total *ad libitum* FI for all treatments at 120 min was lower in female participants compared to male participants (P=0.03) (Table 5.5). There was no treatment by sex interaction (P=0.6) on *ad libitum* FI at 120 min.

The total cumulative FI over 120 min was lower in female participants compared to male participants (P<0.05) (Table 5.5). There was no treatment by sex interaction (P=0.6) on cumulative FI over 120 min.

Table 5. 5 Mean Food Intake by Treatment with Sex Comparison

		W	C3.3%	C5.5%	Y3.3%	Y5.5%	*P value*
Ad libitum food intake at 120 min (kcal)	Females	753.4±209.3	625.0±182.6	653.1±187.8	670.4±211.2	662.7±191.6	P(trt)=0.02 P(sex)=0.03
	Males	1178.9±630.1	1000.3±561.2	1086.4±583.8	1009.2±456.5	1156.6±572.8	P(trt x sex)=0.6
Cumulative food intake over 120 min (kcal)	Females	753.4±209.3	774.0±182.6	824.1±187.8	793.4±211.2	787.8±201.4	P(trt)=0.02 P(sex)=0.03
	Males	1178.9±630.1	1149.3±561.2	1257.4±583.8	1132.2±456.5	1295.6±572.8	P(trt x sex)=0.6

Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA followed by Tukey–Kramer post hoc test for sex comparison of ad libitum food intake, and cumulative food intake. P indicates P values for the effect of a treatment (P trt), session (P sex), and a treatment by sex interaction (P trt × sex).

5.4 Subjective Appetite

Average appetite (AA) scores were calculated using the scores of four motivation to eat dimensions, including desire to eat, hunger, fullness, and prospective food consumption (Table 5.6). There was an effect of a treatment ($P<0.0001$), time ($P<0.0001$), and a treatment by time interaction ($P=0.01$) on AA over 120 min (Table 5.6). All treatments, C3.3%, C5.5%, Y3.3% and Y5.5%, resulted in a suppression of AA compared to water control over 120 min ($P<0.001$). There was an effect of treatment ($P=0.0003$), time ($P < 0.0001$) and a treatment by time interaction ($P=0.01$) on ΔAA scores (Table 5.6). There was an effect of treatment on appetite $tAUC_{0\text{-}120min}$ ($P=0.0004$). There was no effect of a session ($P=0.9$) and a treatment by session interaction ($P=0.8$) on AA $tAUC_{0\text{-}120min}$. There was no effect of treatment ($P=0.4$), time ($P=0.2$) and a treatment by time interaction ($P=0.4$) on AA satiety quotient (SQ).

There was an effect of treatment ($P < 0.0001$) and time ($P < 0.0001$) on DTE over 120 min (Table 5.6). There was no treatment by time interaction on DTE over 120 min ($P=0.1$). The treatments with C3.3%, C5.5% and Y5.5% suppressed subjective DTE over 120 min compared to water (control) ($p<0.05$). There was an effect of treatment ($P < 0.0001$) and time ($P < 0.0001$) on Δ DTE scores. There was no treatment by time interaction on ΔDTE scores ($P=0.5$). There was an effect of a treatment on DTE tAUC0-120min ($P=0.001$). There was no effect of session ($P=0.6$) and a treatment by session interaction ($P=0.6$) on DTE tAUC. There was no effect of treatment ($P=0.3$), and a treatment by time interaction ($P=0.4$) on average DTE SQ. There was an effect of time on average DTE SQ ($P=0.04$).

There was an effect of treatment ($P=0.003$), and time ($P < 0.0001$) on average fullness over 120 min (Table 5.6). There was no treatment by time interaction ($P=0.1$) on fullness over 120 min. There was a difference between C3.3%, Y3.3% and Y5.5% compared to water control on fullness

over 120 min (P<0.05) (Table 5.6). There was no effect of treatment (P=0.8), time (P=0.8) and a treatment by time interaction (P=0.5) on fullness satiety quotient.

There was an effect of treatment (P< 0.0001), and time (P < 0.0001) on subjective feelings of average hunger over 120 min. There was no treatment by time interaction on the subjective feelings of hunger over 120 min (P=0.4) (Table 5.6). The treatments with C3.3% and Y5.5% resulted in a lower subjective feeling of hunger over 120 min compared to water control (P<0.05). There was no effect of a treatment (P=0.4), time (P=0.3) and a treatment by time interaction (P=0.9) on hunger SQ.

There was an effect of a treatment (P=0.001), time (P < 0.0001) and a treatment by time interaction (P=0.003) on prospective food consumption over 120 min (Table 5.6). There was a difference between C3.3%, C5.5% and Y5.5% compared to water control on prospective food consumption over 120 min (P<0.05). There was no effect of treatment (P=0.6), time (P=0.6) and treatment by time interaction (P=0.6) on prospective food consumption SQ.

Table 5. 6 Mean Subjective Appetite by Treatment

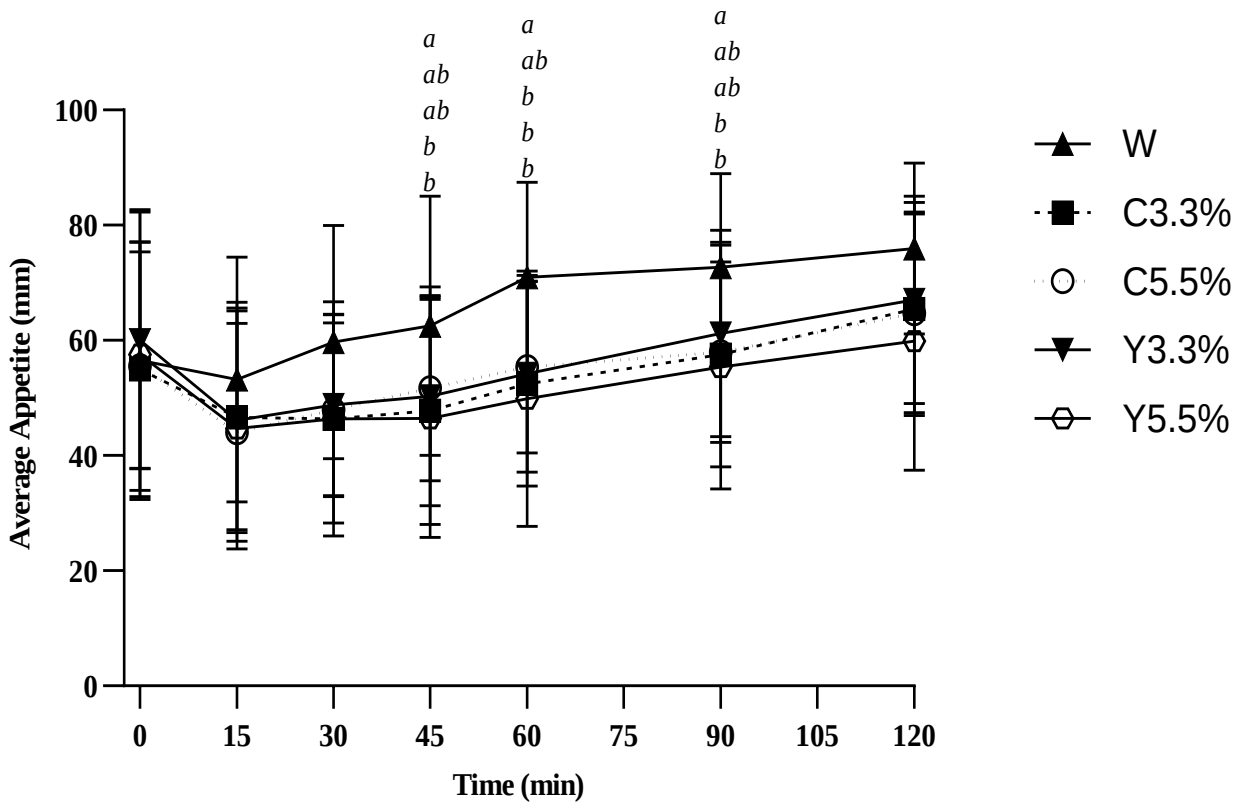
	Treatments					
Measures	W	C3.3%	C5.5%	Y3.3%	Y5.5%	P value
Average Appetite (mm VAS)	64.5±20.1[a]	53.0±20.1 [b]	53.9±17.9[b]	55.4±19.4[b]	51.5±22.3[b]	P(trt) <0.0001 P(time) <0.0001 P(trt × time)=0.01
ΔAverage Appetite (mm VAS)	2.3±26.8[a]	-7.1±22.5[ab]	-6.8±23.2[ab]	-9.7±23.9[b]	-11.01±24.9[b]	P(trt) =0.0003 P(time) <0.0001 P(trt × time)=0.01
Average Appetite tAUC (mm x min)	7972.8±1870.4 [a]	6560.1±2070.9[b]	6527.3±1739.6[b]	6688.38±1874.70 [b]	6176.1±2368.2[b]	P(trt) =0.0004 P(session)=0.9 P(trt × session)=0.8
Average Appetite (SQ)	_	4.3±11.7	-0.2±9	3.5±11.9	4.7±11.5	P(trt) =0.4 P(session)=0.2 P(trt × session)=0.4
DTE (mm VAS)	58.6±25.2[a]	47.1±24.0[b]	48.2±22.4[b]	49.8±23.9[ab]	44.6±26.5[b]	P(trt) <0.0001 P(time) <0.0001 P(trt × time)= 0.1
ΔDTE (mm VAS)	7.2±29.1[a]	-5.0±26.6[b]	-5.0±27.1[b]	-9.2±25.2[b]	-10.6±29.5[b]	P(trt) <0.0001 P(time) <0.0001 P(trt × time)= 0.5
DTE tAUC (mm x min)	7285.8±2420.3[a]	5732.6±2264.2[b]	5897.6±2010.7[b]	5942.3±2406.0[ab]	5335.8±2692.8[b]	P(trt) =0.001 P(session)=0.6 P(trt × session)=0.6
DTE (SQ)	_	0.1±12.8	0.1±11.6	4.5±13.9	5.0±17.5	P(trt) =0.3 P(session)=0.04 P(trt × session)=0.2
Fullness (mm VAS)	20.0±21.4[a]	30.6±21.1[b]	30.3±21.1[ab]	29.7±22.1[b]	31.6±24.2[b]	P(trt) 0.003 P(time) <0.0001 P(trt × time)= 0.1
Fullness (SQ)	_	-4.6±11.8	-4.7±10.8	-6.0±13.6	-5.6±17.0	P(trt) =0.8 P(session)=0.8 P(trt × session)=0.5
Hunger (mm VAS)	57.5±25.6[a]	43.5±26.7[b]	45.1±24.2[ab]	47.1±25.7[ab]	41.9±27.4[b]	P(trt) <0.0001 P(time) <0.0001 P(trt × time)= 0.4
Hunger (SQ)	_	1.6±12.4	2.1±12.1	6.6±19.2	7.7±17.1	P(trt) =0.4 P(session)=0.3 P(trt × session)=0.9
Prospective Food Consumption (mm VAS)	62.0±21.0[a]	52.1±23.7[b]	52.6±20.9[b]	54.3±21.7[ab]	50.9±24.9[b]	P(trt)=0.001 P(time) <0.0001 P(trt × time)= 0.003

Prospective Food Consumption (SQ)	_	-0.8±8.5	-1.7±8.2	1.2±10.1	1.04±9.8	P(trt) =0.6 P(session)=0.6 P(trt × session)=0.6

Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, DTE=Desire to eat, VAS=visual analogue scale, SQ=Satiety quotient, n = 20. Two-way ANOVA followed by Tukey–Kramer post hoc test for average appetite, Δaverage appetite, DTE, ΔDTE, fullness, hunger, and prospective food consumption. One-way ANOVA followed by Tukey–Kramer post hoc test for average appetite tAUC, DTE tAUC, APP SQ, DTE SQ, fullness SQ, hunger SQ, and prospective food consumption SQ. P indicates P values for the effect of a treatment (P trt), time (P time), and a treatment by time interaction (P trt × time) and a treatment (P trt), session (P session), and a treatment by session interaction (P trt × session). Different superscript letters represent significant differences between treatments (P<0.05).

No significant differences were observed for AA between the treatments with C3.3%, C5.5%, Y3.3% and Y5.5% at any the individual time points (Figure 5.10). At 45 min, the treatments with C3.3% and Y5.5% resulted in a lower AA compared to water ($P<0.05$). Treatments with C5.5% and Y3.3% were not different than water control ($P>0.05$). At 60 min, all treatments, except the treatment with C5.5%, showed significantly lower AA than water control. At 90 min, treatments with C3.3% and Y5.5% led to a lower AA compared to water control ($P<0.05$), while treatments with C5.5% and Y3.3% were not different than water control ($P>0.05$).

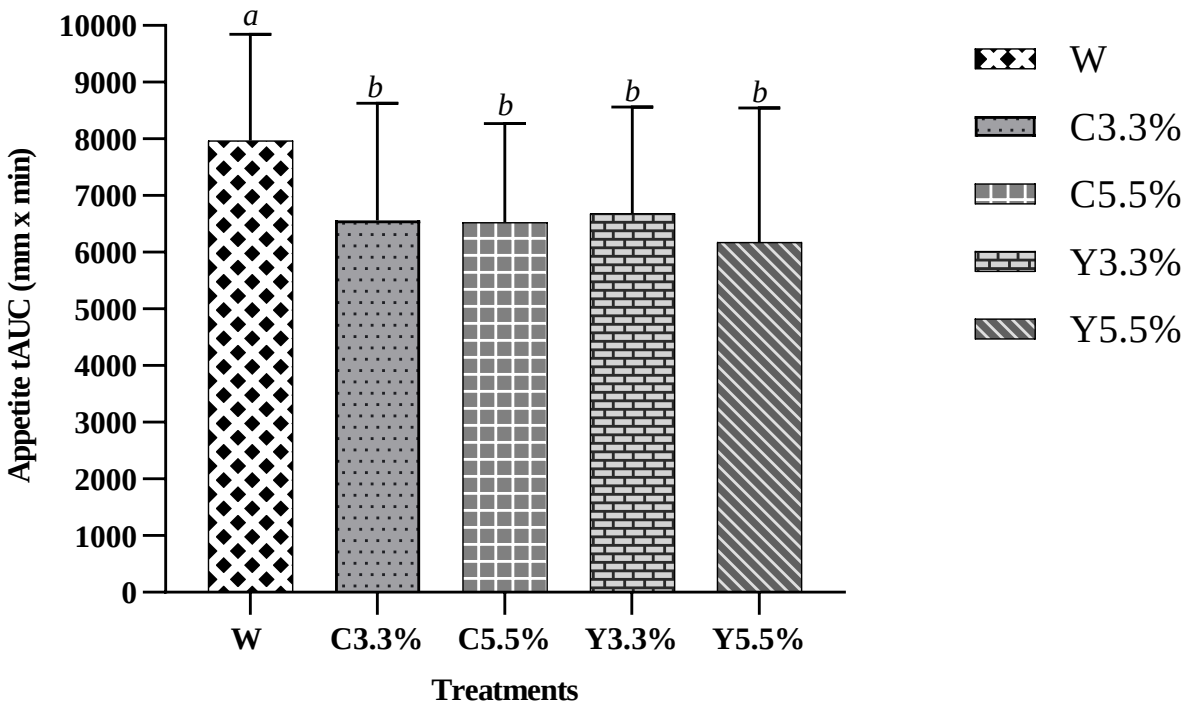
Figure 5. 10 Average Appetite over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. The values with different letters are significantly different (P<0.05).

AA $tAUC_{0\text{-}120min}$ was higher for water control compared to treatments with C3.3%, C5.5%, Y3.3% and Y5.5% ($P<0.05$) (Figure 5.11). No differences were observed between treatments with C3.3%, C5.5%, Y3.3% and Y5.5% on AA $tAUC_{0\text{-}120min}$ ($P>0.05$).

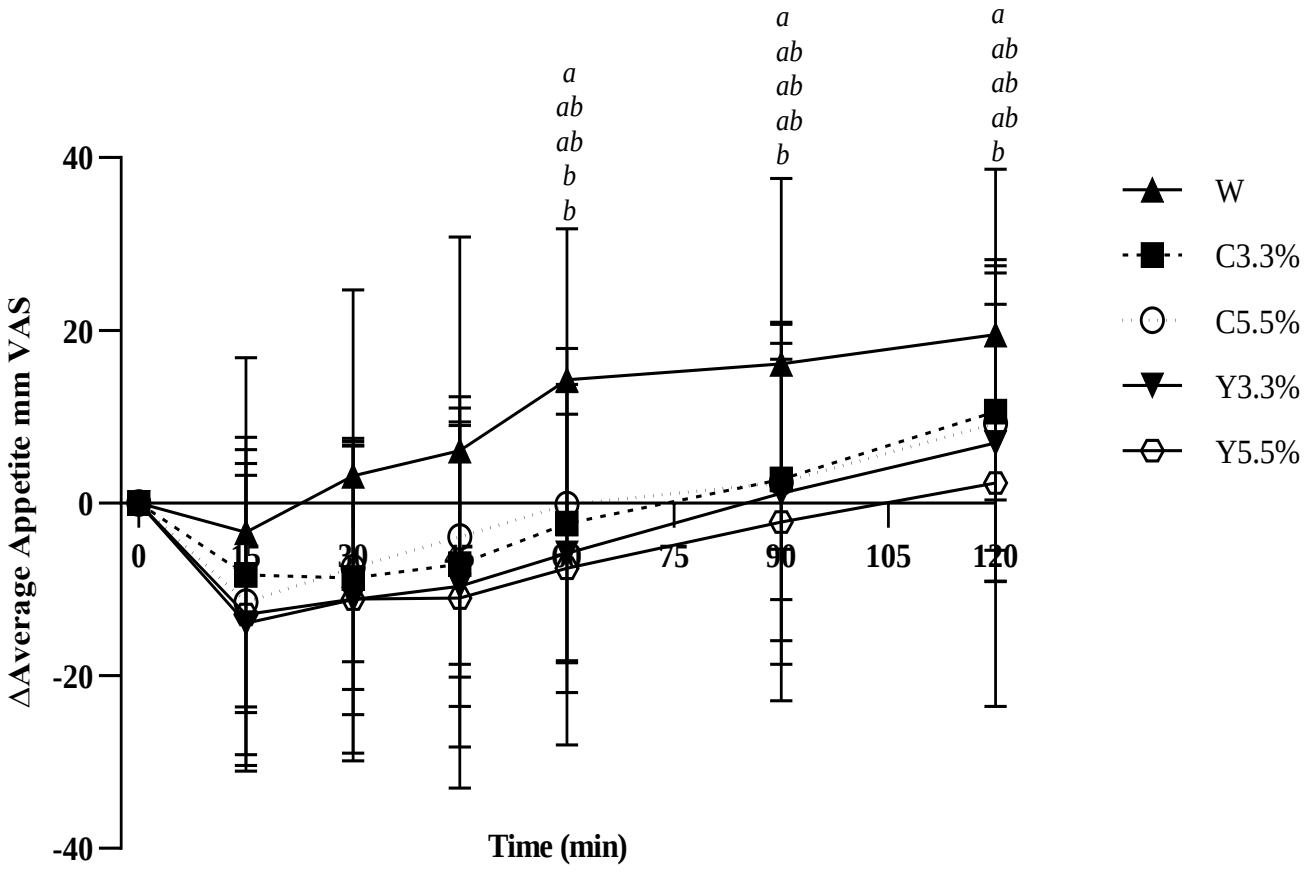
Figure 5. 11 Subjective Average Appetite $tAUC_{0\text{-}120min}$



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

No differences were observed between treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on ΔAverage Appetite at 0, 15, 30 and 45 time points (Figure 5.12). There was no difference on ΔAA between treatments with Y3.3% and Y5.5% compared to water control at 60 min ($P<0.05$). There was difference between Y5.5% and water control on ΔAA at 90 and 120 min ($P<0.05$).

