The Acute Metabolic Effects of Different Post-Exercise Supplements in Young Adults: Implications for Glycemic Control, Insulin Response, and Lipid Mobilization

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Abstract

Exercise induces acute metabolic adaptations that can improve glycemia, insulin sensitivity, and lipid metabolism. However, these responses may be differentially modulated by post-exercise nutrition, and wholefood dairy products are of particular interest. This randomized crossover trial examined whether consumption of 4 separate and different supplements: milk, Greek yogurt (GY), carbohydrate (CHO) or water, following a standardized high-intensity multi-modal exercise bout, differentially influenced acute metabolism in 20 healthy young adults. Plasma glucose, insulin, homeostatic model assessment of insulin resistance (HOMA-IR), and glycerol were assessed over 24h. At 1h post-exercise, insulin followed a hierarchy of CHO>GY>MILK>WATER, and GY elicited lower glucose concentrations than all other conditions. At 24h post-exercise, both dairy conditions exhibited lower HOMA-IR than CHO (males only), whereas WATER sustained lipid mobilization 1h post-exercise. Thus, post-exercise nutrition differentially influences metabolic responses. Wholefood dairy products may favourably improve glycemic control (primarily GY) and insulin sensitivity, whereas water preserves exercise-induced lipid mobilization.

Dedication

To my mother, Natalie, whose drive and perseverance have always inspired me, whose support fostered my passion for science growing up, and who sacrificed and gave me everything so I could pursue an advanced education in the sciences.

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Statement of Contributions

This thesis presents original research based on a secondary analysis conducted by the author, Monika Nguyen, under the supervision of Dr. Andrea Josse. The study was conceived and designed by Dr. Andrea Josse, with contributions from members of the research team to study development, protocol refinement, ethics approval, and pilot testing.

The author (MN) contributed to participant recruitment, familiarization and data collection; conducted the laboratory analyses, data processing, and statistical analyses; and interpreted the findings of the metabolic outcomes presented herein. MN also prepared the data figures and tables; and wrote this thesis.

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List of Abbreviations

AMPK	AMP-Activated Protein Kinase
ANOVA	Analysis of Variance
ATP	Adenosine Triphosphate
AUC	Area Under the Curve
%BF	Body Fat Percentage
BMI	Body Mass Index
BMR	Basal Metabolic Rate
CHO	Carbohydrate
CV	Coefficient of Variation
e1RM	Estimated One-Repetition Maximum
EDTA	Ethylenediaminetetraacetic Acid
ELISA	Enzyme-Linked Immunosorbent Assay
EPOC	Excess Post-Exercise Oxygen Consumption
FDR	False Discovery Rate
FFA	Free Fatty Acid
FPG	Fasting Plasma Glucose
GI	Glycemic Index
GLP-1	Glucagon-Like Peptide-1
GLUT4	Glucose Transporter Type 4
GSV	GLUT4 Storage Vesicle
GV	Glycemic Variability
GY	Greek Yogurt
HIE	High-Intensity Exercise
HIIC	High-Intensity Interval Cycling
HOMA-IR	Homeostatic Model Assessment for Insulin Resistance
HR	Heart Rate
%HRmax	Percentage of Maximal Heart Rate
IR	Insulin Resistance
IU	International Unit
LMM	Linear Mixed-Effects Model
MASLD	Metabolic Dysfunction-Associated Steatotic Liver Disease
MF	Milk Fat
NaHep	Sodium Heparin
NSAID	Non-Steroidal Anti-Inflammatory Drug
PO	Power Output
RCF	Relative Centrifugal Force

REML	Restricted Maximum Likelihood
RPE	Rating of Perceived Exertion
RPM	Revolutions Per Minute
SD	Standard Deviation
SEM	Standard Error of the Mean
SIT	Sprint Interval Training
T2D	Type 2 Diabetes
TAG	Triacylglycerol
TDEE	Total Daily Energy Expenditure

Chapter 1: Introduction

1.1 Background and Rationale

Metabolic health is fundamental to overall physiological well-being, with disturbances in key regulatory pathways serving as early indicators of chronic disease risk. Insulin resistance, a hallmark of early metabolic dysfunction, is often precipitated by hyperinsulinemia and hyperglycemia, and if left unaddressed, can progress into impairments in fasting blood glucose.[1–3] Moreover, both insulin resistance and dyslipidemia are among the primary contributors to metabolic syndrome, a cluster of abnormalities that increases risk for a range of cardiometabolic diseases including type 2 diabetes and atherosclerotic cardiovascular disease.[4] Importantly, a number of young and 'healthy' individuals are exhibiting early signs of dysregulation, such as fasting blood glucose levels within the prediabetic range despite presumably being 'normoglycemic'.[5] This growing prevalence emphasizes the need for widespread practice of preventative measures that improve glycemia, insulinemia, lipid metabolism, and other aspects of metabolism that protect against the trajectory toward type 2 diabetes and broader cardiometabolic complications.

Exercise is well recognized as a management strategy that benefits body composition, glycemic regulation, and overall metabolic health.[2,4-7,52,62] Even a single bout of exercise can yield improvements in both glycemic and lipid responses.[6–9] These effects can partially be attributed to the upregulation of skeletal muscle glucose uptake during exercise, which is facilitated by an increase in the contraction-mediated translocation of glucose transporter type 4 (GLUT4) to the muscle membrane. This confers GLUT4 into a more insulin-responsive organization, thereby promoting insulin sensitivity and glycemic control for up to 48 hours post-exercise.[3,8,10,11] Beyond glucose regulation, exercise can elicit favourable effects on lipid

metabolism by enhancing the clearance of lipid intermediates such as non-esterified fatty acids and glycerol along with chylomicron-triacylglycerols, managing the accumulation of lipid stores in adipose tissue and other ectopic regions, and increasing fat oxidation overall.[12–14]

Importantly, the extent and duration of these beneficial effects can be differentially modulated by nutritional intake in the post-exercise period. Factors such as macronutrient composition and quality (i.e., carbohydrate content and glycemic index), and caloric value can influence the acute metabolic response.[1] While many post-exercise nutritional strategies are designed for rapid energy and glycogen restoration through high-carbohydrate sports drinks and calorie-dense foods, these approaches are largely tailored toward athletes prioritizing performance recovery. For broader populations engaging in exercise for health-related benefits, and particularly for those with some metabolic dysregulation, such strategies may not be optimal, and alternative approaches should be considerate of the aforementioned factors.

Wholefood dairy products, such as milk and Greek yogurt, are of particular interest in this context. Both are nutrient-rich, low-to-moderate glycemic index foods that provide high-quality protein with insulinotropic properties that improve glucose handling and promote satiety.[15–17] Greek yogurt (GY) differs from milk in its macronutrient profile as it provides a high amount of protein but substantially lower carbohydrate. This distinction may be of particular relevance in the post-exercise period, as maintaining a carbohydrate deficit has been shown to preserve exercise-induced improvements in glycemic control and insulin sensitivity by delaying glycogen replenishment.[1,3]

Despite these features, wholefood dairy products in the post-exercise context have been scarcely researched with respect to metabolic outcomes (i.e., glucose, insulin, and lipid responses). Moreover, related work often focuses on single nutrients or isolated components

(e.g., whey protein isolate) rather than intact dairy wholefoods, overlooking the influence of the structural and nutritional environment (i.e., the food matrix) in which the nutrients interact. Since the dairy matrix determines how nutrients are digested, absorbed, and ultimately processed by the body, it likely plays a role in modulating the potential nutritional benefits. [18,19]

Ultimately, the distinct macronutrient compositions, matrices, and physicochemical forms of wholefood dairy products present plausible mechanisms by which post-exercise metabolism may be modulated, yet these remain under-investigated. Our lab has previously conducted longer-term intervention studies on the metabolic effects of habitually high vs. low dairy diets combined with exercise training in females with overweight/obesity. These studies observed improvements in body composition (i.e., reduction in body fat percentage) as well as changes in serum fatty acid profiles, specifically a reduction in linoleic acid within the triacylglycerol fraction, with high dairy intake.[17-18] However, we have yet to investigate the acute metabolic effects of wholefood dairy supplements following a single exercise bout in young and healthy individuals which would provide important evidence regarding the mechanisms through which wholefood dairy supplementation may influence post-exercise metabolism.

1.2 Objectives

The overall objective of this study was to assess whether the acute metabolic response to a single bout of high-intensity exercise was differentially modulated by the post-exercise consumption of two distinct wholefood dairy products (milk and Greek yogurt), a carbohydrate (CHO) beverage, or a water control, with specific attention to glycemia, insulinemia, and lipid mobilization.

Specific Objectives were to characterize and compare:

1) the insulinemic responses of post-exercise milk, GY, CHO and water;
2) the glycemic responses of post-exercise milk, GY, CHO, and water;
3) measures of rested, fasted 24h insulin sensitivity, estimated by HOMA-IR, between the milk, GY, CHO and water conditions, and;
4) the effects on post-exercise lipid mobilization, reflected by circulating glycerol, between the milk, GY, CHO, and water conditions.

1.3 Hypothesis

The post-exercise consumption of milk, Greek yogurt, CHO, and water will differentially modulate exercise-induced metabolic responses, with the wholefood dairy products (GY vs. milk) exerting more favourable effects vs. CHO or water but to different degrees.

Specific hypotheses included that:

1) the post-exercise consumption of the CHO beverage will elicit the greatest acute insulin response, with both dairy products producing an intermediate response approaching CHO due to their insulinotropic amino acid content (but lower carbohydrate content), and water producing the smallest effect. GY will produce a more sustained insulin response than milk due to its nutrient content and food form.
2) the CHO beverage will produce the largest early glucose response, followed by milk and then GY, with water yielding the lowest glucose response in the immediate recovery period. Over the 24h period, the dairy conditions will support more favourable glucose excursions (i.e., attenuated spikes) than CHO or water due to their more balanced delivery of carbohydrate and the effects of insulinogenic protein.

3) the dairy conditions will produce lower 24h HOMA-IR due to their insulinotropic amino acids and lower carbohydrate loads, with GY demonstrating even lower HOMA-IR than MILK. CHO will demonstrate the highest HOMA-IR, and water will have an intermediate response.
4) circulating glycerol, an indicator of lipid mobilization, will be highest in the water condition due to it providing no immediate energy or carbohydrate post-exercise. All three caloric conditions will blunt the response, with CHO suppressing glycerol most strongly and the dairy conditions to a lesser degree. GY will suppress glycerol levels less than MILK.

Chapter 2: Literature Review

2.1 Overview and Effects of Substrate Metabolism and Regulation

2.1.1 Glucose Metabolism

Glucose is the primary carbohydrate serving as the central energy substrate for most tissues. Beyond its role in energy provision, glucose supports nerve impulse transmission, cellular proliferation, and the synthesis of key structural and signalling molecules.[20] Certain tissues, such as that of the central nervous system, are especially dependent on a continuous glucose supply, making its availability essential for both function and long-term metabolic stability.[21]

Accordingly, glucose metabolism is highly dynamic as it adapts to fit the demands of the human body throughout various dietary intake patterns and energetic states. In healthy individuals, the average fasting blood glucose concentration ranges from 4.4-5.0 mmol/L (80-90 mg/dL). In response to feeding, concentrations can transiently peak at 6.7-7.8 mmol/L (120-140 mg/dL) during the post-prandial period, before returning to baseline within approximately two hours *via* tightly regulated feedback mechanisms.[20] While glucose levels from exogenous sources are still abundant in the fed state, the body deposits a portion into reserves for later use through the synthesis of glycogen, ensuring a readily accessible backup supply when needed.[22] During periods of increased demand and/or when glucose levels are low, such as fasting or elevated energy turnover, alternate pathways are activated. Glycogenolysis breaks down hepatic and skeletal muscle glycogen stores to supply glucose for both systemic and local needs. In the event that glycogen stores have been depleted, such as during states of prolonged fasting or starvation, gluconeogenesis synthesises glucose from non-carbohydrate precursors including

gluconeogenic amino acids, particularly alanine and glutamine, along with lactate and glycerol, to maintain substrate availability for glucose-dependent tissues and functions.[22–26]

Both glycogenolysis and gluconeogenesis are stimulated by glucagon, a hormone secreted by pancreatic α-cells that increases circulating glucose when levels have fallen. Alongside insulin (secreted by pancreatic β-cells), glucagon forms the core of the regulatory system that maintains glucose homeostasis. Through the coordination of hepatic glucose output and peripheral tissue glucose uptake, this system ensures blood glucose remains within a narrow physiological range, throughout fluctuating conditions.[23]

2.1.2 Insulin: Secretion and Action

As glucagon acts to increase blood glucose availability, insulin counters this effect by promoting glucose clearance into tissues such as skeletal muscle and adipose. Stimulated by rises in circulating glucose, insulin is not only responsible for the maintenance of glucose homeostasis but is needed for entry into targeted cells so that glucose can execute its purpose, whether its fate is to be oxidized for ATP production, stored as glycogen in the liver and muscle, or converted to triglycerides in adipose tissue.[23]

Once released into circulation, insulin binds to surface transmembrane tyrosine kinase receptors found on designated tissues and cells. The binding of the insulin-receptor complex initiates intracellular signalling cascades that ultimately facilitate glucose uptake, primarily through the recruitment of glucose transporters to the cell surface. Of particular importance is glucose transporter type 4 (GLUT4), the primary transport protein responsible for the majority of glucose uptake in fat and skeletal muscle.[27,28] In the absence of insulin, GLUT4 resides within intracellular GLUT4 storage vesicles (GSVs), which are found within large sequestered GSV

pools. In response to insulin stimulation, GSVs translocate towards the plasma membrane. Upon arrival, the vesicles fuse with the membrane as their contents are released in a rapid exocytosis event.[27,29] Once at the membrane, GLUT4 facilitates glucose uptake from the bloodstream into the cell *via* facilitated diffusion.[28,30] In the sustained presence of insulin, GLUT4 endocytosis is reduced. The rate of glucose transport into respective tissues is ultimately determined by both the surface abundance of GLUT4 and the duration of its membrane localization. As blood glucose levels normalize and insulin concentrations fall, GLUT4 is endocytosed back into GSVs and sequestered to its cytosolic pools.[31] Importantly, GLUT4 translocation can be stimulated through insulin-independent mechanisms (i.e., skeletal muscle contractions), a key adaptation underlying exercise-induced glucose uptake that is later discussed in Section 2.2.1.1.

Importantly, impairments in the insulin signalling pathway can diminish glucose uptake efficiency, a state referred to as insulin resistance (IR). In such cases, individuals with diminished insulin sensitivity exhibit hyperinsulinemia to facilitate the same degree of glucose uptake as an individual with normal insulin sensitivity. The compensatory elevation in insulin maintains near-normal glycemic levels in the early stages but places an increased secretory demand on pancreatic β-cells. Over time, the pancreatic secretion of insulin fails to meet the heightened demands of progressing insulin resistance.[32] This may result in glucose disposal becoming insufficient, leading to overt hyperglycemia, and underscoring the critical role of insulin in maintaining glucose homeostasis.

2.1.3 Clinical Implications of Glycemic Control on Metabolic Health

The importance of efficient glucose homeostasis and having the capacity to dynamically regulate blood glucose lies within the clinical implications of glycemic control on metabolic

health. It is apparent and well established that hyperinsulinemia and hyperglycemia contribute to IR, which is indicative of metabolic syndrome risk and often precedes the onset of type 2 diabetes (T2D). T2D itself (and higher hemoglobin A1C) is associated with greater risks of retinal or renal disease, myocardial infarction, heart failure, peripheral vascular disease, mortality and more.[33]

Although much clinical focus is allocated to T2D risk, evidence indicates that glycemic variability (GV) itself, defined as fluctuations in blood glucose levels over both the short and long term, also confer significant health risks. Increased GV elevates the likelihood of hyperglycemic excursions, which independently carry adverse consequences. Moreover, individuals with greater GV and fasting plasma glucose (FPG) display an increased incidence of severe hypoglycemic events.[34]

Elevated/prolonged hyperglycemia contributes to a range of pathophysiological outcomes, including microvascular and macrovascular complications, increased oxidative stress, inflammation, and endothelial dysfunction. These processes underlie the development of cardiovascular disease, stroke, kidney failure, vision impairments, premature mortality, and more.[32,34] Collectively, these findings underscore the importance of maintaining stable and well-regulated glycemic control as a key strategy for preserving long-term cardiometabolic health.

2.1.4 Lipid Oxidation & Mobilization: Overview and Effects on Metabolic Health

Building on the dynamic capacity of glucose metabolism, it is important to recognize that these carbohydrate-based pathways function within the broader framework of metabolic flexibility. Metabolic flexibility refers to the body's capacity to efficiently adapt to conditional changes by shifting between fuel sources depending on nutrient availability and energetic

demands. [35] At the core of this is the intrinsic process of substrate switching between one primary fuel source to another. Along with carbohydrates, lipids play a primary role in this substrate network serving as a predominant energy reservoir.

The majority of stored fat exists in the form of triacylglycerols (TAGs), which are molecules composed of a glycerol backbone and three fatty acid chains. These molecules are predominantly stored in adipose tissue where they serve a range of purposes including structural support and insulation.[36] When equipped as the appropriate metabolic substrate, fat can be broken down into its constituents to supply alternate energy provision pathways, which is partially coordinated by the attenuated antilipolytic effect of insulin.[36,37] This process is known as lipolysis and involves the hydrolytic cleaving of the three ester bonds in TAGs, yielding three free fatty acids (FFAs) for every glycerol molecule released.[36] This catabolic process enables the constituents to be released into circulation and is otherwise referred to as lipid mobilization.[37] Accordingly, degrees of fat breakdown or lipid mobilization can be measured by analyzing circulating concentrations of FFAs and glycerol. Once in the bloodstream, the lipid constituents can be taken up by designated tissues to act as fuel sources. Glycerol enters the liver and serves as a gluconeogenic precursor, while FFAs are transported to tissues such as skeletal muscle where they can undergo beta-oxidation to produce ATP.[36] Under certain circumstances, FFAs can be re-esterified within adipose tissue, thereby recycling them into TAGs rather than being released into the bloodstream. Therefore, the measurement of glycerol, which cannot be recycled locally given the lack of glycerol kinase activity in adipocytes, presents a more accurate proxy of lipid mobilization rather than the measurement of FFAs.[38,39]

The regulation of substrate switching between carbohydrate and lipid metabolism is intricately linked. In the fed state, abundant glucose and elevated insulin levels promote

carbohydrate metabolism. This simultaneously leads to the suppression of lipolysis while contributing to the anabolic storage of glucose and dietary lipids as fat.[40,41] On the other hand, during periods of low carbohydrate and energy availability, especially when glycogen stores have depleted, the body resorts to lipid metabolism to meet energetic demands, therefore activating lipolysis and the downstream processes.[42] Ultimately, this contingent shift between carbohydrate and lipid metabolism enables the body to maintain energy supplies under diverse physiological conditions.

The dysregulation of lipid metabolism is associated with a wide range of pathologies and elevated disease risk, serving as a key contributor to metabolic syndrome. Dysregulated lipid metabolism often manifests as aberrant circulating triglyceride and FFA levels, along with the accumulation of lipid intermediates (i.e., diacylglycerols), which can drive lipotoxicity. On a cellular level, excess lipids promote endoplasmic reticulum stress, oxidative stress, mitochondrial dysfunction, and impaired autophagy. On a systemic level, impaired clearance and excess deposition of FFAs in non-adipose tissues leads to tissue damage, chronic inflammation, and ectopic fat accumulation. This includes excessive fat deposition in the liver, contributing to the progression of Metabolic Dysfunction-Associated Steatoic Liver Disease (formerly Non-Alcoholic Fatty Liver Disease).[43] Lipid accumulation in the muscle and liver can also impair insulin signalling, thereby increasing the risk of IR and T2D development.[44] Moreover, dysregulated lipid metabolism contributes to a plethora of CVD pathologies through a series of mechanisms, including but not limited to the buildup of fat/plaque in arterial walls.[45]

The clinical implications of lipid dysmetabolism extend far beyond these examples. Further adding to the complexity are the interactions of different components to substrate metabolism and their clinical manifestations. Altogether, this underscores the importance of

regulating lipid metabolism, along with improving metabolic flexibility in the prevention of chronic disease and the maintenance of long-term metabolic health.

2.2 Interventions for Metabolic Health Improvement

Lifestyle interventions are central to the prevention and management of metabolic dysfunction. Among these, exercise and dietary strategies stand as cornerstones yielding favourable outcomes that enhance metabolic regulation, including improvements in glycemia, insulin sensitivity, and metabolic flexibility.

2.2.1 Effects of Acute Exercise on the Metabolic Response

2.2.1.1 Glycemic Control in Response to Exercise

Exercise induces conditions that alter glucose homeostasis. While the responses may vary depending on factors such as intensity, duration, and nutritional state, they can ultimately provoke favourable outcomes in glycemic control. The effects of exercise on acute blood glucose dynamics are the function of two governing factors: (1) skeletal muscle glucose uptake, and (2) hepatic glucose production. Together, these processes interact in a way that supports energy demands during activity while promoting sequential improvements in metabolic regulation.

With approximately 60-80% of glucose being transported into skeletal muscle, this tissue proves to be a key player in glucose homeostasis serving as a dominant site of disposal.[28,35] During exercise, skeletal muscle glucose uptake rises substantially to meet energy demands and to support ATP turnover. This is facilitated through various mechanisms largely independent of insulin, which increases the muscles' ability to extract glucose from circulation. At the onset of high-intensity exercise (HIE) in the post-absorptive state, intramuscular glycogen is the

predominant fuel source. Circulating glucose contributes little during the early stages of HIE but becomes increasingly important as muscle glycogen stores deplete.[46] Accordingly, the reliance on glucose within the bloodstream increases with duration, as it is progressively equipped to sustain prolonged carbohydrate metabolism.

The increase in skeletal muscle glucose uptake during exercise is driven by several adaptive mechanisms. These include intracellular metabolic flux, as well as hemodynamic alterations that enhance glucose delivery through local vasodilation in the muscle, contraction-induced stress on venous return, and an overall increase in cardiac output.[47] Of particular importance is the amplification of GLUT4 translocation to the sarcolemma and transverse-tubules of muscle fibers. Unlike conventional insulin-stimulated translocation seen at rest, this process is specifically orchestrated by muscle contractions during exercise. Energy-sensing signals, such as AMP-activated protein kinase (AMPK) activation in response to shifts in AMP-to-ATP ratio, have been implicated in upregulating this process, particularly as AMPK activity increases with high-intensity contractions.[46,47] Regardless, this contraction-mediated signalling mirrors insulin's effects by recruiting GSVs from cytosolic pools, and achieving the same end-goal of increasing membrane permeability that ultimately allows for more glucose entry into the cell.

While exercise increases the rate at which circulating glucose is disposed via muscle glucose uptake, it also stimulates hepatic glucose production to provide sufficient sources of fuel while attempting to maintain euglycemia.[48,49] Following the principles of glucose homeostasis, hepatic glucose output during exercise generally increases with intensity and duration, with glycogenolysis dominating during HIE, and gluconeogenesis becoming more prominent with prolonged activity.[49] In moderate intensity exercise, hepatic glucose output increases at a similar

rate as muscle glucose uptake,[50] and is primarily governed by the glucagon-to-insulin ratio.[51,52] In contrast, regulation during HIE strays from conventional glucoregulatory principles, and more closely resembles a stress response.[50] A spike in glycemia displayed in the early stages of HIE despite minimal changes in insulin suggests that glucoregulation here may be primarily governed by catecholamines.[50,51] Catecholamines, such as epinephrine and norepinephrine, are a class of hormones and neurotransmitters stimulated by the sympathetic nervous system to act on adrenergic receptors throughout the body when under physical, environmental, and behavioural stress.[51,53] Accordingly, the HIE environment stimulates the secretion of these catecholamines, which bind to α and β-adrenergic hepatocyte receptors to upregulate hepatic glucose output, regardless of insulin activity but working in concert with glucagon. Ultimately, this catecholamine-driven effect magnifies the output-to-uptake ratio, producing a transient rise in blood glucose despite the already substantial increase in muscle uptake.[50,51]

The role of insulin may subside during HIE as studies show that even when insulin secretion is increased using α-blockades and pancreatic clamp techniques, the elevated output-to-uptake ratio still persists.[51] However, insulin activity dominates in the post-exercise recovery period as it resumes its role as a primary mediator. Upon cessation, glucose uptake declines more quickly than the rate at which output declines, resulting in yet another hyperglycemic spike. Catecholamine activity subsides around the same time, and any respective inhibition of the β-cell response is relieved. As a result, a period of hyperinsulinemia ensues to restore balance in glucose homeostasis while simultaneously promoting muscle repair and replenishment of glycogen stores.[51] At this stage, insulin acts rapidly, and a hallmark aftereffect of exercise is an increase in insulin sensitivity. This enhanced sensitivity largely stems from the elevated presence of GLUT4 transporters, which remain in a more insulin-responsive organization following

exercise (**Figure 1**).[10] This works in parallel with glycogen depletion and activated energy-sensing signals which may further promote the capacity for insulin-mediated glucose uptake.[46,54] Collectively, these adaptations enhance insulin's action in the recovery period.[8,10,11]

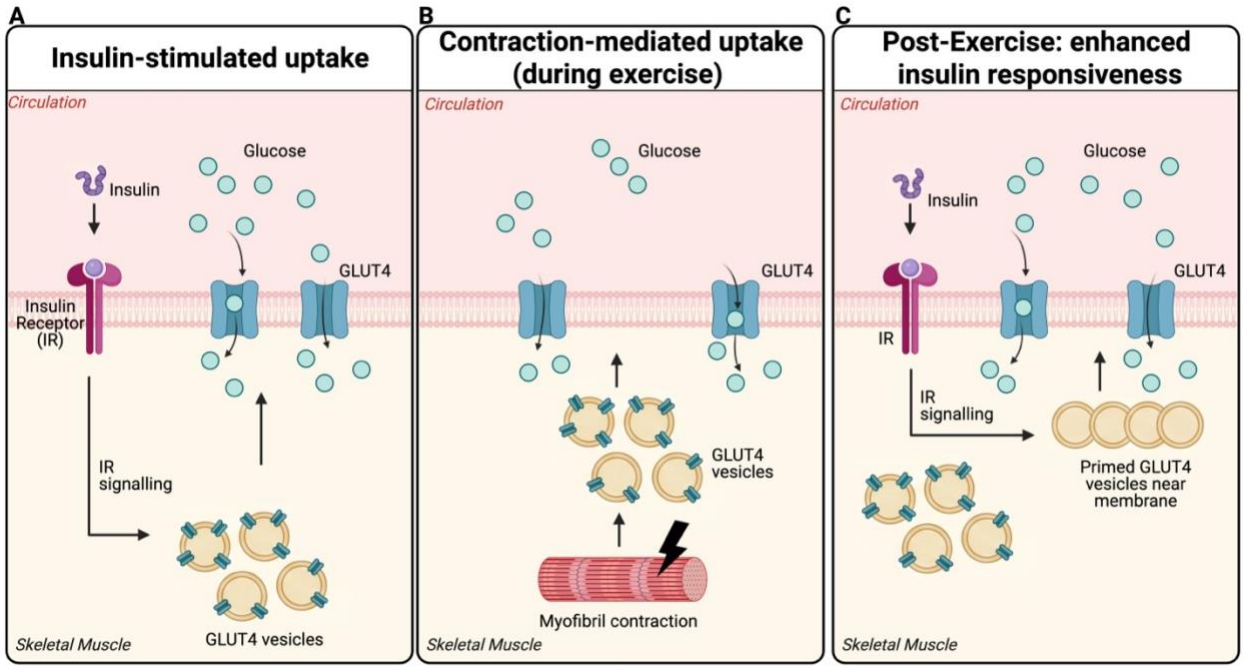


Figure 1. At rest, insulin stimulates GLUT4 translocation to the cell membrane through activation of insulin receptor signaling pathways (Panel A). During exercise, skeletal muscle contractions independently promote GLUT4 recruitment to the membrane, increasing glucose uptake despite reduced reliance on insulin (Panel B). Following exercise, prior contraction-mediated adaptations result in enhanced insulin responsiveness, with GLUT4 vesicles remaining in closer proximity to the membrane, whereby insulin signaling more efficiently promotes GLUT4-mediated glucose uptake, contributing to the transient increase in insulin sensitivity observed for up to 48h post-exercise (Panel C). Created with Biorender.com.

