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ACUTE EFFECTS OF EXERCISE TIMING AND BREAKFAST MEAL GLYCEMIC INDEX ON EXERCISE-INDUCED FAT OXIDATION

by

PATRICK V. BENNARD

B.Sc., University of Ottawa, 2000

THESIS

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ABSTRACT

To examine the acute effects of exercise timing and meal glycemic index (GI) on fat oxidation and glycemic response, five apparently healthy young men participated in four randomly ordered morning trials during which measurements were made at rest, during exercise, and for two hours post-exercise. A factorial design [exercise timing (pre-prandial, post-prandial) x meal GI (low-GI, high-GI)] was used for repeated measures of energy expenditure and whole-body fat oxidation, as well as of plasma glucose and insulin levels after an overnight fast. Subjects were required to perform 400 kcal of moderate treadmill exercise either before consuming a 400 kcal low-GI (ELG) or high-GI (EHG) oatmeal breakfast, or after consuming the low-GI (LGE) or high-GI (HGE) meal. Exercising fat oxidation was significantly greater during ELG and EHG (14.7 ± 1.4 and 14.8 ± 3.2 g, respectively) than during LGE and HGE (8.9 ± 3.1 and 9.8 ± 2.7 g, respectively) ($p < .001$), as was total fat oxidation beyond rest and (ELG: 21.3 ± 3.7 g; EHG: 20.2 ± 5.9 g; LGE: 18.1 ± 6.0 g; HGE: 17.1 ± 3.4 g) ($p < .05$), although energy expenditure was unaffected by experimental conditions. No significant effect of meal GI on fat oxidation was observed and, unexpectedly, the glycemic response was not significantly different across experimental conditions. Total whole-body fat oxidation for the entire morning period is therefore greatest when exercise is performed in the post-absorptive state, a strategy that could help maximize acute exercise-induced fat oxidation.

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PART ONE: EMPIRICAL, THEORETICAL AND METHODOLOGICAL CONSIDERATIONS

CHAPTER I

INTRODUCTION

In order to derive energy for biological activity, the human body can draw upon multiple fuel sources, the partitioning of which depends on the nature of a particular activity. During moderate physical activity, for example, exercising muscle primarily uses endogenous carbohydrate and lipid fuels for energy demands, although protein can occasionally supplement these fuels. Endogenous lipids, which are stored primarily in adipose tissue as well as in proximity to mitochondria throughout muscle tissue (Vock et al., 1996), can provide a substantial portion of energy for exercise, although its utilization is mediated differently than that of carbohydrate. The differences in the utilization of these two primary fuel sources during exercise are as important as a sprint is different from a marathon. When physical activity is initiated, carbohydrate is the principal fuel source, particularly during more intense activity, whereas fat makes a gradual contribution as the duration of exercise progresses, particularly during less intense activity. Although fat-derived energy contributes substantially to the demands of prolonged exercise, it is important to understand that the availability of fat to exercising muscle during such activity is at least partly dependent on carbohydrate utilization (Coyle, Jeukendrup, Wagenmakers, & Saris, 1997; Romijn et al., 1993).

Dietary fatty acids (FA) are absorbed through the gut and are transported in the blood by chylomicrons and lipoproteins towards adipose tissue, where FA can be stored by esterification into triglycerides. Fatty acids can also be released from storage by lipolysis, and these non-esterified fatty acids (NEFA) can be taken up and used in liver, kidney, heart and skeletal muscle tissues (Coppack, Jensen & Miles, 1994). The process of lipolysis is generally stimulated by catecholamines and growth hormone during metabolic activation (Saltin & Astrand, 1993), such

as during physical activity, whereas insulin acts largely to inhibit lipolysis, particularly in the post-prandial period (Coppack et al., 1994). The balance of these metabolic processes is therefore largely dependent on the metabolic state of the individual, and it seems intuitive that if an increased lipid flux into the body occurs as a result of excessive dietary intake then the excess fat would be stored, thus inducing weight gain. Obesity, which is generally characterized by high levels of body fatness, may develop particularly from reduced fat utilization (Wade, Marbut & Round, 1990), a factor that may be more important than reduced energy expenditure (EE) (Zurlo et al., 1990). Thus, preventing the accumulation of body fat, as well as reducing fat stores, apparently necessitates both a negative lipid balance and a negative energy balance over time (Shah & Garg, 1996).

Although the factors involved in the development of obesity are generally a combination of genetics, behaviour, and lifestyle, the exact physiological mechanisms involved are not precisely understood. More importantly, the ideal means to treat obesity have not been precisely identified. When physical activity is used to decrease body fat, the desired effect is essentially the attainment of a repeated state of acute negative lipid balance, whereby total FA oxidation exceeds total FA esterification across the exercise and post-exercise periods (e.g. 24-hour period), despite ingested fat. However, the effectiveness of exercise in eliciting a decrease in fat mass is inconsistent (for review, see Votruba, Horvitz, & Schoeller, 2000). Dietary manipulation is also used to affect a state of negative lipid balance, and although it may be intuitive to consume fewer fat calories than it is to increase fat utilization, its effectiveness is also inconsistent (for review, see Avenell et al., 2004). There is undoubtedly considerable clinical interest for maximizing the effectiveness of these strategies, particularly when diet and exercise can both be used to promote EE and fat utilization. An example of a combined strategy involves

the timing of physical activity around food intake (i.e. a pre- versus post-exercise meal) in order to maximize acute negative lipid balance. However, the literature is unclear about the effectiveness of such a strategy, as comparative studies of pre-meal versus post-meal exercise are scarce.

Despite little supportive research (e.g. Zhu, Shi, & Shang, 1997), many people choose to exercise during their first waking hours. This is typically done with the belief that while blood sugar is low after the overnight fast, the mobilization and utilization of fats is increased, thus making fat a more important substrate for working muscles. Furthermore, it is unclear how the concomitant ingestion of a breakfast meal influences the exercise-induced increase in fat metabolism. Individuals who exercise in the early morning will typically consume a carbohydrate-rich breakfast meal sometime after the exercise session, and yet such a meal is known to inhibit fat oxidation (Coyle et al, 1997; Wee, Williams, Gray & Horabin, 1999). Certain studies have examined the effects of either a pre-exercise (Horowitz, Mora-Rodriguez, Byerley, & Coyle, 1997; Sidossis, Stuart, Shulman, Lopaschuk, & Wolfe, 1996) or post-exercise (Dionne, van Vugt, & Tremblay, 1999) meal on fat metabolism, yet very little research has compared these two scenarios, and the conclusions offered about exercise timing around meal intake are discordant (Matsuo & Suzuki, 1999; Welle, 1984). Furthermore, it is unclear how these conditions would affect fat metabolism when the glycemic index (GI) of the meal is altered. Consequently, if an ideal strategy of exercise timing can be elucidated, then addressing this gap in the literature would be clinically important for weight reduction, or for the prevention of weight gain. Thus, in the context of maximizing the acute increase in fat metabolism induced by exercise, it remains unclear how pre-exercise and post-exercise ingestion of different breakfast meals after overnight fasting affects exercise-induced fat oxidation.