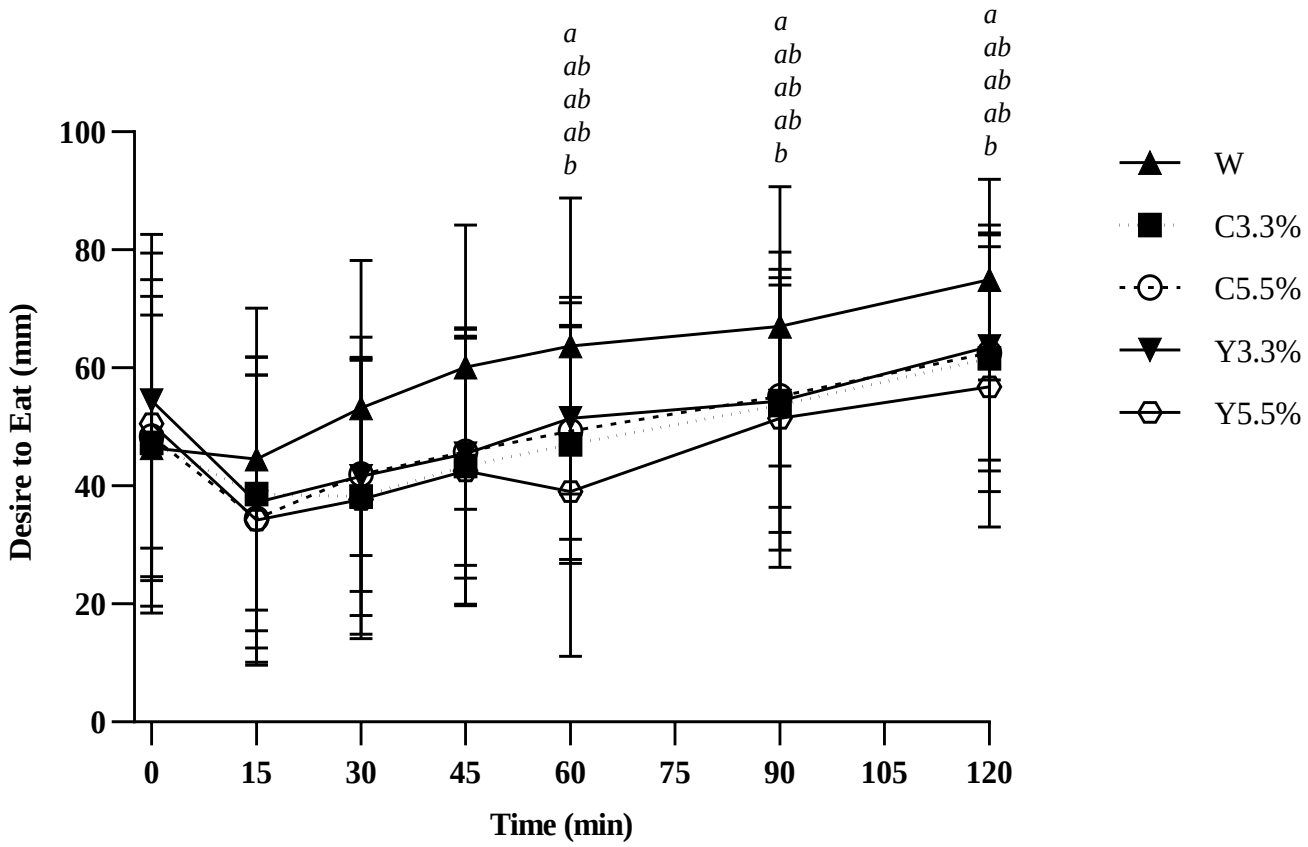
Figure 5. 12 ΔAverage Appetite (mm VAS) over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There were no differences between treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on DTE at 0, 15, 30 and 45 min (P>0.05) (Figure 5.13). There was a difference between the treatment with Y5.5% and water control on DTE at 60, 90 and 120 min (P<0.05).

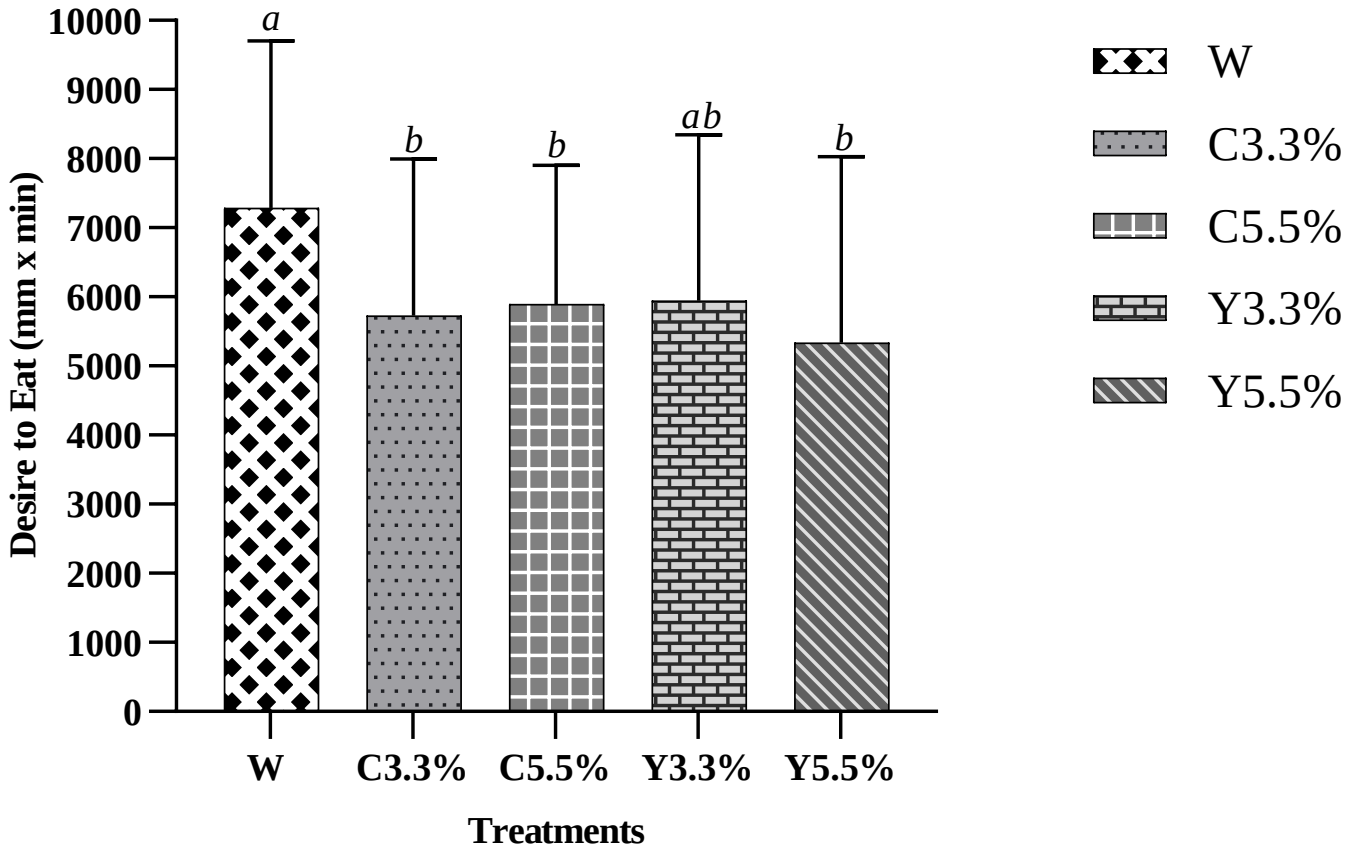
Figure 5. 13 Desire to Eat over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

DTE $tAUC_{0\text{-}120min}$ was higher for the treatment with Y3.3% and water control compared to C3.3%, C5.5%, and Y5.5% ($P<0.05$) (Figure 5.14). There was no difference on DTE $tAUC_{0\text{-}120min}$ between treatment with Y3.3% and water control ($P>0.05$). There was no difference on DTE $tAUC_{0\text{-}120min}$ between treatment with C3.3%, C5.5%, and Y5.5% ($P<0.05$).

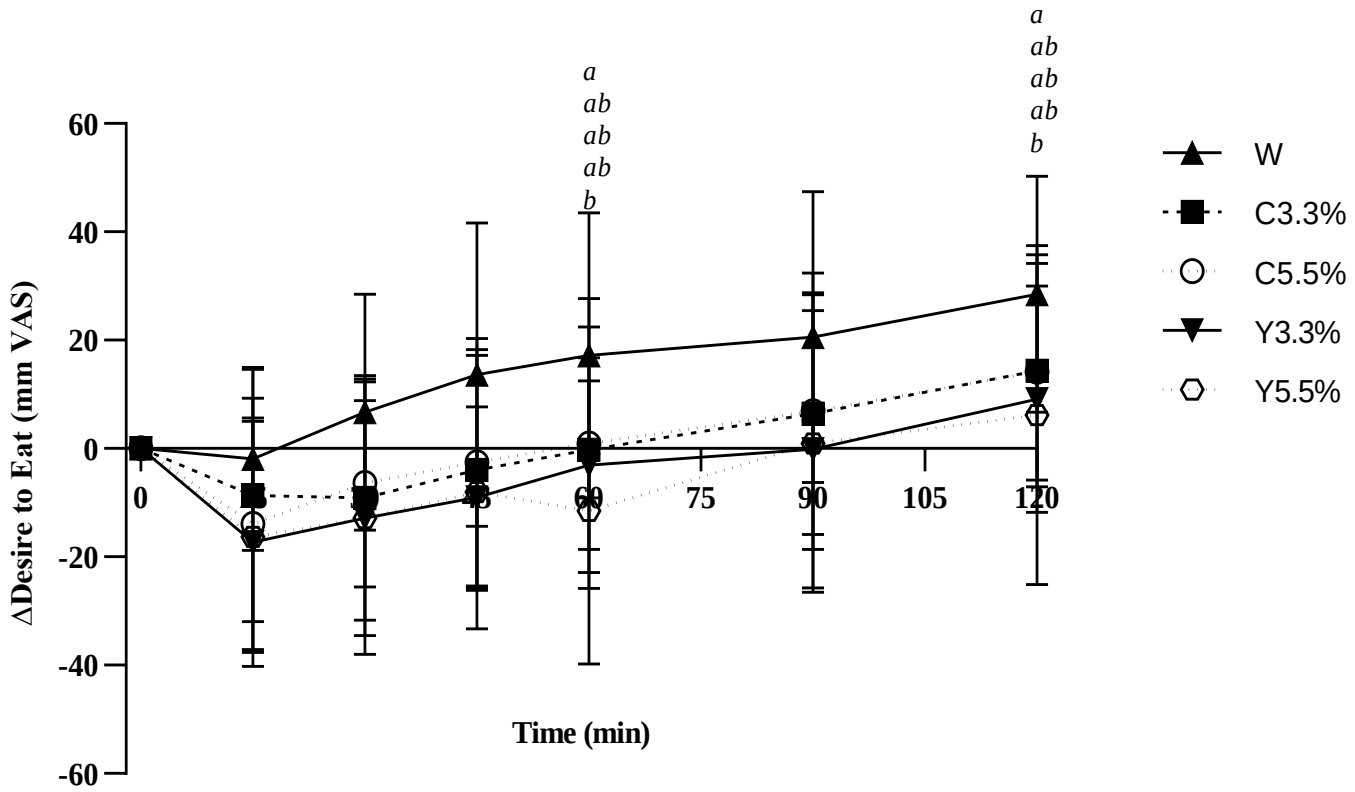
Figure 5. 14 Desire to Eat $tAUC_{0\text{-}120min}$



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

No significant differences observed between treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on ΔDTE at 0, 15, 30, 45 and 90 time points (Figure 5.15). There was a difference between the treatment with Y5.5% compared to water control ($P<0.05$) on ΔDTE at 60 min. There was a difference between the treatment with Y5.5% and water control on ΔDTE at 60 and 120 min ($P<0.05$).

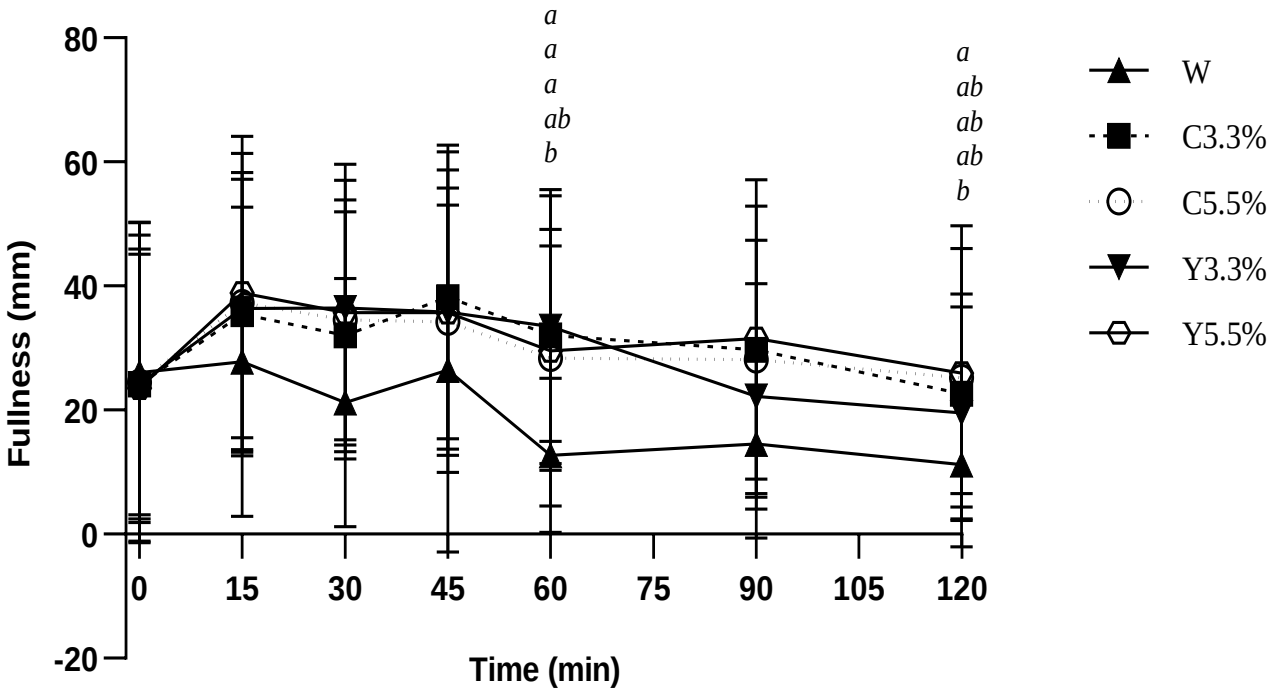
Figure 5. 15 ΔDesire to Eat (mm VAS) over 120 min



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There were no significant differences between treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on subjective feelings of fullness at 0, 15, 30, 45 and 90 time points (Figure 5.16). There was a difference between treatments with C3.3%, Y3.3% and Y5.5% compared to water control ($P<0.05$) on subjective feelings of fullness at 60 min. There was a difference between treatments with Y5.5% and water control on subjective feelings of fullness at 120 min ($P<0.05$).

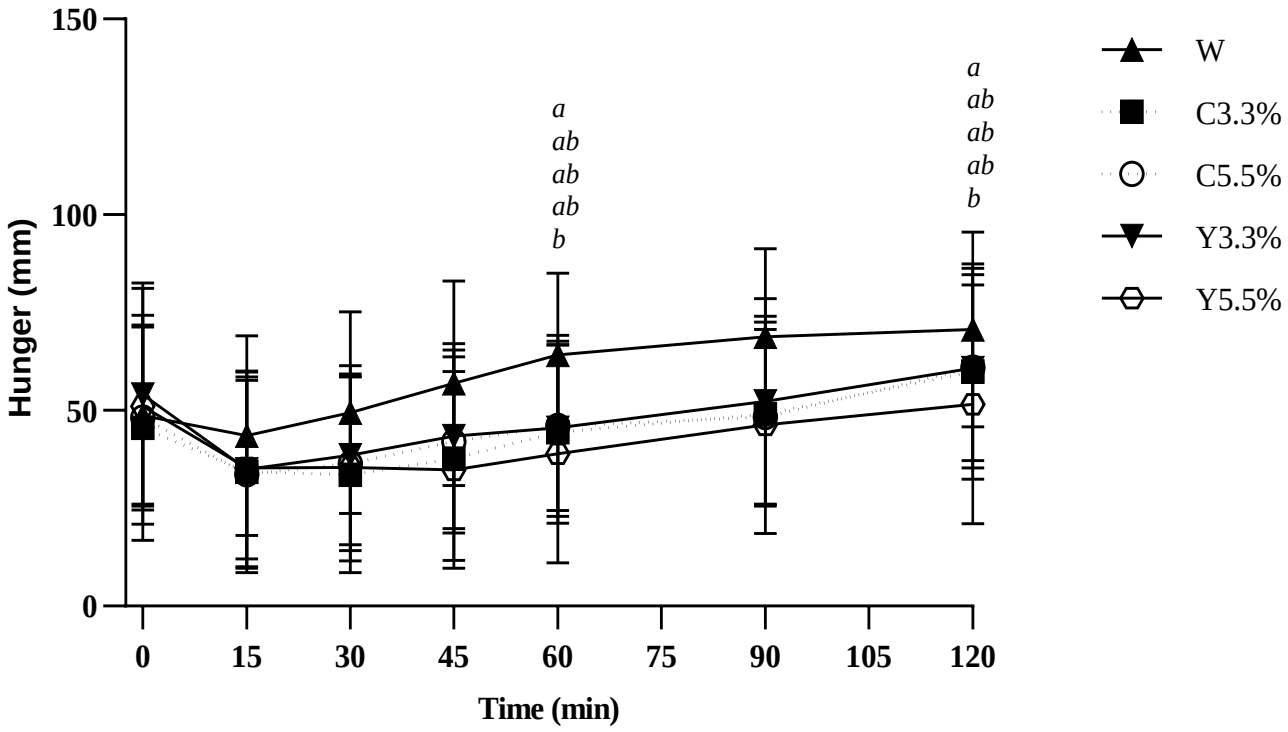
Figure 5. 16 Subjective Feeling of Fullness (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. The values with different letters are significantly different (P<0.05).