In summary, exercise provokes multiple, dynamic responses that together promote glycemic control. Glucose uptake increases during exercise,[46] and although this can cease immediately post-exercise, as well as be outweighed by hepatic glucose production,[51] exercise increases insulin sensitivity which can last up to 48 hours post-exercise.[3] Thus, despite the complex interplay between muscle glucose uptake and hepatic glucose output, the heightened

post-exercise insulin sensitivity and improvements in glucose handling highlight exercise as a uniquely powerful regulator of metabolic health.

2.2.1.2 Lipid Mobilization and Oxidation in Response to Exercise

Increasing lipid oxidation through exercise is a key intervention for enhancing metabolic flexibility, limiting the accumulation of lipid intermediates, and improving body composition. During exercise, carbohydrates and fats serve as the two primary substrates for energy provision. Both substrates can be oxidized simultaneously but their relative contributions depend on several factors such as nutritional state, duration of exercise, and especially exercise intensity. In essence, exercising in the fed state results in an overall favour towards carbohydrate utilization given its increased and ready availability. With prolonged exercise, however, fat contribution rises as glycogen depletion shifts reliance toward plasma FFAs. Interestingly, this progressive adaptation is only observed in prolonged HIE, whereas at lower intensities sustained beyond two hours, fat utilization patterns remain relatively unaltered.[55] Thus, exercise intensity is a principal determinant of lipid metabolism.

Lipid contribution rises from low to moderate intensities. At low exercise intensities of approximately 25% VO_2max, 85% of energy provided via lipid oxidation is derived from plasma FFAs. At this level, the rate of appearance of plasma FFA closely matches the rate of their oxidation.[55] As intensity increases, reliance on plasma FFA diminishes while intramuscular lipids progressively play a larger role. At moderate intensities, around a range of 48-65% VO_2max, fat oxidation reaches its peak.[55,56] During this time, there is a relatively equal contribution from plasma FFAs and intramuscular triglycerides. However, lipid energy alone is insufficient to meet total energetic demand, so carbohydrate and lipid sources each contribute

substantially and roughly equal proportions during this state. At high intensities of approximately 85% VO_2max, fat contribution declines and carbohydrate metabolism becomes the dominant energy source,[55] given its higher capacity for rapid ATP generation.[55,57] The limited fat contribution at this intensity shifts away from plasma FFAs toward intramuscular triglycerides, likely to circumvent the slower rate-limiting process of transmembrane fatty acid transport.[55] This is supported by seminal work from Sidossis et al.[58] demonstrating that lipid oxidation is reduced during HIE despite high FFA availability, and specifically may arise from the inhibition of long-chain fatty acid oxidation. Overall, the effects of exercise on lipid metabolism follow a pattern in which fat oxidation is maximized at moderate intensities, suppressed at high intensities in favor of carbohydrate use, and progressively elevated with longer exercise durations.

Despite the dominance of carbohydrate metabolism during HIE, the acute post-exercise lipid response rises with increasing intensity, likely reflecting greater metabolic perturbations.[59] Previous work has consistently reported elevations in post-exercise fat oxidation following high-intensity protocols such as sprint interval training (SIT).[9,60] Extending this work, Islam et al.[61] examined post-exercise fat utilization and excess post-exercise oxygen consumption (EPOC) in healthy young males following three graded exercise intensity protocols: (1) moderate-intensity continuous training (30 min at 65% VO_2max), (2) vigorous-intensity continuous training (30 min at 85% VO_2max), and (3) SIT (4 × 30 s "all-out" supramaximal sprints with 4-min rests). Their findings demonstrated that both post-exercise fat utilization and EPOC increased in an intensity-dependent manner, with SIT being the most effective despite its shorter duration. This elevation in fat reliance after exercise is thought to reflect an adaptive mechanism that increasingly equips lipid metabolism to support energy demands while muscle glycogen is being resynthesized.[61,62] It is also suggested that this mechanism is further facilitated by catecholamine-induced lipolysis

having increased circulating lipid availability (i.e., glycerol) after intense exercise and thus supporting fat oxidation trends during recovery.[61,63–65]

2.2.2 Effects of Nutrition/Feeding on the Metabolic Response to Exercise

While it is well established that exercise yields favourable improvements in glycemic control and insulin sensitivity, it is important to recognize that post-exercise nutrition can substantially promote the metabolic responses induced by exercise, though the mechanisms remain less clearly defined. A recurring theme in the literature is that carbohydrate consumption post-exercise blunts improvements in insulin sensitivity and glycemic control.[32] For instance, while exercise can induce a 48-hour window of increased insulin sensitivity, Taylor et al.[66] demonstrated in a sample of healthy adults that consuming a carbohydrate drink immediately after a high intensity run (70% VO_2max) attenuated this effect, leading to reduced insulin sensitivity and poorer glucose handling by the next morning compared to a non-caloric placebo drink. These findings align with previous reports showing that restricting carbohydrate intake after exercise helps sustain the favourable alterations in glucose metabolism elicited by exercise,[1,67] and is suggested to be mechanistically attributed to withholding muscle and liver glycogen replenishment, which may further enhance the upregulation of GLUT4 and other proteins involved in muscle glucose uptake.[68]

In considering the effects of post-exercise nutrition on glucose metabolism, it is important to distinguish whether observed changes are driven by carbohydrate restriction specifically, or simply by reduced energy intake overall, as the latter has also been shown to enhance peripheral and hepatic insulin sensitivity.[1] In essence, Schleh et al.[69] noted deteriorations in glucose handling when the amount of energy burned during a moderate

intensity cycling session was replaced by increasing energy intake at a subsequent dinner. Importantly, they recognized that the additional energy was substantially derived from high carbohydrate sources, making it difficult to separate the effects of energy surplus from those of carbohydrate intake. However, some efforts have been made to examine the effects of energy content independent of carbohydrates, and vice versa. In a crossover trial involving healthy young males, Newsom et al.[67] compared three post-exercise nutrition strategies: (1) high-carbohydrate meal with sufficient energy to fully replace exercise expenditure, (2) an isoenergetic meal low in carbohydrate (increased fat in order to provide the same amount of energy), and (3) a low-energy meal that maintained an energy deficit after exercise. Their findings demonstrated that low-carbohydrate content was the most potent factor in enhancing insulin sensitivity post-exercise, an effect that coincided with significantly lower muscle glycogen stores. Collectively, these results suggest that carbohydrate restriction without an energy deficit provides additive benefits for insulin sensitivity beyond energy deficits alone.

The role of post-exercise energy intake extends to lipid metabolism as well. In general, exercise increases adipose tissue lipolysis and constituent release during the recovery window, but these effects are significantly blunted by food intake within 1 hour of exercise cessation, reducing circulating glycerol and FFA concentrations.[70] When investigating the nutritional properties that may be of influence, both carbohydrate and energy restriction have been shown to encourage the exercise-induced lipid response.[71,72] However, when the amount of carbohydrate (i.e., content) is taken into consideration, the isolated effect of energy status prevails as the dominant factor. In the aforementioned study by Newsom et al.,[67] lipid mobilization and oxidation were only enhanced when subjects consumed a post-exercise low-energy meal, rather than carbohydrate restriction alone. This aligns with previous work, including Horowitz et al.[73]

who matched carbohydrate and protein intake in post-exercise meals but modified fat content to create either an energy balance or deficit. The deficit condition produced 3-4 fold higher plasma FFA and glycerol, reduced carbohydrate oxidation, and demonstrated a substrate shift toward fat oxidation. The mechanisms explaining these effects remain complex and varied, with some of the literature proposing they be attributed to low energy availability prolonging catecholamine-driven lipolysis while reducing insulin's anti-lipolytic action[73], while others reference the delay of glycogen resynthesis causing insulin signalling to remain more active and supporting a milieu conducive to fat oxidation.[67] Nevertheless, the ultimate implications presented are that low-energy post-exercise feeding is a dominant driver of lipid metabolism, while carbohydrate restriction does contribute to this effect but to a lesser degree.

Most studies focus on carbohydrate and energy content, however, other nutritional factors may also play a role in shaping the acute post-exercise metabolic response, although these remain less well researched. One such factor is glycemic index (GI), a measure of the postprandial glucose excursion induced by a food. Given its direct influence on circulating glucose availability, GI may reasonably influence how strongly exercise-induced benefits are expressed. Kaviani et al.[74] investigated whether low- versus high-GI meals consumed after evening exercise would alter next-day metabolic outcomes. Following evening exercise, participants consumed isoenergetic meals matched for macronutrients but differing in GI. When responses to a standardized high-fat breakfast the following morning were assessed, they found that postprandial glucose AUC did not significantly differ across conditions. However, the insulin response with the low-GI condition was significantly lower, whereas the high-GI condition displayed no difference from the no-exercise control. These findings may suggest that low-GI consumption post-exercise may sustain the benefits on insulin sensitivity as there was

less insulin demand the next day, without compromising glucose handling. In contrast, high-GI consumption may blunt the acute metabolic benefits of exercise as metabolic function the following morning was similar to when no exercise was performed at all. Importantly, this study was conducted in a clinical population (females with overweight and obesity), and further work is warranted in diverse healthy populations. Gaitan et al.[1] also noted in their relevant review that many studies demonstrating improved insulin sensitivity after post-exercise carbohydrate restriction provided high-GI carbohydrate sources, further supporting the possibility that carbohydrate quality, not just quantity, may have an influence on post-exercise outcomes.

In addition to improving glucose handling, the study by Kaviani et al.[74] also demonstrated that the ingestion of low-GI meals in the post-exercise period sustained alterations in lipid metabolism into the next day. Respiratory exchange ratio values measured after the standardized high-fat breakfast on day 2 were lower (indicating greater fat oxidation) in the low-GI post-exercise and exercise-only conditions compared with high-GI post-exercise intake. Triglycerides were also significantly lower in the low-GI post-exercise and exercise-only conditions, while FFA concentrations in the low-GI group trended higher than in the high-GI group, but did not reach statistical significance. Together, these findings suggest that low-GI feeding in the post-exercise period helps preserve aspects of lipid mobilization and metabolism, while high-GI feeding attenuates it.

In summary, various characteristics of post-exercise nutrition can shape metabolic outcomes (**Figure 2**). Energy deficits are the strongest driver of enhanced lipid mobilization and oxidation,[67,73] while carbohydrate restriction provides additional benefits for glycemic control.[1,66,67] Beyond quantity, carbohydrate quality (particularly GI) modulates these responses, with low-GI intake helping sustain improvements in insulin sensitivity and fat oxidation, and

high-GI intake diminishing them.[1,74] Together, these findings highlight the critical role of nutrient composition and quality in determining how effectively exercise translates into lasting metabolic benefits.

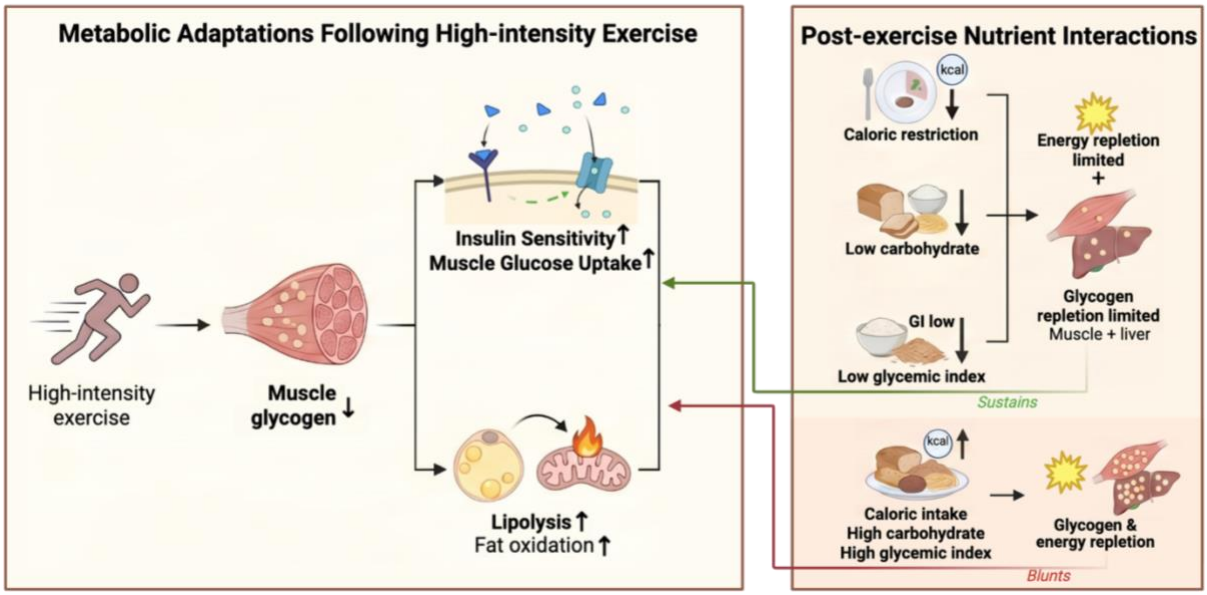


Figure 2. Conceptual model illustrating exercise-nutrient interactions following a high-intensity exercise bout that influence exercise-induced metabolic adaptations. Lower energy availability, reduced carbohydrate intake, and lower glycemic index foods may help sustain exercise-induced improvements in insulin sensitivity, glycemic control, and fat utilization, whereas high glycemic index foods and greater energy and carbohydrate repletion may attenuate these responses. Created with Biorender.com.

2.3 Dairy Products and Metabolism

2.3.1 Dairy Feeding and Metabolism at Rest

Dairy products are nutrient-dense, providing protein, carbohydrate (primarily lactose), and fat, alongside key micronutrients such as calcium, vitamin D, potassium, and phosphorus. Beyond their contribution to dietary nutrient adequacy, higher dairy consumption has been consistently associated with a reduced risk of T2D and cardiometabolic disease, highlighting the potential role of specific nutritional components in these benefits.[75,76]

One of the most notable features of dairy foods is its protein content. Dairy is an easily digestible, high-quality, animal protein source that provides a high-quality amino acid profile. Dairy proteins are composed of whey and casein.[77] Whey is rapidly digested as it remains soluble under acidic gastric conditions. This facilitates rapid gastric emptying and further digestion and absorption in the small intestine, resulting in a quick appearance of amino acids into the bloodstream.[78,79,80,81] In contrast, casein exists in a micellar form that readily coagulates in gastric conditions, therefore slowing down its digestion and producing a more gradual and sustained release of amino acids.[78–82] Consumed together in dairy products, whey and casein can provide both rapid and prolonged aminoacidemia, with responses varying depending on the whey-casein ratio of a product.

Dairy proteins are particularly rich in essential amino acids, which is not only a key factor important for muscle protein synthesis but also for metabolic effects beyond muscle growth. Several amino acids harbour the ability to enhance insulin release and its action independent of glucose.[83] Among the amino acids, leucine, which is rich in whey, along with phenylalanine, arginine, and lysine, have been identified as some of the most potent insulin secretagogues.[84,85] Nevertheless, a more diverse combination of essential amino acids is most effective in potentiating insulin secretion.[84] This balanced amino acid composition explains why dairy proteins display stronger insulinotropic effects compared with other protein sources.

Nilsson et al.[16] sought to further investigate the insulinogenic action and capacity of dairy proteins by comparing the postprandial effects of milk and whey to other isonitrogenous, carbohydrate-matched sources of protein (i.e., cod, gluten, cheese). Their findings demonstrated that dairy protein sources, particularly whey and milk, had the most pronounced effects on attenuating glucose excursions. Insulin responses varied as whey elicited a significantly higher

insulin AUC, whereas milk and cheese induced moderate (relative to whey) but still greater responses than non-dairy proteins. Correlation analyses also revealed that insulinotropic effects were strongly associated with the amino acids especially seen to rise with whey and milk consumption.[16] Taken together, it can be interpreted that the specific amino acid composition of different dairy proteins, rather than protein quantity alone, underpins their insulinogenic capacity. This may be why milk, which contains both whey and casein, elicited a differential (i.e., attenuated AUC) insulin response relative to whey, although it still effectively improved glycemia.

Building on this, substantial research has investigated the effects of dairy consumption for metabolic health, with a specific emphasis on glycemic control. El Khoury et al.[15] conducted a randomized crossover study in healthy young males, comparing the postprandial effects of dairy snacks (yogurt, milk, and cheese) against higher-carbohydrate alternatives such as fruit juice. Dairy snacks consistently produced a lower postprandial glycemic response compared to juice, with reductions of approximately 30-50%. Not only did dairy products yield the lowest glycemic responses during the post-snack period, but also further attenuated the glycemic response to a subsequent meal consumed later in the day. This carryover effect suggests that dairy may enhance glucose handling beyond the immediate postprandial window, potentially by augmenting insulin efficiency. Later work conducted by Sun et al.[86] further supported this notion as they investigated the impact of dairy intake timing relative to high-carbohydrate food intake. In their crossover trial within healthy males, preloading milk 30 minutes before bread consumption halved the postprandial glycemic excursion and reduced the insulin demand. Together, these findings reinforce the concept that dairy ingestion can act as a metabolic primer, capable of enhancing glucose tolerance and insulin action across consecutive meals.

Recent work has also demonstrated the benefits of incorporating dairy directly into meals. Hilkens et al.[17] conducted a randomized crossover study in healthy young adults where carbohydrate-rich breakfast items were progressively replaced with dairy products (i.e., milk and cheese). Across three isoenergetic meal conditions that differed by dairy content (i.e., no dairy items, 1 dairy item, 2 dairy items), they observed a clear dose-dependent effect on attenuating glycemia. The inclusion of dairy products attenuated peak postprandial glucose concentrations and reduced total AUC. These glycemic improvements occurred without significant differences in insulin concentrations compared with the carbohydrate-rich condition. The glycemic improvements were likely multifactorial, reflecting not only the reduced carbohydrate load achieved through substitution with mixed-macronutrient dairy foods, but also the protein and fat content of dairy, which slow gastric emptying and prolong nutrient digestion and absorption. Concurrently, comparable insulin secretion was maintained through stimulation by insulinotropic amino acids rather than glucose alone. Higher dairy intake also led to dose-dependent rises in circulating amino acids and glucagon-like peptide-1 (GLP-1), consistent with the insulinogenic properties of dairy proteins and further illustrating the role of dairy in modulating postprandial glucose metabolism.

Apart from its documented capacity to favourably improve glycemic control, dairy presents as a rich source of calcium which has itself been implicated in lipid metabolism. It is suggested that higher calcium intakes are inversely correlated with adiposity, with possible mechanisms relating to higher calcium lowering intracellular adipocyte calcium and calcitropic hormones, thereby favoring fat oxidation over storage.[87] Consistent with this, Cummings et al.[88] illustrated a rise in postprandial fat oxidation and attenuation in the suppression of circulating non-esterified fatty acids following high-calcium meals sourced from dairy products. Though

modest and not consistently observed, these effects suggest that the inherent calcium content in dairy products may add to its capacity in modulating metabolic responses.

In summary, dairy consumption at rest attenuates glycemic excursions through the insulinotropic actions of its proteins as well as by reducing glycemic load and thus the glycemic response when substituting for carbohydrate-rich alternatives.[15–17] It also primes metabolic responses to subsequent meals and accordingly enhances glucose handling. Furthermore, its calcium provision may favour postprandial lipid metabolism.[15,86,88] These effects highlight dairy's role as a nutritional strategy for improving metabolic function throughout the day.

2.3.2 Dairy Consumption and Acute Exercise: Existing Work and Gaps

Given dairy's favourable effects at rest, growing interest lies in its potential to promote a favourable metabolic response following exercise. From a nutritional standpoint, both milk and Greek yogurt harbour characteristics that position them as beneficial post-exercise snacks, especially in the context of sustaining improvements in insulin sensitivity and glycemia. Skim milk provides approximately 90 kcal in a 250 mL serving, 13 g of carbohydrate, minimal fat (0.2 g), and 9 g of high-quality protein. In contrast, a 100 g serving of 0% MF plain Greek yogurt contains an isonitrogenous amount of protein (9 g) but with lower carbohydrate (8.5 g), and no fat (0 g). Thus, while both milk and Greek yogurt supply high-quality amino acids that could potentially enhance insulin activity post-exercise, Greek yogurt provides it in a lower-carbohydrate matrix, which is an important consideration given that maintaining a carbohydrate deficit has been shown to prolong exercise-induced metabolic benefits.[32]

Although direct research examining dairy products in this context is limited, some work has begun to emerge. Gao et al.[89] conducted a randomized crossover study in adults with

overweight performing 90 minutes of moderate-intensity exercise at 50% VO_2peak in the evening. After exercising, participants consumed either skim milk or an isoenergetic carbohydrate-based sports drink. The following morning, a standardized high-calorie breakfast was provided, and postprandial glucose responses were measured. Results showed that skim milk consumption significantly attenuated the glucose excursion to the subsequent breakfast compared to the sports drink, suggesting that milk preserved the exercise-induced improvements in glycemic control, whereas the carbohydrate-rich drink blunted them. In addition, there were trends indicating that milk consumption promoted fat oxidation, although this did not reach statistical significance.

Moreover, as dairy products are a source of calcium, though the provision varies between products (higher in a 250 mL serving of milk vs. 100 g serving of GY), they may further leverage lipid metabolism responses following exercise through the proposed effects of calcium on lipolysis.[87,88] However, research that investigates the effects of the two products in a post-exercise context remains scarce.

To date, the metabolic effects of Greek yogurt as a post-exercise snack remain especially unexplored, nor has it been directly compared with other dairy products such as milk. This represents a notable gap in the literature, as dairy products can differ substantially in their impact depending on their nutrients, as well as food form and matrices. For instance, Law et al.[19] demonstrated that when consumed with a high-GI carbohydrate meal, semi-solid dairy products (yogurt) and solid dairy products (cheese) improved glycemic control and satiety to a greater extent than liquid milk or soy beverages. Interestingly, these benefits occurred without differences in insulin secretion, suggesting mechanisms such as slowed gastric emptying due to gastric casein gel formation, fermented nature, or textural properties may mediate postprandial

responses. In the exercise context, where nutrient timing and substrate availability are critical, such differences may be especially relevant.

2.3.3 Summary

Overall, the current evidence positions dairy as a nutrient-dense food capable of favourably modulating postprandial metabolism through its unique protein composition, insulinotropic amino acid profile and lower carbohydrate content. At rest, dairy consumption consistently attenuates glycemic excursions,[15–17] enhances insulin efficiency,[17,86] and extends glycemic handling benefits across subsequent meals.[15,86] Preliminary work in the exercise context suggests that dairy may preserve the favorable metabolic effects induced by exercise perturbations, though findings thus far are limited and focused on milk.[89] Importantly, not all dairy products are equivalent as differences in food form, matrix, and macronutrient distribution can meaningfully alter responses.[18,19] These findings reinforce the recognized role of dairy foods in metabolic regulation and emphasize the need for further investigation into the extent to which distinct dairy products differentially modulate the acute post-exercise metabolic response.

Chapter 3: Methods

3.1 Participants & Eligibility

A total of 20 young and healthy participants, both male and female between 18 and 45 years of age were recruited for the "Bone and Inflammatory Outcomes – Nutrition and Exercise Trials" (BIONEX) study. Recruitment was conducted in Toronto (Ontario, Canada) using approved posters posted around campus (York University), social media advertisements, in-class announcements, and word-of-mouth referrals. Upon expressing interest, potential participants completed an initial online screening appointment with a research coordinator to determine their eligibility which was contingent on the inclusion and exclusion criteria outlined in **Table 1**. Participants deemed eligible were provided with study information and written informed consent was collected prior to study commencement. This study has been approved by the York University Research Ethics Board (ORE File #2019-045) and is registered at www.clinicaltrials.gov (NCT06041087).

Table 1. Inclusion and exclusion criteria for participant eligibility.

Inclusion Criteria	Exclusion Criteria
• Age: 18-45 years • Male or female • BMI:18.5-24.9 kg/m^2 • Low levels of structured exercise activity (≤2/week)	• Existing medical conditions, including inflammatory, autoimmune, musculoskeletal disorders • Use of Vitamin D and/or calcium supplements • Allergies to dairy products and/or lactose intolerance • Current smoker

3.2 Pre-experimental Trial Testing

3.2.1 Baseline Measurements

Baseline anthropometric and body composition measurements were collected during an initial laboratory visit prior to the participant's first experimental trial. Height (cm) and weight (kg) were measured without shoes using a stadiometer and digital scale, respectively. Participants' body composition was assessed utilizing the Bodpod (COSMED USA Inc., Concord, CA, USA) which required them to step into a chamber in minimal formfitting clothes and a swim cap. The system uses air displacement as an estimate of body volume and subsequently calculates body density and %BF based on the Siri equation.

After body composition testing, participants completed a maximal vertical jump test as a measure of power, using a vertical jump challenger (Tandem Sport, Louisville, KY, USA). Standing reach was first measured by having participants stretch their dominant arm upward and walk through the device. They then performed a counter-movement jump, where they swung their arms to generate momentum and jumped as high as possible with both arms extended. Vertical jump height was calculated by subtracting standing reach from the highest touch recorded. Each participant was given three attempts to achieve their maximum jump.

3.2.2 Familiarization Session

The remainder of the initial laboratory visit focused on collecting baseline exercise data to inform plans for the individualised exercise bout while simultaneously exposing participants to the exercise movements they were to complete at each trial. Along with determining nuances such as seat height, a certified personal trainer demonstrated and guided participants through a chest press, seated row, and leg press on selectorized resistance machines. After demonstrating proper technique, for each exercise, an estimated 1-repetition maximum (e1RM) test was executed. Participants began the e1RM test with 3 warm-up sets of 8 repetitions, with each set

increasing in load contingent on achieving targeted ratings of perceived exertion (RPE) of about 3, 5, and 7, respectively. Following the warmup, the load increased by intervals of 20-40 lbs and participants were to attempt 5 repetitions of the movement. If successful, the process continued following a short rest period until the participant failed to perform 5 repetitions. The Brzycki formula was then used to estimate the participant's e1RM for the respective exercise.

Once all three resistance exercises were familiarized and tested for their e1RMs, participants completed a maximal power output (PO) test on a stationary cycle ergometer (Monark 894E, Monark Sports & Medical, Vansbro, Sweden). Prior to test commencement, their seat heights were adjusted to their comfort and recorded, and their targeted pedal speed in revolutions per minute (RPM) was calculated based on their body weight and rounded down to the nearest 10 RPM. The test began with a 4-minute warm-up at 0 kg resistance, producing approximately 50–70 watts (W). After the warm-up, 0.5 kg was added to the cycle basket every minute until the participant could no longer maintain their set RPM, and the highest workload completed was recorded as their maximum PO.