Statement of the Problem

The purpose of the present study was to examine the exercise-induced variations in fat oxidation (i.e. during exercise and post-exercise) in lean men when a low- or high-GI breakfast meal was ingested before a morning exercise session, and to examine the variations in fat oxidation when these meals were ingested after exercise. In addition, the study proposed to examine variations in plasma levels of both glucose and insulin hormone across these conditions.

Hypotheses

The primary hypothesis for the present study postulated that total fat oxidation observed across the exercise and recovery periods (i.e. until the lunch meal) would be greatest when a low-GI meal was ingested before the exercise session. Previous studies have found that pre-exercise feeding does not affect the pattern of substrate utilization during exercise (Calles-Escandon, Devlin, Whitcomb, & Horton, 1991; Whitley et al., 1998). Furthermore, the findings of Dionne et al. (1999) suggested that a post-exercise meal could abolish the increased post-exercise fat oxidation. Finally, Matsuo and Suzuki (1999) found that post-prandial exercise, particularly after a high-fat rather than a high-carbohydrate diet, resulted in greater post-prandial fat utilization, while Davis, Sargent, Brayboy, and Bartoli (1992) found that post-prandial exercise elicited greater post-exercise EE than pre-prandial exercise. It was therefore predicted that the post-exercise meal conditions, particularly after a high-GI meal, would attenuate the increased post-exercise fat mobilization and utilization induced by moderate exercise.

The secondary hypothesis postulated that the pre-exercise, low-GI meal conditions would elicit the lowest levels of plasma glucose and insulin hormone during the post-exercise period relative to resting levels, whereas they would be highest under the post-exercise, high-GI meal conditions. Relatively higher insulin levels following a high-GI meal would elicit an exaggerated

response in plasma glucose uptake (Ludwig, 2002; Walton & Rhodes, 1997; Wu, Nicholas, Williams, Took, & Hardy, 2003), and this effect would be expected to be more pronounced post-exercise, as exercise would be expected to attenuate elevated insulin levels elicited by the pre-exercise meal (Jeukendrup, Saris, & Wagenmakers, 1998).

Definitions

For the present study, total fat oxidation was defined as the total grams of oxidized lipid fuel (i.e. from NEFA derived from plasma, and from triglycerides in all adipose tissue deposits) that contributed to energy requirements during the exercise and post-exercise periods. This measurement was performed by the analysis of expired oxygen and carbon dioxide concentrations, with whole-body protein oxidation assumed to be constant and negligible across experimental conditions, thus representing an estimate of whole-body fat oxidation. Fat oxidation was calculated in grams per minute, using stoichiometric equations described by Frayn (1983).

Exercise was defined as a single bout of continuous, moderate-intensity treadmill work. The actual intensity of the bout was determined from the assessment of each subject's level of maximal oxygen consumption (VO_2max), and from the assessment of each subject's capacity of maximal fat oxidation during exercise (FATmax, i.e. the individual percentage of maximal oxygen consumption at which the rate of fat oxidation is maximal, as described by Achten, Gleeson, & Jeukendrup, 2002). The volume of exercise was equivalent to the caloric content of the experimental breakfast meals (i.e. 400 kcal). This exercise bout was performed in the morning following an overnight fast, in order to represent early morning exercise under free-living conditions.

The experimental breakfast meals were defined as either low- or high-GI meals, based on the glycemic index with white bread as the reference food (see Foster-Powell, Holt, & Brand-Miller, 2002). The low-GI meal was composed of large-flake porridge oats, unsweetened apple juice, and fructose; the high-GI meal was composed of one-minute porridge oats, Gatorade sport drink, and sucrose. The calculated glycemic indices of the low- and high-GI meals differed from each other by a margin of 55 units. Furthermore, the meals were isocaloric (400 kcal), and contained the same quantity of dietary carbohydrate.

Finally, subjects were defined as lean, young, moderately active men; lean participants were defined as those with a body mass index of 25 kg/m^2 or less, and with a waist girth of < 90 cm (World Health Organization, 1998); young participants were defined as those of 20 to 29 years of age; and moderately active participants were defined as those who perform 30 to 45 minutes of moderately intense physical activity, three to five times per week.

Assumptions, Delimitations and Limitations

The present study assumed that the participants answered honestly to the pre-screening questions and criteria, and that they did not exhibit any metabolic dysfunctions. It was also assumed that the selected volunteers followed the pre-experimental protocols, such as fasting before experiments, maintaining normal dietary and physical activity habits, and following the prescribed dietary guidelines for macronutrient intake. It was also assumed that the test protocols for the screening visits were valid measurements of VO_2max (Ellestad, 2003) and of FATmax (Achten et al., 2002), and that indirect measurement of fat oxidation by expired gas analysis is a valid measurement of whole-body fat oxidation. The measurement of fat metabolism was limited to the analysis of expired gas for determining whole-body fat oxidation, as opposed to the measurement of localized fat oxidation in working muscles (i.e. arterio-venous difference

methods were unavailable for this study). Finally, the blood analysis techniques employed by the laboratories were assumed valid for the measurement of plasma glucose and insulin levels.

In order to answer the research question proposed in this study, the experimental conditions were delimited to the morning, post-absorptive period between 0730 and 1230. Within this timeframe, the post-exercise period was limited to two hours, between the end of the exercise bout or breakfast meal, and the beginning of the lunch meal. The exercise performed was delimited to treadmill work, because brisk walking or light jogging is a natural movement for almost all individuals. In addition, the experimental breakfast meals were delimited to two levels of glycemic index: low-GI (GI = 48.3), and high-GI (GI = 103.3), indexed against a white bread standard. In order to minimize inter-individual variance in metabolism, the selection of participants was delimited to lean young men whose body mass indices met the criteria for normal weight established by the WHO (1998). In addition, the ages of participants were delimited to the range of 20 to 29 years.