No significant differences were observed between the treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on subjective feelings of hunger at 0, 15, 30, 45 and 90 time points (Figure 5.17). There was a difference between the treatment with Y5.5% and water control on hunger at 60 and 120 min ($p<0.05$).

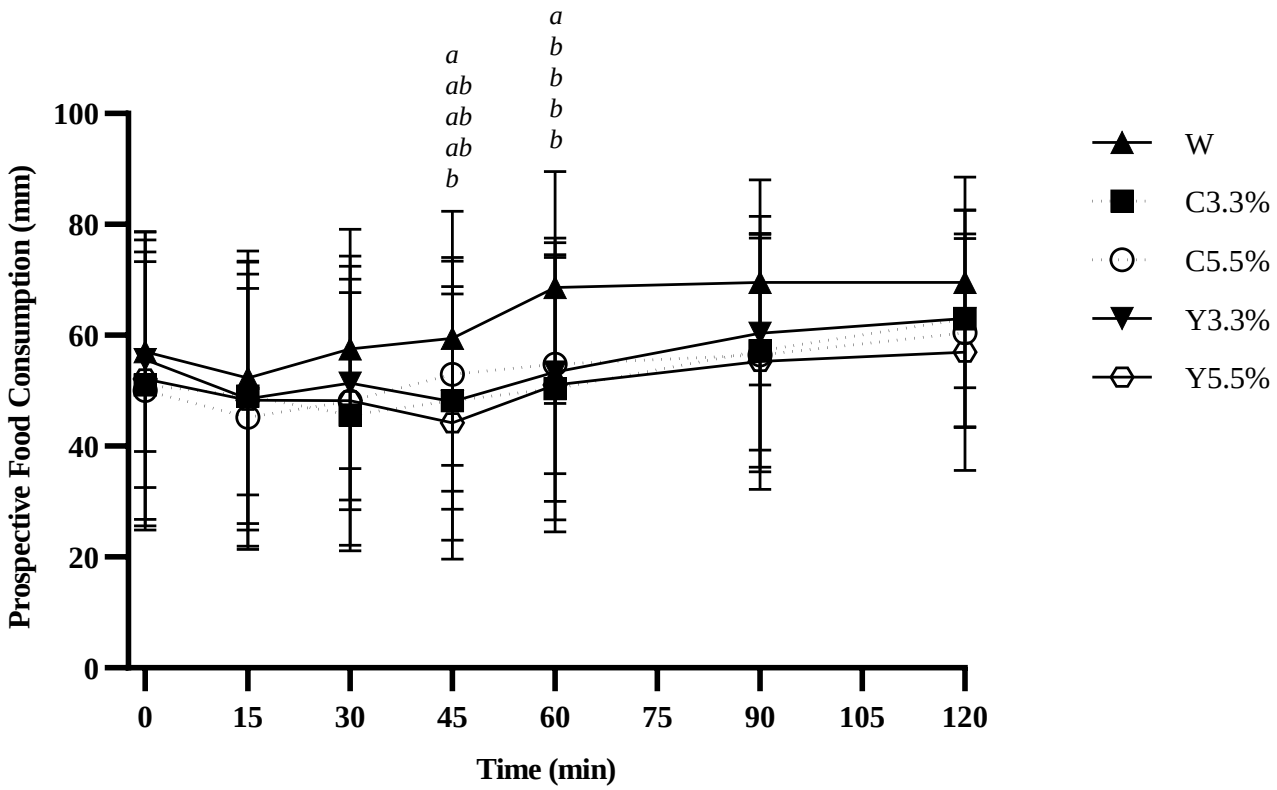
Figure 5. 17 Subjective Feeling of Hunger (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was no significant difference between C3.3%, C5.5%, Y3.3%, Y5.5% and water control on prospective food consumption at 0, 15, 30, 90 and 120 min (Figure 5.18). There was a difference between the treatment with Y5.5% and water control on prospective food consumption at 45 min ($P<0.05$). There was significant difference between C3.3%, C5.5%, Y3.3%, and Y5.5% compared to water (control) on prospective food consumption at 60 min ($P<0.05$).

Figure 5. 18 Subjective Feeling of Prospective Food Consumption (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. The values with different letters are significantly different (P<0.05).

5.5 Thirst, Energy, Fatigue and Physical Comfort Parameters over 120 min

There was an effect of time (P<0.0001) but not treatment (P=0.5) or time by treatment interaction (P=0.4) on subjective feelings of thirst (Table 5.7). There was an effect of time (P < 0.0001) but not treatment (P=0.9) or time by treatment interaction (P=0.7) on perceived feeling of energy. There was no effect of treatment (P=0.8), time (P=0.2), and time by treatment interaction (P=0.4) on perceived fatigue. There was no effect of treatment (P=0.8), time (P=0.4), and time by treatment interaction (P=0.5) on perceived feeling of wellness. There was no effect of treatment on parameters of gastrointestinal comfort such as the feeling of diarrhea or gas (P=0.9) and no time by treatment interaction (P=0.5). There was an effect of time (P=0.01) on feeling of diarrhea, but there was no effect of time (P=0.5) on feeling of gas. There was no effect of treatment (P = 0.7), time (P=0.4), and time by treatment interaction (P=0.6) on feeling of stomach pain. There was no effect of treatment (P = 0.9), time (P=0.5), and time by treatment interaction (P=0.9) on subjective feelings of nausea.

Table 5. 7 Mean Thirst, Energy, Fatigue and Physical Comfort Parameters over 120 min

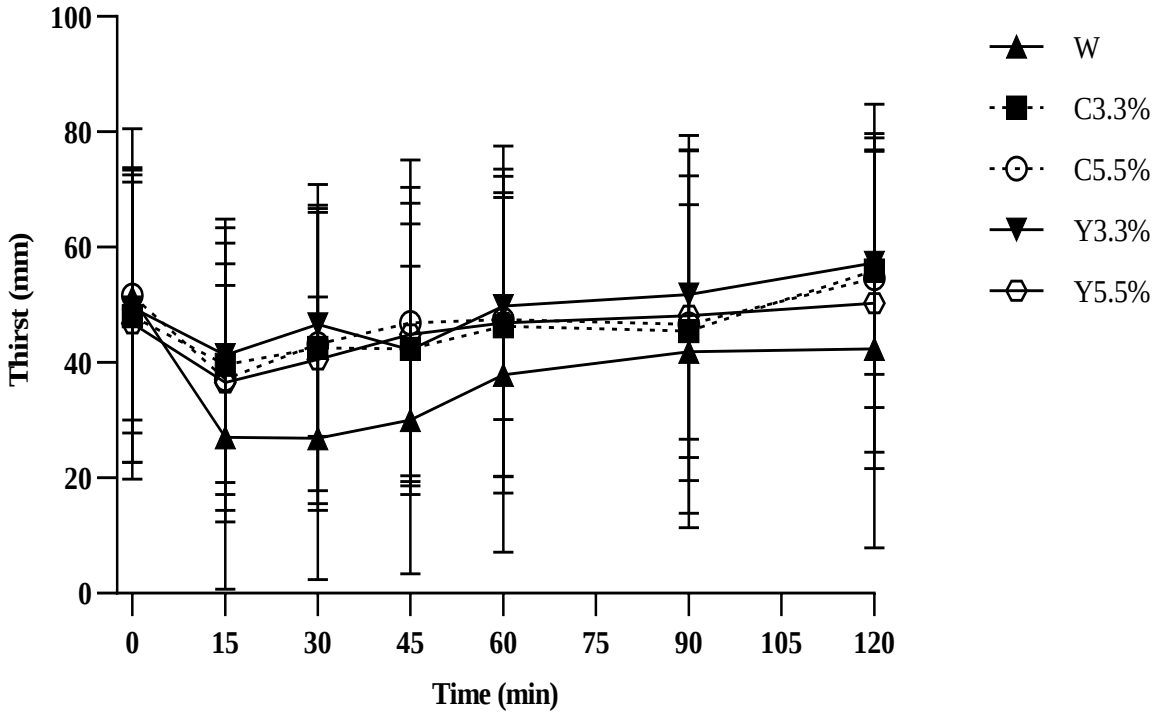
	Treatments					
Measures	W	C3.3%	C5.5%	Y3.3%	Y5.5%	P value
Thirst (mm VAS)	36.7±28.7	45.7±24.6	46.7±28.4	48.4±21.6	44.8±26.5	P(trt)=0.52 P(time) <0.0001 P(trt × time)=0.4
Energy (mm VAS)	60.9±24.6	62.1±21.5	61.1±23.7	56.4±23.7	61.2±25.4	P(trt)=0.9 P(time) <0.0001 P(trt × time)= 0.7
Fatigue (mm VAS)	24.8±21.1	31.5±22.1	31.3±24.9	34.2±23.8	30±26.2	P(trt)=0.8 P(time)= 0.2 P(trt × time)= 0.4
Wellness (mm VAS)	72.1±26.3	74.8±24.4	73.4±24.9	71±25.1	72.9±23.9	P(trt)=0.8 P(time)= 0.4 P(trt × time)= 0.5

Diarrhea (mm VAS)	1.8±6.5	1.5±3.5	1.6±4.2	1.9±5.3	3.2±9.2	P(trt)=0.9 P(time)=0.01 P(trt × time)=0.5
Gas (mm VAS)	2.6±9.3	3.6±10.9	2.5±5.5	4.1±9.9	3.8±11.2	P(trt)=0.9 P(time)=0.5 P(trt × time)=0.9
Stomach pain (mm VAS)	5±11.8	2.4±6.7	2.6±5.5	3.5±8.4	2.9±6.2	P(trt)=0.7 P(time)=0.4 P(trt × time)=0.6
Nausea (mm VAS)	3.8±8.6	2±4.3	3±7.2	3.9±8.6	3.5±8.3	P(trt)=0.9 P(time)=0.5 P(trt × time)=0.9

Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, VAS=visual analogue scale, n = 20. Two-way ANOVA followed by Tukey–Kramer post hoc test for thirst and physical comfort measures (energy, fatigue, wellness, diarrhea, gas, stomach pain, and nausea) over 120 min. P indicates P values for the effect of a treatment (P trt), time (P time), and a treatment by time interaction (P trt × time).

No statistical differences were observed on subjective feelings of thirsty at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.19).

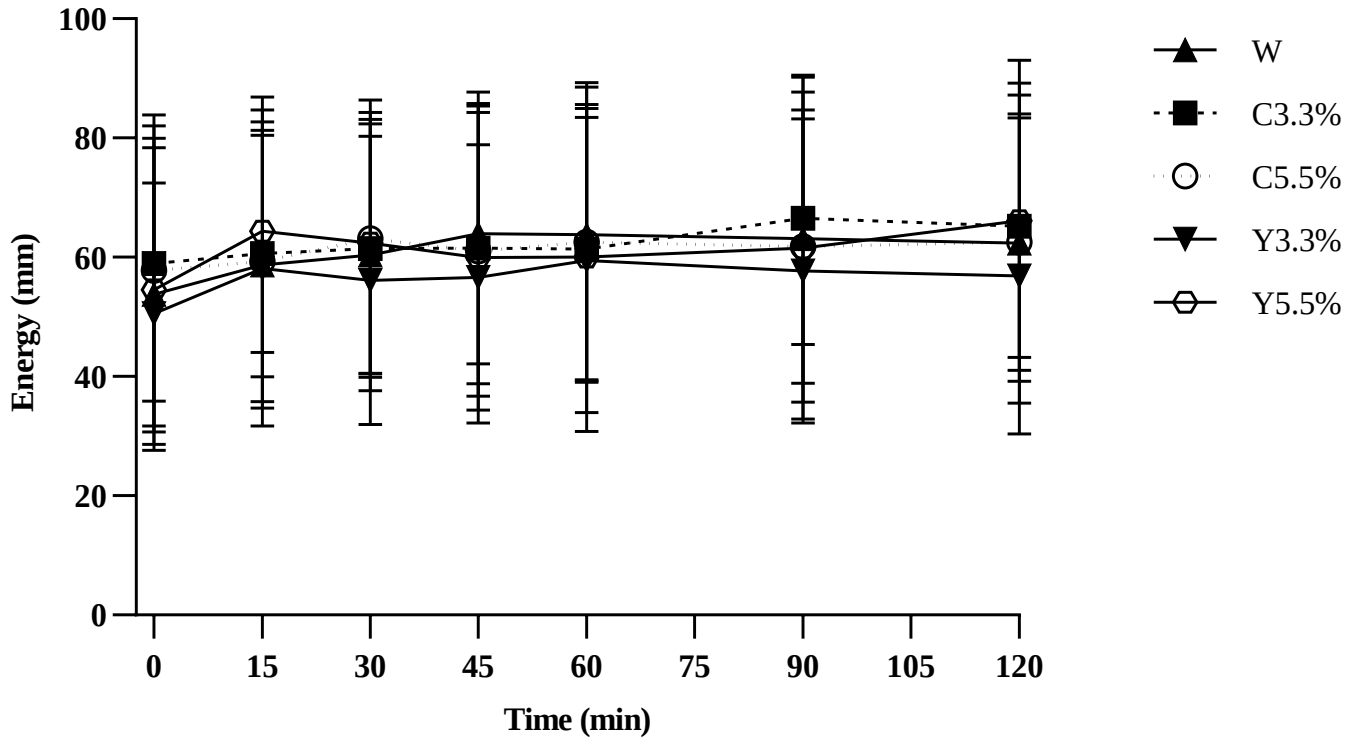
Figure 5. 19 Subjective Feeling of Thirst over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on subjective feelings of energy at 0, 15, 30, 45, 60, 90, and 120 min (P>0.05) (Figure 5.20).

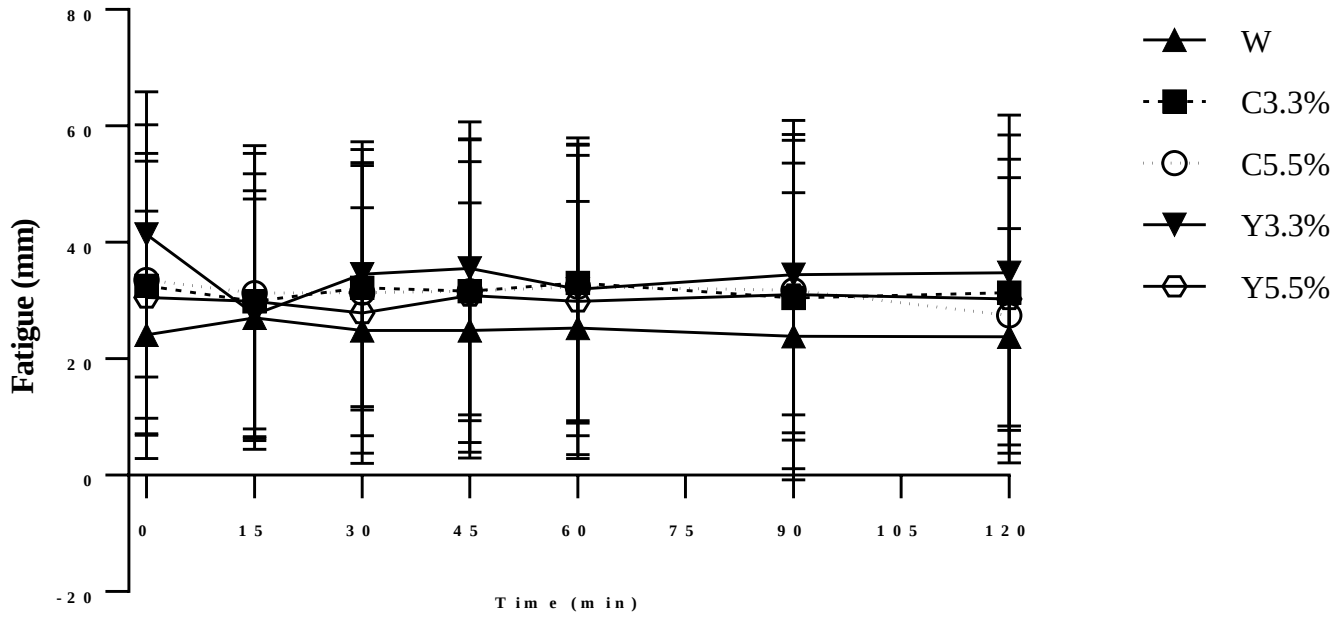
Figure 5. 20 Subjective Feeling of Energy over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on perceived fatigue at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.21).