3.3 Experimental Trials

This study utilized a randomized crossover design to evaluate the outcomes of four different trial arms: (1) CARBOHYDRATE (CHO), (2) MILK, (3) GREEK YOGURT (GY), and (4) WATER. Each trial featured an identical exercise bout but differed by the post-exercise supplement. Following the familiarization session, a randomized sequence of trial arms was generated for each participant. Accordingly, they underwent all four interventions, but in a predetermined, randomized order, serving as their own controls. To mitigate potential carryover effects between arms, a minimum 2-week washout was implemented in between each trial.

Given the influence of menstrual cycle on basal metabolic function, female participants who were not using hormonal contraceptives had their trials scheduled exclusively during the early follicular phase. Thus, their trials were separated by a minimum of one full menstrual cycle (approximately 4 weeks). To reduce potential confounding effects caused by the preceding nutritional environment, participants were instructed to eat similarly each day before a trial (see Section 3.3.4.1). They were also asked to abstain from non-steroidal anti-inflammatory drugs (NSAIDS) for 12 hours, alcohol for 24 hours, and exercise for 48 hours prior to each session.

On the day of each trial, participants arrived at the laboratory in the morning (around 0800h), following a minimum 8-hour overnight fast. Upon arrival, a questionnaire was administered regarding the aforementioned information to confirm adherence to pre-trial instructions (i.e., medication consumption, time-elapsed since last meal and engagement in exercise). Also featured in the questionnaire were inquiries about recent illnesses and ailments. Each trial began with the collection of a baseline pre-exercise blood sample. Body weight was also measured at each visit to monitor weight stability across all trials, followed by baseline assessments of maximum vertical jump performance and perceived muscle soreness. Vertical jump and muscle soreness assessments were repeated several times after the exercise bouts. These measures serve to confirm that the exercise bouts elicited consistently intense and sufficient stimuli across trials. Once all these measurements were collected, participants immediately proceeded to the exercise portion of their trial.

3.3.1 Exercise Protocol

As previously mentioned, the exercise bouts were standardized in structure across all trials and participants, meaning that the type, order, and duration remained consistent. However,

the protocols were individualized based on each participant's maximal strength and physical capacity, as determined by their 1RM and PO tests conducted at the familiarization visit. Each exercise bout was approximately 60 minutes long and was led by a personal trainer at our designated laboratory gym located at York University's Life Sciences Building.

Each session began with a standard warm-up, featuring a series of both dynamic and static stretches to promote safety and prepare participants for the exercise bout.

Participants then proceeded to the plyometric portion of the exercise bout, which consisted of four jumping movements, each performed for 3 sets of 10 repetitions. The protocol began with d*epth jumps,* using aerobic steppers adjusted to an appropriate height for the participant. To perform the movement, participants stepped onto the elevated platform, leaped off, and immediately exploded upward upon ground contact. Once all three sets were completed, participants moved onto *tuck jumps,* where they were instructed to explosively execute 10 consecutive jumps, bringing their knees upwards towards their chest, while minimizing contact time with the floor. The third plyometric exercise was a *split jump.* This movement required the participant to jump upwards and land in a lunge position, alternating their lead and trailing legs with each repetition. Lastly, participants performed *seated jumps*. Starting from a seated position on a chair, participants swung their arms to generate momentum, and explosively jumped up and outwards from the seat, landing on both feet.

Following the plyometric exercises, participants completed the three resistance exercises: (1) *chest press,* (2) *leg press*, and (3) *seated row*. All of which were performed for 3 sets of 8-12 repetitions, done to approximately 70% of the individual's e1RM. These movements were executed on the same selectorized resistance machines from the familiarization. If the individual

failed to complete 3 sets with the same repetition count due to fatigue, the number of repetitions or load volume was adjusted, and this change was maintained accordingly across all trial arms.

The exercise segment concluded with a high intensity interval cycling (HIIC) bout. The cycling protocol lasted 10 minutes, using the same cycle ergometer as during the familiarization. It began with a 4-minute ramp up as the load gradually increased to 50% of their maximum PO. Within the last 10 seconds of this phase, the participant's heart rate (HR) and RPE were recorded. At the 5-minute mark, the load increased to 90% of their max PO for 1 minute. This was followed by a brief 1-minute recovery period where participants cycled at the decreased load of 50% of their max PO. HR and RPE measurements were collected in the last 10 seconds of each high-intensity and recovery period. These 2 periods repeated for 2 more cycles, completing a total of 3 working intervals. However, if participants failed to achieve 85% of their age-predicted HR max, an additional working interval was added to the protocol. Once completed, participants cooled down on the bike for 2 minutes, cycling with no resistance, to gradually decrease their HR. Immediately afterward, participants returned to the laboratory for their post-exercise blood sample collection.

3.3.2 Post-exercise Supplements

3.3.2.1 Dosing Schedule

On each trial day, participants consumed three separate doses of the assigned supplement. The first dose was ingested 5 minutes after exercise, immediately following the post-exercise blood draw. A second dose was provided 1 hour following the consumption of first supplement. The third and final dose was packaged and taken home for consumption later in the evening, before bed. This dosing schedule was designed to reflect realistic dietary behaviours and

complements the timing of our blood sampling to capture both immediate and extended effects of ingestion during the post-exercise period.

3.3.2.2 Supplements: Types and Dosages

This randomized crossover design composed of four different trial arms, each defined by a distinct post-exercise supplement. All liquid based supplements (milk, carbohydrate beverage, and water) were isovolumetric, standardized to 500 mL per dose. Specifically, the milk supplement consisted of 500 mL of Lactantia or Natrel 0% MF milk. The carbohydrate beverage contained 48 g of maltodextrin (Brand: Canadian Protein), dissolved in ~500 mL of water. Notably, the milk and carbohydrate supplements were isocaloric at 180kcals, allowing for further insight into effects including but not limited to the influence of energy versus the complex nutrient matrix of milk on the post-exercise metabolic response. Although water was consumed ad libitum throughout each trial, the WATER arm specifically required the consumption of 500 mL at each dosing interval. To improve palatability, participants had the option to mix in a calorie-free fruit flavoured sweetener (e.g., Mio or Crystal light) to both their carbohydrate and water supplements. As for the GY supplement, each dose was 200 g of 0% MF Oikos high protein Vanilla GY. Although this semi-solid supplement was not matched for volume on a practicality basis, the GY was isonitrogenous to its dairy counter supplement (milk) at 18 g of protein. This detail was implemented to control for the potential mechanistic influence protein may have on the metabolic outcomes, increasing confidence that observed effects can be attributed to matrix, digestive, and other nutritional effects, rather than differences in the amount of protein. Further details regarding the key nutrient breakdown of each supplement are provided in **Table 2**.

Table 2. Key nutrient breakdown for one dose of each trial supplement.

	Milk	GY	CHO	Water
Serving size	500 mL	200 g	500 mL	500 mL
Calories (kcal)	180	149	180	0
Carbohydrate (g)	26	17	45	0
Protein (g)	18	19	0	0
Fat (g)	0.4	0	0	0
Calcium (mg)	600	257	0	0
Vitamin D (µg)	10	0	0	0

3.3.3 Blood Sample Collection

Venous blood samples were collected by a trained phlebotomist using standard venipuncture techniques from a vein in the antecubital fossa. Blood samples were collected at five timepoints across each trial: (1) baseline (pre-exercise, fasted), (2) immediately post-exercise (approximately 5 minutes following conclusion of the exercise bout to allow for travel time between the laboratory gym and core facility), (3) 1 hour following consumption of the first supplement dose, (4) 4 hours following consumption of the first supplement dose, and (5) 24 hours post-exercise (fasted). Notably, the 5-min post-exercise sample captured the immediate effect of exercise only, and the 1-hour sample captured the metabolic environment following exercise and a singular dose of the supplement. The 4-hour sample captured the environment following exercise, the consumption of 2 supplement doses and a light standard lunch, while the 24-hour sample captured the extended metabolic period following a day of consuming 3 supplement doses along with a standard lunch, snack, and dinner. **Figure 3** displays an overview of a trial day, highlighting sampling intervals, in relation to exercise stimuli, supplement dosing and feeding time.

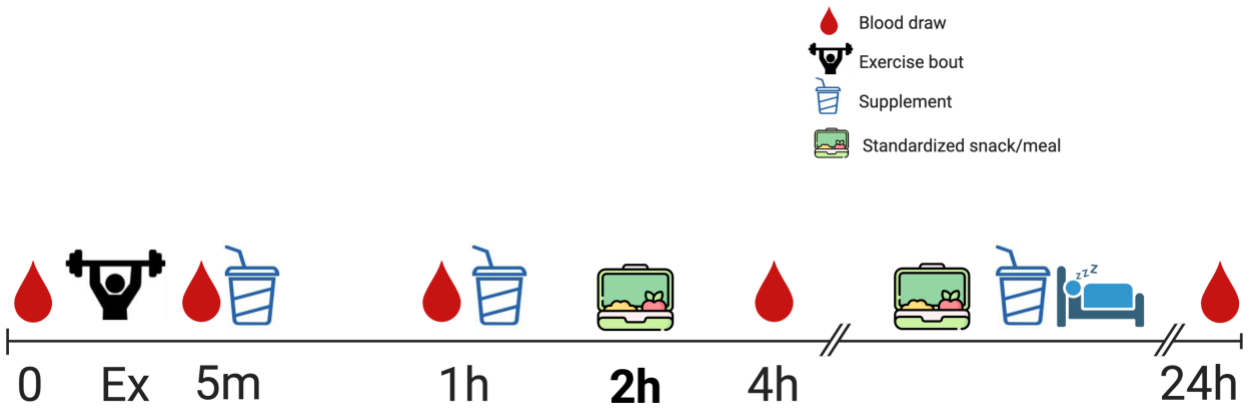


Figure 3. Schematic overview of the experimental trial day, including exercise, supplement dosing, meals, and blood sampling time points. Participants completed a high-intensity exercise bout, followed by the consumption of a trial-specific nutritional supplement 5 minutes post-exercise. Blood samples were collected at five time points: pre-exercise, immediately post-exercise, 1 hour post-initial supplement, 4 hours post-initial supplement, and 24 hours post-exercise. Additional supplement doses were provided 1 hour post-initial supplement and again in the evening (before bed). Standardized meals were consumed 2 hours post-exercise (lunch) and later in the day (snack and dinner).

With the exception of the 5-min post-exercise sample, approximately 22 mL of whole blood was drawn into five vacutainers at each time point. Specifically, 6 mL was drawn into one coated red top vacutainer for serum, 4 mL was drawn into one coated purple-top vacutainer with EDTA (ethylenediaminetetraacetic acid) for plasma, and three coated green-top vacutainers were each filled with 4 mL for sodium heparin (NaHep) plasma. For the immediate post-exercise blood sample, a reduced volume of 16 mL was collected. The 16 mL was distributed as follows: 1 x 6 mL red-top for serum, 1 x 6 mL green top for NaHep plasma, and 1 x 4 mL purple-top for EDTA plasma.

Following collection, plasma samples were processed immediately by centrifuging the respective vacutainers at a relative centrifugal force (RCF) of 1300 g for 10 minutes at 4°C using a benchtop centrifuge (Rotina 38R, Hettich, Tuttlingen, Germany). Serum vacutainers sat at

room temperature for 30 minutes to allow for clotting before undergoing the same spinning protocol (1300 g for 10 minutes at 4°C). After centrifugation, all samples were aliquoted into 1.5 mL microcentrifuge cryotubes and stored at -80°C until further analysis.

3.3.3.1 Hematocrit Assessment

To account for any exercise-induced shifts in plasma volume, hematocrit measurements were assessed at the pre-exercise, immediately post-exercise, 1 hour, and 4 hours post-exercise marks. Pre-calibrated heparinized microhematocrit tubes were filled with a small volume of whole blood drawn from one of the EDTA vacutainers. They were then sealed and centrifuged at 20000 RCF for 5 minutes using a microhematocrit centrifuge (Haematokrit 210, Hettich, Tuttlingen, Germany). This process separates red blood cells from plasma, allowing for visual measurement using a standard evaluation disk. For each timepoint, hematocrit assessments were measured in triplicate, and the average of all three values was recorded.

3.3.4 Standardized Nutrition & Dietary Analysis

3.3.4.1 Pre-trial Nutrition

To control (as best as possible) for the potential influence of differences in pre-trial dietary intake, participants were instructed to replicate their nutritional intake on the day prior to each experimental visit. To monitor this, participants recorded and uploaded their food intake using the Keenoa app. This digital dietary tracking app generates detailed nutritional information based on photo and descriptive inputs, thereby allowing the research team to further analyze intakes and consistency across trials.

3.3.4.2 Trial Day Standardized Meals

On experimental days, standardized meals were provided for lunch and dinner, along with some light snacks. Each meal plan was individualised to the participant based on their estimated total daily energy expenditure (TDEE). TDEEs were calculated using the Harris Benedict equation to determine basal metabolic rate (BMR), which was then multiplied by a physical activity factor of 1.375, indicating a "lightly active" lifestyle which aligned with our sample of individuals who do not regularly engage in structured exercise. **Table 3** outlines the macronutrient profile of a standard meal plan (e.g., for an individual requiring ~2000kcal/day) for the MILK trial day. Adjustments to each meal plan were made in the snack and dinner contents which were given after the 4-hour blood draw, keeping the lunch meal standard across all participants. To ensure a balanced protein intake across the day, an additional protein bar was provided as a snack on the CHO and WATER arms. This accommodated for the lack of protein from the non-dairy supplements, while also facilitating a more even distribution of protein feeding throughout the day. Participants were also instructed to log their dietary intake on trial days using Keenoa to allow for further monitoring.

Table 3. Macronutrient profile of a standardized trial meal plan for the MILK trial day (for participants requiring an approximately 2000kcal/day intake).

Food	Qty	Calories (kcal)	Carbohydrates (g)	Protein (g)	Fat (g)
Supplement					
Milk	3 x 500mL	540	78	54	1.2
Lunch					
Great Value Vegetable Stir Fry	1	190	36	8	2
Banana	1	105	27	1	0
Strawberry Cereal Bar	2	260	48	2	6
Snacks					
Apple	1	104	28	1	0
Banana	1	105	27	1	0
Snack'n Go Hummus & Crackers	2	380	52	12	16
Dinner					
Healthy Choice Steamers Grilled Balsamic Chicken	1	270	37	18	6
Total		1954	333	97	31.2

3.4 Metabolic Biomarker Analysis

All metabolic biomarker analyses were performed on EDTA-treated plasma samples, with each measurement conducted in duplicate. Plasma glycerol concentrations, used as a proxy for lipid mobilization, were measured using commercially available colorimetric enzymatic assay kits (Novus Biologicals, Centennial, CO, USA). Absorbances were read and quantified on a generated standard curve with the use of a BioTek Synergy LX plate reader (Agilent Technologies, Santa Clara, CA, USA). Glucose levels were measured using a YSI 2500 Glucose Analyzer (Xylem Analytics, Yellow Springs, OH, USA) at Centricity Research Toronto LMC (Toronto, Ontario). Insulin concentrations were quantified using commercially available

enzyme-linked immunosorbent assay (ELISA) kits (Sigma-Aldrich, St. Louis, MO, USA) that utilized a sandwich-format with capture and detection antibodies specific to human insulin. Optical density was read using the same BioTek Synergy LX plate reader, and values were calculated from a corresponding standard curve. Standard curves were optimized using Belysa Immunoassay Curve Fitting Software (EMD Millipore Corporation, Etobicoke, ON, Canada) for all biomarkers analysed using assays (insulin and glycerol), ensuring consistent concentration calculations across plates. As an index of insulin resistance, homeostatic model assessment for insulin resistance (HOMA-IR) values were calculated post-analytically using glucose and insulin concentrations obtained from the fasted blood samples (pre-exercise, and 24-hours). Intra-assay coefficients of variation (CVs) were calculated for all assay-measured analytes (insulin and glycerol), and CVs for glucose, measured in duplicate using the YSI analyser, were calculated to assess analytical precision. The average CVs were 0.41% for glucose, 6.6% for insulin, and 4.3% for glycerol. When duplicates demonstrated a CV above 20%, individual values were inspected in a blinded manner to reduce potential bias, and a single replicate that best reflected the participant's overall pattern was retained for analysis. In total, 32/2384 singlets were removed from the analysis (1.3% of the dataset).

3.5 Statistical Analysis

Prior to statistical analysis, all analytes underwent plasma volume correction. Following correction, outlier treatment was performed. Data points were visually inspected and evaluated, and values ±>2 standard deviations (SD) from the mean were replaced with the corresponding upper or lower 2 SD limit. In total, 45 of 1193 measurements across all analytes (3.8% of the dataset) underwent this process. HOMA-IR values were calculated following outlier treatment of

the raw glucose and insulin concentrations to minimize propagation of extreme values into the derived variable.

Data for each analyte were subsequently assessed for normality using skewness and kurtosis z-scores. Variables with z-scores ±>3 were transformed prior to statistical analysis. Insulin concentrations were square-root transformed, as log transformation did not sufficiently normalize the distribution. Following HOMA-IR calculation, the distribution was also revealed to be non-normal, and therefore the data were log transformed. Transformed data were used exclusively for statistical analyses, whereas the Figures present non-transformed data.

Across all trials, only two blood samples were missed during collection. During the glycerol analysis, one additional sample was not analyzed due to insufficient analytical supplies, resulting in final sample sizes of 398 for glucose, 398 for insulin, and 397 for glycerol. For HOMA-IR, one value was excluded due to potential non-compliance with the overnight fasting criteria regarding the 24h blood sample, which is strictly required for proper interpretation of HOMA-IR. Missing datapoints were treated as missing at random and accommodated within the linear mixed-effects models using restricted maximum likelihood (REML) estimation.

For familiarization versus post-study testing outcomes (i.e., e1RM, maximal PO, body weight, and %BF), paired-samples t-tests were performed using GraphPad Prism version 10.5.0 (GraphPad Software Inc., San Diego, CA, USA). Statistical significance was defined at $\alpha < 0.05$.

For dietary intake data, repeated-measures one-way analyses of variance (ANOVA) were performed separately for pre-trial and trial-day intake, with trial (CHO, MILK, GY, WATER) as the independent variable. In cases where the assumption of sphericity was violated, Greenhouse-Geisser corrections were applied. Statistical significance was defined at $\alpha < 0.05$. Statistical analyses of dietary intake data were conducted using SPSS (SPSS Inc., Armonk, NY, USA).

Similarly, exercise data were analysed using repeated-measures one-way ANOVAs with trial (CHO, MILK, GY, WATER) included as the independent variable. Multiple comparisons were adjusted using the false discovery rate (FDR) method. Statistical significance was defined at $\alpha < 0.05$. Statistical analyses of exercise data were conducted using GraphPad Prism version 10.5.0 (GraphPad Software Inc., San Diego, CA, USA).

Plasma glucose, insulin, glycerol, and HOMA-IR were analysed using linear mixed-effects models (LMMs). For glucose, insulin, and glycerol, fixed main effects included trial (CHO, GY, MILK, WATER), time (Pre, 5m, 1h, 4h, 24h), and sex, with participant ID included as a random effect. For HOMA-IR, which was assessed at only two timepoints (Pre and 24h), two complementary models were employed. The first model included trial, time, and sex as fixed main effects, with participant ID as a random effect. This model was used to assess within-trial changes over time from Pre to 24h. The second model used baseline HOMA-IR as a covariate to assess the 24h timepoint for baseline-adjusted between-trial differences at 24h. When significant main effects or interactions were demonstrated, post-hoc pairwise comparisons were performed and adjusted using the FDR method. Statistical significance was defined at $\alpha < 0.05$. All statistical analyses of the metabolic biomarkers and HOMA-IR were conducted using R (R Foundation for Statistical Computing, Vienna, Austria) and corresponding figures were generated using GraphPad Prism version 10.5.0 (GraphPad Software Inc., San Diego, CA, USA).

For insulin and glucose, incremental net area under the curve (AUC) was calculated from immediately post-exercise (5m) to 4h post-exercise, with the 5m timepoint defined as baseline. As each participant completed all four trials, differences in AUC between trials were assessed using one-way repeated measures ANOVA, with multiple comparisons adjusted using the FDR method. Statistical significance was defined at $\alpha < 0.05$. The generation and statistical analyses of

all AUCs, along with the creation of corresponding graphs were conducted using GraphPad Prism version 10.5.0 (GraphPad Software Inc., San Diego, CA, USA).

Chapter 4: Results

4.1 Participant Characteristics

A total of 148 individuals initially contacted the study team expressing interest in participating in the BIONEX study. Sixty-one individuals were excluded due to not meeting the eligibility criteria, 29 did not respond to follow-up correspondence, and 3 declined participation. The remaining 57 individuals completed a thorough eligibility screening assessment. Following this screening, an additional 15 individuals were excluded for not meeting eligibility criteria, 3 did not respond to follow-up, and 2 were waitlisted due to capacity limitations. A total of 37 individuals attended the in-person familiarization session, after which 8 additional individuals were excluded due to not meeting eligibility criteria (i.e., measured anthropometric values resulted in BMIs outside of the criteria) (n=4), declining participation (n=2), or scheduling conflicts (n=2). Subsequently, 29 participants were enrolled and randomized into the study. Of these, 9 participants did not complete the study due to scheduling conflicts that had later emerged (n=5), inability to complete the study protocol (n=3), or relocation to another province (n=1). Therefore, the final sample included 20 participants who completed the study protocol, with 18 participants completing all post-study testing procedures. The full flow of participant recruitment, eligibility, enrollment and completion is illustrated in **Figure 4**. Participants were on average 22.7 ± 4.0 years of age, with a mean body mass index of 22.0 ± 2.3 kg/m^2. The sample consisted of 8 males and 12 females. All participant characteristics at the familiarization visit are summarized in **Table 4**.

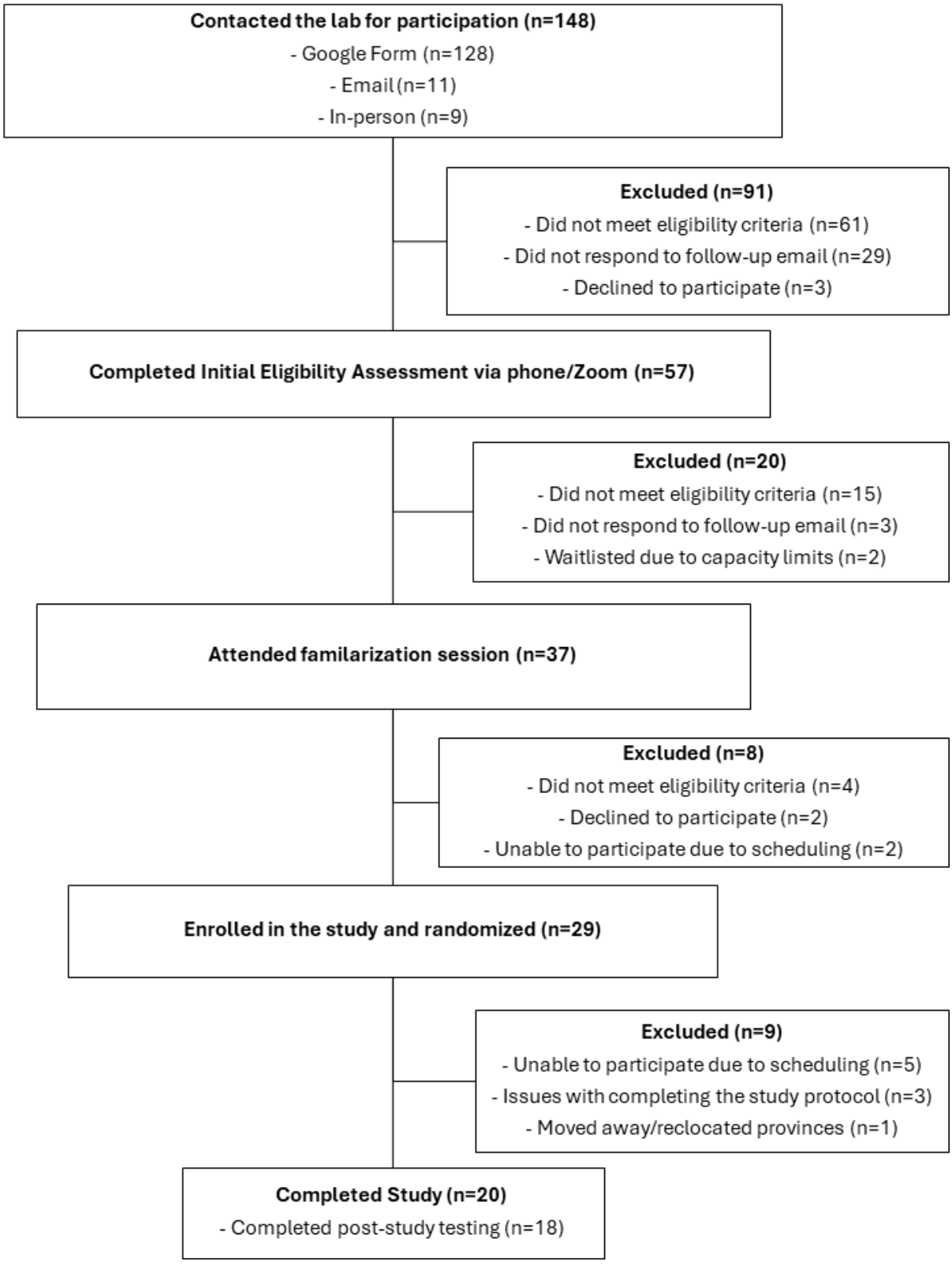


Figure 4: CONSORT flow diagram of participant recruitment, screening, enrollment, and study completion.

Table 4: Baseline participant characteristics (n=20).

Characteristics	Mean ± SD
Sex (F/M)	12/8
Age (years)	22.7 ± 4.0
Height (cm)	165.5 ± 8.8
Weight (kg)	60.3 ± 8.3
BMI (kg/m^2)	22.0 ± 2.3
Body Fat (%)	23.4 ± 9.6
Peak Power Output (W)	166 ± 41

Abbreviations: BMI: body mass index.

4.2 Exercise Data

Exercise performance variables were comparable across trials. For the resistance exercises, no significant differences were observed between trials for exercise intensity (%e1RM), volume load, or RPE for the chest press ($p>0.05$ for all), leg press ($p>0.05$ for all), and seated row ($p>0.05$ for all). Moreover, no significant differences were observed for the HIIC bouts between trials in %HRmax ($p>0.05$), cycling volume load ($p>0.05$), or RPE ($p>0.05$). Additional exercise details across trials are presented in **Table 5**.

Table 5: Comparison of number of impacts, resistance exercise volume load, and cycling volume load between all trials (n=20).