Significance of the Study

The present study attempted to add to the existing literature by addressing incongruent or lacking conclusions regarding the effects of pre- versus post-prandial exercise timing on fat oxidation. Only two known studies have compared exercise-induced fat oxidation in the presence of pre-exercise and post-exercise meals (see Matsuo & Suzuki, 1999; Welle, 1984). These two studies have provided discordant conclusions as to exercise timing around meal intake, and it is thus difficult to derive clinical applications for weight loss or weight maintenance from these conclusions. Therefore, in the context of maximizing the increase in whole-body fat oxidation induced by exercise, the present study has attempted to add to the literature by making such a comparison under conditions of free-living exercise and food intake, and by introducing

differences in the GI of the pre- or post-exercise breakfast meal. There also appears to be contradictory findings on the influence of a pre-exercise meal on fat mobilization and utilization, as well as contradictory findings on the influence of a post-exercise meal. For example, studies by Horowitz et al. (1997) and by Whitley et al. (1998) differed in their conclusions about the influence of a pre-exercise carbohydrate meal on the exercise-induced increase in fat utilization. Furthermore, studies by Dionne et al. (1999) and by Thompson, Townsend, Boughey, Patterson, and Bassett (1998) differed in their conclusions about the influence of a post-exercise meal on post-exercise substrate utilization. In light of these discordant findings, it was deemed necessary to pursue the investigation of the implications of exercise and concomitant food ingestion on fat oxidation.

CHAPTER II

REVIEW OF LITERATURE

This review of the literature pertinent to the submitted thesis has been revised from its original version, and upon suggestion of the thesis committee, has been submitted to and accepted for publication in the Canadian Journal of Applied Physiology prior to submittal of this thesis. The review article, entitled Maximizing Acute Fat Utilization: Effects of Exercise, Food, and Individual Characteristics (Bennard, Imbeault, & Doucet, in press), has therefore been included here in its final form as accepted by the journal's referees because it remains, in the thesis author's opinion, a most appropriate review of literature for the submitted thesis.

ABSTRACT: In discussion of the physiological mechanisms that regulate fat metabolism, and with consideration of the metabolic stimuli that modulate substrate metabolism, the issue of how an acute state of negative lipid balance can be maximized is addressed. The regulation of lipolysis by catecholamines and insulin is addressed, and the mechanisms of fatty acid mobilization and uptake by muscle are also briefly discussed. The implications of substrate availability and the hormonal response during physiological states such as fasting, exercise, and after food intake are also addressed, with particular regard to the influences on fatty acid mobilization and/or oxidation from eliciting these stimuli conjointly. Finally, a brief discussion is made of both the nature of exercise and the exercising individual, and how these factors influence fat metabolism during exercise. It is also a primary thrust of this paper to underline gaps in the existing literature with regard to exercise timing around food ingestion for maximizing acute lipid utilization.

Key words: Lipid balance, catecholamines, insulin, exercise, glycemic index

RÉSUMÉ: Les conditions selon lesquelles l'atteinte d'un bilan lipidique négatif est rendue possible sont discutées en mettant l'emphase sur les mécanismes physiologiques et les stimuli métaboliques modulant le métabolisme des lipides. La régulation de la lipolyse par les catécholamines et l'insuline est abordée, et les mécanismes qui influencent la mobilisation et l'entrée des acides gras dans le muscle sont aussi brièvement discutés. La disponibilité des substrats et la réponse hormonale lors de situations physiologiques telles le jeûne, l'exercice et après la prise alimentaire ainsi qu'une discussion de leurs effets combinés sur le métabolisme des lipides est présentée. Une brève discussion quant au type d'exercice ainsi qu'aux différences interindividuelles qui peuvent influencer l'utilisation des lipides au cours de l'exercice fait également l'objet de ce travail. Enfin, un objectif important de cet article est de mettre en évidence les lacunes qui persistent dans la littérature en ce qui a trait aux effets de la prise alimentaire sur les variations de métabolisme lipidique engendrées par l'exercice.

Mots clefs : Balance lipidique, catécholamines, insuline, exercice, indice glycémique

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Post-exercise substrate compensation

Nature of Exercise, and Characteristics of the Individual

Exercise duration and intensity

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Conclusion

Introduction

Aside from its other important physiological roles, body fat constitutes a major source of stored energy for human beings. Purposeful physical activity accounts for the second largest utilization of fat-derived energy after resting metabolism. However, body fat mass can often increase because energy intake exceeds energy needs over prolonged periods of time. As individuals become aware of this, they often purposely engage themselves in regular sessions of physical activity in order to attenuate, and ideally to reverse, the accumulation of body fat. The ruling principal of any strategy that aims at inducing a reduction in body energy reserves is the achievement and maintenance of a negative energy balance over extended periods of time. Nonetheless, lipid balance remains an important determinant of energy balance (Flatt, 1988). Indeed, low whole-body lipid oxidation has been shown to be predictive of weight gain (Zurlo et al., 1990) and regain (Valtuena et al., 1997). Furthermore, the literature suggests that the acute energy and lipid deficits created by purposely engaging oneself in exercise can be completely compensated when the diet is rich in lipids (Tremblay et al., 1994; King & Blundell, 1995). Therefore, an important dimension of weight control is lipid balance (Tremblay, 1995). Thus, lipid balance will be the focus of this paper, and it is assumed herein that a negative energy balance must also be achieved and maintained through exercise and/or dietary manipulations in order to reduce body energy reserves and hence induce a negative lipid balance.

Lipid balance indicates the sum of all metabolic activity resulting in either the esterification (i.e. storage) or oxidation (i.e. utilization) of ingested or synthesized fatty acids (FA). A negative lipid balance is achieved under conditions when more fat is oxidized than is ingested from the diet during negative energy balance (Shah & Garg, 1996). The available literature suggests that achieving and maximizing negative lipid balance is an important aspect

for consideration of the effectiveness of diet and exercise in decreasing fat mass, and with good reason: Numerous factors related to food intake and physical activity influence both the oxidation and esterification of FA. It is thus within the context of such factors that this paper proposes to address the issue of achieving and maximizing a state of acute negative lipid balance.

Recent studies have examined the ideal volume (Bond Brill et al., 2002) or intensity (Achten et al., 2002; Bergman & Brooks, 1999; Deriaz et al., 2001) of exercise for maximizing fat utilization. The effects of caloric content (Martin et al., 2000) and macronutrient composition (Whitley et al., 1997) of a meal on fat metabolism have also been the subject of recent study. However, fewer studies have assessed the interactions between food intake and exercise on fat oxidation. Research efforts have examined such interactions during one metabolic state or another (e.g. fasting, post-prandial, exercising), but few have comparatively examined them across a combination of metabolic conditions (e.g. exercise timing around meal ingestion). Ultimately, it is of clinical interest to the exercising individual to take advantage of factors that increase fat oxidation, although the literature appears to provide little in the way of concrete strategies on how to maximize acute fat utilization through diet and exercise interventions.