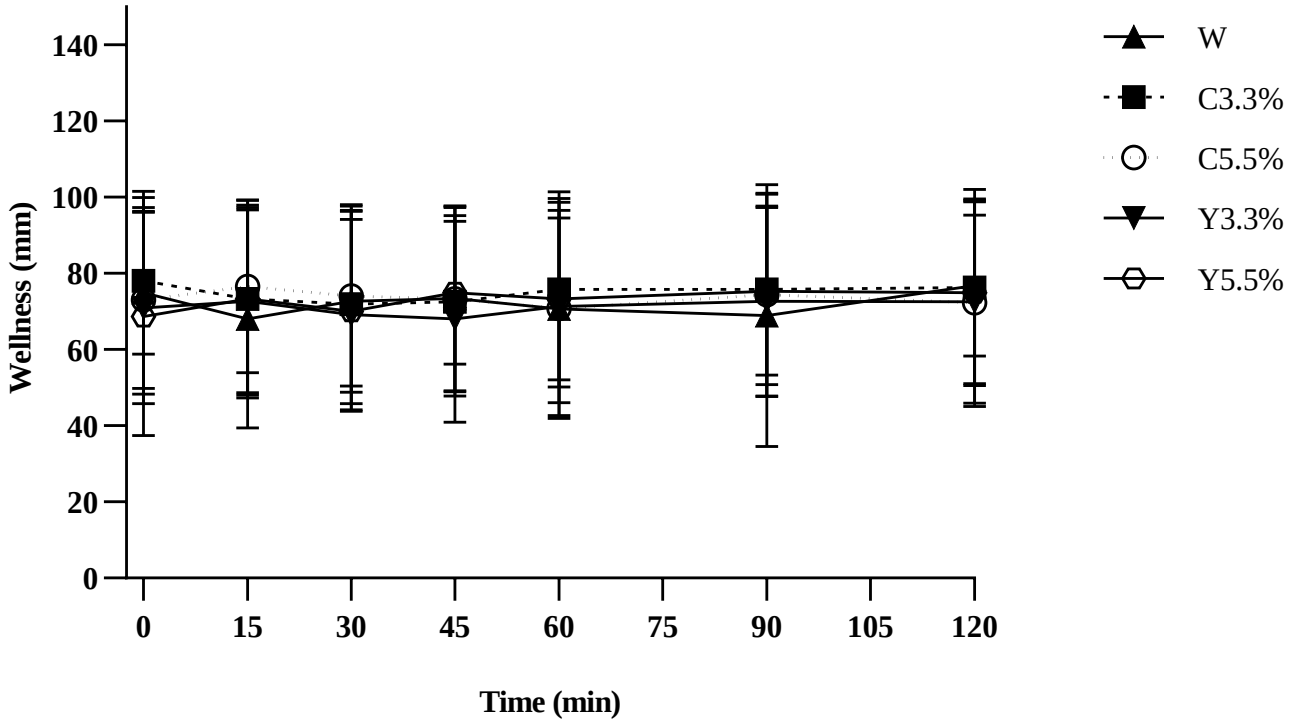
Figure 5. 21 Subjective Feeling of Fatigue over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on perceived feeling of wellness at 0, 15, 30, 45, 60, 90, and 120 min (P>0.05) (Figure 5.22).

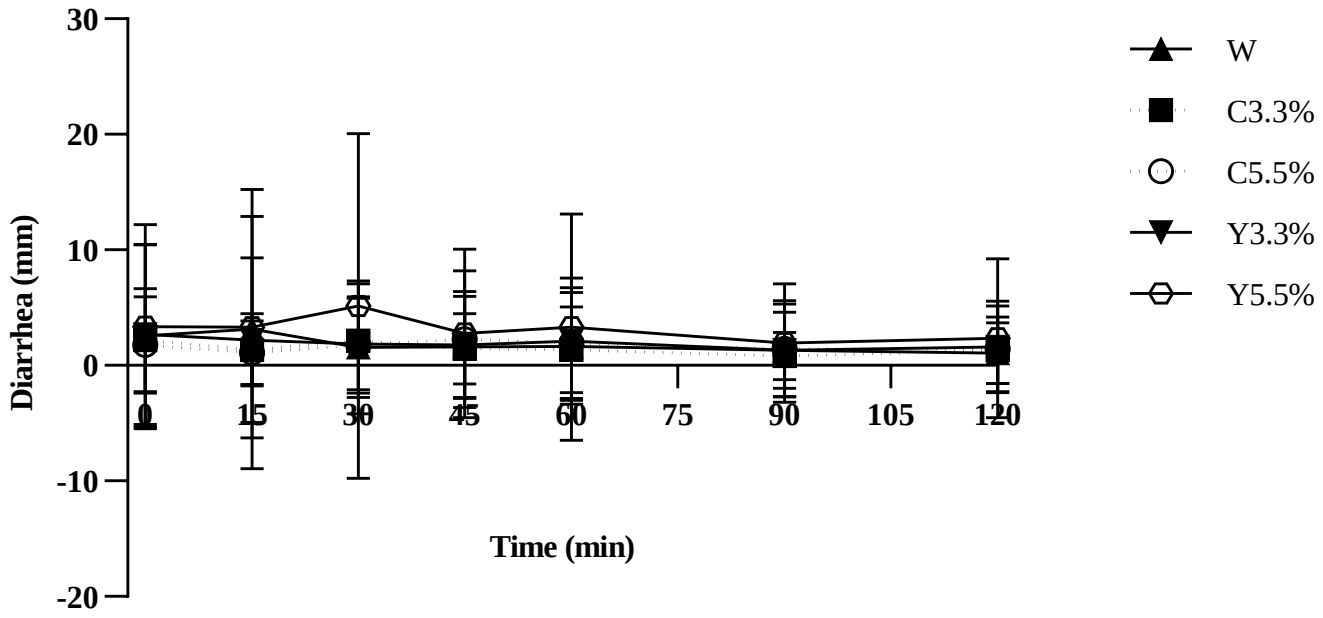
Figure 5. 22 Subjective Feelings of Wellness over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on subjective feelings of diarrhea at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.23).

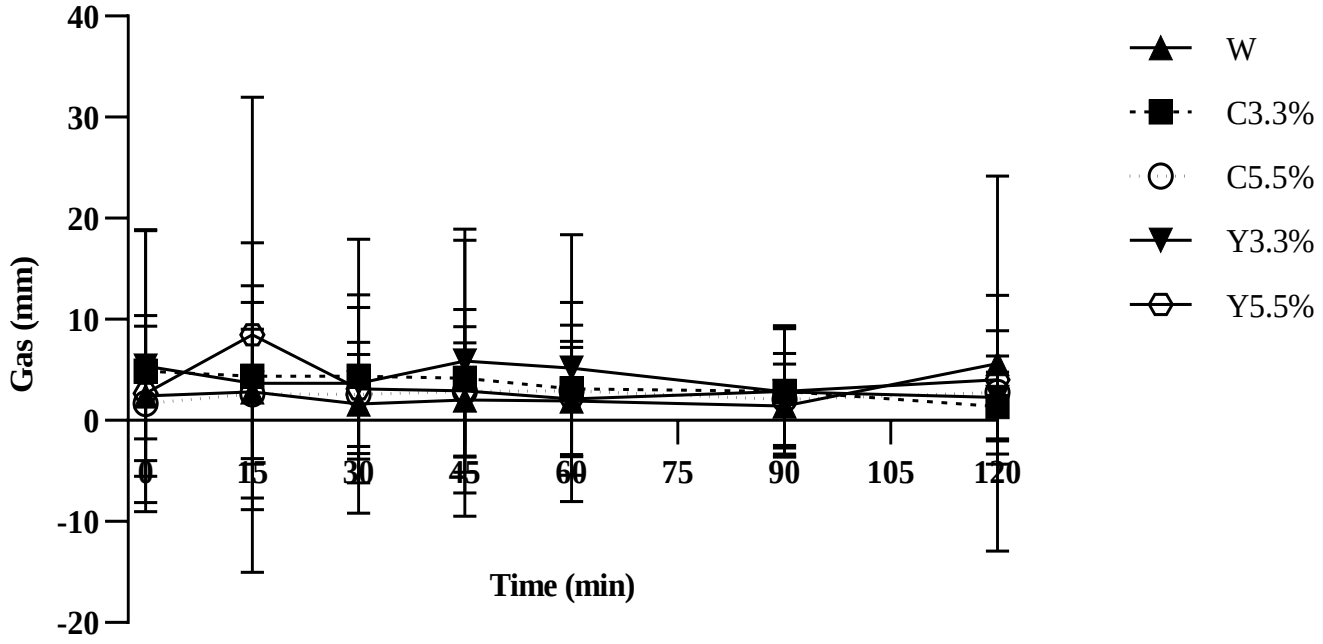
Figure 5. 23 Subjective Feeling of Diarrhea over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on subjective feelings of gas at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.24).

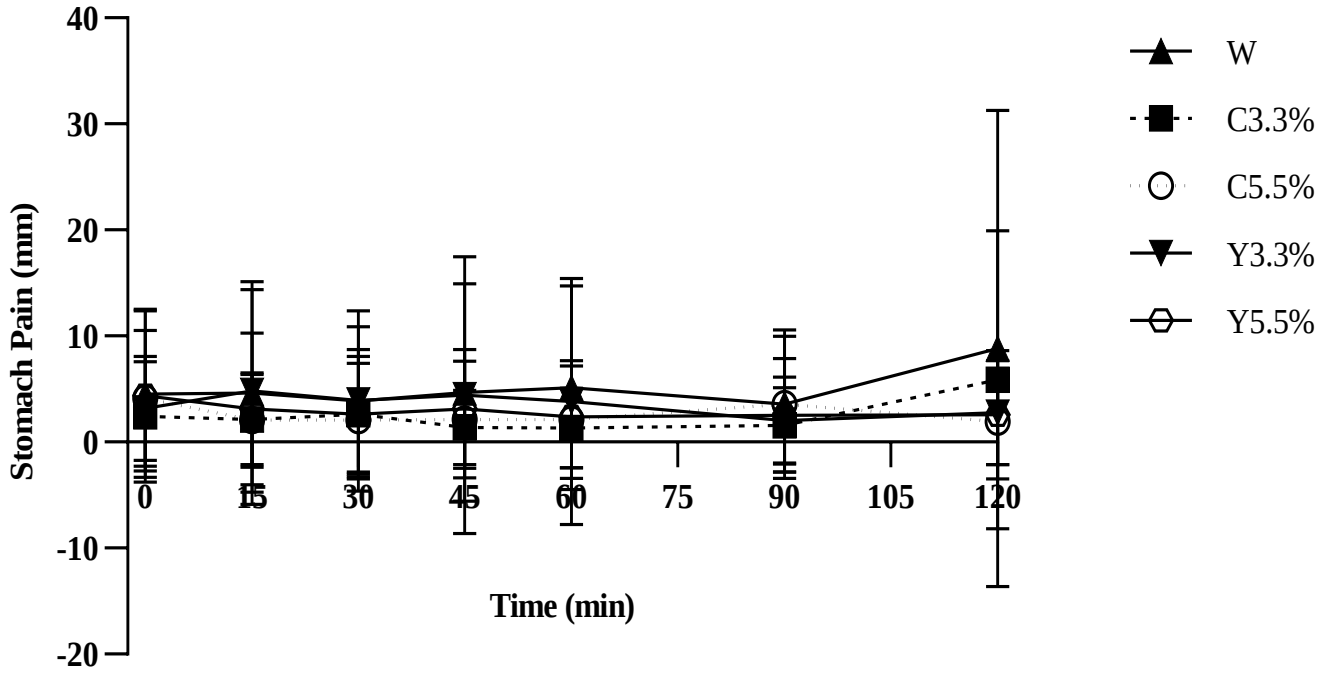
Figure 5. 24 Subjective Feelings of Gas over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test.

No statistical differences were observed on subjective feelings of stomach pain at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.25).

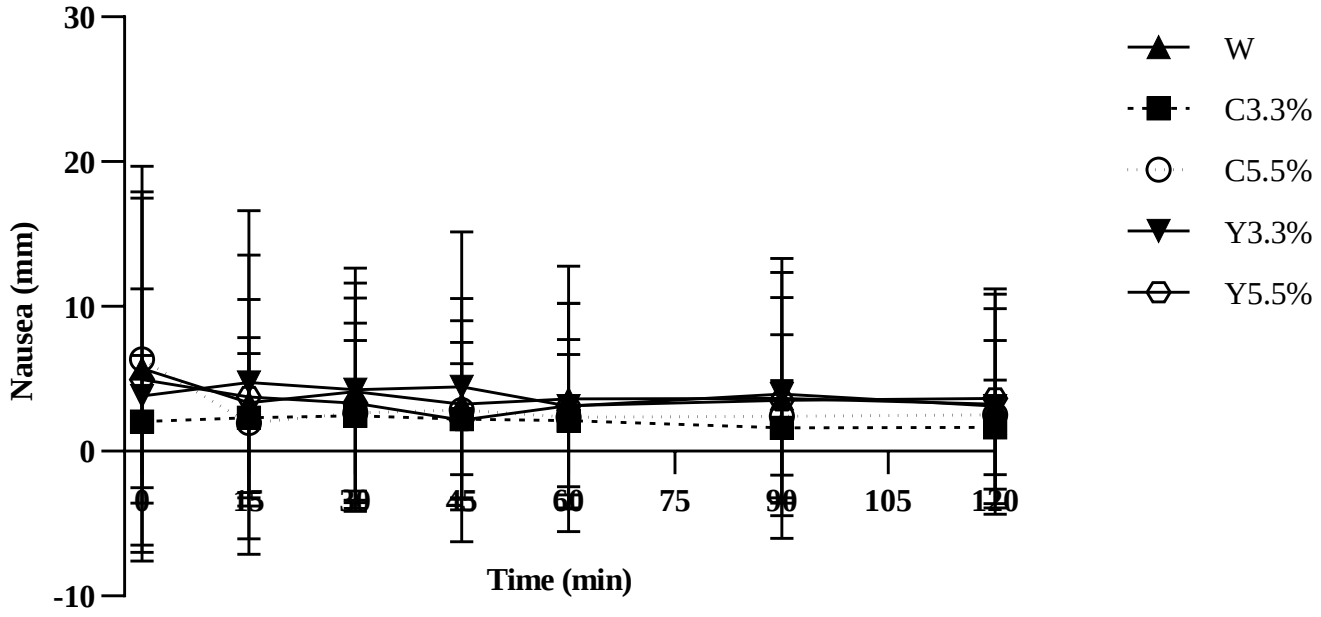
Figure 5. 25 Subjective Feeling of Stomach Pain over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with same letters are not significantly different (P>0.05).

No statistical differences were observed on subjective feelings of nausea at 0, 15, 30, 45, 60, 90, and 120 min (Figure 5.20).

Figure 5. 26 Subjective Feeling of Nausea over 120 min (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. Two-way ANOVA with Tukey's-Kramer post-hoc test. Values with same letters are not significantly different (P>0.05).

5.6 Sensory Characteristics of Treatments

The intensities of the taste stimuli were measured with VAS while their acceptability with 9-point hedonic scale. There was an effect of a treatment on perceived taste (hedonic), flavor (hedonic), sweetness (VAS), sweetness (hedonic), sourness (VAS), sourness (hedonic), bitterness (VAS), creaminess (VAS), creaminess (hedonic), and mouthfeel (hedonic) ($P<0.05$) (Table 5.8). There was no effect of a treatment on perceived pleasantness (VAS), pleasantness (hedonic), and aftertaste (hedonic) ($P>0.05$). There was an effect of a session on perceived taste (hedonic), flavor (hedonic), and bitterness (VAS) ($P<0.05$). There was no effect of session on perceived pleasantness (VAS), pleasantness (hedonic), sweetness (VAS), sweetness (hedonic), sourness (VAS), sourness (hedonic), creaminess (VAS), creaminess (hedonic), mouthfeel (hedonic), and aftertaste (hedonic) ($P>0.05$). There was a treatment by session interaction on bitterness (VAS), and creaminess (hedonic) ($P<0.05$). There was no a treatment by session interaction on pleasantness (VAS), pleasantness (hedonic), taste (hedonic), flavor (hedonic), sweetness (VAS), sweetness (hedonic), sourness (VAS), sourness (hedonic), creaminess (VAS), mouthfeel (hedonic), and aftertaste (hedonic) ($P>0.05$).

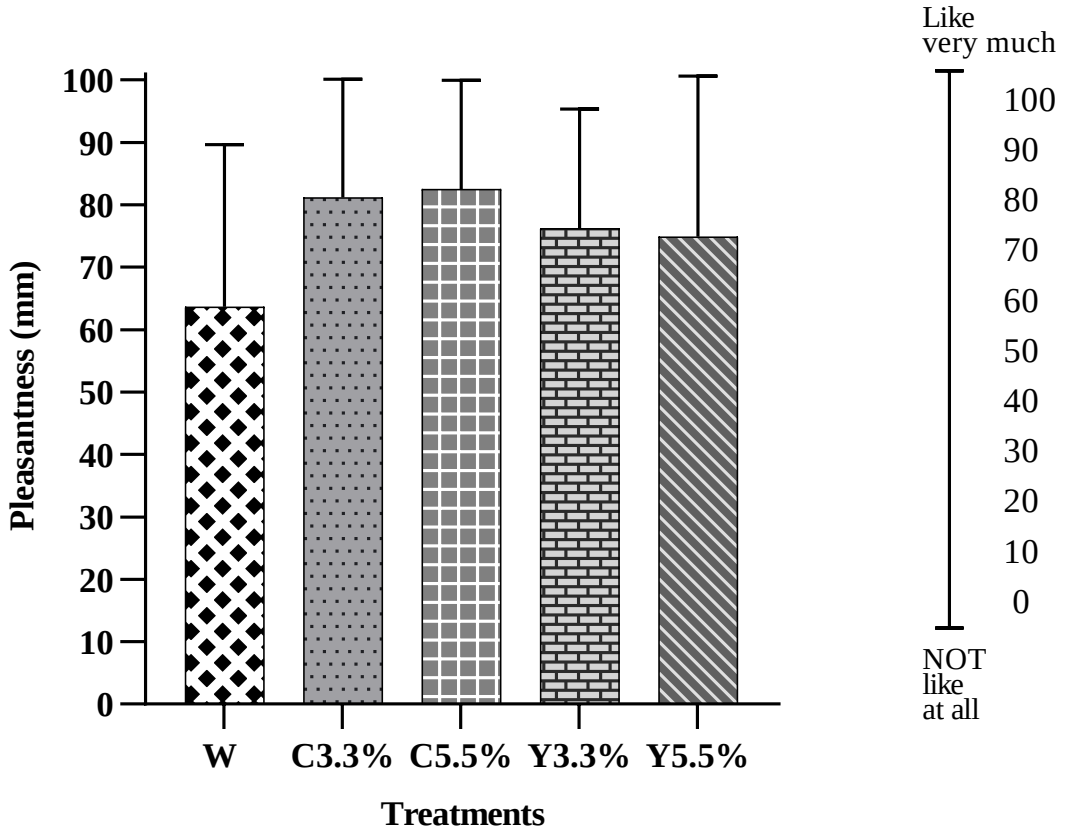
Table 5. 8 Mean Food Sensory Characteristic of Treatments