Exercises	CHO	MILK	GY	WATER	p-value
Impacts	120	120	120	120	1.00
Chest Press					
%e1RM	69 ± 5	68 ± 5	69 ± 5	70 ± 4	0.27
Volume Load (kg)	1075 ± 460	1066 ± 458	1075 ± 460	1080 ± 454	0.49
RPE	5 ± 1	5 ± 1	5 ± 1	5 ± 2	0.11
Leg Press					
%e1RM	68 ± 9	68 ± 9	68 ± 9	68 ± 9	0.33
Volume Load (kg)	2190 ± 1097	2190 ± 1097	2189 ± 1096	2190 ± 1097	0.33
RPE	5 ± 2	5 ± 1	6 ± 2	5 ± 2	0.58
Seated Row					
%e1RM	68 ± 6	68 ± 6	68 ± 6	68 ± 6	1.00
Volume Load (kg)	939 ± 320	939 ± 320	939 ± 320	939 ± 320	1.00
RPE	5 ± 2	6 ± 2	6 ± 1	5 ± 2	0.44
Cycling					
%HR max	90 ± 8	92 ± 5	93 ± 4	92 ± 6	0.078
Volume Load (kJ)	28 ± 7	28 ± 7	28 ± 7	28 ± 7	0.28
RPE	8 ± 2	8 ± 2	8 ± 2	8 ± 2	0.29

Note: Volume load was determined by sets*repetitions*weight for resistance exercises and sets*time*Watts for cycling. RPE was measured using Borg's 0-10 Category Ratio scale.[90] %HRmax and cycling RPE were taken from the last working interval (third or fourth). HRmax was estimated using 220-age.[91] Abbreviations: CHO: carbohydrate; GY: Greek yogurt; %e1RM: Percentage of estimated one repetition maximum; RPE: ratings of perceived exertion; %HRmax: percentage of heart rate maximum.

4.3 Post-Study Exercise Data & Body Composition

When strength and cycling performance measures were compared between pre-study (familiarization visit) and post-study, significant increases were observed in strength for the leg press ($p<0.001$), chest press ($p=0.002$), and seated row ($p=0.002$), as well as cycling maximal power output ($p=0.023$) (**Figure 5**). Body mass increased from pre- to post-study by 1.39 ± 1.54 kg ($p=0.001$), with no corresponding change in body fat percentage ($p=0.43$) (**Figure 6**).

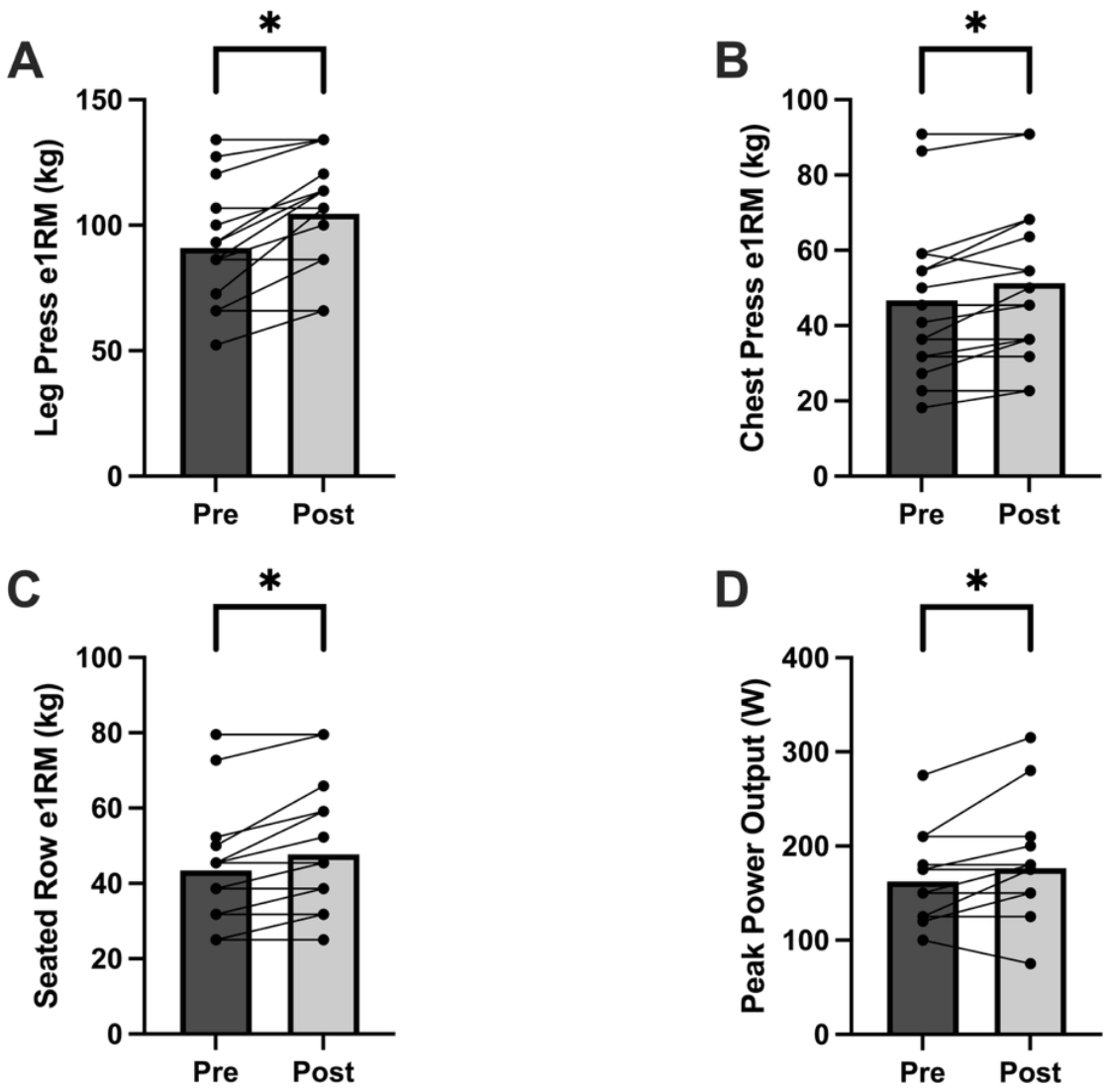


Figure 5. Changes in strength and power from familiarization (Pre) to post-study testing (n=18). Outcomes presented are e1RMs for leg press (A), chest press (B), seated row (C), and max PO during cycling (D). Individual data points are shown, with lines connecting paired values from pre to post for each participant. Bars represent group means. Symbols denote statistical significance ($p<0.05$): * = difference between pre- and post-study. Abbreviations: e1RM: estimated 1-repetition maximum; PO: power output.

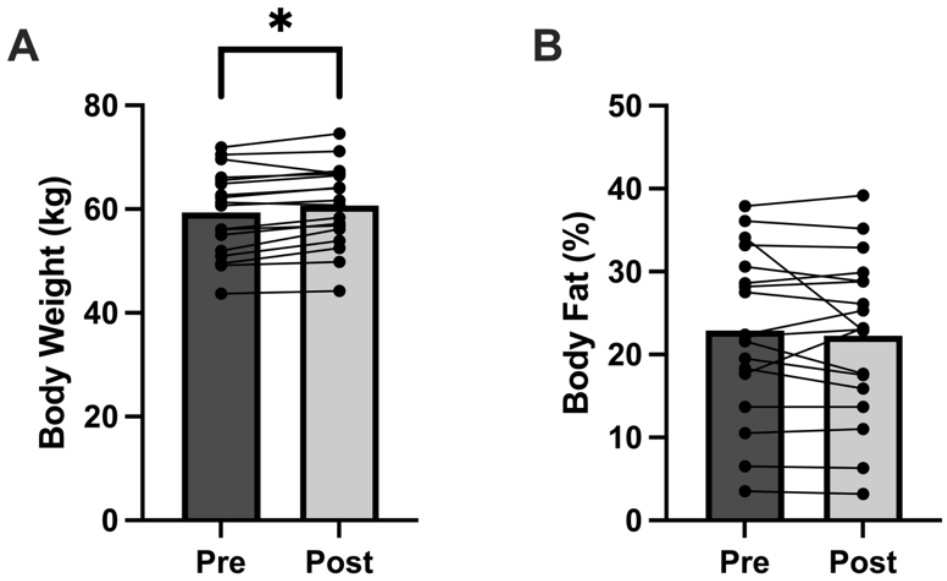


Figure 6. Changes in body weight (A) and body fat percentage (B) from familiarization (Pre) to post-study testing (n=18). Individual data points are shown, with lines connecting paired values from pre to post for each participant. Bars represent group means. Symbols denote statistical significance ($p<0.05$): * = difference between pre- and post-study.

4.4 Dietary Data

4.4.1 Pre-Trial Dietary Intake

There were no significant differences observed in energy, macronutrient, or micronutrient intake on the days prior to each trial ($p>0.05$ for all), except for sodium intake ($p=0.036$) (**Table 6**). Sodium intake differed between trials, with higher intake observed during pre-trial days for the MILK and GY trials compared to CHO ($p=0.044$ and $p=0.048$, respectively), and higher intake during pre-trial days for GY compared to WATER ($p=0.035$).

Table 6: Dietary intake from the day before the trial (*n*=20).

Dietary Variable	CHO	MILK	GY	WATER	p-value
Energy (kcal)	1533 ± 544	1620 ± 633	1498 ± 519	1443 ± 543	0.37
Protein (g)	64 ± 29	68 ± 30	65 ± 31	58 ± 24	0.46
Fat (g)	57 ± 28	61 ± 34	59 ± 30	55 ± 28	0.80
Saturated Fat (g)	17 ± 9	20 ± 12	21 ± 12	18 ± 12	0.39
Trans Fat (g)	0 ± 0	1 ± 1	1 ± 1	1 ± 0	0.70
Carbohydrate (g)	194 ± 67	203 ± 77	182 ± 59	182 ± 68	0.24
Sugar (g)	66 ± 35	75 ± 49	62 ± 30	72 ± 35	0.28
Fibre (g)	11 ± 6	12 ± 6	12 ± 5	11 ± 5	0.70
Calcium (mg)	437 ± 275	536 ± 348	536 ± 400	452 ± 220	0.35
Iron (mg)	9 ± 5	9 ± 5	9 ± 5	9 ± 5	0.87
Sodium (mg)	2049 ± 884 [a]	2659 ± 1453 [bc]	2697 ± 1479 [c]	2190 ± 1181 [ab]	**0.036**
Potassium (mg)	1569 ± 711	1697 ± 731	1586 ± 789	1694 ± 743	0.68
Vitamin D (IU)	98 ± 130	129 ± 146	119 ± 134	134 ± 147	0.33
Magnesium (mg)	166 ± 92	172 ± 100	168 ± 99	177 ± 86	0.90
Phosphorus (mg)	699 ± 381	738 ± 418	735 ± 479	673 ± 308	0.75

Note: Values are mean ± SD. Abbreviations: CHO: carbohydrate; GY: Greek yogurt. *p*-values <0.05 are bolded. Different letters represent significant differences between trials.

4.4.2 Trial Day Dietary Intake

On the day of the trial, total energy intake did not differ between trial arms ($p>0.05$). However, as expected, due to differences in the nutritional compositions of the post-exercise supplements, significant differences were observed in macronutrient and micronutrient intakes across arms ($p<0.001$ for all) except for trans-fat ($p>0.05$). Details pertaining to dietary intake on trial days are presented in **Table 7**.

Table 7: Comparison of dietary intake for the day of the trial (i.e., the day of exercise; *n*=20).

Dietary Variable	CHO	MILK	GY	WATER	p-value
Energy (kcal)	2017 ± 237	1969 ± 262	1979 ± 214	1975 ± 242	0.23
Protein (g)	58 ± 10 [a]	101 ± 14 [b]	107 ± 9 [c]	78 ± 12 [d]	**<0.001**
Fat (g)	35 ± 7 [a]	34 ± 7 [b]	33 ± 7 [ab]	46 ± 7 [c]	**<0.001**
Saturated Fat (g)	7 ± 1 [a]	5 ± 1 [b]	5 ± 1 [c]	8 ± 1 [d]	**<0.001**
Trans Fat (g)	0 ± 0	0 ± 0	0 ± 0	0 ± 0	0.11
Carbohydrate (g)	385 ± 38 [a]	332 ± 39 [b]	333 ± 33 [b]	334 ± 39 [b]	**<0.001**
Sugar (g)	88 ± 14 [a]	161 ± 17 [b]	149 ± 13 [c]	111 ± 18 [d]	**<0.001**
Fibre (g)	27 ± 4 [a]	28 ± 5 [b]	31 ± 5 [c]	35 ± 6 [d]	**<0.001**
Calcium (mg)	242 ± 27 [a]	1937 ± 241 [b]	988 ± 30 [c]	319 ± 36 [d]	**<0.001**
Iron (mg)	12 ± 2 [a]	11 ± 2 [bc]	11 ± 2 [c]	15 ± 2 [d]	**<0.001**
Sodium (mg)	1839 ± 315 [a]	2379 ± 355 [bd]	2045 ± 291 [c]	2343 ± 313 [d]	**<0.001**
Potassium (mg)	1981 ± 365 [a]	4511 ± 548 [b]	3280 ± 280 [c]	2622 ± 426 [d]	**<0.001**
Vitamin D (IU)	0 ± 0 [a]	1032 ± 267 [b]	0 ± 0 [a]	0 ± 0 [a]	**<0.001**
Magnesium (mg)	64 ± 15 [ad]	234 ± 25 [b]	128 ± 4 [c]	67 ± 16 [d]	**<0.001**
Phosphorus (mg)	63 ± 14 [ad]	1537 ± 210 [b]	939 ± 9 [c]	66 ± 14 [d]	**<0.001**

Note: Values are mean ± SD. Abbreviations: CHO: carbohydrate; GY: Greek yogurt. *p*-values <0.05 are bolded. Different letters represent significant differences between trials.

4.5 Blood Volume and Hematocrit Determination

A significant main effect of time was observed for hematocrit (p=0.001). Across all trial arms, hematocrit increased at 5m compared to pre-exercise (p<0.001), followed by reductions at 1h and 4h compared to both pre-exercise and 5m (p<0.001 for all) (**Figure 7**). No significant effects of trial nor interactions were observed (p>0.05 for all).

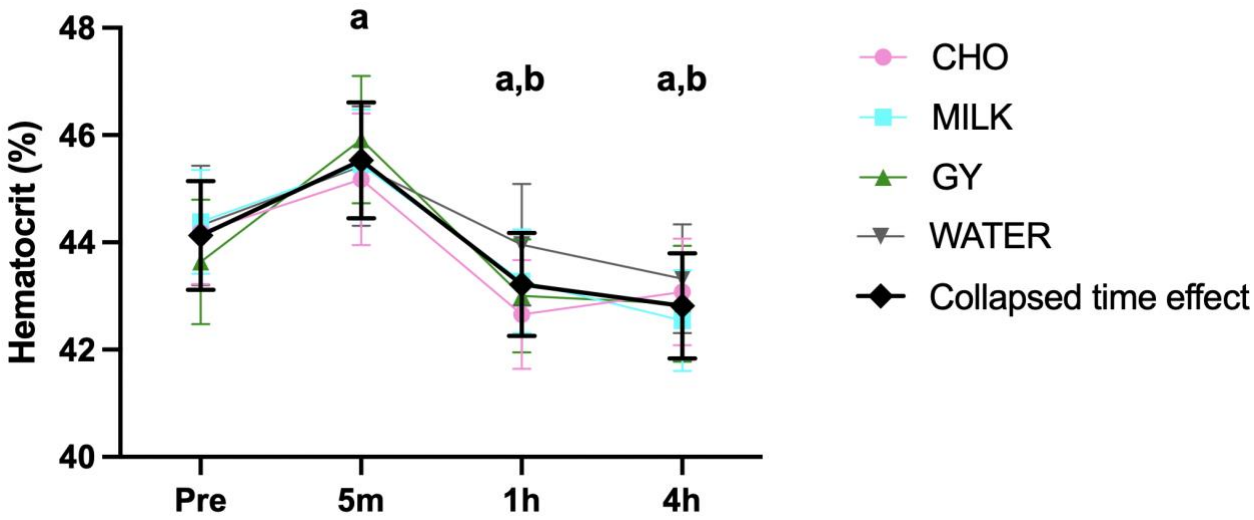


Figure 7. Changes in hematocrit percentage across timepoints on the day of exercise (pre-exercise [Pre], 5m, 1h, and 4h; n=20). Values from CHO, MILK, GY, and WATER trials are shown and statistical comparisons reflect data collapsed across trials. Letters denote statistical significance ($p<0.05$): a = different from Pre, and b = different from 5m.

4.6 Metabolic Biomarker Responses

4.6.1 Plasma Glucose Concentrations

Plasma glucose demonstrated significant main effects of time ($p<0.001$) and trial ($p=0.036$), with no effect of sex ($p=0.81$). A significant trial-by-time interaction was also observed ($p=0.041$) (**Figure 8**).

Regarding between-trial differences over time, at 1h post-exercise, plasma glucose was lower in the GY trial compared to CHO ($p=0.003$), MILK ($p=0.006$), and WATER ($p=0.002$). At 4h post-exercise, plasma glucose was lower in the CHO trial compared to MILK ($p=0.037$) and WATER ($p=0.041$). No differences were observed at Pre, 5m, or 24h.

Within-trial changes over time were observed in select conditions. In the CHO trial, plasma glucose decreased at 4h compared to both Pre and 5m (4h vs. Pre [$p=0.037$], 4h vs. 5m [$p=0.037$]) and returned to baseline by 24h ($p=0.95$). The GY trial demonstrated a drop at 1h

compared to all other timepoints (1h vs. Pre [p<0.001], 5m [p<0.001], 4h [p<0.001], 24h [p<0.001]), with values returning to baseline at 4h and remaining similar at 24h (Pre vs. 4h [p=0.75], Pre vs. 24h [p=0.62]). In the WATER and MILK trial, plasma glucose remained unchanged across all timepoints.

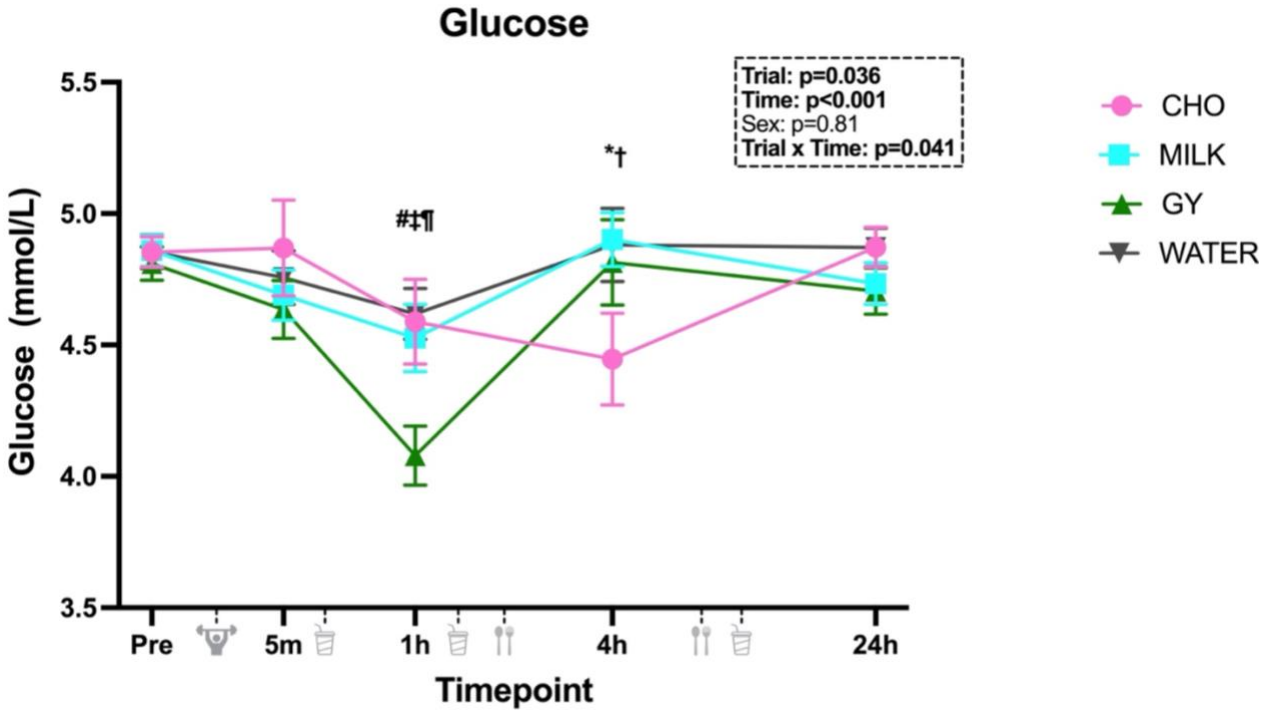


Figure 8. Absolute concentrations of plasma glucose over time (n=20). Values are mean±SEM. When a significant trial-by-time interaction was present, only symbols that denote significant differences between trials at a given timepoint are shown (p<0.05): * = CHO vs. MILK, # = CHO vs. GY, † = CHO vs. WATER, ‡ = MILK vs. GY, § = MILK vs. WATER, ¶ = GY vs. WATER.

4.6.2 Plasma Insulin Concentrations

Plasma insulin demonstrated significant main effects of trial (p<0.001) and time (p<0.001), with no significant effect of sex (p=0.50). Significant trial-by-time (p<0.001) and time-by-sex (p=0.004) interactions were also observed (**Figure 9**).

Regarding between-trial differences over time, at 1h post-exercise, plasma insulin was highest in CHO compared to GY (p=0.001), MILK (p<0.001), and WATER (p<0.001).

Additionally, plasma insulin was higher in GY compared to MILK ($p<0.001$) and WATER ($p<0.001$), and higher in MILK compared to WATER ($p<0.001$). At 4h post-exercise, plasma insulin was lower in WATER compared to CHO ($p=0.001$), and higher in CHO compared to GY ($p=0.036$).

Within-trial changes over time were observed across all conditions. In the CHO trial, plasma insulin peaked at 1h (1h vs. Pre [$p<0.001$], 5m [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]), decreased at 4h (1h vs. 4h [$p<0.001$]) while remaining elevated relative to all other timepoints (4h vs. Pre [$p<0.001$], 5m [$p<0.001$], 24h [$p<0.001$]), followed by a return to baseline at 24h ($p=0.87$). In the MILK trial, plasma insulin increased at 4h (4h vs. Pre [$p<0.001$], 5m [$p=0.006$], 1h [$p=0.032$], 24h [$p<0.001$]), followed by a return to baseline at 24h ($p=0.26$). In the GY trial, plasma insulin peaked at 1h (1h vs. Pre [$p<0.001$], 5m [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]) and remained above baseline at 4h (4h vs. Pre [$p<0.001$]), followed by a return to baseline at 24h ($p=0.53$). In the WATER trial, plasma insulin dropped from 5m to 1h ($p=0.047$), followed by an increase at 4h above both Pre and 1h (4h vs. Pre [$p=0.047$], 1h [$p<0.001$]), and a return to baseline at 24h (Pre vs. 24h [$p=0.80$]).

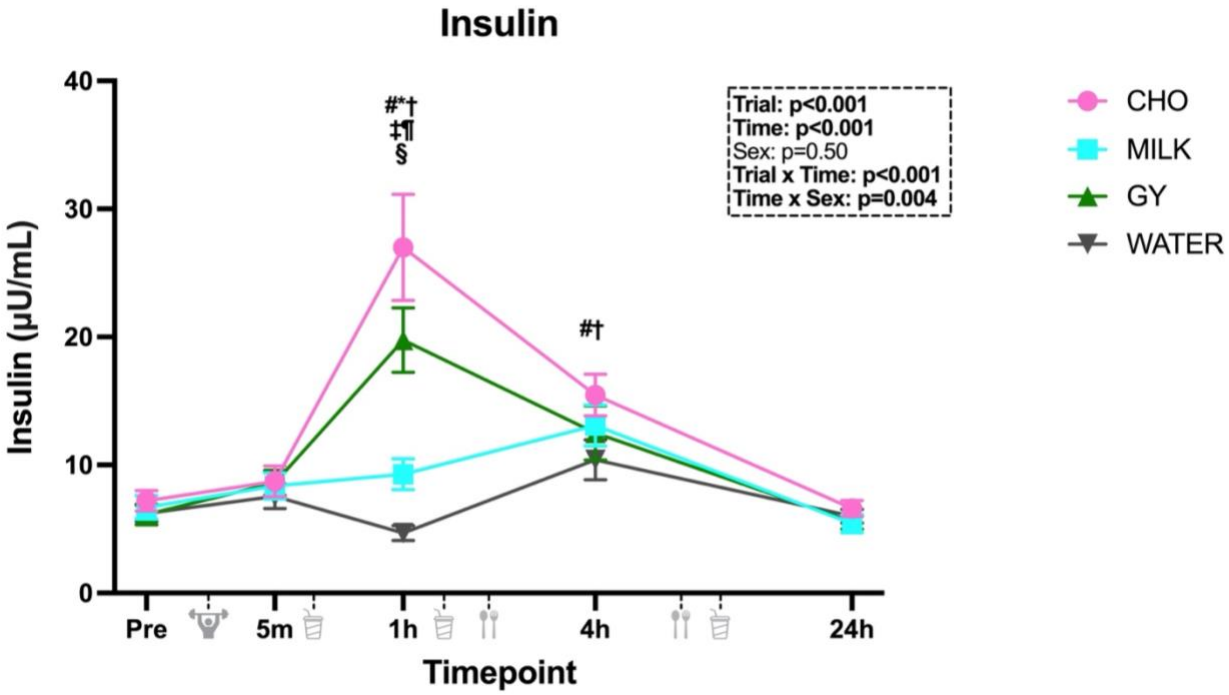


Figure 9. Absolute concentrations of plasma insulin over time (n=20). Values are mean±SEM. When a significant trial-by-time interaction was present, only symbols that denote significant differences between trials at a given timepoint are shown ($p<0.05$): * = CHO vs. MILK, # = CHO vs. GY, † = CHO vs. WATER, ‡ = MILK vs. GY, § = MILK vs. WATER, ¶ = GY vs. WATER.

Analyses of net incremental AUC revealed no significant between-trial differences for glucose ($p=0.08$) (**Figure 10a**). For insulin, a significant effect of trial was observed ($p<0.001$) (**Figure 10b**), with post-hoc comparisons reflecting the same between-trial hierarchy as seen in the insulin concentrations at 1h in **Figure 9**. Insulin AUC was greater in CHO compared to GY ($p=0.017$), MILK ($p<0.001$), and WATER ($p<0.001$), greater in GY compared to MILK ($p<0.001$) and WATER ($p<0.001$), and greater in MILK compared to WATER ($p=0.004$).