Thus, the first part of this paper will highlight the principal physiological functions that regulate fat metabolism, from hormonal regulation of mobilization, to enzymatic breakdown of non-esterified fatty acids (NEFA). Specifically, the regulation of fat metabolism by catecholamines and insulin, the mediation of FA mobilization by lipolytic enzymes, and the mechanisms of FA uptake will be addressed. Secondly, the various influences of substrate availability on fat metabolism across fasting, post-prandial, and exercising conditions will be explored. A critical synopsis will be made of the literature pertaining to the food and respiratory quotient, as well as to the glycemic index of a meal, while the discussion will also address the

metabolic effects of post-absorptive, exercising, and post-prandial states. The final part will explore how fat utilization is influenced by the nature of exercise, as well as by characteristics of the exercising individual. The focus of this paper is then to examine how fat metabolism can be manipulated. The hormonal and enzymatic control of FA mobilization and oxidation is, naturally, the physiological basis upon which fat metabolism is regulated, and so should be addressed first.

Regulation of the Mobilization and Uptake of FA

Numerous hormones and enzymes take part in the regulation of substrate metabolism, but this part of the discussion will focus on those which impact most heavily upon FA mobilization from adipose tissue, rather than from skeletal muscle, as well as those which impact upon NEFA oxidation in muscle tissue. The metabolic responses to these hormones and enzymes in lipolysis and fat oxidation will be addressed in the contexts of post-absorptive, post-prandial, and exercising states. Although a number of hormones can partake in the regulation of the supply of NEFA under resting conditions, insulin and catecholamines appear to be the most important that affect lipolysis, i.e. the process of triglyceride breakdown and subsequent release of NEFA from adipose tissue (Coppack et al., 1994). It should be noted that while the liver and kidneys play important roles in controlling blood lipid levels, this section will focus on FA fuel availability from adipose tissue, where fat metabolism is primarily regulated by these hormones. The efflux of NEFA supplied by lipolysis exceeds the rate of fat oxidation under both post-absorptive resting (Wolfe & Peters, 1987) and exercising (Wolfe et al., 1990) conditions, and because the surplus is reesterified into triglycerides, it appears that supply overcomes demand (Coppack et al., 1994), although percent reesterification of released NEFA decreases from rest to exercise (Wolfe et al., 1990). The effects of both fasting and food ingestion on NEFA supply are

undoubtedly pertinent to the discussion of lipid balance, as these stimuli induce daily variations in fat metabolism. The influence of physical activity on the hormonal control of fat oxidation, meanwhile, is quite particular, and it will equally be addressed in this section.

Catecholamines. Circulating epinephrine and norepinephrine (secreted by the adrenal medulla), as well as sympathetic nerve-released norepinephrine, are collectively referred to as the catecholamines. These hormones are part of the "fight or flight" autonomic response towards stress, and are major stimulatory factors for lipolysis in adipose tissue cells (Coppack et al., 1994; Galster et al., 1981; Saltin & Astrand, 1993). At rest, fat mobilization is stimulated during fasting, an effect of a more readily epinephrine-stimulated lipolysis (Jensen et al., 1987), once increasing plasma epinephrine levels have reached a lipolytic threshold (Galster et al., 1981), although other mechanisms may increase lipolysis during prolonged (i.e. 84-hour) fasting, as evidenced during a blockade of beta-adrenergic receptors (Klein et al., 1989) (N.B. adrenergic mechanisms are discussed in the next section). Catecholamine action also plays an important part in the stimulation of lipolysis during exercise, along with other factors such as decreased insulin secretion and increased adipose tissue blood flow, whereby catecholamine secretion increases at the onset of exercise and stimulates FA mobilization during the activity (Arner, 1995b), although the lipolytic action of exercise-induced epinephrine secretion is attenuated at 45% compared to 25% VO_2max (Mora-Rodriguez & Coyle, 2000). The advantage of a catecholamine-stimulated increase in NEFA availability is apparent when, as the availability of NEFA is increased, the extraction of NEFA from the blood by working muscle increases as well, and therefore uptake by muscle is at least partially affected by the rate of lipolysis (Groop et al., 1991). However, this relationship no longer holds at higher exercise intensity (i.e. 85% of VO_2max), where plasma NEFA utilization is observed to decrease even while NEFA supply is maintained (Romijn et al.,

1995). Catecholamine secretion is also involved in the mobilization of NEFA liberated from intramuscular triglycerides during exercise (Langfort et al., 1999). In general, catecholamine action is a major regulatory factor of fat metabolism during periods of stress to the body, such as fasting and exercise, as well as during cold exposure, such that these hormones are largely responsible for promoting fat metabolism by increasing the supply of NEFA to tissues for oxidation.

Adrenergic regulation of lipolysis. In addressing the influences of hormonal action on FA mobilization, a brief discussion should be made of the sites of activation for epinephrine, and how the regulation of lipolysis at these sites influences fat utilization. In adipose tissue cells, adrenergic receptors respond to catecholamine action, whereby α-adrenergic inhibitory effects modulate lipolysis at rest, and β-adrenergic stimulatory effects modulate lipolysis during exercise (Arner et al., 1990). Furthermore, although β_1-adrenoceptor activation in vitro is weakly counteracted by α_2-adrenoceptor activation (Lafontan et al., 1983), α_2-adrenergic receptors appear to exhibit an antilipolytic effect during exercise in untrained individuals, particularly with higher catecholamine levels during increasing exercise intensity (De Glisezinski et al., 2001; Stich et al., 1999). It should also be noted that regional fat depot heterogeneity exists in the sensitivity to adrenergic stimulation, and thus to α_2-adrenoceptor activation (Lafontan et al., 1979), a function of variations in the distribution of β- and α_2-adrenoceptor binding sites (Mauriège et al., 1987). Increased adipocyte sensitivity to β-adrenergic stimulation, along with increased epinephrine concentrations, may account for increased FA mobilization during fasting (Jensen et al., 1987), whereas in vitro evidence suggests that exercise increases this β-adrenoceptor-mediated responsiveness to catecholamine action (Wahrenberg et al., 1987). It therefore appears that adrenergic mechanisms provide a rather tight control of FA mobilization

between resting and exercising conditions, at least under conditions of normal dietary intake and normal body weight, although, the degree of adipocyte responsiveness appears to be regionally dependent (Arner, 1995a; Bouchard et al., 1993; Portillo et al., 2000).

As previously noted, increased extraction of NEFA from the blood by working muscle is associated with increased plasma NEFA availability (Groop et al., 1991), which underlines the importance of adrenergic stimulation by exercise in promoting an acute negative lipid balance. However, recent research has shown that whole body lipolytic activity of abdominal adipose tissue is impaired in upper body obese women (Horowitz & Klein, 2000), and that impaired β-adrenergic-mediated lipolysis is determinant of low fat oxidation rates in obese men (Imbeault et al., 2000; Snitker et al., 1998). The obese state may then render it difficult to achieve an acute state of negative lipid balance independently of the effects of physical activity.