	Treatments					
Measures	W	C3.3%	C5.5%	Y3.3%	Y5.5%	P value
Pleasantness (mm VAS)	63.7±26.0	81.2±19.0	82.5±17.5	76.2±19.1	74.9±25.7	P(trt)=0.08 P(session)=0.3 P(trt × session)=0.6
Pleasantness (Hedonic)	6.4±1.7	7.4±1.5	7.6±0.9	7.2±1.5	7.5±1.8	P(trt)=0.09 P(session)=0.08 P(trt × session)=0.4
Taste (Hedonic)	6.1±1.8^{a}	7.6±1.2^{b}	7.6±1.0^{b}	7.1±1.6^{ab}	7.4±1.8^{ab}	P(trt)=0.02 P(session)=0.01 P(trt × session)=0.5
Flavor (Hedonic)	5.8±1.9 ^{a}	7.5±1.3^{b}	7.6±1.3^{b}	7.1±1.5^{ab*}	7.5±1.8^{b}	P(trt)=0.004 P(session)=0.01 P(trt × session)=0.3
Sweetness (VAS)	6.6±15.7^{a}	62.6±22.1^{b}	68.4±16.2^{b}	48.5±28.0^{b}	67.2±23.5^{b}	P(trt) <.0001 P(session)=0.8 P(trt × session)=1.0
Sweetness (Hedonic)	5.7±1.7^{a}	7.3±1.5^{b}	7.4±1.2^{b}	7.0±1.7^{ab}	6.9±2.1^{ab}	P(trt)=0.006 P(session)=0.3 P(trt × session)=0.8
Sourness (VAS)	2.7±4.1^{a}	5.9±7.0^{b}	7.0±9.2^{b}	30.8±23.4^{c}	34.7±27.9^{c}	P(trt) <.0001 P(session)=0.7 P(trt × session)=0.9
Sourness (Hedonic)	5.8±1.5^{ab}	6.4±1.7^{ab}	5.4±2.0^{a}	6.7±1.5^{b}	6.7±1.8^{ab}	P(trt)=0.01 P(session)=0.1 P(trt × session)=0.7
Bitterness (VAS)	4.3±10.6^{a}	3.9±5.1^{a}	4.8±7.3^{ab}	11.1±12.6^{ab}	13.6±25.1^{b}	P(trt)=0.004 P(session)=0.03 P(trt × session)=0.006
Creaminess (VAS)	2.9±4.1^{a}	59.5±25.8^{b}	60.1±25.4^{b}	69.5±26.6^{bc}	79.5±22.6^{c}	P(trt) <.0001 P(session)=0.09 P(trt × session)=0.09
Creaminess (Hedonic)	5.7±1.5^{a}	7.4±1.3^{b}	7.3±1.2^{b}	7.5±1.2^{b}	7.3±1.7^{b}	P(trt) <.0001 P(session)=0.5 P(trt × session)=0.04
Mouthfeel (Hedonic)	5.7±2.0^{a}	7.5±1.3^{b}	7.3±1.3^{ab*}	7.3±1.4^{b}	7.2±2.1^{ab}	P(trt)=0.01 P(session)=0.3 P(trt × session)=0.9
Aftertaste (Hedonic)	5.9±1.7	6.8±1.7	6.9±1.6	6.5±1.9	6.7±2.0	P(trt)=0.2 P(session)=0.4 P(trt × session)=0.4

*Means ± SD, Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, VAS=visual analogue scale, n = 20. One-way ANOVA followed by Tukey–Kramer post hoc test for all food sensory characteristics of treatments measures (pleasantness, taste, flavor, sweetness, sourness, bitterness, creaminess, mouthfeel, aftertaste). P indicates P values for the effect of a treatment (P trt), session (P session), and a treatment by session interaction (P trt × session). Different superscript letters represent significant differences between treatments (P<0.05). *P=0.06 compared to W.*

There were no differences between all the treatments on pleasantness (mm VAS) (P>0.05) (Figure 5.27).

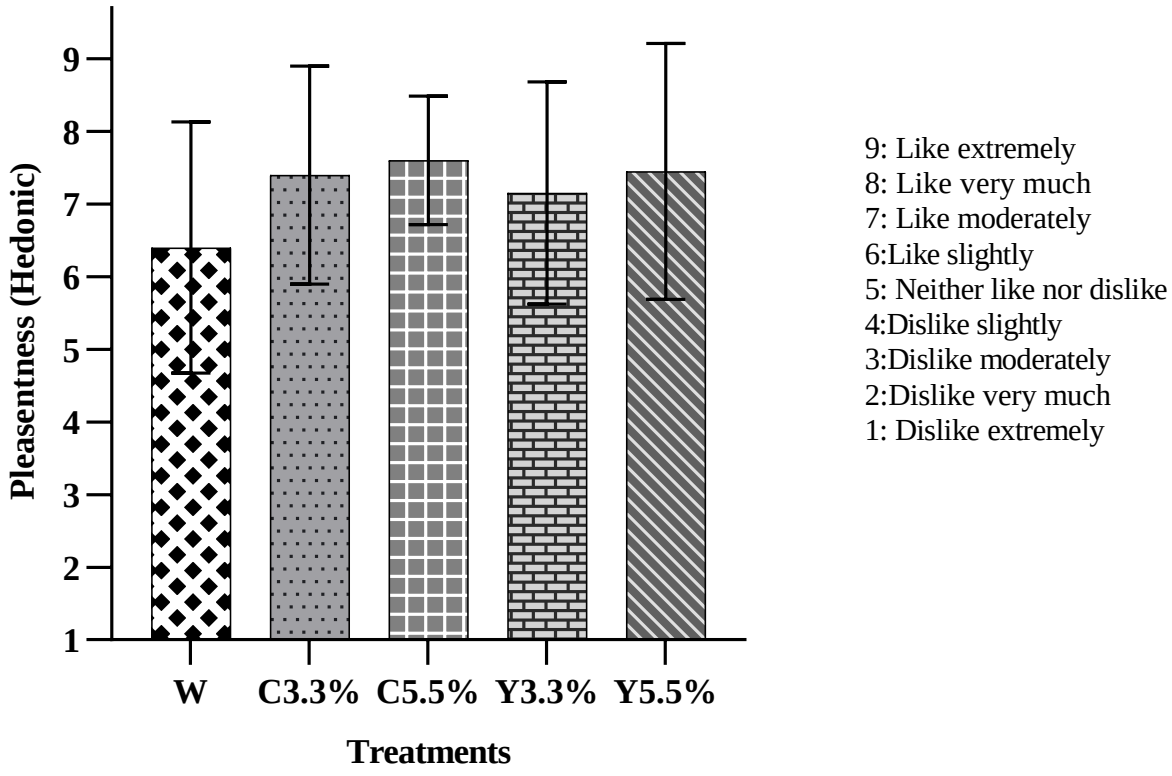
Figure 5. 27 Pleasantness (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test.

There were no significant differences between the treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on pleasantness (Hedonic scale) (P>0.05) (Figure 5.28).

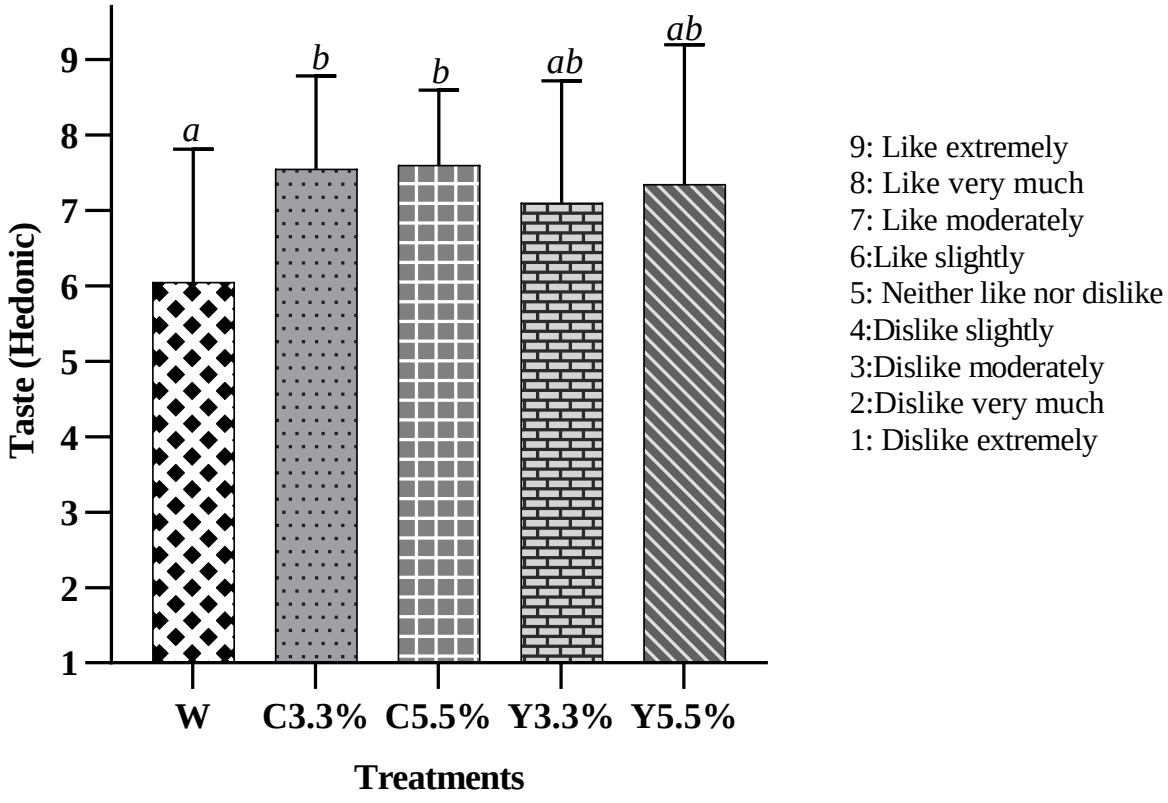
Figure 5. 28 Pleasantness (Hedonic Scale)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test.

There was a difference between the treatment with C3.3%, and C5.5% compared to water control on taste (Hedonic Scale) ($P<0.05$) (Figure 5.29).

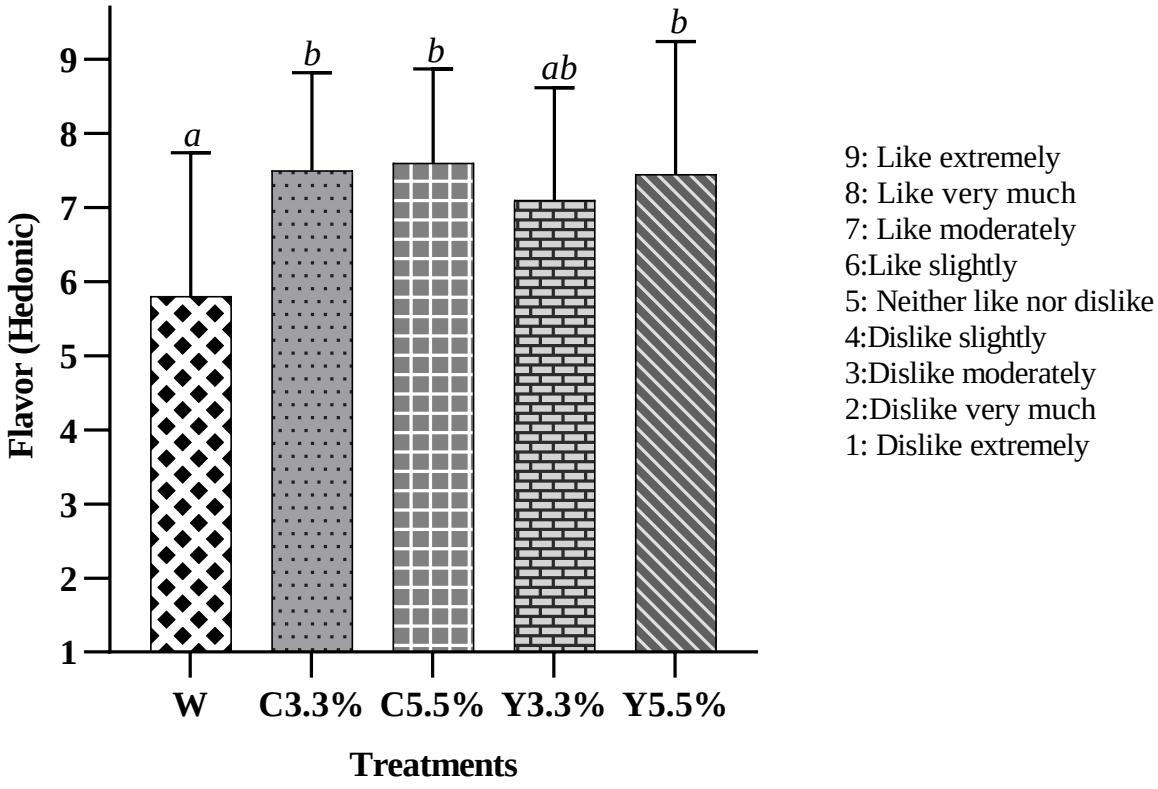
Figure 5. 29 Taste (Hedonic Scale)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a difference between the treatments with C3.3%, C5.5%, and Y5.5% compared to water control on flavor of treatments (Hedonic Scale) ($P<0.05$) (Figure 5.30).

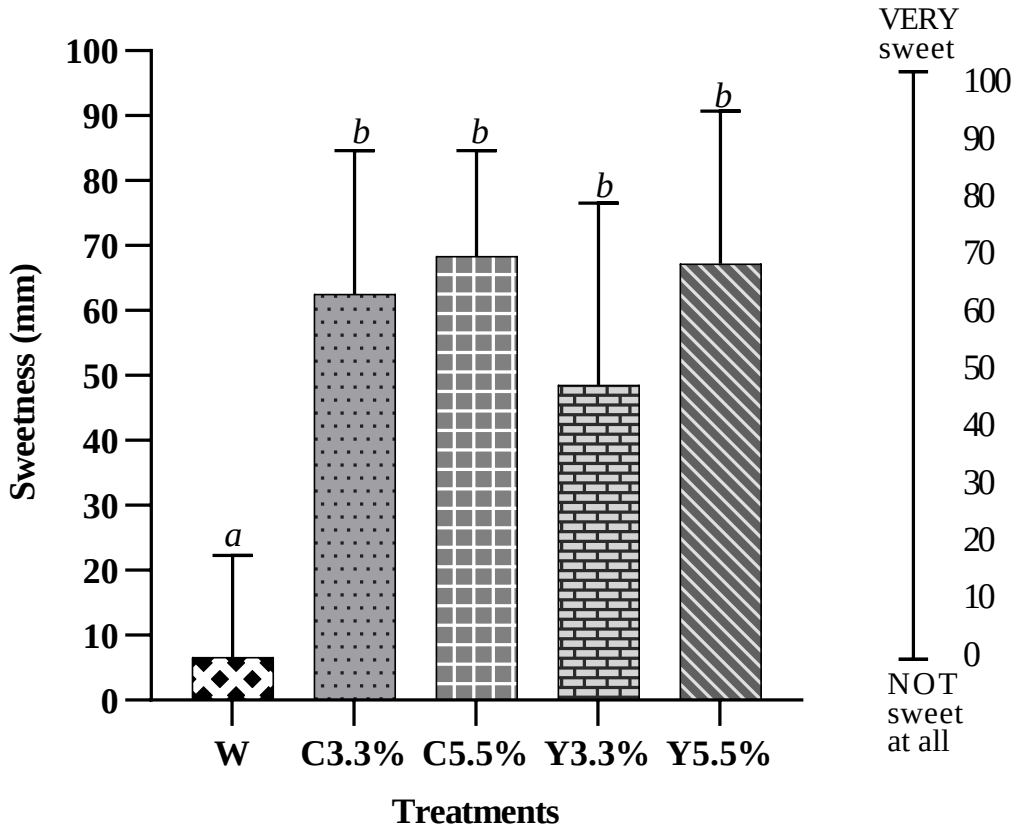
Figure 5. 30 Flavor (Hedonic Scale)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a difference between the treatments with C3.3%, C5.5%, Y3.3%, and Y5.5% compared to water control on sweetness (VAS) ($P<0.05$) (Figure 5.31).

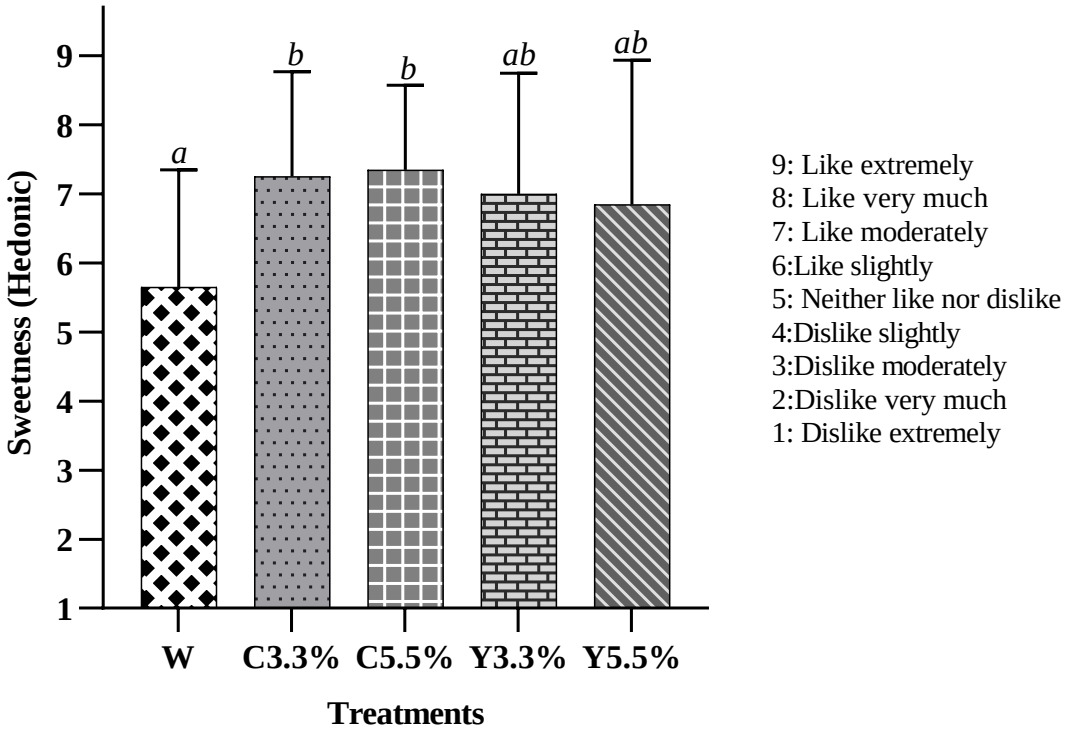
Figure 5. 31 Sweetness (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a difference between the treatments with C3.3%, and C5.5% compared to water control on sweetness (hedonic) ($P<0.05$).