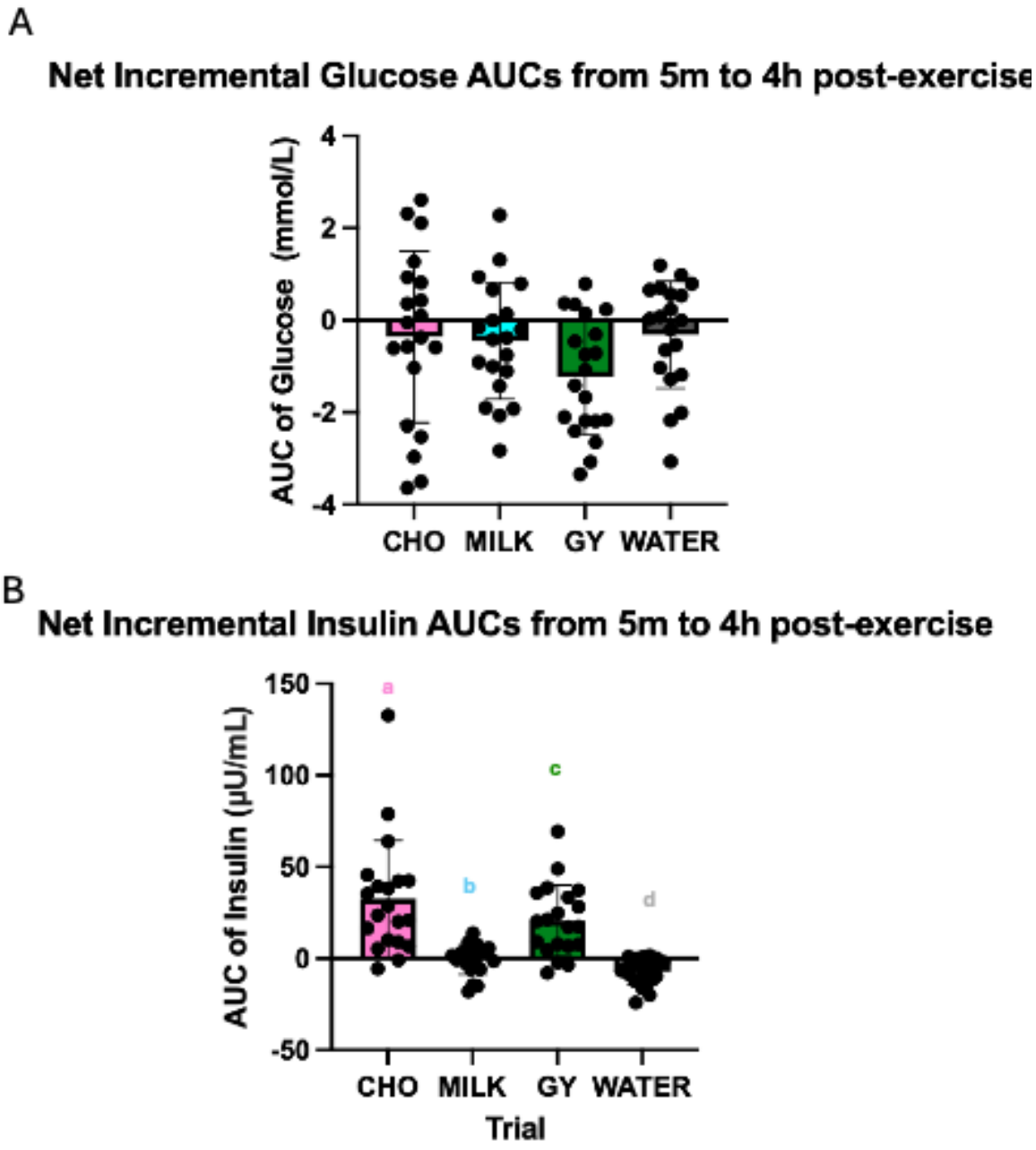


Figure 10. Net incremental area under the curve (AUC) from 5min to 4h post-exercise for (A) plasma glucose and (B) plasma insulin (n=20). Bars represent group means±SD, and dots represent individual participant values. Different letters denote significant differences between trials ($p<0.05$).

4.6.3 HOMA-IR

In the first model assessing within-trial change from Pre to 24h, HOMA-IR demonstrated a significant three-way trial*time*sex interaction (p=0.043), with no significant main effects of trial (p=0.058), time (p=0.10), or sex (p=0.45). Regarding within-trial changes over time, in males, HOMA-IR decreased from Pre to 24h during the GY trial only (Pre vs. 24h [p=0.034]). No significant within-trial changes were observed in females.

In the second model assessing baseline-adjusted between-trial differences at 24h, a significant main effect of trial (p=0.014) and a trial-by-sex interaction (p=0.036) were observed (**Figure 11**). Between-trial differences at 24h were also observed only in males. HOMA-IR was lower during the GY trial compared to CHO (p=0.007), and lower during the MILK trial compared to CHO (p=0.007). No significant between-trial differences were observed in females.

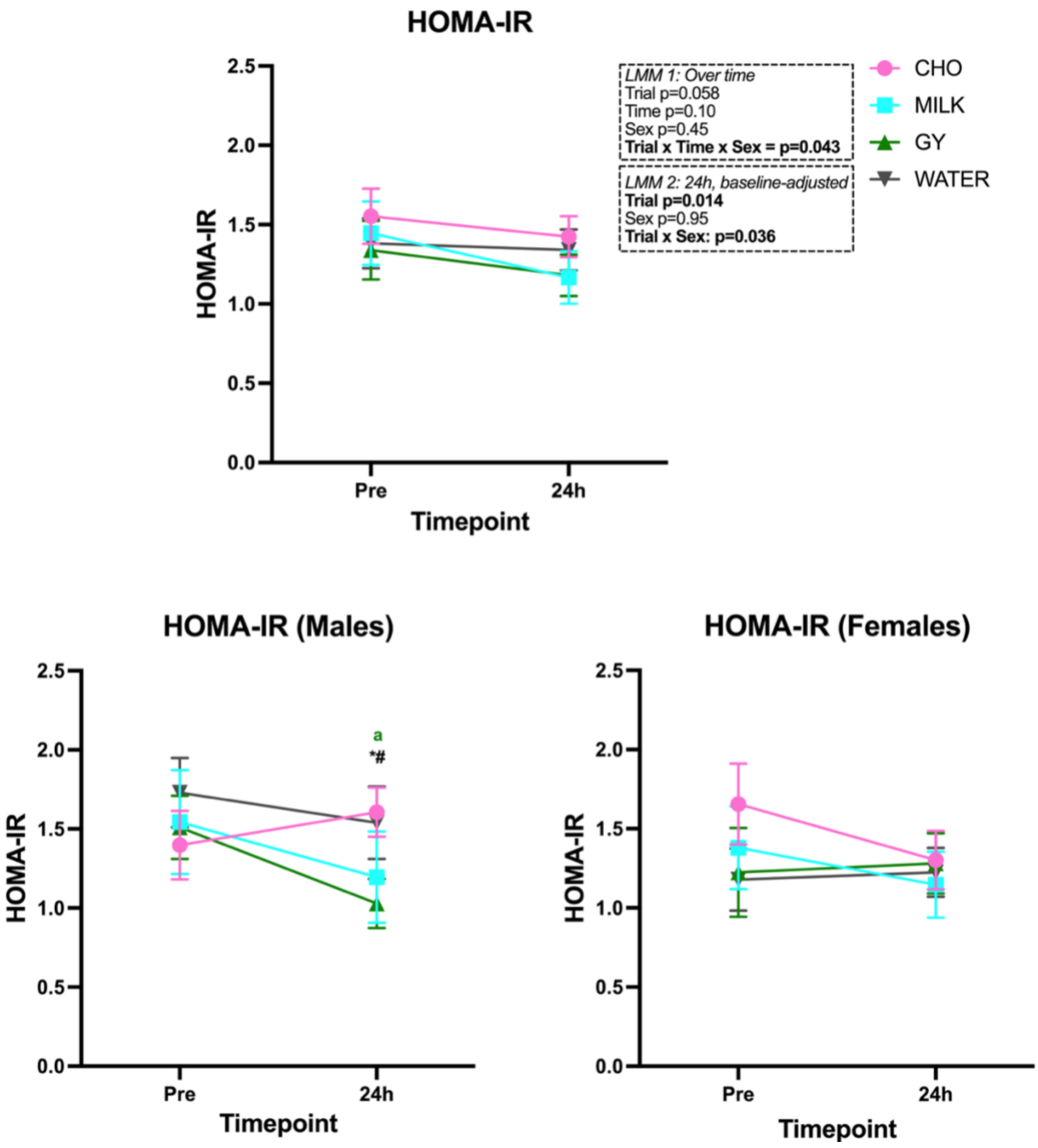


Figure 11. HOMA-IR values at Pre and 24h (n=19). Overall responses across trials are presented in (A), with sex-specific responses displayed for males (B) and females (C). Values are mean±SEM. Within-trial changes over time were assessed using Pre and 24h without baseline adjustment, whereas between-trial differences at 24h only were assessed using a baseline-adjusted model (with baseline HOMA-IR as a covariate). Letters denote significant within-trial differences over time ($p<0.05$): a = difference from Pre. Symbols denote significant between-trial differences at 24h within sex ($p<0.05$): * = CHO vs. MILK, # = CHO vs. GY, † = CHO vs. WATER, ‡ = MILK vs. GY, § = MILK vs. WATER, ¶ = GY vs. WATER. Abbreviations: HOMA-IR: homeostatic model assessment for insulin resistance.

4.6.4 Glycerol

Plasma glycerol demonstrated a significant main effect of time ($p<0.001$) and sex ($p=0.009$), with no significant effect of trial ($p=0.12$). A significant trial-by-time interaction was observed ($p<0.001$) (**Figure 12**).

Regarding between-trial differences over time, at 1h post-exercise, plasma glycerol was higher in WATER compared to CHO ($p<0.001$), MILK ($p<0.001$), and GY ($p<0.001$). No between-trial differences were observed at Pre, 5m, 4h, or 24h.

Within-trial changes over time were observed across all conditions. In the CHO trial, plasma glycerol peaked at 5m (5m vs. Pre [$p<0.001$], 1h [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]), decreased to baseline at 1h ($p=0.48$), then dipped below baseline at 4h ($p=0.001$), before returning to baseline at 24h ($p=0.19$). In the MILK trial, plasma glycerol peaked at 5m (5m vs. Pre [$p<0.001$], 1h [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]), returned to baseline at 1h ($p=0.60$), followed by a dip below baseline at 4h ($p=0.025$), and subsequently returned to baseline at 24h ($p=0.29$). In the GY trial, plasma glycerol peaked at 5m (5m vs. Pre [$p<0.001$], 1h [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]), returned to baseline at 1h ($p=0.21$), decreased below baseline at 4h ($p=0.011$), and subsequently returned to baseline at 24h ($p=0.078$). In the WATER trial, plasma glycerol peaked at 5m (5m vs. Pre [$p<0.001$], 4h [$p<0.001$], 24h [$p<0.001$]), and remained elevated at 1h (1h vs. Pre [$p<0.001$], 5m [$p=0.050$], 4h [$p<0.001$], 24h [$p<0.001$]), then returned to and remained at baseline at 4h (4h vs. Pre [$p=0.19$], 24h vs. Pre [$p=0.065$]).

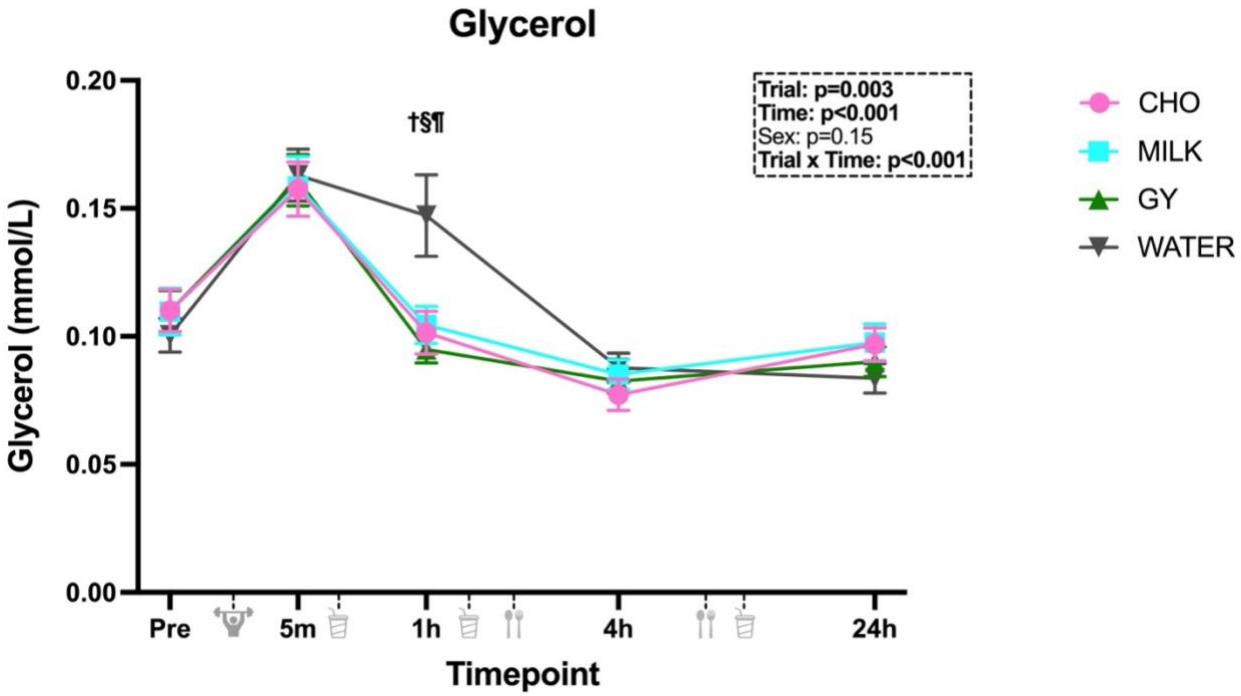


Figure 12. Absolute concentrations of plasma glycerol over time (n=20). Values are mean±SEM. Given the significant trial-by-time interaction, symbols denote significant differences between trials at a given timepoint ($p<0.05$): * = CHO vs. MILK, # = CHO vs. GY, † = CHO vs. WATER, ‡ = MILK vs. GY, § = MILK vs. WATER, ¶ = GY vs. WATER.

Chapter 5: Discussion

5.1 Summary of Results

The present crossover study investigated the acute metabolic response to a high-intensity multimodal exercise bout followed by the consumption of milk, Greek yogurt (GY), carbohydrate (CHO), or water in healthy young adults, with the aim of determining whether different post-exercise nutritional supplements, including wholefood dairy products, confer distinct and potentially metabolically advantageous responses. Several key findings emerged. Post-exercise insulin responses differed between all four supplements, following a hierarchy of CHO > GY > MILK > WATER at 1h. Meanwhile, GY elicited lower plasma glucose concentrations compared to all other conditions at the same time point. In addition, GY reduced rested/fasted HOMA-IR over 24hrs, and both dairy conditions demonstrated greater next-day insulin sensitivity compared to CHO, although this effect was driven by male participants. Finally, WATER was the only condition to sustain lipid mobilization following exercise, evidenced by the maintenance of elevated plasma glycerol concentrations at 1h compared to all other supplements. Collectively, these findings indicate that the acute metabolic response following exercise is differentially influenced by post-exercise nutritional intake. The potential mechanisms underlying these responses are discussed below.

5.2 Insulin Secretion Following Post-Exercise Nutrition

The hierarchal insulin response at 1h (CHO> GY> MILK> WATER) appears to reflect the mechanisms of two distinct insulin secretagogues. The pronounced response to CHO likely reflects its large, rapidly available carbohydrate load, which elevates plasma glucose and directly stimulates glucose-dependent insulin secretion. The dairy conditions, by contrast, increased insulin despite supplying comparatively less carbohydrate. This finding is consistent with

previous research demonstrating that dairy products stimulate insulin secretion out of proportion with their glycemic index, an effect attributed to the postprandial appearance of insulinotropic amino acids, such as leucine, valine, and isoleucine (the branched chain amino acids), as well as the potential additive stimulation of incretin hormones such as GLP-1.[16,92,93] The attenuated insulin response observed with WATER, which supplied neither carbohydrate nor protein (or any energy), is consistent with this framework.

The diverging insulin response between the two dairy conditions, despite providing an isonitrogenous protein dose, is likely explained by differences in protein composition and physical form of the food rather than protein quantity. The straining process characteristic of GY production results in a slower-digesting, casein-rich product containing proportionally less of the rapidly digested whey in milk.[94] Together with its semi-solid fermented matrix, this may slow gastric emptying and promote a more gradual release of amino acids into circulation,[95] such that the insulin response remained at or near its peak at 1h. Milk, in contrast, has a greater proportion of whey protein and, as a liquid, is digested more rapidly, resulting in a faster appearance of circulating amino acids and insulin responses that have been shown to peak as early as 30 minutes postprandially in previous studies.[16,96] Indeed, despite providing a greater carbohydrate load than GY, which would be expected to augment insulin secretion, MILK elicited lower concentrations at 1h. Accordingly, it is possible that the insulin response to MILK peaked during the unsampled interval between 5m and 1h and had already begun to decline by the time of the 1h measurement, thereby explaining its comparatively modest level relative to GY **(Figure 13**). However, as no insulin samples were collected within this interval, this interpretation cannot be confirmed and remains speculative.

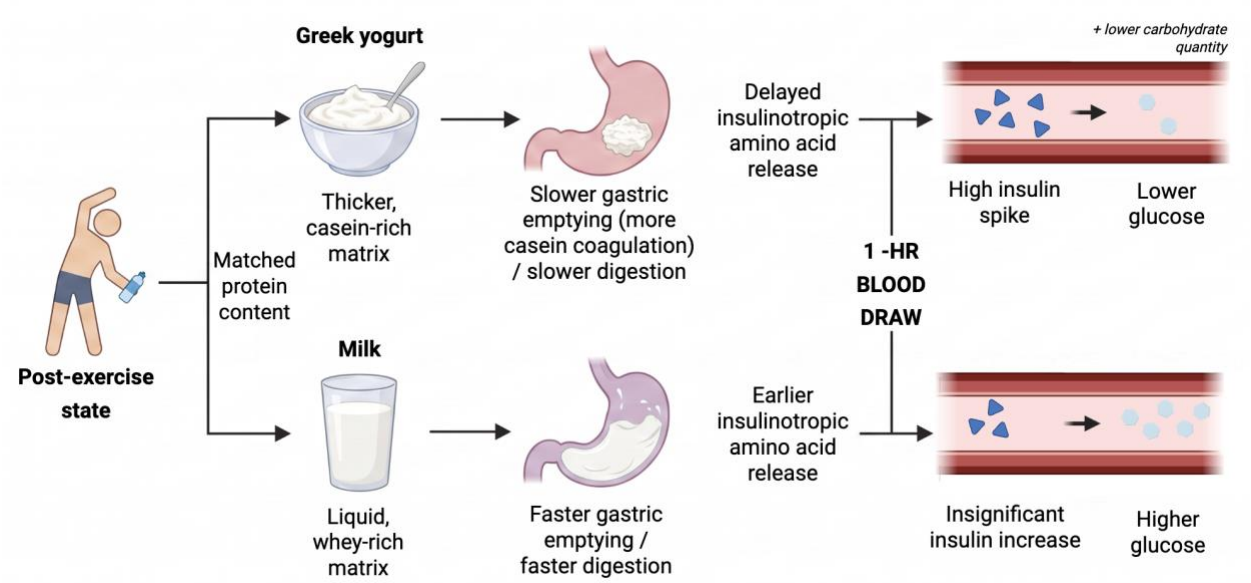


Figure 13. Milk is a liquid with a higher whey-to-casein ratio than Greek yogurt, promoting more rapid gastric emptying and nutrient absorption. In contrast Greek yogurt is a semi-solid with a higher casein-to-whey ratio relative to milk, resulting in slower gastric emptying and a more gradual sustained release of insulinotropic amino acids and glucose into circulation this lower absorption profile, together with the lower carbohydrate content of Greek yogurt, may explain the attenuated glycemic response captured at the 1h blood sample. Created with Biorender.com.

5.3 Glycemic Responses Following Post-Exercise Nutrition

Between-trial differences in plasma glucose were most apparent at 1h where plasma glucose concentrations in GY fell below all other conditions, including the CHO and energy-free WATER condition. This may suggest that the attenuation did not merely reflect the carbohydrate load, but enhanced insulin-mediated glucose disposal as well (**Figure 14**). GY supplied the lowest carbohydrate load of the caloric supplements yet elicited the second-highest insulin spike, such that insulin-stimulated glucose uptake may have exceeded the rate of glucose appearance in the circulation, transiently lowering plasma glucose below baseline. Similar findings have been reported when dairy protein is consumed without a commensurate rise in glycemia.[16,92] Notably, and consistent with previous reports of mild postprandial hypoglycemia following dairy protein ingestion,[92] nine of twenty participants in the present study recorded glucose concentrations just

below 3.9 mmol/L at 1h following GY consumption. Although these excursions were transient and occurred in young, healthy individuals (none of whom reported perceiving any symptoms at the time), and are therefore unlikely to carry the clinical weight they might in a population with impaired glucose control, they reinforce the interpretation that the reduction in glucose represented a genuine physiological response rather than an artifact of group averaging. Whether similar responses would be observed in populations with impaired glucose regulation or greater susceptibility to reactive hypoglycemia warrants further investigation. Taken together, the GY trial displayed the most favourable balance between glycemic burden and insulin secretion, consistent with our hypothesis that its lower-carbohydrate, protein-rich composition would confer an acute glycemic advantage. This also aligns with previous work demonstrating reductions in postprandial glycemia as the protein-to-carbohydrate ratio of dairy products increases.[15]

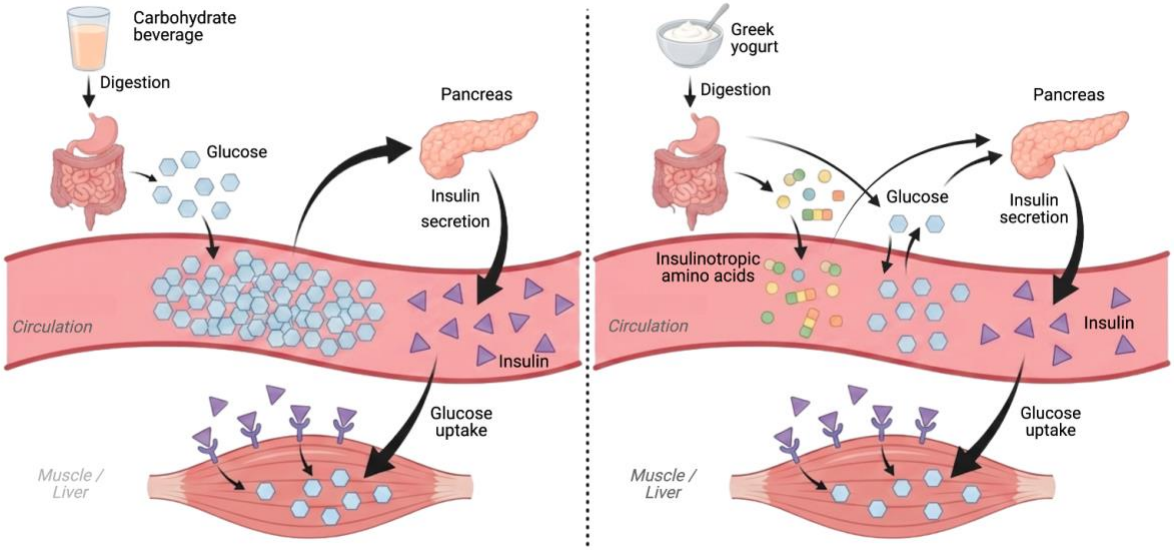


Figure 14. The high glycemic load of the carbohydrate beverage (left panel) primarily relied on glucose-mediated insulin secretion to facilitate the clearance of substantial circulating glucose. In contrast, Greek yogurt (right panel) with a higher protein and lower carbohydrate fraction, provided two insulin secretagogues (i.e., insulinotropic amino acids and glucose), which worked in concert to simulate insulin secretion and facilitate the clearance of less circulating glucose. Created with Biorender.com.

Another important finding was the lower 4h glucose concentration demonstrated in CHO relative to MILK and WATER. This may reflect a carryover effect of the robust CHO insulin response observed at 1h, which could have enhanced disposal of the glycemic load presented by the standardized lunch consumed shortly thereafter (at 2h) (**Figure 15**). These findings diverge from previous studies demonstrating that dairy consumption attenuates glycemic excursions at subsequent meals.[15,86,97] However, if MILK elicited an earlier and more transient insulin response, as proposed above, its glucose-disposing effects may have diminished by the time lunch was consumed (at 2h), even with the second supplement dose provided at 1h. In contrast, the more robust and sustained insulin response observed in CHO at 1h may have persisted towards lunch and was further compounded by the second supplement dose, thereby contributing to enhanced glucose clearance and the attenuation seen at 4h. The same reasoning can be inferred regarding the comparison with WATER, which, having provided neither carbohydrate nor an insulinotropic stimulus, elicited no additional insulin response to facilitate glucose disposal at the subsequent meal.

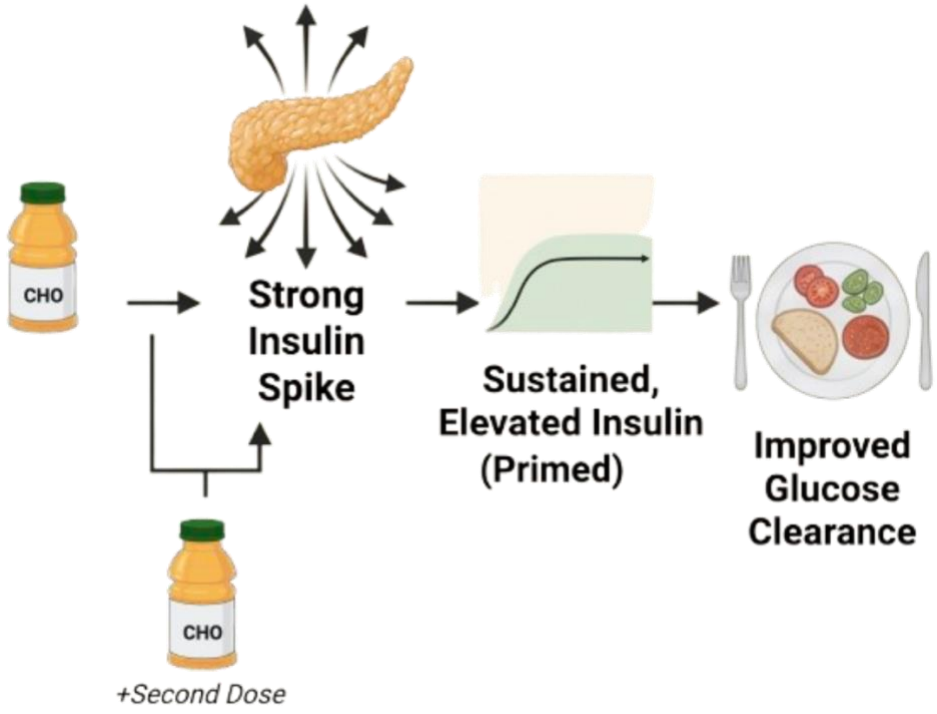


Figure 15. The decrease in circulating glucose in the carbohydrate (CHO) trial at 4h may reflect the pronounced insulin response observed at 1h following the initial supplement dose, which was likely further amplified by the second dose consumed immediately after the 1h blood sample. The standardized lunch was consumed 1h after the second supplement dose and was therefore digested in a high-insulin milieu, likely facilitating more efficient glucose clearance from the circulation. Created with Biorender.com.

5.4 Effects of Post-Exercise Nutrition on 24-Hour Insulin Sensitivity

While HOMA-IR is a surrogate measure of glucose-insulin homeostasis, whereby higher values denote greater insulin resistance and lower values denote greater insulin sensitivity, for clarity, all changes in HOMA-IR are discussed herein in terms of insulin sensitivity. The findings from the present study suggest that, in male participants, the two wholefood dairy supplements were the only interventions associated with preservation of the exercise-induced improvement in insulin sensitivity into the following day. The divergence between the dairy and CHO conditions, whereby GY and MILK yielded greater insulin sensitivity at 24h, aligns with a recurring theme in the post-exercise nutrition literature that replenishing carbohydrate after exercise can blunt the

insulin-sensitizing adaptation otherwise observed for up to 48h following exercise.[32] The CHO beverage delivered the largest carbohydrate load with rapid absorption, replenishing glycogen stores and reducing the post-exercise deficit, and with it the stimulus for enhanced next-day insulin sensitivity. In contrast, the dairy supplements, GY in particular, maintained a greater carbohydrate deficit which limited the degree of glycogen replenishment during the acute post-exercise period and thereby better promoted next-day insulin sensitivity. This interpretation is consistent with broader evidence suggesting that dairy consumption may favourably influence insulin sensitivity, including a meta-analysis of randomized controlled trials reporting a net reduction in HOMA-IR following higher dairy intake, albeit with substantial heterogeneity and outside of an exercise context.[98] It is worth noting, however, that the reduction in HOMA-IR from Pre to 24h reached statistical significance only in the GY trial, whereas MILK exhibited a non-significant trend in the same direction ($p=0.055$). This small difference may reflect the even lower carbohydrate content of GY relative to MILK.