Insulin. Just as important to the regulation of fat metabolism is the involvement of insulin hormone in the regulation of FA mobilization and oxidation. Plasma NEFA reesterification is particularly sensitive to insulin action (Campbell et al., 1992), such that even low concentrations of plasma insulin can be sufficient to suppress adipose tissue lipolysis at rest (Bonadonna et al., 1990). Thus, whereas catecholamine-activated α-adrenoceptors modulate lipolysis at rest, insulin action modulates lipolysis post-prandially. The problem then in regards to fat oxidation is that, as both plasma NEFA and insulin levels are major determinants of total lipid oxidation (Groop et al., 1991), suppression of plasma NEFA oxidation following a high-carbohydrate meal may induce an acute positive lipid balance. Furthermore, research by Sidossis et al. (1996) demonstrated that insulin in conjunction with glucose, in addition to inhibiting lipolysis, appear to regulate the entry of long chain fatty acids into the mitochondria, and thus concluded that glucose availability determines the nature of substrate oxidation. This is rather contradictory to

the early proposition that glucose uptake and phosphorylation is reduced by NEFA oxidation inducing citrate accumulation (Randle et al., 1963), and while the precise regulation mechanism of a glucose-fatty acid cycle lacks supporting evidence, the conclusion of Sidossis and colleagues (1996) appears congruent with the literature on insulin addressed thus far, and therefore may be pertinent in terms of achieving and maximizing an acute negative lipid balance, or rather in terms of avoiding a positive lipid balance. Thus, the sensitivity of carbohydrate metabolism to alteration by carbohydrate intake and insulin action signifies that food choices are important for consideration of their potential to acutely inhibit fat catabolism. However, the implications of a typical mixed meal, rather than a high-carbohydrate meal, on insulin-mediated substrate oxidation have not been sufficiently investigated in the context of lipid balance.

During food deprivation, insulin levels decrease just as epinephrine levels increase, and so lipolysis is stimulated (Wolfe et al., 1987). A similar effect is observed during exercise, along with a lesser inhibition by norepinephrine of pancreatic insulin release (Jeukendrup et al., 1998). This reciprocal interaction between these two hormones is paramount to the up-regulation of fat metabolism, particularly during periods of stress such as exercise and fasting, as opposed to food ingestion, which promote an increased fat mobilization. Research by Jensen et al. (1987) showed that the increased lipolysis induced by short-term fasting is less completely suppressed by an equivalent insulin infusion. This would suggest that the effects of epinephrine action may be predominant over insulin action, perhaps in relation to the aforementioned responsiveness of β-adrenoceptors. However, Klein et al. (1990) showed that even when plasma glucose was maintained throughout prolonged fasting by glucose infusion, the lipolytic response to epinephrine still increased with fasting, and plasma insulin still declined. This suggests then that some mechanism related to fasting other than plasma glucose level is helping maintain the

lipolytic response to epinephrine. Increased cortisol secretion during prolonged fasting may partially account for this effect, as it is known to stimulate lipolysis at physiologically high levels (Djurhuus et al., 2002).

Finally, the issue of insulin-inhibited fat metabolism is not fully addressed until the influence of this hormone is examined in the context of pre-exercise food intake. Montain et al. (1991) demonstrated that 6 hours of fasting were required after ingestion of a 500- to 600-kcal carbohydrate meal before carbohydrate oxidation and plasma glucose homeostasis during exercise were similar to values after an overnight (i.e. 8- to 12-hour) fast. This is an effect of insulin levels rising in response to meal ingestion, and then decreasing as fasting is prolonged. It was also demonstrated in the latter study that lipolysis during exercise increases in direct proportion to the length of fasting. Furthermore, Horowitz et al. (1997) also demonstrated that a pre-exercise glucose meal induces an insulin-mediated suppression of lipolysis during exercise. In this manner, they showed that the elevated fat mobilization elicited by exercise can be attenuated by prior carbohydrate ingestion to the point at which lipolysis equaled and apparently limited fat oxidation. These findings, along with the aforementioned evidence by Sidossis et al. (1996), suggest that a session of exercise performed after ingestion of a carbohydrate meal may not be conducive to maximizing an acute state of negative lipid balance incurred by the exercise bout alone. In consideration of this evidence, however, it remains unclear how insulin-mediated inhibition of post-exercise fat metabolism from a pre-exercise meal compares to that from a post-exercise meal.

Growth hormone. Another hormone that appears to have an important influence on fat metabolism is growth hormone (GH), which has been shown to have a stimulatory effect on lipolysis and fat oxidation (Bak et al., 1991; Goodman & Grichting, 1983; Leung & Ho, 1997).

GH may have a role in increasing lipolysis during starvation (Coppack et al., 1994), as well as during exercise (Gibney et al., 2003), but it appears to have a more important and direct role in the elevation of fat oxidation during the recovery period (Pritzlaff et al., 2000). Although fat mobilization and oxidation are increased by GH, the effect on oxidation is indirect (i.e. mediated by the hormone's lipolytic activity) (Piatti et al., 1999), and these effects have been shown to be dose-dependent (Moller et al., 1992). Thus, the greatest potential for GH towards affecting a negative lipid balance may involve the elevation of post-exercise fat oxidation. Furthermore, exercise-induced secretion of GH is also apparently enhanced following a low-carbohydrate pre-exercise diet (Galbo et al., 1979), which suggests that exercise performed in the fasted state may elicit a greater GH-mediated increase in fat metabolism. However, although GH stimulates FA mobilization during exercise, it may be of secondary importance or priority after the secretion of epinephrine. Furthermore, it is unknown how the elevation of post-exercise fat oxidation induced by GH is affected by post-exercise food intake. More research is required in order to fully elucidate the effects of GH on fat metabolism across various metabolic states.

Lipolytic enzymes. Although the discussion thus far has encompassed the implications of lipolytic and antilipolytic hormones on FA mobilization and oxidation, a brief discussion should be made of the enzymes in adipose tissue that are responsible for hydrolyzing fatty acids from triglycerides and from circulating lipoprotein-triglyceride complexes. Catecholamine action is transferred at the cellular level by adrenergic receptors; subsequently, the hydrolysis of FA from triglycerides is initiated by the activation of intracellular hormone-sensitive lipase (HSL), whereas insulin inhibits the action of this enzyme (Coppack et al., 1994). Lipoprotein lipase (LPL), on the other hand, is located primarily on the capillary walls, and is stimulated by the presence of circulating lipoprotein complexes as well as by post-prandial insulin secretion to

promote NEFA uptake by adipocytes for reesterification (Coppack et al., 1994; Jeukendrup et al., 1998). The action of HSL is considered a rate-limiting step in the mobilization of FA because the balance of catecholamine and insulin actions regulates the enzyme, and because most measurable post-absorptive lipolysis is mediated by adipose tissue HSL (Coppack et al., 1994; Jeukendrup et al., 1998). HSL appears then to be a common mediator for epinephrine- and insulin-mediated fat metabolism. The relationships between epinephrine, insulin, and the lipases, and their involvement in FA turnover, are illustrated in Figure 1. Furthermore, HSL becomes reactivated in the starved state as insulin activation is suppressed, resulting in stimulated lipolysis, and thus increasing the availability of NEFA for use as fuel (Coppack et al., 1994). It follows that this effect may be compounded if exercise is performed during the fasted state, when lipolysis is more sensitive to epinephrine (Jensen et al., 1987).