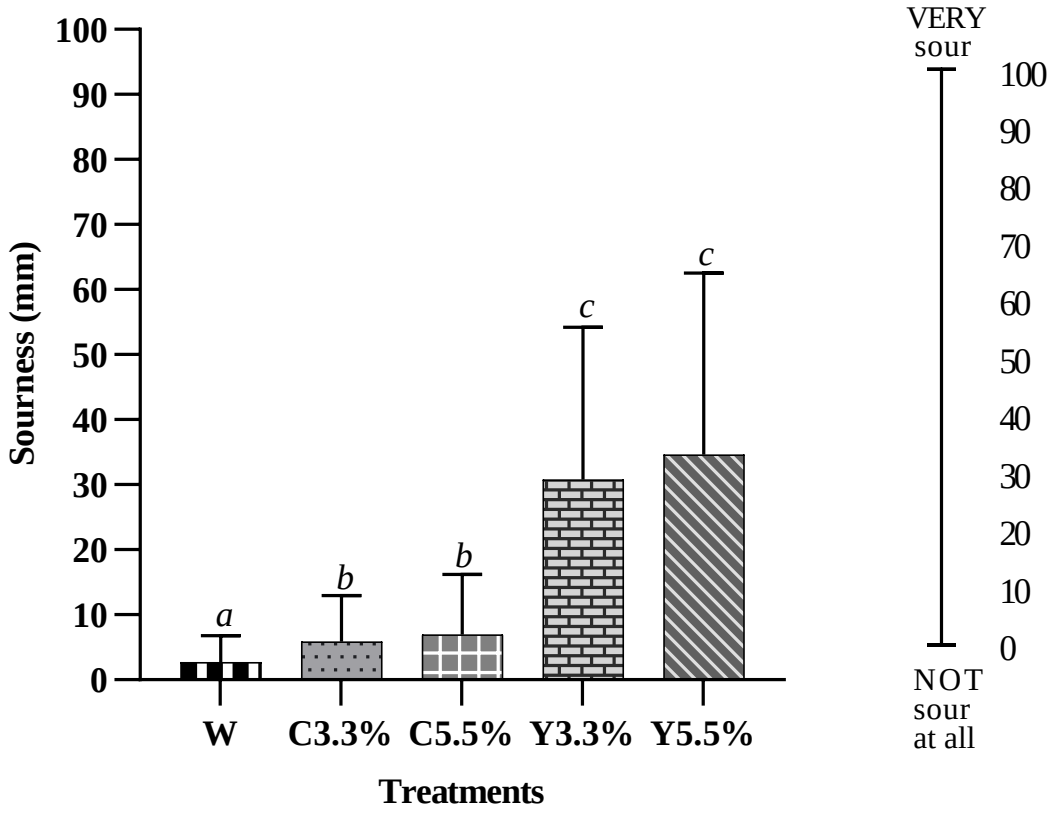
Figure 5. 32 Sweetness (Hedonic Scale)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a significant difference between C3.3%, and C5.5% compared to water control on sourness. There was a difference between Y3.3%, and Y5.5% compared to water control on sourness (VAS) ($P<0.05$) (Figure 5.33). The yogurt treatments, Y3.3%, and Y5.5%, resulted in higher intensity for sourness compared to the chocolate milk treatments, C3.3%, and C5.5% ($P<0.05$).

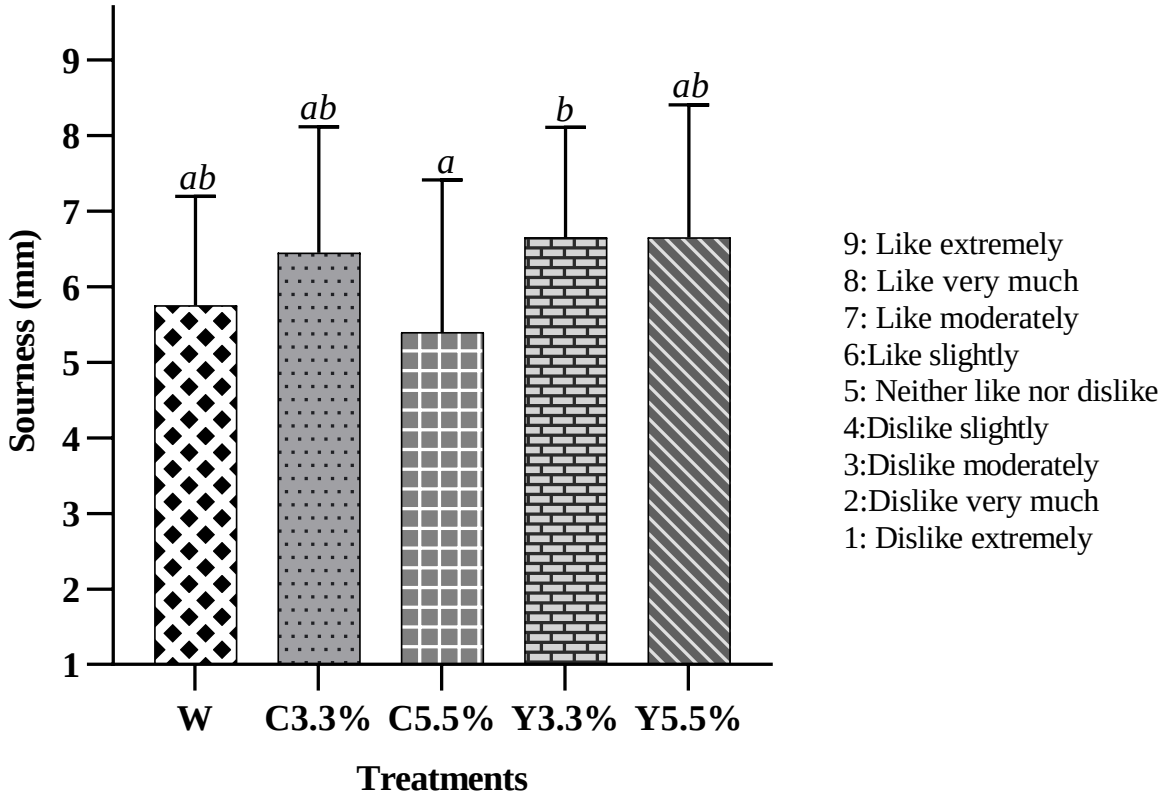
Figure 5. 33 Sourness (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a significant difference between the treatments with C5.5% and Y3.3% on sourness (hedonic) (Figure 5.34).

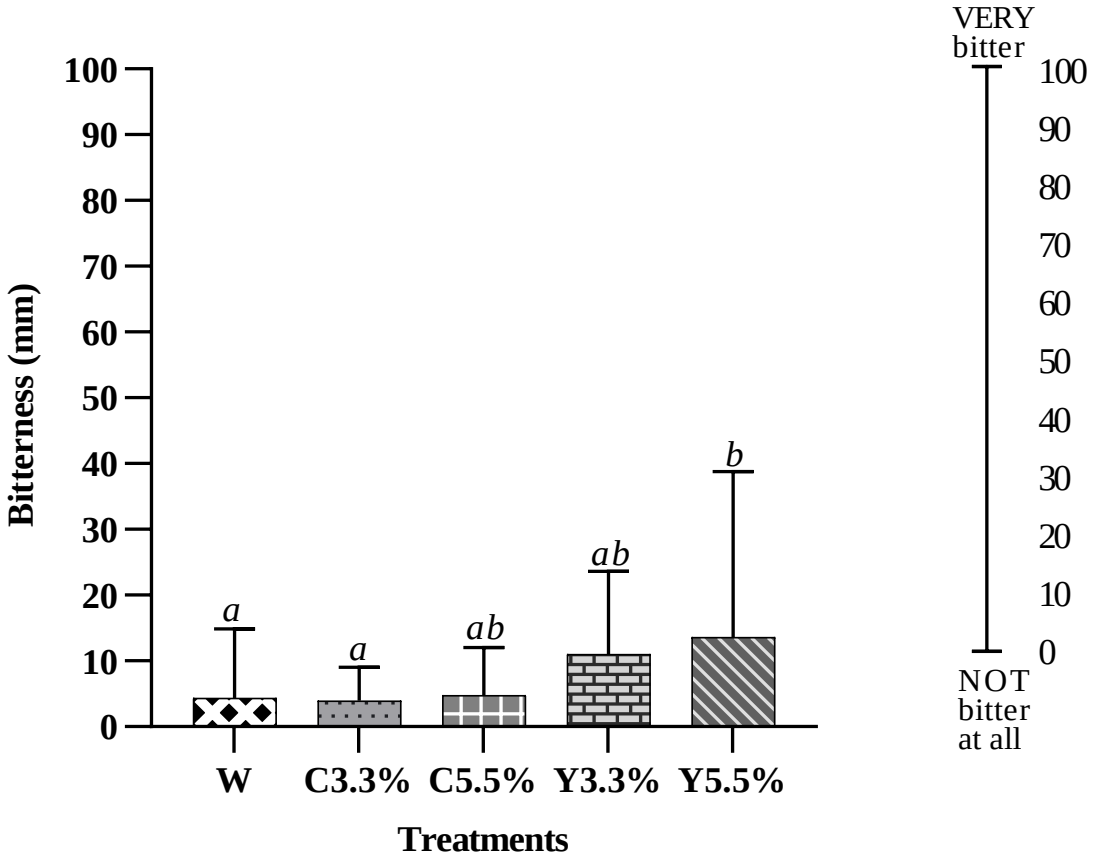
Figure 5. 34 Sourness (Hedonic Scale)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a significant difference between the treatments with C3.3%, and water control compared to Y5.5% on bitterness (VAS) (Figure 5.35).

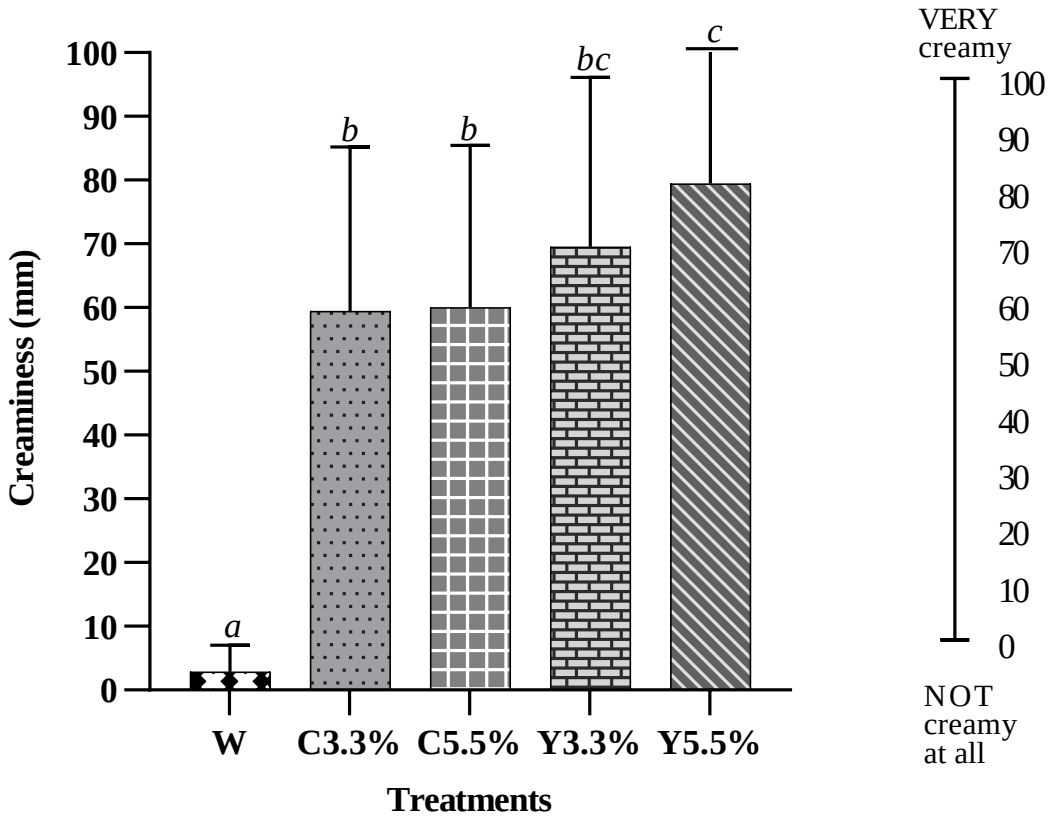
Figure 5. 35 Bitterness (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a difference between the treatments with C3.3%, C5.5%, Y3.3%, and Y5.5% compared to water control on creaminess (VAS) (P<0.05). There was a difference between C3.3%, C5.5% compared to the treatment with Y5.5% on creaminess (VAS) (P<0.05) (Figure 5.36).

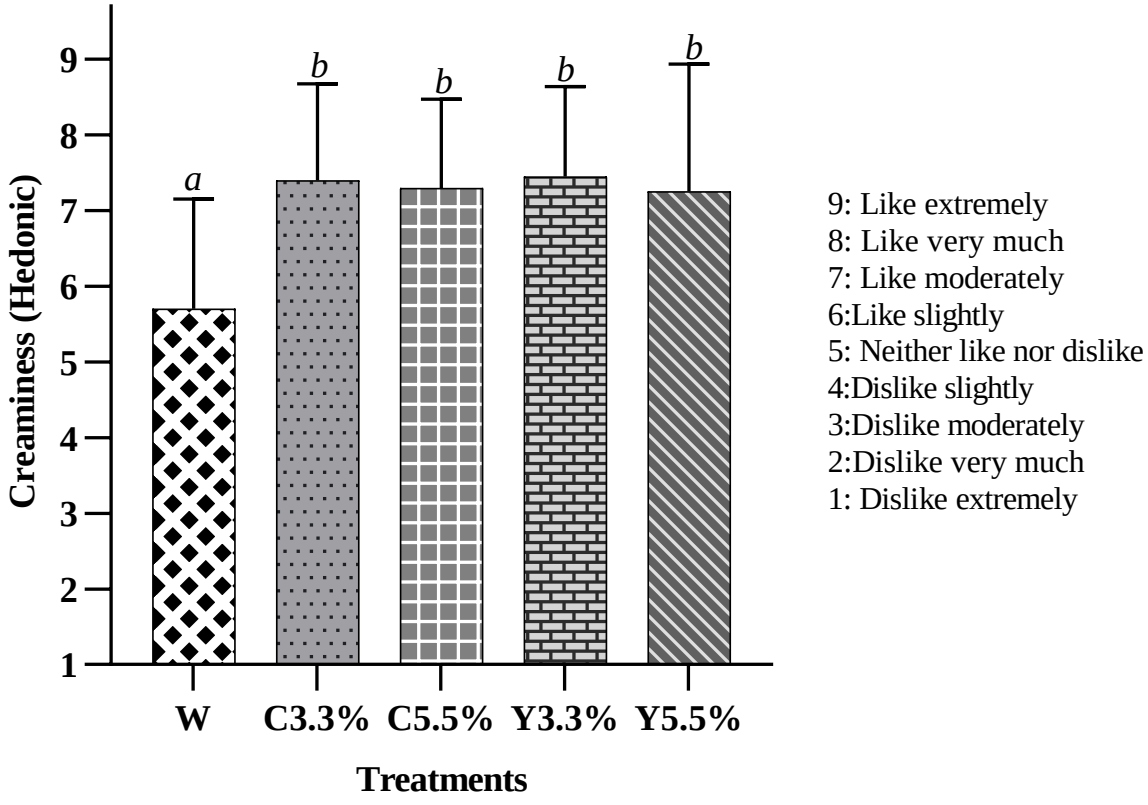
Figure 5. 36 Creaminess (mm VAS)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20. One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a significant difference between the treatments with C3.3%, C5.5%, Y3.3%, and Y5.5% compared to water on creaminess (Hedonic) (Figure 5.37).

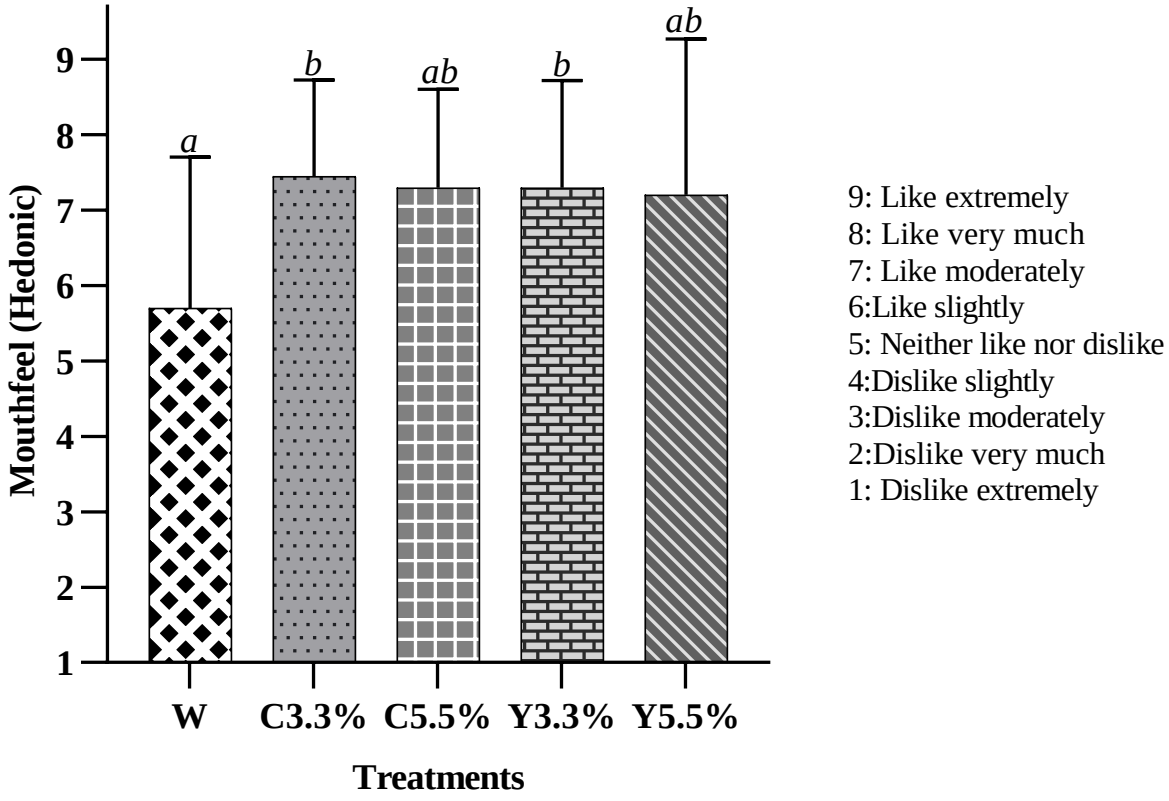
Figure 5. 37 Creaminess (Hedonic)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was a difference between the treatments with C3.3%, and Y3.3% compared to water control on mouthfeel (Hedonic) ($P<0.05$) (Figure 5.38).