Interestingly, WATER, which represented the greatest post-exercise carbohydrate deficit did not demonstrate an improvement in insulin sensitivity. This suggests that carbohydrate/energy restriction alone cannot fully explain the present findings and reinforces the notion that dairy supplements likely engaged additional mechanisms that preserved or bolstered exercise-induced insulin sensitivity. Potential contributors include amino acid stimulation of insulin secretion, however, further research deciphering the underlying mechanisms is warranted. These findings also carry practical significance. In real-world settings, abstaining from food following exercise proves difficult, unless perhaps performed before an extended fasting period, such as prior to sleep. Moreover, post-exercise nutrient intake is commonly encouraged to support recovery processes. The pertinent question is therefore not whether to eat after exercise,

but rather what to consume. Viewed in this context, the present findings suggest that appropriately selected post-exercise nutrition, such as dairy, can preserve and potentially enhance exercise-induced improvements in insulin sensitivity while providing energy and nutrients that support recovery.

Although research examining post-exercise dairy consumption in an acute context remains scarce, it is important to situate the present findings within the broader dairy literature, where the relationship between dairy consumption and insulin sensitivity is not uniformly concordant. A systematic review of weight-stable chronic dairy intervention trials reported mixed findings across a range of study populations, with four of ten studies demonstrating improvements in insulin sensitivity, five reporting no effect, and one observing a reduction.[99] The present findings align with the former. They are less consistent with null and adverse findings, including a multi-week crossover study in adults with overweight in which a high-dairy diet increased fasting insulin resistance.[100] Nevertheless, these studies lack an exercise component and are chronic in nature. Thus, they do not necessarily conflict with the present findings but rather suggest that the metabolic effects of dairy may depend on factors such as intervention duration and the surrounding exercise context.

Among males, both dairy supplements improved insulin sensitivity, whereas no corresponding effects reached significance in females. However, this should not be interpreted as evidence of an absence of response in females, nor as evidence that the sexes responded in opposing directions. Nevertheless, a sex-dependent response to dairy is biologically plausible, as prior crossover work examining dairy across age and sex has demonstrated that these factors can influence glycemic and insulinemic responses.[101] More broadly, biological sex is an established determinant of insulin sensitivity, with direct measures showing that females exhibit greater

whole-body and skeletal-muscle insulin sensitivity than males.[102] However, the present findings neither replicate nor contradict this body of work, as these studies describe baseline differences in insulin sensitivity rather than responses to an acute nutritional intervention. Moreover, the present analyses reflect within-sex responses rather than a direct comparison between males and females: post-hoc comparisons indicated that males exhibited significant changes over time in select conditions, while no significant corresponding changes in females were exhibited. A likely explanation for the differential findings between the sexes is limited statistical power. The present analysis was conducted as a secondary outcome within a study originally designed and powered for bone and inflammatory endpoints. Moreover, sex was included as an exploratory term rather than an a priori comparison. This limitation is compounded by the greater physiological variability inherent in female metabolic data, including cyclical fluctuations in insulin sensitivity that are attenuated, but not eliminated, by scheduling trials within the early follicular phase of the menstrual cycle. Collectively, these factors may have increased variability in females and reduced the ability to detect effects of sufficient magnitude. Thus, the present findings provide preliminary evidence of a favourable response in males, whereas the corresponding response in females remains undetermined.

5.5 Nutritional Modulation of Post-Exercise Lipid Mobilization

In terms of plasma glycerol, used as a proxy for lipid mobilization, all four conditions demonstrated a pronounced increase immediately following exercise, reflecting the lipolytic stimulus of the high-intensity exercise bout. WATER was the only condition to sustain elevated glycerol concentrations thereafter. Despite substantial differences in carbohydrate content, CHO and both dairy supplements suppressed glycerol to a comparable degree and returned lipid

mobilization toward baseline, whereas the energy-free WATER condition did not. This finding is consistent with evidence suggesting that maintenance of an energy deficit is a primary driver of the post-exercise lipolytic response, with carbohydrate restriction contributing secondarily.[67,73,103] Mechanistically, the post-exercise increase in lipolysis is thought to be driven largely by catecholamine-mediated stimulation, a response that is sustained when energy remains limited. In the present study, all three caloric supplements provided energy as well as elicited an insulin response, the combination of which compounds anti-lipolytic effects. The converging responses at 4h further support this interpretation. Following consumption of the standardized lunch at 2h, glycerol concentrations in the WATER condition returned toward baseline and converged with the other trials, suggesting that the introduction of energy, albeit later (at 2h), blunted lipid mobilization. This observation aligns with previous reports where general post-exercise feeding, likely as a function of caloric replenishment, suppressed circulating glycerol and free fatty acid concentrations.[70]

Taken together, these findings partially support our hypothesis: while WATER most effectively preserved the exercise-induced lipolytic response, neither dairy supplement (with a lower carbohydrate content than the CHO arm) conferred an advantage over CHO. Rather, the present findings suggest that, in the context of post-exercise lipid mobilization, energy is a more important determinant than specific macronutrient or micronutrient composition (**Figure 16**).

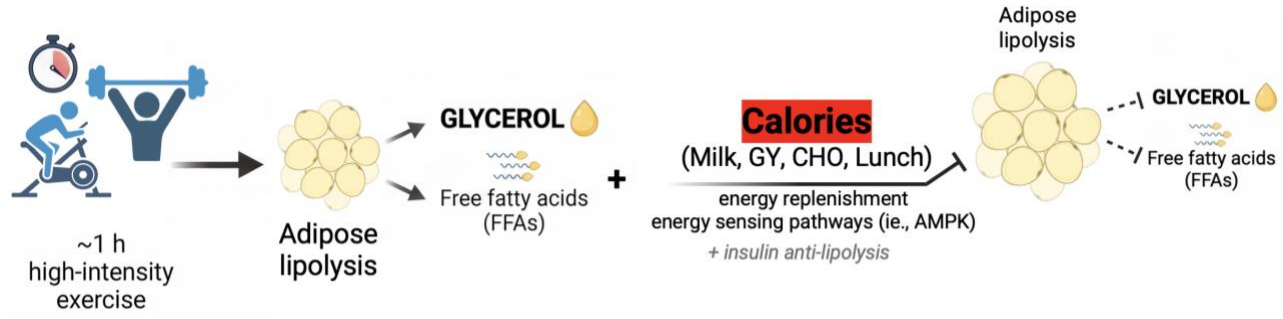


Figure 16. All trial supplements apart from water (milk, Greek yogurt, and carbohydrate) blunted lipid mobilization indifferently, and glycerol responses in the water trial converged with the other arms (i.e., blunted) following the standardized lunch consumption at 2h, suggesting that caloric repletion may be the primary mediator of post-exercise lipid mobilization. Created with Biorender.com.

5.6 Strengths and Limitations

This study has several strengths. To our knowledge, it is the first to directly compare milk and Greek yogurt in a post-exercise context in healthy young adults, assessing glycemic, insulinemic, insulin sensitivity, and lipid mobilization outcomes. The randomized crossover design minimized interindividual variability, and the inclusion of a standardized meal plan throughout the study day helped control dietary intake and reduce potential confounding from differences in nutrient intake between trials.

Several limitations should also be acknowledged. The sampling schedule may not have captured peak or nadir responses for all outcomes, limiting interpretation of the full profiles of glucose, insulin, and glycerol over the 24-hour period. Additionally, glycerol was used as a proxy for lipid mobilization and therefore is not indicative of actual fat oxidation responses. Finally, although the study was adequately powered for the primary analyses targeting its original objectives (i.e., bone and inflammatory endpoints), the study was not technically powered for the secondary analysis of metabolic outcomes nor the relative exploratory sex-specific outcomes.

5.7 Implications

Several implications emerge from the findings of this study. First, the consumption of wholefood dairy products post-exercise, particularly Greek yogurt, may promote more balanced glycemic control in the immediate recovery period through mechanisms attributable to its high protein content together with the provision of a lower quantity of carbohydrate than the other nutritional supplements. In contrast, repeated doses of a carbohydrate supplement following exercise may elicit insulin-mediated priming effects that facilitate the clearance of circulating glucose following a subsequent meal. Moreover, the consumption of dairy products post-exercise may promote sustained exercise-induced improvements in next-day HOMA-IR, indicative of a greater insulin-sensitive environment. Furthermore, high-intensity exercise yields substantial increases in post-exercise lipid mobilization, which appears to be attenuated by energy repletion of any kind. Collectively, these findings demonstrate that post-exercise nutrition is an important determinant of the acute metabolic response following exercise and further corroborate the integration of nutritional strategies alongside exercise to support metabolic health. Moreover, because post-exercise nutrition is difficult to avoid in real-world settings, these findings shift the focus from whether individuals should eat after exercise to what they should consume to maximize the metabolic benefits of exercise. Importantly, by examining wholefood dairy products rather than isolated nutritional supplements, the present study extends beyond traditional post-exercise nutrition strategies (e.g., whey protein isolate, carbohydrate beverages, electrolyte drinks) and demonstrates that readily available wholefoods that can be easily integrated into a diet, particularly Greek yogurt and milk, may present as practical and effective options for supporting favourable post-exercise metabolic responses. As such, these findings may help inform future evidence-based nutritional recommendations for individuals seeking to

promote their metabolic health through the combined application of exercise and dietary strategies.

5.8 Future Directions

The present study contributes to a growing body of literature examining the role of post-exercise nutrition in shaping acute metabolic responses. Future research should explore these findings in larger cohorts to improve statistical power and permit more robust examination of potential sex-specific metabolic responses. More thorough and direct measures of the metabolic outcomes of interest should also be carried out. Additional sampling timepoints would provide a more comprehensive characterization of the temporal insulin and glucose responses, enabling any transient fluctuations between the current sampling intervals to be captured. Furthermore, the use of continuous glucose monitoring could provide a more detailed assessment of glycemic responses throughout the post-exercise period. Similarly, incorporation of more direct measures of insulin sensitivity and substrate utilization, such as hyperinsulinemic-euglycemic clamps and indirect calorimetry (for respiratory exchange ratio measurements), would strengthen the interpretation of the observed responses. Additionally, assessment of other hormones involved in glycemic regulation, including glucagon and GLP-1, may provide a more comprehensive understanding of the integrated mechanisms underlying the observed responses. Finally, longer-term interventions are warranted to determine whether the acute metabolic responses observed translate into meaningful adaptations following habitual dairy consumption and exercise training, and whether these effects differ between dairy products over time.

5.9 Conclusion

In conclusion, the present study demonstrated that post-exercise nutrition differentially modulated acute metabolic responses following a high-intensity multimodal exercise bout in healthy young adults. Distinct effects were observed across glycemic and insulin excursions, HOMA-IR, and lipid mobilization. Notably, the dairy supplements supported favorable responses in several domains, including postprandial glucose and insulin excursions, as well as HOMA-IR. The consumption of Greek yogurt following exercise yielded a substantial increase in insulin that was coupled with a marked reduction in circulating glucose, suggesting improved glycemic control during the early post-exercise period. Among males, the consumption of Greek yogurt facilitated a decrease in HOMA-IR over time, while both Greek yogurt and milk resulted in lower HOMA-IR compared to post-exercise carbohydrate supplementation, concluding that post-exercise dairy consumption may help sustain reductions in insulin resistance, promoting a more insulin-sensitive metabolic environment during recovery. Meanwhile, exercise elicits substantial increases in lipid mobilization in the immediate recovery period, which appears to be primarily attenuated by energy repletion. Collectively, these findings highlight the importance of post-exercise nutrition in shaping acute metabolic responses in healthy people and provide a foundation for future research exploring the role of wholefood dairy products in supporting metabolic health.

References

1. Gaitan JM, Weltman A, Malin SK. Enhancing Exercise Responsiveness across Prediabetes Phenotypes by Targeting Insulin Sensitivity with Nutrition. *J Diabetes Res*. 2017;2017:1-8. doi:10.1155/2017/8314852

2. Islam H, Gillen JB. Skeletal muscle mechanisms contributing to improved glycemic control following intense interval exercise and training. *Sports Med Health Sci*. 2023;5(1):20-28. doi:10.1016/j.smhs.2023.01.002

3. Gillen JB, Estafanos S, Govette A. Exercise-nutrient interactions for improved postprandial glycemic control and insulin sensitivity. *Appl Physiol Nutr Metab*. 2021;46(8):856-865. doi:10.1139/apnm-2021-0168

4. Swarup S, Ahmed I, Grigorova Y, Zeltser R. Metabolic Syndrome. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 2, 2025. http://www.ncbi.nlm.nih.gov/books/NBK459248/

5. Hall H, Perelman D, Breschi A, et al. Glucotypes reveal new patterns of glucose dysregulation. Locasale J, ed. *PLOS Biol*. 2018;16(7):e2005143. doi:10.1371/journal.pbio.2005143

6. Van Dijk JW, Manders RJF, Tummers K, et al. Both resistance- and endurance-type exercise reduce the prevalence of hyperglycaemia in individuals with impaired glucose tolerance and in insulin-treated and non-insulin-treated type 2 diabetic patients. *Diabetologia*. 2012;55(5):1273-1282. doi:10.1007/s00125-011-2380-5

7. Burr JF, Rowan CP, Jamnik VK, Riddell MC. The Role of Physical Activity in Type 2 Diabetes Prevention: Physiological and Practical Perspectives. *Phys Sportsmed*. 2010;38(1):72-82. doi:10.3810/psm.2010.04.1764

8. Wojtaszewski JFP, Nielsen JN, Richter EA. Invited Review: Effect of acute exercise on insulin signaling and action in humans. *J Appl Physiol*. 2002;93(1):384-392. doi:10.1152/japplphysiol.00043.2002

9. Chan HH, Burns SF. Oxygen consumption, substrate oxidation, and blood pressure following sprint interval exercise. *Appl Physiol Nutr Metab*. 2013;38(2):182-187. doi:10.1139/apnm-2012-0136

10. Jensen TE, Richter EA. Regulation of glucose and glycogen metabolism during and after exercise. *J Physiol*. 2012;590(5):1069-1076. doi:10.1113/jphysiol.2011.224972

11. Henriksen EJ. Invited Review: Effects of acute exercise and exercise training on insulin resistance. *J Appl Physiol*. 2002;93(2):788-796. doi:10.1152/japplphysiol.01219.2001

12. Akins JD, Crawford CK, Burton HM, Wolfe AS, Vardarli E, Coyle EF. Inactivity induces resistance to the metabolic benefits following acute exercise. *J Appl Physiol Bethesda Md 1985*. 2019;126(4):1088-1094. doi:10.1152/japplphysiol.00968.2018

13. Ashcroft SP, Stocks B, Egan B, Zierath JR. Exercise induces tissue-specific adaptations to enhance cardiometabolic health. *Cell Metab*. 2024;36(2):278-300. doi:10.1016/j.cmet.2023.12.008

14. Brouwers B, Hesselink MKC, Schrauwen P, Schrauwen-Hinderling VB. Effects of exercise training on intrahepatic lipid content in humans. *Diabetologia*. 2016;59(10):2068-2079. doi:10.1007/s00125-016-4037-x

15. El Khoury D, Brown P, Smith G, et al. Increasing the protein to carbohydrate ratio in yogurts consumed as a snack reduces post-consumption glycemia independent of insulin. *Clin Nutr*. 2014;33(1):29-38. doi:10.1016/j.clnu.2013.03.010

16. Nilsson M, Stenberg M, Frid AH, Holst JJ, Björck IM. Glycemia and insulinemia in healthy subjects after lactose-equivalent meals of milk and other food proteins: the role of plasma amino acids and incretins. *Am J Clin Nutr*. 2004;80(5):1246-1253. doi:10.1093/ajcn/80.5.1246

17. Hilkens L, Praster F, Van Overdam J, et al. Graded Replacement of Carbohydrate-Rich Breakfast Products with Dairy Products: Effects on Postprandial Aminoacidemia, Glycemic Control, Bone Metabolism, and Satiety. *J Nutr*. 2024;154(2):479-490. doi:10.1016/j.tjnut.2023.12.012

18. Thorning TK, Bertram HC, Bonjour JP, et al. Whole dairy matrix or single nutrients in assessment of health effects: current evidence and knowledge gaps ,. *Am J Clin Nutr*. 2017;105(5):1033-1045. doi:10.3945/ajcn.116.151548

19. Law M, Lee YT, Vien S, Luhovyy BL, Anderson GH. The effect of dairy products consumed with high glycemic carbohydrate on subjective appetite, food intake, and postprandial glycemia in older adults. *Appl Physiol Nutr Metab*. 2017;42(11):1210-1216. doi:10.1139/apnm-2017-0210

20. Nakrani MN, Wineland RH, Anjum F. Physiology, Glucose Metabolism. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 2, 2025. http://www.ncbi.nlm.nih.gov/books/NBK560599/

21. Ritter S. Monitoring and Maintenance of Brain Glucose Supply: Importance of Hindbrain Catecholamine Neurons in This Multifaceted Task. In: Harris RBS, ed. *Appetite and Food Intake: Central Control*. 2nd ed. CRC Press/Taylor & Francis; 2017. Accessed September 2, 2025. http://www.ncbi.nlm.nih.gov/books/NBK453140/

22. Han HS, Kang G, Kim JS, Choi BH, Koo SH. Regulation of glucose metabolism from a liver-centric perspective. *Exp Mol Med*. 2016;48(3):e218. doi:10.1038/emm.2015.122

23. Röder PV, Wu B, Liu Y, Han W. Pancreatic regulation of glucose homeostasis. *Exp Mol Med*. 2016;48(3):e219-e219. doi:10.1038/emm.2016.6

24. Sanvictores T, Casale J, Huecker MR. Physiology, Fasting. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 7, 2025. http://www.ncbi.nlm.nih.gov/books/NBK534877/

25. Holeček M. Origin and Roles of Alanine and Glutamine in Gluconeogenesis in the Liver, Kidneys, and Small Intestine under Physiological and Pathological Conditions. *Int J Mol Sci*. 2024;25(13):7037. doi:10.3390/ijms25137037

26. Melkonian EA, Asuka E, Schury MP. Physiology, Gluconeogenesis. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 7, 2025. http://www.ncbi.nlm.nih.gov/books/NBK541119/

27. Stöckli J, Fazakerley DJ, James DE. GLUT4 exocytosis. *J Cell Sci*. 2011;124(24):4147-4159. doi:10.1242/jcs.097063

28. Vargas E, Podder V, Carrillo Sepulveda MA. Physiology, Glucose Transporter Type 4. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 2, 2025. http://www.ncbi.nlm.nih.gov/books/NBK537322/

29. Olson AL. Regulation of GLUT4 and Insulin-Dependent Glucose Flux. *ISRN Mol Biol*. 2012;2012:856987. doi:10.5402/2012/856987

30. Richter EA, Hargreaves M. Exercise, GLUT4, and Skeletal Muscle Glucose Uptake. *Physiol Rev*. 2013;93(3):993-1017. doi:10.1152/physrev.00038.2012

31. Chang L, Chiang SH, Saltiel AR. Insulin signaling and the regulation of glucose transport. *Mol Med Camb Mass*. 2004;10(7-12):65-71. doi:10.2119/2005-00029.Saltiel

32. Gillen JB, Estafanos S, Govette A. Exercise-nutrient interactions for improved postprandial glycemic control and insulin sensitivity. *Appl Physiol Nutr Metab*. 2021;46(8):856-865. doi:10.1139/apnm-2021-0168

33. Gerstein HC. Dysglycemia and Cardiovascular Risk in the General Population. *Circulation*. 2009;119(6):773-775. doi:10.1161/CIRCULATIONAHA.108.834408

34. Zhou Z, Sun B, Huang S, Zhu C, Bian M. Glycemic variability: adverse clinical outcomes and how to improve it? *Cardiovasc Diabetol*. 2020;19(1):102. doi:10.1186/s12933-020-01085-6

35. Shoemaker ME, Gillen ZM, Fukuda DH, Cramer JT. Metabolic Flexibility and Inflexibility: Pathology Underlying Metabolism Dysfunction. *J Clin Med*. 2023;12(13):4453. doi:10.3390/jcm12134453

36. Edwards M, Mohiuddin SS. Biochemistry, Lipolysis. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 2, 2025. http://www.ncbi.nlm.nih.gov/books/NBK560564/

37. Berlan M, Barbe P, Stich V, Galitzky J, Bousquet-Mélou A, Lafontan M. [Lipid mobilization, physiopathological and pharmacological aspects]. *Ann Endocrinol*. 1995;56(2):97-100.

38. Edens NK, Leibel RL, Hirsch J. Mechanism of free fatty acid re-esterification in human adipocytes in vitro. *J Lipid Res*. 1990;31(8):1423-1431.

39. Klein S, Weber JM, Coyle EF, Wolfe RR. Effect of endurance training on glycerol kinetics during strenuous exercise in humans. *Metabolism*. 1996;45(3):357-361. doi:10.1016/s0026-0495(96)90291-7

40. Palmer BF, Clegg DJ. Metabolic Flexibility and Its Impact on Health Outcomes. *Mayo Clin Proc*. 2022;97(4):761-776. doi:10.1016/j.mayocp.2022.01.012

41. Santoro A, McGraw TE, Kahn BB. Insulin action in adipocytes, adipose remodeling, and systemic effects. *Cell Metab*. 2021;33(4):748-757. doi:10.1016/j.cmet.2021.03.019

42. Gipson EZ, Deru LS, Graves PG, Jacobsen CG, Peterson NE, Bailey BW. The effects of initiating a 24-hour fast with a low versus a high carbohydrate shake on glycemic control in older adults: a randomized crossover study. *Nutr Metab*. 2025;22(1):32. doi:10.1186/s12986-025-00920-5

43. Yoon H, Shaw JL, Haigis MC, Greka A. Lipid metabolism in sickness and in health: Emerging regulators of lipotoxicity. *Mol Cell*. 2021;81(18):3708-3730. doi:10.1016/j.molcel.2021.08.027

44. Jahdkaran M, Sistanizad M. From lipids to glucose: Investigating the role of dyslipidemia in the risk of insulin resistance. *J Steroid Biochem Mol Biol*. 2025;250:106744. doi:10.1016/j.jsbmb.2025.106744

45. Dakal TC, Xiao F, Bhusal CK, et al. Lipids dysregulation in diseases: core concepts, targets and treatment strategies. *Lipids Health Dis*. 2025;24(1):61. doi:10.1186/s12944-024-02425-1

46. Rose AJ, Richter EA. Skeletal Muscle Glucose Uptake During Exercise: How is it Regulated? *Physiology*. 2005;20(4):260-270. doi:10.1152/physiol.00012.2005

47. Sylow L, Kleinert M, Richter EA, Jensen TE. Exercise-stimulated glucose uptake — regulation and implications for glycaemic control. *Nat Rev Endocrinol*. 2017;13(3):133-148. doi:10.1038/nrendo.2016.162

48. Trefts E, Williams AS, Wasserman DH. Exercise and the Regulation of Hepatic Metabolism. In: *Progress in Molecular Biology and Translational Science*. Vol 135. Elsevier; 2015:203-225. doi:10.1016/bs.pmbts.2015.07.010

49. Kjreri M. HEPATIC GLUCOSE PRODUCTION DURING EXERCISE.

50. Wasserman DH. REGULATION OF GLUCOSE FLUXES DURING EXERCISE IN THE POSTABSORPTIVE STATE. Published online 2025.

51. Marliss EB, Vranic M. Intense Exercise Has Unique Effects on Both Insulin Release and Its Roles in Glucoregulation. *Diabetes*. 2002;51(suppl_1):S271-S283. doi:10.2337/diabetes.51.2007.S271

52. Kreisman SH, Mew NA, Halter JB, Vranic M, Marliss EB. Norepinephrine Infusion during Moderate-Intensity Exercise Increases Glucose Production and Uptake. 86(5).