Circulation and uptake of NEFA. Although the regulatory hormones and the lipolytic enzymes of fat metabolism have been extensively researched throughout the past several decades, recent research has permitted an understanding of the mechanisms involved in the circulation and uptake of NEFA. Ultimately, it is at the site of oxidation where a positive or negative lipid balance is created, assuming a consistent dietary fat intake. Whereas the lipolytic hormones regulate the supply of NEFA to be released into the plasma, most released NEFA circulate while bound to and forming a complex with albumin, and are thus made available to tissues for use as fuel (Spector, 1975), although the rate of NEFA uptake from the circulation is still dependent in part upon the NEFA plasma-tissue gradient (Groop et al., 1991). In addition, Spector (1975) suggested that fatty acids of different chain length (i.e. short, medium, or long) might compete with each other for the binding with albumin; otherwise, unbound NEFA are incorporated into lipoproteins in the liver. However, short- and medium-chain fatty acids

represent only a small portion of total fat oxidation (Jeukendrup, 2002), and the literature does not suggest that the actual circulation of NEFA constitutes a limiting factor for whole-body fat oxidation.

Finally, whereas albumin is responsible for transporting NEFA to muscle cells, which are a major site of fat oxidation, NEFA are taken up into the cells by special carrier proteins known as fatty acid binding proteins, specifically in the form of FABPpm and FAT/CD36 (Bonen et al., 1998; Bonen et al., 2002). It is within the cell where fatty acids are activated by conversion into fatty acyl-CoA, and then translocated across the mitochondrial membrane through the action of the enzyme carnitine palmitoyl transferase 1 (CPT1), after which fatty acyl-CoA is converted (through β-oxidation) into the oxidizable acetyl-CoA (Jeukendrup, 2002). CPT1, however, appears to be strongly inhibited by malonyl-CoA, which is formed by the catalysis of acetyl-CoA (Jeukendrup, 2002; Rasmussen et al., 2002), and is probably regulated by the presence of glucose and insulin (Saha et al., 1997). The precise nature and importance of this intracellular regulatory mechanism of fat metabolism, compared with extracellular regulation, remains to be verified.

In final consideration of the evidence presented on the physiological regulation of fat metabolism, lipid balance is indeed very dependent on hormonal control. The responses of catecholamine and insulin hormones to exercise or food ingestion are reciprocally strong enough to serve as initiators of changes in substrate partitioning, specifically in regards to their effects on fat metabolism. Much of the literature discussed here addresses the stimulatory action of catecholamines, as well as the inhibitory action of insulin, on fat metabolism during various metabolic states (i.e. post-absorptive and exercising states). It is clear that an acute negative lipid balance can be achieved when exercise is performed in the fasted state. However, as food must be ingested at some time under free-living conditions, and in light of the evidence of suppressed

fat oxidation following carbohydrate intake, it is currently unclear whether a meal should precede or follow the exercise period in order to maximize the acute exercise-induced increase in fat oxidation. It is also important to consider that energy balance must remain negative in order to benefit from an acute negative lipid balance induced by exercise. A discussion of the available evidence that pertains to fat oxidation during various metabolic states may therefore provide more insight for maximizing an exercise-induced negative lipid balance.

Substrate Availability and Oxidation Across Various Metabolic States

Although food is essential for biological activity, what is important to this discussion is the composition of ingested food, because substrate metabolism responds differently to the availability of different fuel mixtures. In the first part of this paper, the effects of carbohydrate metabolism on fat metabolism were addressed, including the effects of ingested carbohydrate on fat metabolism. Therefore, two pertinent and measurable concepts here are the food quotient and the respiratory quotient. The food quotient (FQ) of ingested foodstuffs indicates the ratio of carbon dioxide produced to oxygen consumed during the complete oxidation of a representative sample of the food, and indicates the contributable provision of energy from carbohydrate and lipid, as well as from protein (Flatt, 1987). The respiratory quotient (RQ), in the context of metabolic activity, indicates the ratio of carbon dioxide production to oxygen consumption at the cellular level, and indicates the contributions of carbohydrate, fat and protein to oxidative metabolism. These ratios exist and are physiologically important because, per mole of adenosine triphosphate produced, the oxidation of fat requires more oxygen than that required for carbohydrate oxidation (Astrand & Rodahl, 1986). Carbohydrate and fat are the two main oxidative substrates for a given energy demand under normal circumstances, and there is no known evidence of either substrate being completely shunted from oxidative metabolic pathways

under normal conditions. As such, the focus of this part of the paper is to address how variations in the oxidation of one substrate are seemingly linked to the availability of the other during different metabolic states.

Respiratory quotient. Fat oxidation is most often measured by changes in RQ (i.e. substrate turnover at the cellular level), or estimated by changes in respiratory exchange ratio (RER, or non-protein RQ) (i.e. concentrations of inspired and expired gasses at the mouth). It should be noted that the term RQ is often used interchangeably with RER, particularly when referring to bouts of extended exercise, and it is used in this paper in respect of its use in the literature. After an overnight fast, fat is the main oxidative substrate in individuals who consume a diet that is balanced for energy requirements and weight maintenance, and RQ during this time is typically ~0.80 (Acheson et al., 1984), with the possibility of lower regional (i.e. leg) values (Kelley et al., 1990). After a carbohydrate-rich meal (Acheson et al., 1984), however, or during physiological hyperinsulinemia (Kelley et al., 1990), glucose becomes the principal oxidative substrate, whereupon RQ increases to values near 1.0. Individual variances exist in fasting RQ regardless of fat intake, such that factors other than diet may influence the ratio of fat to carbohydrate oxidized (e.g. exercise trained state, obesity) (Acheson et al., 1984). Nevertheless, it appears that the maintenance of a negative lipid balance can be facilitated in normal individuals if increases in RQ to values near 1.0 are avoided, i.e. by avoiding high-carbohydrate intake, and also if a low RQ is maintained by regular light or moderate exercise, by reduced caloric intake, or by fasting. Indeed, lipid oxidation after an overnight fast can account for more than 70% of total energy expenditure, during which time the rate of appearance (Ra) of plasma NEFA exceeds lipid oxidation, and continues to do so as fasting is prolonged (Coppack et al., 1994).