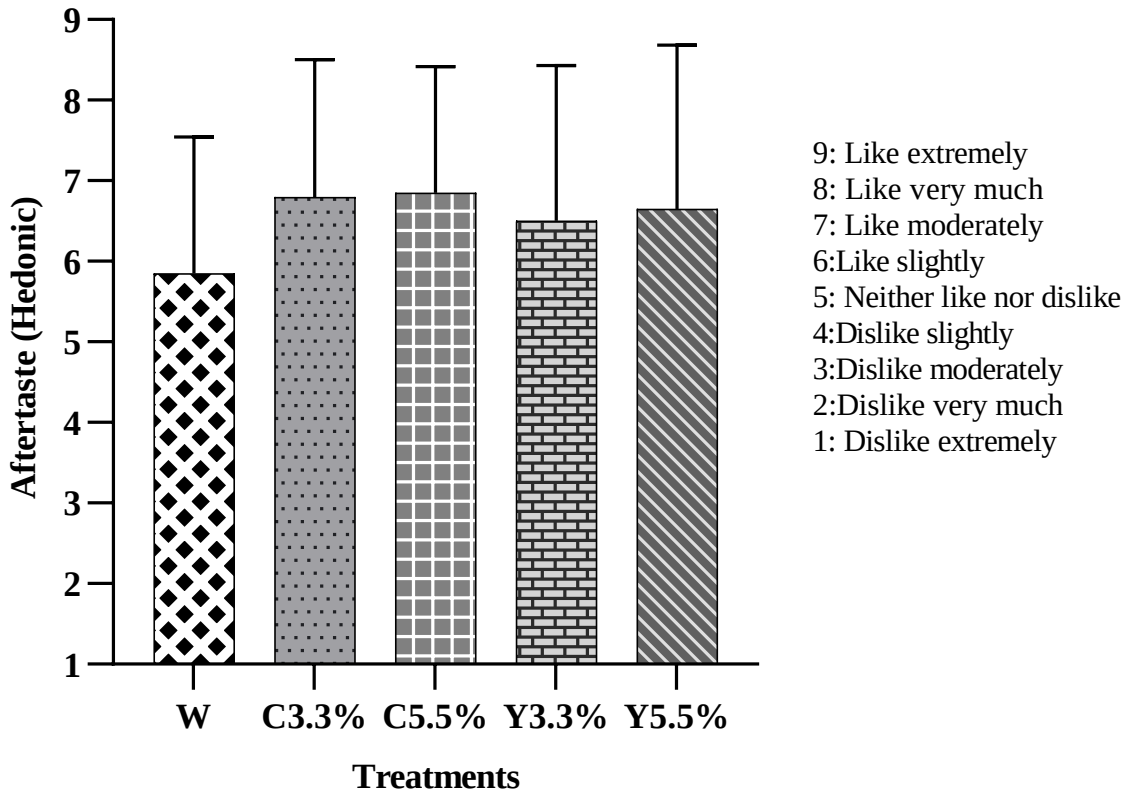
Figure 5. 38 Mouthfeel (Hedonic)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test. Values with different letters are significantly different (P<0.05).

There was no difference between the treatments with C3.3%, C5.5%, Y3.3%, Y5.5% and water control on aftertaste (hedonic scale) (P>0.05) (Figure 5.39).

Figure 5. 39 Aftertaste (Hedonic)



Mean ± SD. Abbreviations: W=water, C3.3%=3.3% added sugar chocolate milk, C5.5%=5.5% added sugar chocolate milk, Y3.3%= 3.3% added sugar yogurt, Y5.5%= 5.5% added sugar yogurt, n = 20., One-way ANOVA with Tukey's-Kramer post-hoc test.

5.7 Relations Among Dependent Measures

A strong positive relationship was found between *ad libitum* food intake and average appetite scores over 120 min (r=0.7, P=0.0002) (Table 5.9). A weak negative relationship was found between subjective feelings of fullness and BG (r=-0.2, P=0.04). No relationship was found between ad libitum FI and BMI (P=0.3); between *ad libitum* FI and FFM (P=0.1); between *ad libitum* FI and BG (0.7); between average appetite score and BG (P=0.9), and between subjective feelings of hunger and BG (P=0.6).

Table 5. 9 Correlation Between the Variables

Variable 1	Variable 2	Pearson's r	P value
Ad libitum Food Intake at 120 min	Average Appetite Score	0.7	0.0002
Ad libitum Food Intake at 120 min	BMI	0.27179	0.3
Ad libitum Food Intake at 120 min	Fat Free Mass	0.38358	0.1
Ad libitum Food Intake at 120 min	Blood Glucose	-0.08534	0.7
Average Appetite Score	Blood Glucose	0.04229	0.9
Subjective Feelings of Hunger	Blood Glucose	-0.05849	0.6
Subjective Feelings of Fullness	Blood Glucose	-0.20830	0.04

Pearson's correlation coefficient test for relationship between variables. P value <0.05 represents significant difference.

6 Discussion

The primary objective of this study was to investigate the effect of partial reduction of added sugar in chocolate milk and yogurt on postprandial blood glucose (BG). We hypothesized that chocolate milk and yogurt formulated with the reduced level of added sugar will result in reduced blood glucose response over two hours. Overall, the partial reduction of added sugar from 5.5% to 3.3% led to a similar postprandial glycemia over two hours; however, the benefits of the sugar reduction were observed at the blood glucose response peak at 30 min showing that the ingestion of yogurt with reduced added sugar content did not result in a significantly higher BG compared to water control. Similarly, BG $iAUC_{0\text{-}120min}$ was not different between Y3.3% and water control; although Y3.3% was not different from other dairy treatments with full and reduced added sugar content. The rationale for the study was to evaluate the risks related to dairy products with added sugar in terms of their effects on blood glucose, satiety and energy intake. In the past decade, the multiple attempts were made to remove SSB, including chocolate milk, from school cafeterias due to their high sugar content (247,248). Also, Dietitians of Canada considered chocolate milk among other SSBs in their position paper on taxation of SSBs (223,224). The chocolate milk may contain up to 6% of added sucrose and sweetened yogurt has added sucrose (~5g per 100g) to tone down the sour taste (249,250). Previous studies completed in our laboratory with chocolate milk and yogurt containing 0%-5.5% added sugar demonstrated that the reduction of added sugar to 3.3% led to the retention of acceptable sensory characteristics of both products in children, adults and senior adults, while any further reduction of added sugar resulted in inferior sensory characteristics (238,251–255). However, it was not clear whether the reduction of added sugar to 3.3% results in any benefits for blood glucose control. The present study clearly demonstrated that there are no risks associated with chocolate milk and yogurt with 5.5% added sugar content: they result in a

similar glycemic response to their counterparts with reduced added sugar, similarly, suppress appetite and do not contribute to an excessive cumulative short-term energy intake.

Health Canada Draft Guidance Document on Food Health Claims Related to the Reduction in Post-Prandial Glycaemic Response specifies that the amounts of reference and test food given in the study must be consistent with its serving size and intended pattern of consumption (246). According to the sections D11 and D12 of the Health Canada's *Table of Reference Amounts for Food*, one serving of chocolate milk is one cup and one serving of yogurt is 3/4 cup (256). The present study compared the effects of one serving of chocolate milk and yogurt. The difference in sucrose between one serving of full and reduced level of added sugar chocolate milk and yogurt was 7g and 4g, respectively. The results of this study indicate that BG response was similar for all treatments with chocolate milk and yogurt. Health Canada recommends using iAUC for the expression of blood glucose response (246). All methods of BG calculation used in the present study including mean 2-h BG, tAUC, iAUC and niAUC resulted in non-significant difference between dairy products with full and reduced added sugar content, although Y3.3% demonstrated additional benefits as discussed above. The maximum difference in sugar content between dairy treatments in the present study was 10g total (sucrose and lactose). The dose of 10 g appears to be insufficient to cause a difference in blood glucose response. In another study conducted by Panahi and colleagues using a similar design, two dairy treatments, infant formula and chocolate milk, did result in significant difference in BG at 30 min; however, the difference in their sugar content was 16g (141). On the other hand, in Panahi's study, there was no difference in BG at 30 min between white milk and infant formula, although the difference in their sugar content was 15g (141). The lack of the difference in BG at 30 min in Panahi's study between white milk and infant formula is possible due to two factors: all sugar in white milk is low-glycemic lactose, and a ×2.4 higher

content of milk protein in white milk, compared to infant formula, that may result in a higher insulin release and utilization of blood glucose. On the other hand, two treatments in Panahi's study, infant formula and orange juice, that had only 6.4g difference in sugar resulted in a significant difference in BG at 30 min (141). These observations suggest that the type and the amount of sugar, as well as the amount of milk protein are the factors that determine postprandial blood glucose. In the present study, the treatments had a similar protein content (7-9g), the maximum difference in total sugar was 18g and in added sugar was 10g, and all treatments included both lactose and sucrose, although in different proportions. While there was no difference in BG between the dairy treatments, the fact that glycemic response was not different between Y3.3% and water suggest that 12g of total carbohydrate, including 6g of added sugar and 6g of lactose, and 7g of milk protein may position yogurt with reduced added sugar content as the product of choice for the individuals required a strict blood glucose control. The other important conclusion drawn from the obtained results is that the reduction of added sugar in chocolate milk and yogurt does not lead to changes in postprandial glycaemia over two hours. However, the reduction of sugar has other benefits such as the reduction of energy density of chocolate milk and yogurt contributing to the lower energy intake.

The secondary objective of this study was to investigate short-term food intake (FI) and subjective appetite after consuming chocolate milk and yogurt with reduced sugar. This study demonstrated that the treatment with C3.3% reduced *ad libitum* FI at 120 min compared to water ($P<0.05$) (Table 5.4). The results also have shown that Y3.3% tended to reduce *ad libitum* FI at 120 min compared to water ($P=0.05$). However, there was no difference between caloric treatments on *ad libitum* FI at 120 min ($P>0.05$). The lack of the difference in *ad libitum* FI at 120 min between caloric treatments might be explained by a two-hour time interval between the ingestion of liquid caloric

treatments and *ad libitum* pizza meal. Panahi and colleagues has shown that liquid treatments affected *ad libitum* FI at 30 min but not at 120 min (141). Rolls and colleagues have demonstrated that between three time intervals (30, 90 and 180 min), the interval between the preload and subsequent FI provides the most accurate caloric compensation (257). Since caloric treatments contained different amount of energy (C5.5% - 177 kcal, C3.3% - 149 kcal, Y5.5% - 139 kcal and Y3.3% - 123 kcal), the cumulative FI was calculated as the sum of the energy consumed with the treatment and pizza meal. There was no difference in cumulative FI between the treatments ($P>0.05$). This is important observation suggesting that a serving of chocolate milk or yogurt consumed two hours before subsequent meal will not contribute to an excessive energy intake. The fact that only two treatments with reduced sugar content, C3.3% and Y3.3%, resulted in a lower *ad libitum* FI at 120 min compared to water control may be explained that the chocolate milk and yogurt with reduced added sugar resulted in 103% and 102.7% caloric compensation, respectively. The caloric compensation after full sugar treatments, C5.5% and Y5.5% was 56.3% and 40.6%, respectively. Similar observation was reported by Woodend and Anderson (2001) in the study with young men who were given drinks (300ml) containing 25, 50, or 75g sucrose (129). The treatment with 25g of sucrose led to 123% caloric compensation compared to 62% and 90% caloric compensation after the treatments with 50 and 75g of sucrose, respectively (125). In the present study, the cumulative energy intake after the treatments with reduced added sugar and water control was the same ($P>0.05$), the consumption of pizza meal was less after C3.3% and Y3.3% than after water control ($P<0.05$). This finding suggests that both chocolate milk and yogurt with reduced added sugar content may provide a better compensation and be products of choice for targeting energy intake.

All caloric treatments resulted in reduced subjective AA over 120 min compared to water control ($P<0.05$). Subjective AA scores did not show significant differences among all treatments until 45 min. At 45 min, the treatments with C3.3% and Y5.5% resulted in lower AA compared to water control ($P<0.05$). At 60 min, all treatments, except C5.5%, resulted in lower AA than water control ($P<0.05$). The lack of the difference in AA at 15-30 min period could be explained by the slow digestion of casein which is a main protein (80% of total protein) in chocolate milk and yogurt; thus, it takes longer time for casein to be digested to peptides and amino acids in order to trigger satiety signals (258–260). There was a strong relationship between subjective AA over 120 min and *ad libitum* FI at 120 min ($r=0.7$, $P=0.0002$). A serving of water was significantly less filling than a serving of C3.3% ($P=0.001$) and Y3.3% ($P=0.01$) over 120 min, and *ad libitum* FI at 120 min compared was also higher after water control compared to C3.3% ($P=0.02$) and Y3.3% (*$P=0.05$). Although, subjective AA was higher after water control compared to the treatments with C5.5% ($P=0.02$) and Y5.5% ($P<.0001$), there was no difference between C5.5% and Y5.5% for *ad libitum* FI at 120 min.

Previous experimental studies mentioned that subjective appetite is not always a reliable predictor of food intake (140,251). Similarly, *Health Canada Draft Guidance Document on Satiety Health Claims on Food* stated that although visual analogue scales have an acceptable degree of reproducibility, when used within a single eating occasion, they might not always correlate with food/energy intake at the next meal (114,239). Although appetite-related sensations do not always predict the amount of energy consumed at the next meal (114), the current study demonstrates the strong positive correlation between subjective AA over 120 min and *ad libitum* FI at 120 min.

This subjective feelings of thirst over 120 min did not differ between the treatments ($P>0.05$). This could be explained by liquid and semi-solid consistency of chocolate milk and yogurt, respectively.

It was reported that water suppressed thirst more effectively than sucrose-sweetened lemonade containing 20g of sucrose per 8 oz (127); however, this dose of sucrose was higher than the dose of sucrose containing in the treatments used in the present study. On the other hand, water intake there was higher after the treatments with C5.5% and Y5.5% compared to control and C3.3%. It is known that the taste is a sensory component of thirst (261). The negative correlation was shown between the intensity rating of thirst and food intake (262). The observed discrepancy between the thirst sensation and actual water intake in this study requires further investigation.

All treatments resulted in a similar subjective rating of energy, and fatigue ($P>0.05$). Physical comfort parameters such as the subjective feelings of wellness, diarrhea, gas, stomach pain and nausea did not differ among the treatments at any individual time points over 120 min ($P>0.05$). It is also important to note that lactose intolerant individuals were not included in the study. Therefore, the subjective ratings of gastrointestinal discomfort were low after all treatments. Mather and colleagues showed that the caloric treatments consumed at the breakfast resulted in a higher perceived energy, lower feelings of tiredness, higher perceived wellness and lower feelings of stomach pain compared to water in young females (263). The caloric treatments in Mather's study were either dairy or non-dairy-based breakfast meals formulated with granola cereal. In contrast, the liquid (i.e., chocolate milk) and semi-solid (i.e., yogurt) treatments were employed in the present study. Therefore, not only nutrient composition, but also the texture and consistency of the food may have also played a role in the perception of physical comfort feelings.

The results of the food acceptance showed no difference between the dairy treatments. These results reproduce our previous studies that demonstrated that the reduction of added sugar from 5.5% to 3.3% did not result in a lower acceptance of chocolate milk and yogurt (253,254). However, the novel component of this study is that the participants rated their sensory perception

after consuming the whole serving of a product, while participants tested a small volume of a food sample in previous studies (253,254).

6.1 Limitations

There are several limitations of this study. The time span for assessment of appetite and subsequent food intake was two hours as the study was following the recommendations set in Health Canada's guidelines on food claims for satiety and the reduction of postprandial glycaemia (114,246). However, considering the fluid and semi-solid state of chocolate milk and yogurt, their effect on FI could be more pronounced in the first 30-60 min after ingestion. Another limitation is that the serving size and macronutrient composition of the treatments was different; however, the goal of this work was to compare the effect of a single serving of each product on BG, satiety and FI. Finally, a sample size of less than 40 showed less repeatability in food sensory studies (264,265).

7 Future Directions

This study investigated the short-term effects of partially reduced sugar in chocolate milk and yogurt. This study suggests further studies are required to investigate long-term effects as commercially available chocolate milk and yogurt products might not be as harmful as thought before banning the flavored dairy products from high schools. Additionally, further studies are required with focusing on more serving size of treatments because the energy and sugar levels of treatments in study was similar to each other.

One of unexplored and potential areas for this research is to investigate the combined effect of dairy products with reduced sugar content and other foods high in available carbohydrate on postprandial glycaemia, appetite, and food intake. Potentially, both chocolate milk and yogurt with reduced sugar content may improve glycaemic control after the consumption of meals high in glycaemic carbohydrates (sugars and starches). Another unexplored area is to explore the effect of dairy products with reduced sugar on physical performance, including exercises of various intensities. Finally, the long-term effects of dairy products with reduced sugar content need to be explored in various groups of the population, including those with metabolic disorders such as overweight and obesity, and T2M.

8 Conclusion

The treatments with chocolate milk and yogurts with full and reduced added sugar content result in a similar glycaemic response over two hours. However, the reduction of added sugar in yogurt results in BG response similar to water control and tended to lower *ad libitum* FI compared to water control, while chocolate milk with reduced added sugar content results in a lower *ad libitum* FI compared to water control. Both chocolate milk and yogurt with reduced sugar content similarly suppress subjective appetite over two hours as their full sugar counterparts, and do not cause any physical discomfort. The reduction of added sugar does not negatively impact the sensory properties of chocolate milk and yogurt. Although the reduction of added sugar in chocolate milk and yogurt does not cardinally impact postprandial glycaemic response, both chocolate milk and yogurt with reduced added sugar content possess with unique metabolic characteristics that may position them as potential functional products for metabolic control.