53. Khalil B, Rosani A, Warrington SJ. Physiology, Catecholamines. In: *StatPearls*. StatPearls Publishing; 2025. Accessed September 8, 2025. http://www.ncbi.nlm.nih.gov/books/NBK507716/

54. Wojtaszewski JFP, Richter EA. Effects of acute exercise and training on insulin action and sensitivity: focus on molecular mechanisms in muscle. Wagenmakers AJM, ed. *Essays Biochem*. 2006;42:31-46. doi:10.1042/bse0420031

55. Melzer K. Carbohydrate and fat utilization during rest and physical activity. *E-SPEN Eur E-J Clin Nutr Metab*. 2011;6(2):e45-e52. doi:10.1016/j.eclnm.2011.01.005

56. Venables MC, Achten J, Jeukendrup AE. Determinants of fat oxidation during exercise in healthy men and women: a cross-sectional study. *J Appl Physiol*. 2005;98(1):160-167. doi:10.1152/japplphysiol.00662.2003

57. Brosnan JT. Comments on metabolic needs for glucose and the role of gluconeogenesis. *Eur J Clin Nutr*. 1999;53 Suppl 1:S107-111. doi:10.1038/sj.ejcn.1600748

58. Sidossis LS, Gastaldelli A, Klein S, Wolfe RR. Regulation of plasma fatty acid oxidation during low- and high-intensity exercise. *Am J Physiol*. 1997;272(6 Pt 1):E1065-1070. doi:10.1152/ajpendo.1997.272.6.E1065

59. Laforgia J, Withers RT, Gore CJ. Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. *J Sports Sci*. 2006;24(12):1247-1264. doi:10.1080/02640410600552064

60. Townsend LK, Couture KM, Hazell TJ. Mode of exercise and sex are not important for oxygen consumption during and in recovery from sprint interval training. *Appl Physiol Nutr Metab*. 2014;39(12):1388-1394. doi:10.1139/apnm-2014-0145

61. Islam H, Townsend LK, Hazell TJ. Excess Postexercise Oxygen Consumption and Fat Utilization Following Submaximal Continuous and Supramaximal Interval Running. *Res Q Exerc Sport*. 2018;89(4):450-456. doi:10.1080/02701367.2018.1513633

62. Kiens B, Richter EA. Utilization of skeletal muscle triacylglycerol during postexercise recovery in humans. *Am J Physiol*. 1998;275(2):E332-337. doi:10.1152/ajpendo.1998.275.2.E332

63. McCartney N, Spriet LL, Heigenhauser GJ, Kowalchuk JM, Sutton JR, Jones NL. Muscle power and metabolism in maximal intermittent exercise. *J Appl Physiol Bethesda Md 1985*. 1986;60(4):1164-1169. doi:10.1152/jappl.1986.60.4.1164

64. Trapp EG, Chisholm DJ, Boutcher SH. Metabolic response of trained and untrained women during high-intensity intermittent cycle exercise. *Am J Physiol-Regul Integr Comp Physiol*. 2007;293(6):R2370-R2375. doi:10.1152/ajpregu.00780.2006

65. Phillips SM, Green HJ, Tarnopolsky MA, Heigenhauser GF, Hill RE, Grant SM. Effects of training duration on substrate turnover and oxidation during exercise. *J Appl Physiol Bethesda Md 1985*. 1996;81(5):2182-2191. doi:10.1152/jappl.1996.81.5.2182

66. Taylor H, Wu CL, Chen YC, Wang PG, Gonzalez J, Betts J. Post-Exercise Carbohydrate-Energy Replacement Attenuates Insulin Sensitivity and Glucose Tolerance the Following Morning in Healthy Adults. *Nutrients*. 2018;10(2):123. doi:10.3390/nu10020123

67. Newsom SA, Schenk S, Thomas KM, et al. Energy deficit after exercise augments lipid mobilization but does not contribute to the exercise-induced increase in insulin sensitivity. *J Appl Physiol*. 2010;108(3):554-560. doi:10.1152/japplphysiol.01106.2009

68. Francois ME, Durrer C, Pistawka KJ, Halperin FA, Chang C, Little JP. Combined Interval Training and Post-exercise Nutrition in Type 2 Diabetes: A Randomized Control Trial. *Front Physiol*. 2017;8:528. doi:10.3389/fphys.2017.00528

69. Schleh MW, Pitchford LM, Gillen JB, Horowitz JF. Energy Deficit Required for Exercise-induced Improvements in Glycemia the Next Day. *Med Sci Sports Exerc*. 2020;52(4):976-982. doi:10.1249/MSS.0000000000002211

70. Enevoldsen LH, Simonsen L, Macdonald IA, Bülow J. The combined effects of exercise and food intake on adipose tissue and splanchnic metabolism. *J Physiol*. 2004;561(3):871-882. doi:10.1113/jphysiol.2004.076588

71. Freese EC, Levine AS, Chapman DP, Hausman DB, Cureton KJ. Effects of acute sprint interval cycling and energy replacement on postprandial lipemia. *J Appl Physiol*. 2011;111(6):1584-1589. doi:10.1152/japplphysiol.00416.2011

72. Harrison M, O'Gorman DJ, McCaffrey N, et al. Influence of acute exercise with and without carbohydrate replacement on postprandial lipid metabolism. *J Appl Physiol*. 2009;106(3):943-949. doi:10.1152/japplphysiol.91367.2008

73. Horowitz JF, Kaufman AE, Fox AK, Harber MP. Energy deficit without reducing dietary carbohydrate alters resting carbohydrate oxidation and fatty acid availability. *J Appl Physiol*. 2005;98(5):1612-1618. doi:10.1152/japplphysiol.00936.2004

74. Kaviani M, Chilibeck PD, Yee P, Zello GA. The effect of consuming low- versus high-glycemic index meals after exercise on postprandial blood lipid response following a next-day high-fat meal. *Nutr Diabetes*. 2016;6(7):e216-e216. doi:10.1038/nutd.2016.26

75. Aune D, Norat T, Romundstad P, Vatten LJ. Dairy products and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of cohort studies. *Am J Clin Nutr*. 2013;98(4):1066-1083. doi:10.3945/ajcn.113.059030

76. Astrup A. Yogurt and dairy product consumption to prevent cardiometabolic diseases: epidemiologic and experimental studies. *Am J Clin Nutr*. 2014;99(5):1235S-1242S. doi:10.3945/ajcn.113.073015

77. Ribadeau-Dumas B, Grappin R. Milk protein analysis. *Le Lait*. 1989;69(5):357-416. doi:10.1051/lait:1989527

78. Morr CV. Chemistry of Milk Proteins in Food Processing. *J Dairy Sci*. 1975;58(7):977-984. doi:10.3168/jds.S0022-0302(75)84669-8

79. Mulet-Cabero AI, Mackie AR, Brodkorb A, Wilde PJ. Dairy structures and physiological responses: a matter of gastric digestion. *Crit Rev Food Sci Nutr*. 2020;60(22):3737-3752. doi:10.1080/10408398.2019.1707159

80. Boirie Y, Dangin M, Gachon P, Vasson MP, Maubois JL, Beaufrère B. Slow and fast dietary proteins differently modulate postprandial protein accretion. *Proc Natl Acad Sci*. 1997;94(26):14930-14935. doi:10.1073/pnas.94.26.14930

81. Mahé S, Roos N, Benamouzig R, et al. Gastrojejunal kinetics and the digestion of [15N]beta-lactoglobulin and casein in humans: the influence of the nature and quantity of the protein. *Am J Clin Nutr*. 1996;63(4):546-552. doi:10.1093/ajcn/63.4.546

82. Lacroix M, Bos C, Léonil J, et al. Compared with casein or total milk protein, digestion of milk soluble proteins is too rapid to sustain the anabolic postprandial amino acid requirement. *Am J Clin Nutr*. 2006;84(5):1070-1079. doi:10.1093/ajcn/84.5.1070

83. Van Loon LJC, Kruijshoop M, Menheere PPCA, Wagenmakers AJM, Saris WHM, Keizer HA. Amino Acid Ingestion Strongly Enhances Insulin Secretion in Patients With Long-Term Type 2 Diabetes. *Diabetes Care*. 2003;26(3):625-630. doi:10.2337/diacare.26.3.625

84. Nuttall FQ, Gannon MC. Plasma Glucose and Insulin Response to Macronutrients in Nondiabetic and NIDDM Subjects. *Diabetes Care*. 1991;14(9):824-838. doi:10.2337/diacare.14.9.824

85. Van Loon LJC, Greenhaff PL, Constantin-Teodosiu D, Saris WHM, Wagenmakers AJM. The effects of increasing exercise intensity on muscle fuel utilisation in humans. *J Physiol*. 2001;536(1):295-304. doi:10.1111/j.1469-7793.2001.00295.x

86. Sun L, Tan KWJ, Han CMS, Leow MKS, Henry CJ. Impact of preloading either dairy or soy milk on postprandial glycemia, insulinemia and gastric emptying in healthy adults. *Eur J Nutr*. 2017;56(1):77-87. doi:10.1007/s00394-015-1059-y

87. Zemel MB, Shi H, Greer B, Dirienzo D, Zemel PC. Regulation of adiposity by dietary calcium. *FASEB J Off Publ Fed Am Soc Exp Biol*. 2000;14(9):1132-1138.

88. Cummings NK, James AP, Soares MJ. The acute effects of different sources of dietary calcium on postprandial energy metabolism. *Br J Nutr*. 2006;96(1):138-144. doi:10.1079/bjn20061803

89. Gao R, Rapin N, Elnajmi AM, Gordon J, Zello GA, Chilibeck PD. Skim milk as a recovery beverage after exercise is superior to a sports drink for reducing next-day postprandial blood

glucose and increasing postprandial fat oxidation. *Nutr Res*. 2020;82:58-66. doi:10.1016/j.nutres.2020.08.007

90. Williams N. The Borg Rating of Perceived Exertion (RPE) scale. *Occup Med*. 2017;67(5):404-405. doi:10.1093/occmed/kqx063

91. Fox SM, Naughton JP, Haskell WL. Physical activity and the prevention of coronary heart disease. *Ann Clin Res*. 1971;3(6):404-432.

92. Liljeberg Elmståhl H, Björck I. Milk as a supplement to mixed meals may elevate postprandial insulinaemia. *Eur J Clin Nutr*. 2001;55(11):994-999. doi:10.1038/sj.ejcn.1601259

93. Panahi S, El Khoury D, Kubant R, et al. Mechanism of action of whole milk and its components on glycemic control in healthy young men. *J Nutr Biochem*. 2014;25(11):1124-1131. doi:10.1016/j.jnutbio.2014.07.002

94. Karastamatis S, Zoidou E, Moatsou G, Moschopoulou E. Effect of Modified Manufacturing Conditions on the Composition of Greek Strained Yogurt and the Quantity and Composition of Generated Acid Whey. *Foods*. 2022;11(24):3953. doi:10.3390/foods11243953

95. Sanggaard KM, Holst JJ, Rehfeld JF, Sandström B, Raben A, Tholstrup T. Different effects of whole milk and a fermented milk with the same fat and lactose content on gastric emptying and postprandial lipaemia, but not on glycaemic response and appetite. *Br J Nutr*. 2004;92(3):447-459. doi:10.1079/bjn20041219

96. Brown JK, Perry CGR, Prior ET, Phillips SM, Skelly LE, Josse AR. Differential plasma branched-chain amino acid responses following the consumption of Greek-style yogurt and skimmed milk. *Appl Physiol Nutr Metab Physiol Appl Nutr Metab*. 2023;48(7):544-549. doi:10.1139/apnm-2022-0416

97. Kung B, Anderson GH, Paré S, et al. Effect of milk protein intake and casein-to-whey ratio in breakfast meals on postprandial glucose, satiety ratings, and subsequent meal intake. *J Dairy Sci*. 2018;101(10):8688-8701. doi:10.3168/jds.2018-14419

98. Sochol KM, Johns TS, Buttar RS, et al. The Effects of Dairy Intake on Insulin Resistance: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. *Nutrients*. 2019;11(9):2237. doi:10.3390/nu11092237

99. Turner KM, Keogh JB, Clifton PM. Dairy consumption and insulin sensitivity: a systematic review of short- and long-term intervention studies. *Nutr Metab Cardiovasc Dis NMCD*. 2015;25(1):3-8. doi:10.1016/j.numecd.2014.07.013

100. Eelderink C, Rietsema S, van Vliet IMY, et al. The effect of high compared with low dairy consumption on glucose metabolism, insulin sensitivity, and metabolic flexibility in overweight adults: a randomized crossover trial. *Am J Clin Nutr*. 2019;109(6):1555-1568. doi:10.1093/ajcn/nqz017

101. Vien S, Fard S, El Khoury D, Luhovyy B, Fabek H, Anderson GH. Age and Sex Interact to Determine the Effects of Commonly Consumed Dairy Products on Postmeal Glycemia, Satiety, and Later Meal Food Intake in Adults. *J Nutr*. 2021;151(8):2161-2174. doi:10.1093/jn/nxab122

102. Nuutila P, Knuuti MJ, Mäki M, et al. Gender and insulin sensitivity in the heart and in skeletal muscles. Studies using positron emission tomography. *Diabetes*. 1995;44(1):31-36. doi:10.2337/diab.44.1.31

103. De Glisezinski I, Harant I, Crampes F, et al. Effect of carbohydrate ingestion on adipose tissue lipolysis during long-lasting exercise in trained men. *J Appl Physiol*. 1998;84(5):1627-1632. doi:10.1152/jappl.1998.84.5.1627

Appendices

Appendix A: Recruitment Poster

YORK UNIVERSITÉ UNIVERSITY

PARTICIPANTS NEEDED FOR THE BIONEX STUDY







SCAN FOR MORE INFORMATION!

ARE YOU BETWEEN THE AGES OF 18-35 YEARS WITH LIMITED EXERCISE EXPERIENCE?

HELP US DETERMINE IF POST-EXERCISE NUTRITION CAN INFLUENCE BONE & INFLAMMATION!

STUDY INFORMATION

- 4 TRIALS: 2 VISITS/TRIAL 27 TOTAL HOURS
- 4 POST-EXERCISE SUPPLEMENTS (MILK, GREEK YOGURT, CARBOHYDRATE, WATER)
- 5 BLOOD SAMPLES/TRIAL

BENEFITS OF PARTICIPATING

- 4 EXERCISE SESSIONS WITH A PERSONAL TRAINER
- LUNCH & DINNER MEALS PROVIDED ON STUDY DAYS
- UP TO $100 COMPENSATION

Contact: york.nutrition.research@gmail.com or by phone (416) 736-2100 x33990

Dr. Andrea R. Josse (Principal Investigator): ajosse@yorku.ca or by phone (416) 736-2100(30038)

*should prospective participants choose to interact with this post on Facebook, their privacy and confidentiality may be compromised

Approved by the York University Research Ethics Board (ORE) File# (2019-045)

Appendix B: Ethics Approval

YORK
UNIVERSITÉ
UNIVERSITY

Certificate #:	**2019-045**
Initial Approval:	**01/31/19-01/31/20**
Amendments:	**6th Amendment approved: 08/11/23; 7th Amendment approved: 02/09/24; 8th Amendment approved: 07/15/24**
Renewals:	**02/04/22-02/04/23; 02/03/23-02/03/24 02/09/24-02/09/25; 02/10/25-01/10/26**
Current Approval Period:	**02/10/25-01/10/26 *Final**

OFFICE OF RESEARCH ETHICS (ORE)

4700 Keele St.
Toronto ON
Canada M3J 1P3
ore@yorku.ca
www.research.yorku.ca

ETHICS RENEWAL

To: **Professor Andrea Josse**
Department of Kinesiology
Faculty of Health
ajosse@yorku.ca

From: Alison M. Collins-Mrakas, Director, Research Ethics
(on behalf of Gillian Parekh, Chair, Human Participants Review Committee)

Date: Friday, February 28, 2025

Title: **Cre-Ex Study: Does Creatine Augment the Acute Effect of Exercise and Dairy on Bone Turnover?**

Risk Level: ☒ Minimal Risk ☐ More than Minimal Risk

Level of Review: ☒ Delegated Review ☐ Full Committee Review

I am writing to inform you that this research project, **"Cre-Ex Study: Does Creatine Augment the Acute Effect of Exercise and Dairy on Bone Turnover?"** has received ethics review and renewal by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines.

Note that renewal is granted for one year. Ongoing research – research that extends beyond one year – must be renewed prior to the expiry date.

Any changes to the approved protocol must be reviewed and approved through the amendment process by submission of an amendment application to the HPRC prior to its implementation.

Any adverse or unanticipated events in the research should be reported to the Office of Research ethics (ore@yorku.ca) as soon as possible.

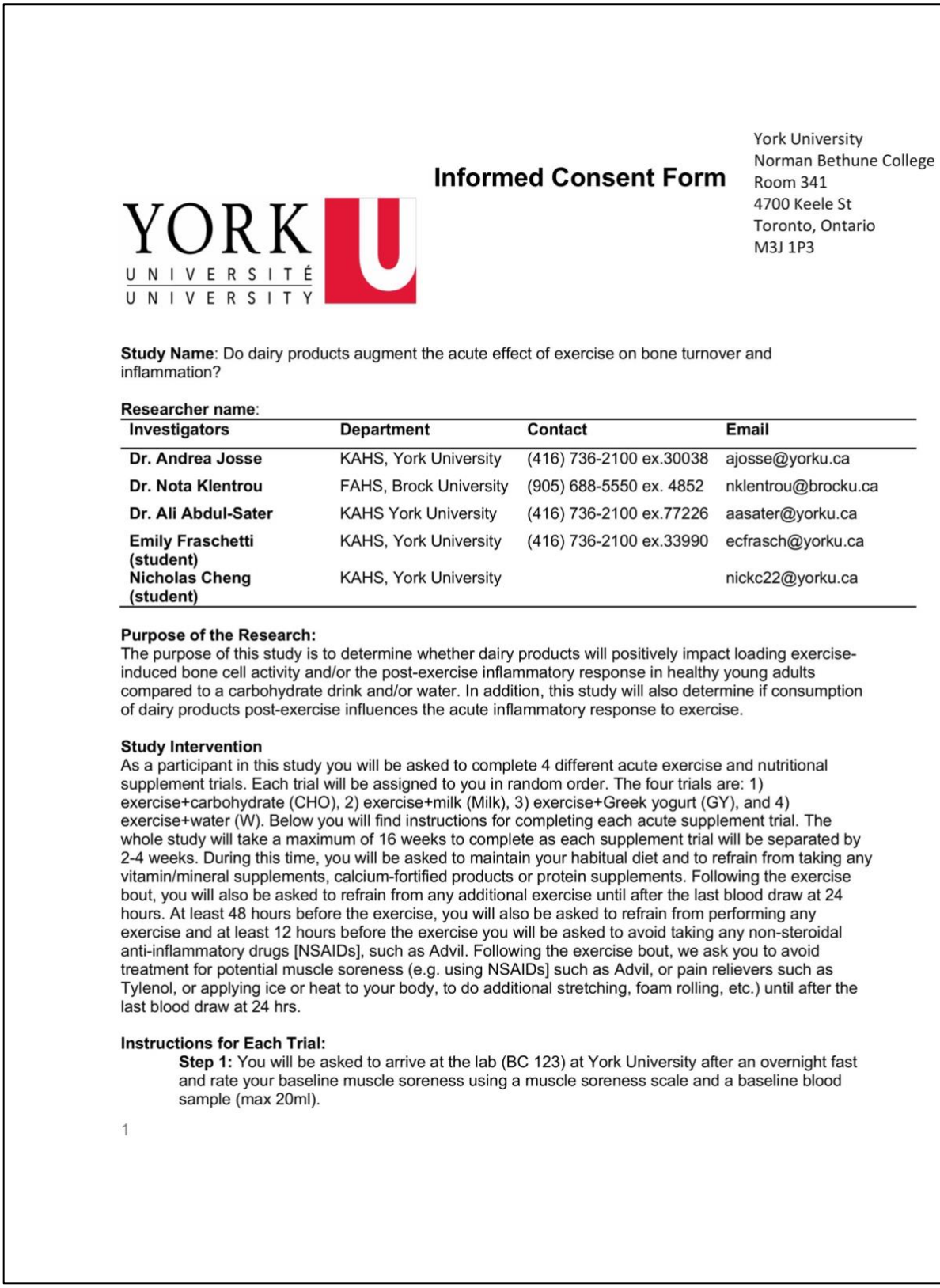
For further information on researcher responsibilities as it pertains to this approved research ethics protocol, please refer to the attached document, **"RESEARCH ETHICS: PROCEDURES to ENSURE ONGOING COMPLIANCE"**.

Should you have any questions, please feel free to contact me at: ore@yorku.ca

Yours sincerely,

Alison M. Collins-Mrakas M.Sc., LLM
Director, Office of Research Ethics

Appendix C: Informed Consent Form



Informed Consent Form

York University
Norman Bethune College
Room 341
4700 Keele St
Toronto, Ontario
M3J 1P3

Study Name: Do dairy products augment the acute effect of exercise on bone turnover and inflammation?

Researcher name:

Investigators	Department	Contact	Email
Dr. Andrea Josse	KAHS, York University	(416) 736-2100 ex.30038	ajosse@yorku.ca
Dr. Nota Klentrou	FAHS, Brock University	(905) 688-5550 ex. 4852	nklentrou@brocku.ca
Dr. Ali Abdul-Sater	KAHS York University	(416) 736-2100 ex.77226	aasater@yorku.ca
Emily Fraschetti (student)	KAHS, York University	(416) 736-2100 ex.33990	ecfrasch@yorku.ca
Nicholas Cheng (student)	KAHS, York University		nickc22@yorku.ca

Purpose of the Research:
The purpose of this study is to determine whether dairy products will positively impact loading exercise-induced bone cell activity and/or the post-exercise inflammatory response in healthy young adults compared to a carbohydrate drink and/or water. In addition, this study will also determine if consumption of dairy products post-exercise influences the acute inflammatory response to exercise.

Study Intervention
As a participant in this study you will be asked to complete 4 different acute exercise and nutritional supplement trials. Each trial will be assigned to you in random order. The four trials are: 1) exercise+carbohydrate (CHO), 2) exercise+milk (Milk), 3) exercise+Greek yogurt (GY), and 4) exercise+water (W). Below you will find instructions for completing each acute supplement trial. The whole study will take a maximum of 16 weeks to complete as each supplement trial will be separated by 2-4 weeks. During this time, you will be asked to maintain your habitual diet and to refrain from taking any vitamin/mineral supplements, calcium-fortified products or protein supplements. Following the exercise bout, you will also be asked to refrain from any additional exercise until after the last blood draw at 24 hours. At least 48 hours before the exercise, you will also be asked to refrain from performing any exercise and at least 12 hours before the exercise you will be asked to avoid taking any non-steroidal anti-inflammatory drugs [NSAIDs], such as Advil. Following the exercise bout, we ask you to avoid treatment for potential muscle soreness (e.g. using NSAIDs] such as Advil, or pain relievers such as Tylenol, or applying ice or heat to your body, to do additional stretching, foam rolling, etc.) until after the last blood draw at 24 hrs.

Instructions for Each Trial:

Step 1: You will be asked to arrive at the lab (BC 123) at York University after an overnight fast and rate your baseline muscle soreness using a muscle soreness scale and a baseline blood sample (max 20ml).

Step 2: You will be asked to perform a jump height test.
Step 3: You will then be asked complete a supervised high-intensity exercise bout consisting of a mix of resistance, plyometric (jumping) and/or aerobic exercise.
Step 4: Immediately after exercise you will be asked to rate your muscle soreness and perform the jump height test again.
Step 5: You will then be asked to consume the trial supplement (either a carbohydrate drink, milk, Greek yogurt or water) and a blood sample will be taken 5 minutes after exercise (max 20ml).
Step 6: Another blood sample will be taken 1 hour after exercise (max 20ml), and you will consume an additional trial supplement.
Step 7: You will be provided with a light lunch meal 2 hours after the exercise bout.
Step 8: Another blood sample will be taken at 4 hours after the exercise bout (max 20ml). You will then rate your muscle soreness again and perform another jump height test.
Step 9: You will be provided with a dinner meal to take home and an additional trial supplement to consume before bed.
Step 10: You will be asked return to the lab (BC 123) at York University after an overnight fast (8h minimum), 24 hours later to rate your muscle soreness once more, perform the jump height tests, and complete a final blood sample~~s~~ (max 20ml).

Acute Exercise Bout
As part of each acute supplement trial, you will be asked to perform one bout of exercise. Exercise will take place in the morning hours between 8 and 11am at York University in Dr. Josse's Exercise Research Lab in the Life Science Building (Rm 002). The bout will consist of a mix of resistance exercise (e.g. bench press, lat pulldown, squats, leg press, free weights) combined with plyometric (jumping) exercise (e.g. box jumps, drop-box jumps, frog jumps) and/or a high-intensity aerobic exercise session (e.g. cycling or running). Each session will last for approximately 60 minutes. Each exercise session will be facilitated by a personal trainer and/or a trained kinesiology student who will ensure safety and correct technique. Please ensure you are properly dressed for the exercise sessions. This includes running shoes, shorts/jogging pants and a t-shirt/tank-top. You may also bring a water bottle. We ask that you maintain your current pre-study physical activity patterns during the study and refrain from starting any new forms of physical activity until the study is complete. Due to the nature of the exercise bout, muscle soreness is likely to occur the evening and day or two following. Researchers will contact you the first and/or second day after each exercise bout to ensure there are no issues or concerns. Please do not treat your muscle soreness as described on the first page of the consent form.

Description of Testing Procedures
1. Questionnaires
You will be asked to complete three short questionnaires at the beginning of the study to see if you meet our inclusion criteria: i) Health and Screening questionnaire to gain insight about your health and lifestyle (this asks questions about your general health and lifestyle including questions on alcohol consumption and smoking). ii) A PAR-Q to determine readiness to participate in physical activity. iii) A physical activity questionnaire to determine your current levels of physical activity. We ask that you complete these questionnaires as truthful as possible and ask us any questions you may have. You may choose to leave questions blank with no penalty if you do not wish to provide an answer. You will also be asked to answer a standard single question about muscle soreness several times throughout the trials to assess the degree of soreness you experience after the exercise and up to 48 hours later.

2. Familiarization Session
You will be asked to come to the lab an additional time prior to beginning the study for an exercise familiarization session to acclimatize yourself to the exercises that will be completed. During this session, we will also determine the workrate for the interval cycling/running component of the exercise and how much weight you can lift/push/pull on certain machines (1 repetition maximums) such as the Leg Press, Leg Curl, Seated Row and Machine Chest Press. This will enable us to determine the appropriate weights that you will use for the following exercise bouts.

3. Food diaries
You will be asked to complete a 2-day food diary for each trial. The 2 days include the day before the exercise bout, and the day of the exercise bout. We ask that you try to consume a similar diet/eating pattern on the day before the exercise bout for all supplement trials. On the day of exercise, we will be providing you with a lunch and dinner meal as well ~~at~~ as the supplements to consume in the lab and at home. This will allow us to accurately assess your current nutrient intake and control the foods you eat on the day of the trial. We ask that these diaries be filled out truthfully and with as much detail as possible. We encourage any questions you may have about these diaries and we will go over how to fill them out with you prior to you filling it out on your own.

4. Blood collection
We would like to collect blood from your arm (front part of the elbow using a standard technique, and taken by a trained professional) 5 times per supplement trial. During each blood sample, a maximum of 20ml of blood will be taken: upon arrival to the lab, 5 minutes post-exercise, 1 hour post-exercise, 4 hours post-exercise and 24 hours post-exercise. This is a total of 100ml of blood (over 2 days) for each supplement trial. For context, those that donate blood provide ~450-500 ml at one time. We ask that you arrive to our laboratory (BC 123) for testing after an overnight fast (minimum 8 hours), but you may/should drink water. We also ask that you refrain from exercise for at least 48 hours prior to your first blood sample and until after the last blood draw at 24 hours. We will use your blood to measure different bone markers that reflect activity inside the bone and/or inflammatory markers that are related to muscle soreness. Once blood is collected, it will be transferred to special tubes. These tubes will then be stored in a freezer for later analysis. **Note**: You may also leave the lab to use the washroom or grab water briefly between the 5 minutes post-exercise and 4 hour post-exercise blood draws.

5. Anthropometry and Body Composition
We would like to measure your body weight and body composition. Body composition will be measured through Bio Electrical Impedance (BIA) or through air displacement plythsmography (Bodpod). Please wear athletic clothing (shorts, t-shirt) for this component. This measurement is routinely done in laboratory and is very safe. This will take a few minutes to complete. If requested, a female researcher can complete these measurements for you.

6. Jump Height Test
We would like you to perform a jump height test at each blood draw (baseline, post-exercise, 4hr, and 24hr post-exercise). We ask that you perform this test to the best of your ability. During the familiarization session we will discuss proper technique for performing the jump and show you how to perform the jump height test.

What You Will Be Asked to Do in the Research:
Maximal Time Commitment

Treatment Number	Acute trial 1	Acute Trial 2	Acute Trial 3	Acute Trial 4
Initial meet, signing of consent form, questionnaires Familiarization Session (Introduce exercises, body composition, 1RM testing)	(1.5 hours)			
Day 1 – Pre-exercise testing (15min), exercise (60 mins), Blood Sample (240 mins)	(5 hours, 15 mins)	(5 hours, 15 mins	(5 hours, 15 mins)	(5 hours, 15 mins)
Day 2 - 24 Hour Blood Sample	(15 mins)	(15 mins)	(15 min)	(15 mins)
Food Diary	(30 mins)	(30 mins)	(30 mins)	(30 mins)

Post-study testing session (body composition, 1RM testing)				(1 hour)
Total Per acute trial:	**7.5 hours**	**6 hours**	**6 hours**	**7 hours**

Estimate of total time commitment in hours to complete initial familiarization session and all 4 acute trials: ~ 26.5 hours.

Upon completion of all trials or withdrawal, you will be compensated $20 for each completed trial and an additional $20 for completing the post-study testing.

Risks and Discomforts:
There is little direct risk to you, you may experience some uneasiness or anxiety due to the personal nature of the questions asked on the questionnaires or due to the measurements taken (i.e., anxiety around the blood sample or body composition testing or exercise). We assure you that this is nothing to be anxious about and we will do our best to minimize these feelings. The only foreseeable physical risks involved in participation include a) potential muscle soreness/discomfort after completing the exercise bout which may take 2-3 days to subside. This is completely normal; b) you may experience slight pain, and/or tingling, and/or bruising in the area after the blood samples. In some instances, you may unexpectedly feel faint or lose consciousness if looking at the needle or blood. It is also possible that a hematoma (bruise) could form under the skin beside the puncture site. Applying adequate pressure following the blood draw will mimimize this. It is also important to know that there is always a risk of infection whenever the skin is punctured. Please inform the researchers if you experience any of the above-mentioned events or you are concerned about them.