Substrate availability and substrate partitioning. RQ reflects the partitioning of oxidized substrates, and one of the factors that influence this partitioning is the mixture of ingested food. For example, it has been demonstrated that the intake of carbohydrate (i.e. a meal with a high FQ) induces an increase in the oxidation of carbohydrate from the fuel mix (i.e. a high RQ) (Schutz et al., 1989), whereas in the same study no increase in fat oxidation was observed after fat was supplemented to the experimental diet. Other studies, however, have demonstrated that a short-term fat-rich, low-carbohydrate diet (i.e. a low FQ diet) results in a relatively increased contribution of fat to the oxidative metabolism (i.e. a low RQ) (Jansson & Kaijser, 1982), although neither the length nor the degree of saturation of fatty acid chains were considered in this study. Later research has since provided evidence of an increased rate of fat metabolism with a dietary substitution of medium-chain over long-chain triglycerides (Stubbs & Harbron, 1996), as well as evidence of an increased rate of basal fat oxidation following a week-long diet with a lower polyunsaturated-to-saturated fatty acid ratio (Jones & Schoeller, 1988). Furthermore, Griffiths et al. (1994) observed an increase in fat oxidation for six hours after fat was added to a carbohydrate meal, despite elevated levels of glucose and insulin. Even if a very large amount of fat is consumed, however, only a meager increase in fat oxidation will occur if a small dose of carbohydrate is ingested as well (Flatt, 1995; Flatt et al., 1985). It therefore appears that the suppression of fat oxidation elicited by a high-carbohydrate (i.e. a high FQ) meal is more important than the increase in fat oxidation elicited by a high-fat (i.e. low FQ) meal. This notion is supported by later evidence that glycolytic flux regulates fat oxidation during exercise (Coyle et al., 1997). It should also be stressed that, even if repeatedly adding fat to a meal increases fat oxidation in the long-term, the addition of fat is intuitively counterproductive to the maintenance of a negative lipid balance unless energy balance remains negative. Furthermore, in the context

of physical activity, a moderate- or high-carbohydrate meal may simply be a more natural choice because adequate levels of muscle glycogen should be maintained for sustained activities.

It has been suggested that because protein and carbohydrate intake primarily determine the rates of amino acid and carbohydrate oxidation, the rate of fat oxidation is set by the difference between total energy expenditure and the energy intake from the other two substrates, and therefore not necessarily set by the intake of fat (Flatt, 1987). Furthermore, a meal with a fixed carbohydrate and protein content apparently elicits no effect on postprandial oxidative substrate mix whether fat is present in the meal or not (Flatt, 1987). However, although lipolysis normally exceeds fat oxidation at rest (Coppack et al., 1994), the utilization of fat can be limited by plasma NEFA availability when exercise is involved. Horowitz et al. (1997) have demonstrated such a limitation whereby a lipid infusion during exercise restored fat oxidation following a glucose-insulin response, although the restoration was only partial. This led them to speculate that the ingestion of carbohydrate had additional effects on fat oxidation above and beyond restricted fat availability. Furthermore, an increased glucose uptake resulting from carbohydrate ingestion during long-duration exercise, rather than before exercise, may account for decreased fat oxidation under conditions where suppressed lipolysis does not appear to limit fat oxidation (Horowitz et al., 1999). Earlier research (Hodgetts et al., 1991) demonstrated that retained NEFA were released from adipose tissue after exercise, leading them to suggest that the supply of NEFA from adipose tissue is the major limiting factor for fat oxidation during exercise, although these implications were ascertained without the measurement of net fat oxidation. In another study, however, plasma NEFA levels were maintained by lipid infusion between moderate and high intensities, although a decrease in fat oxidation was observed during high-intensity exercise (Romijn et al., 1995). This latter evidence suggests that fat oxidation is not

limited strictly by NEFA availability, considering that an increase in glycolytic flux during exercise due to pre-exercise glucose ingestion has been shown to inhibit the mitochondrial uptake of long-chain FA (Coyle et al., 1997). Thus, while these studies have demonstrated that the utilization of NEFA can be limited at the cellular level, the notion that the supply of NEFA to working muscle is a limiting factor for fat oxidation during exercise appears to be irresolute.

Glycemic index. The glycemic index (GI) rates and classifies the blood glucose concentration response, a function of the rates of appearance and disappearance of plasma glucose, following the digestion of a given type of carbohydrate, whereby the glycemic response is indexed against a standard (e.g. glucose or white bread) (Schenk et al., 2003; Wolever, 1990). The ingestion of high-GI carbohydrates produces a sharper rise in blood glucose, which elicits a more pronounced insulin response (Walton & Rhodes, 1997), and thus a sharper rise in carbohydrate uptake and oxidation. The ramifications of this response during subsequent exercise include a decrease in plasma fatty acids levels (Febbraio et al., 2000; Wee et al., 1999), and in fat oxidation (Wee et al., 1999). It should be noted that although the latter study reported no differences in insulin levels after the high-GI meal such as were observed by Febbraio et al. (2000), and although the time of pre-exercise meal ingestion differed greatly between the studies (3 hours vs. 30 minutes), both observed significantly lower blood glucose levels after the high-GI meal. Moreover, both the GI and the quantity of carbohydrate ingested in a mixed meal strongly determine the insulin response (Wolever & Bolognesi, 1996). In particular, a low-GI high-carbohydrate meal has been shown to elicit a greater rate of fat oxidation during exercise than does a high-GI meal (Wu et al., 2003). Taken together, these findings suggest that the consumption of a carbohydrate meal need not dramatically inhibit fat oxidation if the GI is low and the meal is of adequate size.