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Appendices

Appendix 1: Recruitment Letter

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

Dear Participant,

A team of researchers from Mount Saint Vincent University are investigating how dairy products influence the blood sugar level and appetite.

We are asking for male and female participants 19-35-year-old to take part in a research study on four separate days one-week apart. Each session will take up to three hours of your time and if you will participate in the study you will be asked to come to our laboratory at the same time for each of four research sessions.

If you are interested in participating in this study, please contact us through the e-mail or telephone and we will contact you and ask some questions to determine if you are eligible to participate in this study. You will be also invited to attend the Information and Screening session to learn more about the study and complete the questionnaires about your food and eating habits. We will also measure your weight, height and body composition using an analyzer similar to electronic body weight scale. This will not hurt you and will not cause any pain or discomfort. Once we determine that you are eligible to participate in this study, we will schedule you to attend from four up to eight research sessions.

The study will take place in the food research laboratory in the Centre of Applied Research, Department of Applied Human Nutrition (47 College Rd) at MSVU.

There are criteria for participation that you need to be aware of, the participant needs not to be:

- Breakfast skipper
- Smoker (including e-cigarettes) / cannabis consumer
- Be underweight, overweight or obese
- Have chronic diseases including diabetes
- Taking certain medication
- Have lactose intolerance, food allergies or gastrointestinal disorders (e.g., irritable bowel syndrome, or others).

We will review these conditions with you over the telephone and will measure your body weight and height during the Information and Screening session.

To thank you for your participation you will receive $30 for each session with a total of $120 for four sessions. At the end of each research session, you will be served pizza and chocolate milk or fruit juice.

The ethical components of this research study have been reviewed by the University Research Ethics Board and found to be in compliance with Mount Saint Vincent University's Research Ethics Policy.

If you would like to participate, or to get more information about this study, please contact Appetite.Study@msvu.ca or leave a message at 902-457-6568 and we will contact you.

Thank you for your support in this research.

Sincerely,

Dr. Bohdan Luhovyy

Appendix 1a: Recruitment Poster

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

MOUNT SAINT VINCENT UNIVERSITY

WOULD YOU LIKE TO PARTICIPATE IN A RESEARCH STUDY AT THE MSVU APPETITE LAB?

The purpose of this study is to learn more about chocolate milk and fermented dairy products and their effect on blood sugar and appetite.

Participants must be:
- Ages 19-35 years old
- Healthy males and females
- Non-smokers
- Not following any food restrictions or not have any food allergies or intolerances

YOUR PARTICIPATION INVOLVES:

-INFORMATION SESSION, 5 SESSIONS OVER 5 WEEKS, 3 HOURS PER SESSION, BLOOD COLLECTION, EATING SNACKS AND PIZZA AT EACH SESSION

AS A THANK-YOU, PARTICIPANTS WILL BE GIVEN $30 FOR EACH SESSION FOR A TOTAL OF $150 FOR THE FULL STUDY. AFTER EACH VISIT, WE WILL SERVE YOU PIZZA, CHOCOLATE MILK, AND FRUIT JUICE.

IF YOU'RE INTERESTED IN PARTICIPATING, PLEASE EMAIL APPETITE.STUDY@MSVU.CA OR LEAVE A MESSAGE AT 902-457-6568 AND WE WILL CONTACT YOU

Appendix 2: Telephone Screening Questionnaire (part 1)

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

TO BE KEPT SEPARATELY FROM DATA FORMS

Name: _____

Month and year of birth _____/_____ (_____ y: calculated by recruiter)

To be completed by Staff: Eligible to participate: Yes ____ No____

If not eligible, this concludes the conversation and the form is to be shredded.

If eligible, continue with the remaining parts 1 and 2.

Address:

_____

_____

Cell phone: _____ Can we send text messages? _____ Yes _____ No

Home phone: (_____) _____ What time would be convenient to call you? _____

E-mail: _____@_____

Participant ID assigned: _____

Appendix 2a: Telephone Screening Questionnaire (part 2)

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

ID: _____

Weight (circle the correct unit):_____ lbs kg Height:_____cm (Calculated by recruiter) BMI:_____ kg/m^2

Do you regularly consume breakfast? Yes__ No___

Are you physically active? Yes ___ No___

Can you briefly recall your daily normal physical activity routine?

_____

_____

_____

Do you consume cannabis? Yes___ No___ Do you smoke tobacco? Yes__ No__

Do you have any allergies to any foods? Yes __ No__

If yes, are you allergic to any of the following wheat, lentil, dairy, eggs, or other foods? Yes ___ No___

Other foods? _____

Are you on a special diet? Yes ____ No____ If yes, please specify: _____

How many alcoholic beverages do you consume per day? _____ per week? _____

Do you have any major disease or medical conditions? Yes ____ No____ If yes, please specify:

_____

Do you take medications? Yes ____ No____ If yes, please specify:

_____

Do you like snack bars? Yes ____ No____

Do you like pizza? Yes ____ No____ If yes, please rank these types of pizza with (1) the first choice, (2) second choice, and (3) third choice: Pepperoni (Cheese, pepperoni) _____, Deluxe (cheese, pepperoni, peppers, mushrooms) _____, Three Cheese (mozzarella, cheddar, parmesan) _____

To be completed by Staff: Eligible to participate: Yes ____ No____

Screening scheduled at: _____

Appendix 4: Information Sheet and Consent Form

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

Investigators:

Dr. Bohdan Luhovyy, Principal Investigator

Dr. Priya Kathirvel, Co-Investigator, and Dr. Maryam Akbari, Post-Doctoral Fellow

Department of Applied Human Nutrition, Mount Saint Vincent University

E-mails: bohdan.luhovyy@msvu.ca ; priya.kathirvel@msvu.ca ; maryam.akbari@msvu.ca

Students and Research Assistants:

Ms. Duygu Gunaydin, Research Assistant, MSc Student

Ms. Emily MacEachern, Research Assistant, MSc Student

Ms. Megan Churchill, Research Assistant, BSc AHN Honours Student

Ms. Devanshi Desai, Research Assistant, MSc Student

Mr. Tongtong Li, MSc Students

E-mails: duygu.gunaydin@msvu.ca ; emily.maceachern@msvu.ca; tongtong.li1@msvu.ca ; devanshi.desai@msvu.ca ; megan.churchill@msvu.ca

Introduction:

You are invited to participate in the research study listed above. This form provides you with information about the study so you can make an informed decision about if you would like to participate. It will enable you to understand the purpose of the study, the risks and benefits of participating, and what you will be asked to do should you choose to participate. We will keep you informed of any new information that may influence your willingness to continue to participate in the study. A member of the research team will be available to answer any questions you may have. You may decide not to participate, and you may withdraw from the study at any time. Your participation in the study is entirely voluntary.

Funding Source:

Funding for this project is provided by the grants from Dairy Farmers of Canada and Natural Sciences and Engineering Research Council.

Background and Purpose of Research:

Similar to regular milk, chocolate milk and yogurt contain many nutrients such as protein, fat, Vitamin D, Vitamin A, phosphorous and calcium. These nutrients play a role in development and bone health of children and adolescents as it helps to prevent osteoporosis later in life. Although chocolate milk and yogurt provide these benefits, they also contain added sugar not seen in regular milk. The extra calories the sugar adds to the diet and the higher level of sugar intake has been linked to concerns related to obesity and higher risks for certain diseases. Given the risks, previous researchers looked into how removing chocolate milk from school

cafeterias have affected the amount of milk students drink to find that students were drinking less milk. Based on the benefits of drinking chocolate milk versus the disadvantages of not drinking milk, this study aims to look for a way to improve the healthfulness of chocolate milk and yogurt. Different formulations of chocolate milk and yogurt that differ in sugar and/or protein content will be looked at to explore the differences in pleasantness, taste, sweetness, bitterness, creaminess, mouthfeel, flavour, texture and aftertaste. We will also explore whether the reduction of added sugar will result in improved blood sugar and hormone insulin that regulates blood sugar in the human body. The results from this study may help the dairy industry in their development of flavoured milk products such as chocolate milk and yogurt with lower sugar content.

This study will not cost you anything. This study has two experiments aimed at the investigation of (1) chocolate milk and yogurt products varying in their sugar content, yogurt products varying in their sugar, and (2) chocolate milk with added milk proteins. Each experiment includes five sessions. **You may participate in one or both experiments**. This study is funded by NSERC (Natural Sciences and Engineering Research Council of Canada) and Dairy Farmers of Canada. There is no conflict of interests between the investigators and the sponsors.

Invitation to Participate:

You are being invited to take part in this study. If you chose to take part and meet eligibility criteria, you will be asked to drink a treatment (e.g., chocolate milk), or plain water five times (five sessions) with each session one week apart. At each session, your blood will be collected by a registered nurse, and your appetite will be measured using simple scales after eating the treatment. Each session will take up to three hours of your time.

Eligibility:

To participate in this study, you must be considered overall healthy and not being underweight, overweight or obese, and not having any diseases. You must also be between the ages of 19 and 35. You must be a non-smoker (including e-cigarettes), and you cannot be taking certain medications and consume cannabis products. You will not be able to participate if you have lactose intolerance, allergies to any food or if you usually skip breakfast. To find out if you can take part in this study, you will be asked to fill out questionnaires, which ask questions about your age, if you smoke, exercise, your health, if you are on any medications, and your eating habits. Your height and weight will be measured. Female participants will be asked to attend experimental sessions during the same time of their menstrual cycle (i.e. follicular phase) and, therefore, will be asked to provide information about their menstrual cycle at the screening session. The study will take place in the Department of Applied Human Nutrition, Room 211, in the Centre of Applied Research, Mount Saint Vincent University, 47 College Rd, Halifax, NS.

Procedure:

You will be asked to fast for at least 10 hours overnight before each session and arrive at our laboratory between 8 am and 10 am in the morning. However, you may drink water until one hour before coming to the laboratory. Please note that you will have to arrive at exactly the same time at each of five sessions. Upon arrival, we will ask you about your recent food intake, stress level, sleep, and physical activity. You will then be asked to have a blood sample taken to measure your fasting blood glucose levels. A registered nurse will insert a tiny cannula (tubing) into your arm vein, so we will not need to poke you every time we need to take a blood sample. This cannula will stay in your vein for two hours. If your blood glucose is higher than a normal fasting level (higher than 6 mmol/L), we will repeat the test within 30 min, and if it still is high, then we will need to reschedule the session. Similarly, if you had experienced stress, unusual food intake, alcohol consumption, did not sleep enough, or had extra physical activity in the day before your session, we will need to reschedule your session as well. If your fasting blood glucose level is normal, and you did not experience any unusual stress, sleep deprivation, extra physical activity, excessive food intake, and alcohol consumption in the previous day, the session will commence. We will ask you to complete the questionnaire related to your appetite and physical comfort, and then will serve you with a dairy product or just a glass of water (in one session). You will have up to 12 minutes to consume your food and water and evaluate the taste. You will be asked to consume the whole amount of a dairy product or water provided. Then you will be taken back to the Appetite Lab, and you can read, study, or use your computer during the next 112 minutes. Please note that you may not browse any content

related to food or eating. The registered nurse will be taking seven blood samples over the 2 hours, at 0, 15, 30, 45, 60, 90, and 120 minutes. Our research assistants will ask you to complete the same questionnaires related to your appetite, physical comfort and energy and fatigue at the same time points. At 120 min, we will ask you to proceed to the feeding cubicles and will serve you with a pizza meal. You will be asked to eat until you feel comfortably full. The new tray with sliced pizza and a new bottle with spring water will be provided every 8 minutes, and the previous tray and water bottle with the leftover (if any) will be taken back by our research assistant. Once you completed your meal, we will ask you to fill out the questionnaire rating your appetite and physical comfort. Then you will be offered to drink a glass of chocolate milk or fruit juice of your choice, and you are free to go.

An Example of a Potential Time and Activity Schedule for Each Session:

Time	Activity
8:45	Arrive at the laboratory
8:50	Fill in COVID-19 assessment and contact tracing forms, take a finger prick blood sample, Sleep, Stress, and VAS questionnaires, IV insertion and baseline blood collection
9:00 - 9:08	Eat the treatment (0 min)
9:15 - 11:00	Fill out VAS questionnaires and blood collection at 15, 30, 45, 60, 90 and 120 min
11:00 – 11:20	Eat the lunch meal until you feel comfortably full
11:20	The session is completed. You will receive a glass of chocolate milk or fruit juice

VAS= Visual analogue scale

Voluntary Participation and Early Withdrawal:

Participation in this study is voluntary. You may choose to stop being in the study at any time without any negative consequences.

Risks:

There is minimal risk of food poisoning. All of the foods that you will be asked to consume will be prepared using practices to ensure food safety. Therefore, your risk of developing a foodborne illness from participation in this study is very minimal. All blood samples gathered will be done so by a trained Nurse, and all samples will be collected using aseptic techniques in a hygienic environment. Each sample will be 5 ml, for a total of less than 35 ml, far less than that taken when donating blood, which is 450 ml in one session: https://blood.ca/en/blood/donating-blood/donation-process . The intravenous blood draw may cause some bruising (dark blue mark) or hematoma (accumulation of blood deep inside) at the venipuncture site, but it is usually harmless and recovers within a few days. After the overnight fast, you may feel faint or dizzy; however, the risk of this is minimal. If this happens and you feel it will be unsafe to travel the session will be rescheduled.

There is minimal risk of COVID-19 exposure. Social distancing will be followed at all times except during blood withdrawal.

Though COVID-19 poses health risks, the chances of a participant or researcher being infectious/infected is low due to Nova Scotia's low case count, quarantine procedures, and adjusted research protocols.

COVID-19 Safety Measures:

Due to the novel Coronavirus (COVID-19) and the pandemic it has caused, increased safety measures have been put in place to minimize risk. Social distancing will be adhered to at all times, except during blood collection. A mask must be worn at all times inside the building, except when in the sensory booth. You will be alone in the sensory booth and the booth will be sanitized between use. When you arrive at MSVU you will be met by a researcher who will guide you to where the study will take place. Once the study is completed a researcher will guide you back to the buildings entrance.

Benefits:

You will not benefit directly from taking part in this study. However, the study results will advance nutritional science and may lead to practical dietetic recommendations.

Confidentiality and Privacy:

Confidentiality will be respected, and no information that shows your identity will be released or published without your permission unless required by law. Your name, personal information, and signed consent form will be kept in a locked filing cabinet in the investigator's office. Your results will not be kept in the same place as your name. Your results will be recorded on data sheets and in computer records that have an ID number for identification, but will not include your name. Your results, identified only by an ID number, will be made available to the study sponsor if requested. Only study investigators will have access to your individual results. If you withdraw from the study, your consent form and any other paperwork associated with your ID number will be destroyed.

Publication of Results:

The results of the study may be presented at scientific meetings and published in a scientific journal. If the results are published, no information about individuals will be reported.

New Findings:

If anything is found during the course of this research, which may change your decision to continue, you will be told about it.

Incentive for Participation:

You will be paid $30 per experimental session with a total of $150 for the completed experiment. The payment will be in the form of cash. You can be paid either after each session or after the end of the study. Please note that in order to process your compensation, the collection of your personal information, including your full name, current mailing address and signature, is required by the university's financial services department. This information is collected, stored and accessible to the financial services department alone other than the initial collection and is not linked to the current study in any way.

Voluntary Participation and Early Withdrawal:

Your decision to take part in this study is entirely voluntary. We hope that you will finish all sessions; however, you may choose to stop participating in the study at any time without any consequences, and you will be paid for sessions you have completed. The study staff may also withdraw you with a provided explanation if she feels that participation is no longer in your best interest, or if you fail to follow the directions of the staff. If you choose to participate, you will agree to cooperate entirely with the visit schedule and will follow the study staff's instructions. Should you wish to withdraw from the study, you must inform us via email Appetite.Study@msvu.ca or leave a message at 902-457-6568.

Injury Statement:

If you begin to feel sick following participation in the study, please seek medical advice as soon as possible. We will provide your medical specialist with information about the food you have consumed during the session.

You can send the email to Dr. Luhovyy (Bohdan.Luhovyy@msvu.ca), call his cell phone 902-221-3810, and leave your question and contact information, and we will contact you back at the earliest convenience.

Rights of Participants:

Before agreeing to take part in this research study, it is important that you read and understand your role as described here in this study information sheet and consent form. You waive no legal rights by taking part in this study. If you have questions about how this study is being conducted and wish to speak with someone not involved in the study, you may contact the Chair of the University Research Ethics Board (UREB) c/o MSVU Research Office, at 457-6350 or via e-mail at research@msvu.ca

The ethical components of this research study have been reviewed by the University Research Ethics Board and found to be in compliance with Mount Saint Vincent University's Research Ethics Policy.

Voluntary Participation and Early Withdrawal:

Participation in this study is voluntary. You may choose to stop being in the study at any time without any negative consequences. If you wish to withdraw simply inform a member of the research team and you are free to leave.

Dissemination of findings:

A summary of results will be made available for you to pick up in one year after the study is done.

Copy of informed consent for the participant:

You will be given a copy of this informed consent to keep for your records.

Consent form

TO BE KEPT SEPARATELY FROM DATA FORMS

Study title: The Sensory and Metabolic Effects of Functional and Value-Added Dairy Products

Participant ID assigned: _____

PARTICIPANT AUTHORIZATION:

I have read or had read to me this information and authorization form and have had the chance to ask questions which have been answered to my satisfaction before signing my name. I understand the nature of the study, and I understand the potential risks. I understand that I have the right to withdraw from the study at any time without any problems. I have received a copy of the Information and Authorization Form for future reference. I understand that to receive compensation, and I will need to provide personal information including full name, current mailing address, and a signature on the compensation form, which will be returned to the university's financial services department. I freely agree to participate in this research study.

Would you like to receive a summary of the results when they are available? Yes ___ No ____.

Would you like to be contacted for future research? Yes ___ No____.

Name of Participant: (Print) _____

Date: _____ Time: _____ Participant ID: _____

Sex _____ M _____ F, Age ____ (y). Month and year of birth (mm/yyyy) _____

If you would like to receive the summary of the results and/or be contacted for future research, please print your address below:

_____

Participant Signature: _____

Date: _____ Time: _____

STATEMENT BY PERSON PROVIDING INFORMATION ON STUDY AND OBTAINING CONSENT

I have explained the nature and demands of the research study and judge that the participant named above understands the nature and demands of the study. I have explained the nature of the consent process to the participant and judge that they understand that participation is voluntary and that they may withdraw at any time from participating.

Name: (Print) _____ Signature: _____ Position: _____

Date: _____ Time: _____