Benefits of the Research and Benefits to You:
By participating in this research study, you will: (1) become exposed to a research study, (2) contribute to the advancement of science; (3) gain knowledge about your own body, health and nutrition. Your own individual results or group results provided to you upon request.

Voluntary Participation and Withdrawal: Your participation in the study is completely voluntary and you may choose to stop participating at any time. Your decision not to volunteer, to stop participating, or to refuse to answer particular questions will not influence the nature of the ongoing relationship you may have with the researchers or study staff, or the nature of your relationship with York University either now, or in the future. If you decide to stop participating, you may withdraw without penalty, financial or otherwise, and you will still receive the promised inducement. In the event you withdraw from the study, all associated data collected will be immediately destroyed wherever possible. Should you wish to withdraw after the study, you will have the option to also withdraw your data up until the analysis is complete.

Confidentiality:
Once you begin the study, you will be given a personal participant identification number. All research related documents will only use only this ID number to identify you and your data, not your name. This way, those who may be analyzing your online/computer data will not be able to link it to you. You maintain your right to withdraw from the study at any time, including research data (if applicable). However, your personal information will not be linked to your data. The identification key and all information collected about you during this study will remain confidential and will be stored in locked offices and on secured computers to which only the principal investigator, co-investigator and student investigator will have access to. Unless you choose otherwise, all information you supply during the research will be held in confidence and unless you specifically indicate your consent, your name will not appear in any report or publication of the research. Results of this study will be made available to scientists, through publication in scientific journals, but your name and personal data will not appear on its own. We will only report group information. Electronic and paper information will be kept safely for 5 years after publication (~2030), at which time, all information will be destroyed. In addition, you will have access to your own results, as well as results from the group when our analysis is completed and available. Please let us

know if you would be interested in receiving the results. Confidentiality will be provided to the fullest extent possible by law. The data collected in this research project may be used – in an anonymized form – by members of the research team or approved colleagues in subsequent research investigations exploring similar lines of inquiry. Such projects will still undergo ethics review by the HPRC, our institutional REB. Any secondary use of anonymized data by the research team or approved colleagues will be treated with the same degree of confidentiality and anonymity as in the original research project.

Questions About the Research? If you have questions about the research in general or about your role in the study, please feel free to contact Emily Fraschetti ecfrasch@yorku.ca (student investigator) or the main supervisor of this research, Andrea Josse at ajosse@yorku.ca and/or (416) 736-2100 (30038). You may also contact the Graduate Program in Kinesiology and Health Science at kahs@yorku.ca and/or (416) 736-5728.

This research has received ethics review and approval by the Delegated Ethics Review Committee, which is delegated authority to review research ethics protocols by the Human Participants Review Sub-Committee, York University's Ethics Review Board, and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. If you have any questions about this process, or about your rights as a participant in the study, please contact the Sr. Manager & Policy Advisor for the Office of Research Ethics, 5th Floor, Kaneff Tower, York University (telephone 416-736-5914 or e-mail ore@yorku.ca).

Legal Rights and Signatures:

I_____consent to participate in this study conducted by Dr. Andrea Josse. I have understood the nature of this project and wish to participate. I am not waiving any of my legal rights by signing this form. My signature below indicates my consent.

Signature_____ **Date**_____
Participant

Signature_____ **Date**_____
Principal Investigator

Appendix D: Health and Screening Questionnaire

Participant ID#: BIONEX Trials

Researcher:
Date & Time:

1. Participant Details			
Name:			
Telephone #:		Email:	
Preferred Contact method?	| | Telephone	| | Zoom	| | Email
Age:		| | **18 - 30 years old?**	
Date of Birth:			
Sex:			
Self-Identified Ethnicity:			
Height:			
Weight:			
Calculated BMI:		| | **18.5 - 25 kg/m^2?**	
2. Exercise/Activity Level			
How many times per week do you perform structured exercise (excluding walking)?			
		| | **≤ 2 structured weekly sessions?**	
Type of activity and duration (per day or week):			
How many times per week do you engage in any weight-lifting or jumping exercises (plyometrics)?			
		| | **≤ 2 structured weekly sessions?**	
Type of activity and duration (per day or week):			
3. Supplement consumption			

Page 1 of 5

Do you consume nutrition supplements (e.g., Multivitamin, Calcium, Vitamin D, Iron, B12, protein supplements, etc.)	| | Yes*	| | No
If YES, please specify the:		
Types:		
Amount:		
Frequency (per day or average per week):		
Have you been instructed by a physician to take these supplements?		
If yes, would you be willing to not take these supplements during the duration of the study (maximum 18 weeks)?	| | Yes*	| | No

4. Health & Lifestyle Questions		
Do you regularly or occasionally smoke/vape tobacco and/or use cannabis products?	| | Yes	| | No
If yes, please explain (specify if using tobacco or cannabis, smoking/vaping or consuming edibles and frequency of use):		
Do you consume alcohol on a regular basis (at least every couple of days)?	| | Yes	| | No
Are you lactose intolerant (diagnosed by doctor)?	| | Yes	| | No
Do you believe you may be lactose-intolerant or lactose-sensitive?	| | Yes	| | No
Do you have an allergy to dairy/milk protein?	| | Yes	| | No
Do you have any other food allergies, or	| | Yes	| | No

dietary restrictions (gluten free, vegetarian) If yes, explain:		
Do you regularly take any pain management medication, such as Tylenol or Advil? (Please select yes if you usually take pain management medication for menstrual cramps?)	| | Yes	| | No
Do you have Celiac Disease?	| | Yes	| | No
Do you have any other gastrointestinal diseases or condition?	| | Yes	| | No
Have you ever had any bone, joint or muscle injury? If yes, when and how did this injury occur? How was it treated and does it affect your physical ability?	| | Yes	| | No
Have you ever had any major joint instability or ongoing chronic pain such as in the knee, back or elbow? If yes, when and how did this occur? How was this treated and does this affect your physical ability?	| | Yes	| | No
5. Dairy Intake Questionnaire		
Do you regularly consume dairy products (milk, yogurt, cheese)?	| | Yes	| | No
Do you regularly consume dairy-containing products (ice cream, sour cream, whipped cream etc.,)	| | Yes	| | No
What dairy products do you consume?		
What dairy-containing products do you consume?		
On average, how many dairy products do you consume per week?		

On average, how many dairy-containing products do you consume?		
6. Medical History Questions		
Heart disease/condition	| | Yes	| | No
Kidney disease/condition	| | Yes	| | No
Liver disease/condition	| | Yes	| | No
Pancreatic disease/condition	| | Yes	| | No
Hepatitis B	| | Yes	| | No
Hepatitis C	| | Yes	| | No
HIV/AIDS	| | Yes	| | No
Other (please specify):		
Do you take any prescription medication?	| | Yes	| | No
If YES, please specify (anything prescribed by a doctor, including supplements):		
7. Menstrual Cycle and Hormonal Questions		
Do you experience a menstrual cycle?	| | Yes	| | No
If yes, do you experience a regular menstrual cycle?	| | Yes	| | No
Have you ever been diagnosed with any menstrual imbalances or disorders (such as PCOS, endometriosis, etc.)? If yes, please specify, including when you were diagnosed:		

Are you currently taking any type of hormonal contraception? If yes, what kind of contraceptive and how long have you been taking it?	
What was the approximate date of your last period (mm/dd/yy)?	
What day of your cycle are you currently at (with day 0 being the day menstrual bleeding starts then day 1 is the next full 24 hours of bleeding)?	
How many days in duration is your monthly cycle?	
What is the average or most consistent length & if it varies, what is the range?	

Eligible: | | Yes | | No/consult with coordinator

Appendix E: PAR-Q+

2018 PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

The health benefits of regular physical activity are clear; more people should engage in physical activity every day of the week. Participating in physical activity is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

GENERAL HEALTH QUESTIONS

Please read the 7 questions below carefully and answer each one honestly: check YES or NO.	**YES**	**NO**
1) Has your doctor ever said that you have a heart condition ☐ OR high blood pressure ☐?	☐	☐
2) Do you feel pain in your chest at rest, during your daily activities of living, **OR** when you do physical activity?	☐	☐
3) Do you lose balance because of dizziness **OR** have you lost consciousness in the last 12 months? Please answer **NO** if your dizziness was associated with over-breathing (including during vigorous exercise).	☐	☐
4) Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)? **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
5) Are you currently taking prescribed medications for a chronic medical condition? **PLEASE LIST CONDITION(S) AND MEDICATIONS HERE:** _____	☐	☐
6) Do you currently have (or have had within the past 12 months) a bone, joint, or soft tissue (muscle, ligament, or tendon) problem that could be made worse by becoming more physically active? Please answer **NO** if you had a problem in the past, but it ***does not limit your current ability*** to be physically active. **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
7) Has your doctor ever said that you should only do medically supervised physical activity?	☐	☐

If you answered NO to all of the questions above, you are cleared for physical activity.
Please sign the PARTICIPANT DECLARATION. You do not need to complete Pages 2 and 3.

- Start becoming much more physically active – start slowly and build up gradually.
- Follow International Physical Activity Guidelines for your age (www.who.int/dietphysicalactivity/en/).
- You may take part in a health and fitness appraisal.
- If you are over the age of 45 yr and NOT accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional before engaging in this intensity of exercise.
- If you have any further questions, contact a qualified exercise professional.

PARTICIPANT DECLARATION

If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that the community/fitness centre may retain a copy of this form for records. In these instances, it will maintain the confidentiality of the same, complying with applicable law.

NAME _____ DATE _____

SIGNATURE _____ WITNESS _____

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER _____

If you answered YES to one or more of the questions above, COMPLETE PAGES 2 AND 3.

Delay becoming more active if:

- You have a temporary illness such as a cold or fever; it is best to wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ at **www.eparmedx.com** before becoming more physically active.
- Your health changes - answer the questions on Pages 2 and 3 of this document and/or talk to your doctor or a qualified exercise professional before continuing with any physical activity program.

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01-11-2017

2018 PAR-Q+

FOLLOW-UP QUESTIONS ABOUT YOUR MEDICAL CONDITION(S)

1. Do you have Arthritis, Osteoporosis, or Back Problems?

If the above condition(s) is/are present, answer questions 1a-1c — If **NO** ☐ go to question 2

1a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
1b.	Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebra (e.g., spondylolisthesis), and/or spondylolysis/pars defect (a crack in the bony ring on the back of the spinal column)?	YES ☐ NO ☐
1c.	Have you had steroid injections or taken steroid tablets regularly for more than 3 months?	YES ☐ NO ☐

2. Do you currently have Cancer of any kind?

If the above condition(s) is/are present, answer questions 2a-2b — If **NO** ☐ go to question 3

2a.	Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head, and/or neck?	YES ☐ NO ☐
2b.	Are you currently receiving cancer therapy (such as chemotheraphy or radiotherapy)?	YES ☐ NO ☐

3. Do you have a Heart or Cardiovascular Condition? *This includes Coronary Artery Disease, Heart Failure, Diagnosed Abnormality of Heart Rhythm*

If the above condition(s) is/are present, answer questions 3a-3d — If **NO** ☐ go to question 4

3a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
3b.	Do you have an irregular heart beat that requires medical management? (e.g., atrial fibrillation, premature ventricular contraction)	YES ☐ NO ☐
3c.	Do you have chronic heart failure?	YES ☐ NO ☐
3d.	Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?	YES ☐ NO ☐

4. Do you have High Blood Pressure?

If the above condition(s) is/are present, answer questions 4a-4b — If **NO** ☐ go to question 5

4a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
4b.	Do you have a resting blood pressure equal to or greater than 160/90 mmHg with or without medication? (Answer **YES** if you do not know your resting blood pressure)	YES ☐ NO ☐

5. Do you have any Metabolic Conditions? *This includes Type 1 Diabetes, Type 2 Diabetes, Pre-Diabetes*

If the above condition(s) is/are present, answer questions 5a-5e — If **NO** ☐ go to question 6

5a.	Do you often have difficulty controlling your blood sugar levels with foods, medications, or other physician-prescribed therapies?	YES ☐ NO ☐
5b.	Do you often suffer from signs and symptoms of low blood sugar (hypoglycemia) following exercise and/or during activities of daily living? Signs of hypoglycemia may include shakiness, nervousness, unusual irritability, abnormal sweating, dizziness or light-headedness, mental confusion, difficulty speaking, weakness, or sleepiness.	YES ☐ NO ☐
5c.	Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, **OR** the sensation in your toes and feet?	YES ☐ NO ☐
5d.	Do you have other metabolic conditions (such as current pregnancy-related diabetes, chronic kidney disease, or liver problems)?	YES ☐ NO ☐
5e.	Are you planning to engage in what for you is unusually high (or vigorous) intensity exercise in the near future?	YES ☐ NO ☐

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6. Do you have any Mental Health Problems or Learning Difficulties? *This includes Alzheimer's, Dementia, Depression, Anxiety Disorder, Eating Disorder, Psychotic Disorder, Intellectual Disability, Down Syndrome*

If the above condition(s) is/are present, answer questions 6a-6b — If **NO** ☐ go to question 7

6a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
6b.	Do you have Down Syndrome **AND** back problems affecting nerves or muscles?	YES ☐ NO ☐

7. Do you have a Respiratory Disease? *This includes Chronic Obstructive Pulmonary Disease, Asthma, Pulmonary High Blood Pressure*

If the above condition(s) is/are present, answer questions 7a-7d — If **NO** ☐ go to question 8

7a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
7b.	Has your doctor ever said your blood oxygen level is low at rest or during exercise and/or that you require supplemental oxygen therapy?	YES ☐ NO ☐
7c.	If asthmatic, do you currently have symptoms of chest tightness, wheezing, laboured breathing, consistent cough (more than 2 days/week), or have you used your rescue medication more than twice in the last week?	YES ☐ NO ☐
7d.	Has your doctor ever said you have high blood pressure in the blood vessels of your lungs?	YES ☐ NO ☐

8. Do you have a Spinal Cord Injury? *This includes Tetraplegia and Paraplegia*

If the above condition(s) is/are present, answer questions 8a-8c — If **NO** ☐ go to question 9

8a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
8b.	Do you commonly exhibit low resting blood pressure significant enough to cause dizziness, light-headedness, and/or fainting?	YES ☐ NO ☐
8c.	Has your physician indicated that you exhibit sudden bouts of high blood pressure (known as Autonomic Dysreflexia)?	YES ☐ NO ☐

9. Have you had a Stroke? *This includes Transient Ischemic Attack (TIA) or Cerebrovascular Event*

If the above condition(s) is/are present, answer questions 9a-9c — If **NO** ☐ go to question 10

9a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer **NO** if you are not currently taking medications or other treatments)	YES ☐ NO ☐
9b.	Do you have any impairment in walking or mobility?	YES ☐ NO ☐
9c.	Have you experienced a stroke or impairment in nerves or muscles in the past 6 months?	YES ☐ NO ☐

10. Do you have any other medical condition not listed above or do you have two or more medical conditions?

If you have other medical conditions, answer questions 10a-10c — If **NO** ☐ read the Page 4 recommendations

10a.	Have you experienced a blackout, fainted, or lost consciousness as a result of a head injury within the last 12 months **OR** have you had a diagnosed concussion within the last 12 months?	YES ☐ NO ☐
10b.	Do you have a medical condition that is not listed (such as epilepsy, neurological conditions, kidney problems)?	YES ☐ NO ☐
10c.	Do you currently live with two or more medical conditions?	YES ☐ NO ☐

PLEASE LIST YOUR MEDICAL CONDITION(S) AND ANY RELATED MEDICATIONS HERE: _____

_____

GO to Page 4 for recommendations about your current medical condition(s) and sign the PARTICIPANT DECLARATION.

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If you answered NO to all of the FOLLOW-UP questions (pgs. 2-3) about your medical condition, you are ready to become more physically active - sign the PARTICIPANT DECLARATION below:

- It is advised that you consult a qualified exercise professional to help you develop a safe and effective physical activity plan to meet your health needs.
- You are encouraged to start slowly and build up gradually - 20 to 60 minutes of low to moderate intensity exercise, 3-5 days per week including aerobic and muscle strengthening exercises.
- As you progress, you should aim to accumulate 150 minutes or more of moderate intensity physical activity per week.
- If you are over the age of 45 yr and **NOT** accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional before engaging in this intensity of exercise.

If you answered **YES** to **one or more of the follow-up questions** about your medical condition:

You should seek further information before becoming more physically active or engaging in a fitness appraisal. You should complete the specially designed online screening and exercise recommendations program - the **ePARmed-X+ at www.eparmedx.com** and/or visit a qualified exercise professional to work through the ePARmed-X+ and for further information.

Delay becoming more active if:

- You have a temporary illness such as a cold or fever; it is best to wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ **at www.eparmedx.com** before becoming more physically active.
- Your health changes - talk to your doctor or qualified exercise professional before continuing with any physical activity program.

- You are encouraged to photocopy the PAR-Q+. You must use the entire questionnaire and NO changes are permitted.
- The authors, the PAR-Q+ Collaboration, partner organizations, and their agents assume no liability for persons who undertake physical activity and/or make use of the PAR-Q+ or ePARmed-X+. If in doubt after completing the questionnaire, consult your doctor prior to physical activity.

PARTICIPANT DECLARATION

- All persons who have completed the PAR-Q+ please read and sign the declaration below.
- If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that the community/fitness center may retain a copy of this form for records. In these instances, it will maintain the confidentiality of the same, complying with applicable law.

NAME _____ DATE _____

SIGNATURE _____ WITNESS _____

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER _____

For more information, please contact

www.eparmedx.com
Email: eparmedx@gmail.com

Citation for PAR-Q+
Warburton DER, Jamnik VK, Bredin SSD, and Gledhill N on behalf of the PAR-Q+ Collaboration.
The Physical Activity Readiness Questionnaire for Everyone (PAR-Q+) and Electronic Physical Activity Readiness Medical Examination (ePARmed-X+). Health & Fitness Journal of Canada 4(2):3-23, 2011.

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Key References

1. Jamnik VK, Warburton DER, Makarski J, McKenzie DC, Shephard RJ, Stone J, and Gledhill N. Enhancing the effectiveness of clearance for physical activity participation; background and overall process. APNM 36(S1):S3-S13, 2011.
2. Warburton DER, Gledhill N, Jamnik VK, Bredin SSD, McKenzie DC, Stone J, Charlesworth S, and Shephard RJ. Evidence-based risk assessment and recommendations for physical activity clearance; Consensus Document. APNM 36(S1):S266-s298, 2011.
3. Chisholm DM, Collis ML, Kulak LL, Davenport W, and Gruber N. Physical activity readiness. British Columbia Medical Journal. 1975;17:375-378.
4. Thomas S, Reading J, and Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). Canadian Journal of Sport Science 1992;17:4 338-345.

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Appendix F: BIONEX Trial Data Collection Sheet

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials
Trial Data Collection Sheet

ID: _____ Date: _____

Keenoa ID: _____ Time: _____

Trial Number:

Supplement: GY / Milk / CHO / W

Estimated Energy Expenditure:

Meal Type: Regular / Vegetarian

Meal/Snack modifications:

Cycle day (naturally cycling):

Any illness in the last week: Details:		☐ YES	☐ **NO**
Feeling well today:		☐ **YES**	☐ NO
Time of last food/caloric drink:	Fasted 8+ hours?	☐ **YES**	☐ NO
Last exercise session:	Rested state?	☐ **YES**	☐ NO
Caffeine consumed:		☐ YES	☐ **NO**
Any medication taken today: Details:		☐ YES	☐ NO

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials
Trial Data Collection Sheet

Standing Reach (inches):

Timeline	Time of Day	Time post-exercise (h/min)	Mean Hematocrit	Muscle Soreness Rating (0-5)	Jump Height (cm)	Serum and Plasma Aliquots (# and Volume)
Baseline Draw						Serum: Plasma:
Exercise Session	Start: End:					
5min Post Exercise						Serum: Plasma:
Supplement 1						
1h Post Supplement						Serum: Plasma:
Supplement 2						
Lunch (2h post-exercise)						
4h Post Supplement						Serum: Plasma:
Provide Participants with Snacks and Meals, and final supplement for before bed. Remind participant to log food data on Keenoa and note the time of consumption. Remind participants to not treat their muscle soreness in any way (no stretching, Advil/Tylenol, exercise, icing, applying heat, massage etc.,). Remind them to come fasted in the morning, and not eat after ~10pm.						

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials
Trial Data Collection Sheet

Trial Day 2:

Feeling well today:		☐ **YES**	☐ NO
Time of last meal/supplement:	Fasted 8+ hours?	☐ **YES**	☐ NO
Caffeine consumed:		☐ YES	☐ **NO**
Any medication taken today: Details:		☐ YES	☐ NO
Muscle Soreness Treatment: (i.e., stretching, heat, ice etc.) Details:		☐ YES	☐ **NO**

Timeline	**Time of Day**	**Time post-exercise (h/min)**	**Mean Hematocrit**	**Muscle Soreness Rating (0-5)**	**Jump Height (cm)**	**Serum and Plasma Aliquots (# and Volume)**
24h Post Exercise			[redacted]			Serum: Plasma:

Appendix G: Godin-Shephard Leisure-Time Exercise Questionnaire

BIONEX Trials
Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials

ID :_____

GODIN-SHEPHARD LEISURE-TIME EXERCISE QUESTIONNAIRE

1. Considering a **7-day period** (a week), how many times on average do you do the following kinds of exercise for **more than 15 minutes** during your **free-time** (write on each line the appropriate number)?

	Times Per Week
(a) STRENUOUS EXERCISE **(HEART BEATS RAPIDLY)** (i.e. running, jogging, hockey, football, soccer, squash, basketball, cross country skiing, judo, roller skating, vigorous swimming, vigorous long distance bicycling)	_____
Please specify the types of exercise:	
(b) MODERATE EXERCISE **(NOT EXHAUSTING)** (i.e. fast walking, baseball, tennis, easy bicycling, volleyball, badminton, easy swimming, alpine skiing, popular and folk dancing)	_____
Please specify the types of exercise:	
(c) MILD EXERCISE **(MINIMAL EFFORT)** (i.e. yoga, archery, fishing from river bank, bowling, horseshoes, golf, snow-mobiling, easy walking)	_____
Please specify the types of exercise:	

2. Considering a **7-day period** (a week), during your leisure-time, how often do you engage in any regular activity long enough to work up a sweat (heart beats rapidly)?

1. OFTEN (≥4x/week)	**2. SOMETIMES (2-3x/wk)**	**3. NEVER/RARELY (0-1x/wk)**
☐	☐	☐

Appendix H: Familiarization Tracking Sheet

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials
Familiarization Session Tracking Sheet

Participant ID: _____ Date: _____
Age: _____ Time: _____

Trainer: _____
Researchers Present: _____

Body Composition

Height (cm):	Mean Weight:	BMI (kg/m^2):
BodyMetrix	Male Quads (mm): Chest (mm): Waist (mm): Body Fat %:	Female Quads (mm): Triceps (mm): Hip (mm): Body Fat %:
	Weight (kg)	Body Fat %
BIA		
BodPod		

Notes: (For BodPod, please note the clothing worn and any jewelry that remained on during the test. For the BIA, please note an estimate of water consumption.)

Jump Height Test:

Standing Reach: _____ feet _____ inches _____ total inches _____ total cm

Vertical Jump Attempt 1: _____ feet _____ inches _____ total inches _____ total cm

Vertical Jump Attempt 2: _____ feet _____ inches _____ total inches _____ total cm

Vertical Jump Attempt 3: _____ feet _____ inches _____ total inches _____ total cm

Best Vertical Jump – Standing Reach = _____ total inches _____ total cm

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials

Familiarization Session Tracking Sheet

Leg Press

Seat Height:

Intensity	**Weight**	**Reps**	**RPE**
Warm-Up			
RPE 3 (Easy)		8	
RPE 5 (Somewhat Difficult)		8	
RPE 7 (Hard)		8	
Trial Sets Until Failure			
Sets	**Weight**	**Reps**	**RPE**
Set 1			
Set 2			
Set 3			
Set 4			
e1RM: weight/[(1.0278) – (0.0278 x repetitions completed)] e1RM =			

Chest Press

Seat Height:

Intensity	**Weight**	**Reps**	**RPE**
Warm-Up			
RPE 3 (Easy)		8	
RPE 5 (Somewhat Difficult)		8	
RPE 7 (Hard)		8	
Trial Sets Until Failure			
Sets	**Weight**	**Reps**	**RPE**
Set 1			
Set 2			
Set 3			
Set 4			
e1RM: weight/[(1.0278) – (0.0278 x repetitions completed)] e1RM =			

BIONEX: Bone and Inflammatory Outcomes, the Nutrition and Exercise Trials

Familiarization Session Tracking Sheet

Seated Row

Intensity	**Weight**	**Reps**	**RPE**
Warm-Up			
RPE 3 (Easy)		8	
RPE 5 (Somewhat Difficult)		8	
RPE 7 (Hard)		8	
Trial Sets Until Failure			
Sets	**Weight**	**Reps**	
Set 1			
Set 2			
Set 3			
Set 4			
e1RM: weight/[(1.0278) – (0.0278 x repetitions completed)] e1RM =			

Cycling

Resting HR: Estimated Max HR (220-Age): 90% of Max HR:

Seat Height:

Workload	Time	HR	RPE
	4 min		
	1 min		
	1 min		
	1 min		
	1 min		
	1 min		
	1 min		
	1 min		
	1 min		

Warm-up Watts will be roughly their weight in kg, rounded to the lowest 10kg.
Increase by 0.5kg each min.
Watts = RPM*resistance (kg)

Notes: (Any notes about the veins of participants, food preferences and allergies.)

Appendix I: Exercise Trial Tracking Sheet

Exercise Tracking Sheet

Participant ID: _____ Date:_____

Trial: Milk GY CHO W Trainer:_____

Participant Maxes & Seat Height

Exercise	e1RM	60%	Actual Weight	70%	Actual Weight	80%	Actual Weight	Seat Height
Chest Press								
Leg Press								
Seated Row								

Exercise	Sets	Reps	Intensity	Load	Notes (RPE)		
Depth Jumps	3	10		BW			
Tuck Jumps	3	10		BW			
Split Jumps	3	10		BW			
Seated Box Jumps	3	10		BW			
Chest Press	3	10	60-80% 1RM				
Leg Press	3	10	60-80% 1RM				
Seated Row	3	10	60-80% 1RM				

*Take note of how many steppers used for depth jump

*Add RPE for each set for resistance exercises

Completed all 3 sets for the same reps and weight? Yes ☐ No ☐

If no, record the exercise, reps, weight and RPE for the incomplete exercise.

Exercise Tracking Sheet

Age:
Estimated Max HR (220-age):
90% of Max HR:
Cycling Max PO: 50%: 90%: Seat Height:
Weights Required
50%: 90%:

Cycling			RPM:	
Intensity	Time	Workload	HR	RPE
Warm-up Ramping Protocol				
50-70 W	1 min			
	1 min			
50% of max	1 min			
50% of max	1 min			
High Intensity Interval Bouts				
90% of max	1 min			
50% of max	1 min			
90% of max	1 min			
50% of max	1 min			
90% of max	1 min			
50% of max	1 min			
90% of max	1 min			
50% of max	1 min			
50% of max	2 min			

If the HR average of the first 3 intervals is greater than 85% of HR max, they are done.
[(HR1 + HR2 + HR3)/3]/HR max
[(____ + ____ + ____)/3]/____ =
If it is below 85%, do a 4th interval.