Post-prandial and exercising substrate oxidation. The pattern of substrate utilization during exercise has been shown to be resistant to variations in substrate availability induced by a pre-exercise meal, whereas post-prandial (Whitley et al., 1998) and possibly post-exercise (Calles-Escandon et al., 1991) substrate utilization were not. However, the findings of these studies cannot be reconciled with abundant research that has shown otherwise, and that supports the role of physical activity in lowering lipid balance. Aside from previously addressed studies that have demonstrated inhibited fat oxidation during exercise after carbohydrate ingestion (Bergman & Brooks, 1999; Coyle et al., 1997; Wee et al., 1999; Wu et al., 2003), Montain et al. (1991) observed an increase in carbohydrate oxidation and a decrease in plasma NEFA levels during exercise up to 6 hours after a pre-exercise carbohydrate meal, although they speculated that the size of the meal might have been a factor. This was later supported by evidence of fat oxidation limited by FFA availability during exercise following a pre-exercise carbohydrate meal (Horowitz et al., 1997). The notion that emerges from these and previously discussed investigations is that pre-exercise carbohydrate ingestion may affect substrate utilization in such a way as to increase carbohydrate oxidation and concomitantly decrease fat oxidation during the pre- as well as post-exercise resting periods. Thus, a high-FQ pre-exercise meal may counterbalance the effect of increased fat oxidation elicited by fasting or by exercise, which could impose a potential constraint on the use of exercise for fat loss, particularly if caloric intake is not more than compensated by exercise energy expenditure. In final consideration of the influence of a meal on RQ, Welle (1984) compared exercise RQ before and after a high-carbohydrate meal, and demonstrated that post-prandial exercise RQ was higher than pre-prandial exercise RQ. It was thus suggested by the author that post-prandial exercise is no more beneficial for weight maintenance than is pre-prandial exercise. This inference is tenuous,

however, because the experimental exercise protocol was of intermittent nature (i.e. repeated 15-minute bouts of exercise every hour), and therefore not accurately representative of free-living exercise. Furthermore, it is unknown how factors such as the meal's GI affects exercise RQ across these conditions, or how these variations in substrate oxidation progress throughout the post-exercise period. The composition of the meal, however, affects post-exercise RQ, as Matsuo and Suzuki (1999) showed that post-prandial exercise, particularly after a high-fat meal, elicited a lower RQ. Furthermore, post-prandial exercise may be of more benefit to obese individuals, as it has been shown to elicit greater energy expenditure in the first three hours post-exercise than pre-prandial exercise (Davis et al., 1992). Further research is required in order to clarify which conditions of exercise timing around meal ingestion permit the greatest total fat oxidation across the exercise period.

Post-exercise fat oxidation. After prolonged physical activity of moderate intensity, fat utilization is increased in the post-exercise period (Bielinski et al., 1985; Kiens & Richter, 1998). Moreover, the stimulation of lipid oxidation has been observed for as long as 18 hours after the cessation of exercise (Bielinski et al., 1985), and has been shown to account for more than 50% of oxidative metabolism in the 18-hour post-exercise period following exhaustive glycogen-depleting exercise (Kiens & Richter, 1998), despite the intake of a carbohydrate-rich pre-exercise meals in both studies. Therefore, there is great potential for attaining a state of negative lipid balance in the aftermath of long-duration exercise, although the primary working muscles have been observed to take up only a small fraction of the substantial post-exercise lipid mobilization after moderate-intensity exercise (Mulla et al., 2000), which suggests a role for other tissues (e.g. heart, liver). Interestingly, although NEFA reesterification is decreased in transition from rest to exercise, NEFA reesterification appears to dramatically increase at the cessation of exercise

(Wolfe et al., 1990). As previously discussed, however, the rate of fat oxidation is not influenced strictly by NEFA availability, and more importantly, a negative lipid balance is largely determined by increased rates of fat oxidation.

Regardless of precisely how FA mobilization and oxidation are regulated in the post-exercise period, the rates of fat oxidation and oxygen consumption appear to be greater after high-intensity exercise (i.e. 75% VO_2max) compared to low-intensity (i.e. 50% VO_2max) (Phelain et al., 1997). These findings, although observed for only 2 or 3 hours post-exercise, suggest that the intensity of an exercise bout should be carefully considered if exercise is used to stimulate fat oxidation, particular in consideration of the potential duration of stimulated post-exercise lipid utilization (Bielinski et al., 1985). Furthermore, the ingestion of a pre-exercise meal should also be considered, as this has been shown to increase post-exercise substrate utilization (Calles-Escandon et al., 1991) and oxygen consumption (Lee et al., 1999), although the experimental meal of glucose added to milk offered in the latter study may not represent a typical pre-exercise meal. However, because the intensity of exercise influences substrate partitioning during exercise (discussed in the next part), it is unclear if the rate of fat oxidation during the post-exercise period is more important to short-term lipid balance than is fat oxidation during exercise.

Post-exercise substrate compensation. The preceding findings should also be considered alongside the results of Dionne et al. (1999), who found that neither post-exercise energy expenditure nor substrate oxidation differed between two 24-hour trials, one of which was at rest, the other preceded by submaximal exercise and a calorically and compositionally equivalent post-exercise meal. This suggests that, compared to a pre-exercise meal, a post-exercise meal could have a stronger impact on post-exercise energy expenditure, and thus on fat oxidation,

whereby the increase in these parameters that usually follows exercise can be abolished. In support of these findings, Tittelbach et al. (2000) demonstrated a greater attenuation of fat oxidation following a high-fructose vs. high-glucose post-exercise meal in energy-balanced, but not in negative energy-balanced individuals. Moreover, the energy content of a post-exercise meal appears to be an influential factor for the potential impact, as another study in which the meal was not matched to the exercise cost demonstrated no effect of a post-exercise mixed meal on substrate oxidation following low- or moderate-intensity exercise (Thompson et al., 1998). Furthermore, it is unclear whether the meal's effect on post-exercise substrate oxidation observed by Dionne and colleagues is independent of the meal's GI, despite an apparently high GI in that experimental meal. These findings suggest that a post-exercise meal strongly influences post-exercise fat oxidation, although it is unclear whether this influence is stronger than that of a pre-exercise meal. Addressing these issues may permit an elucidation of specific exercise and dietary strategies for maximizing lipid oxidation, specifically by manipulating the temporal sequence of meal intake and exercise practice.

Without dismissing the differences between pre-exercise and post-exercise substrate oxidation, there appears to be discord among findings on substrate utilization during post-prandial or pre-prandial exercise. Thus, it is warranted to examine substrate utilization before, during, and after an exercise session when a meal either precedes or follows exercise, and when said meal differs in FQ or in GI. The influences of these various factors, as discussed herein, are summarized in Figure 2. As this line of inquiry remains largely unexplored within the discipline of exercise physiology, and as the considerations for exercise prescription continue to expand, the interest for the obese person in addressing this gap ultimately lies in maximizing periods of acute negative lipid balance across daily meal and exercise periods while reducing energy

balance. Nevertheless, as both fat mobilization and oxidation vary between types of exercise, as well as between individuals, it remains to be addressed how these factors might influence lipid balance.

Nature of Exercise, and Characteristics of the Individual

As both the nature of an exercise bout and the characteristics of the exercising individual can vary to many degrees, the discussion of factors that may help maximize an acute negative lipid balance is not complete without addressing how such variance influences FA mobilization and oxidation. Differences in the duration and intensity of exercise, as well as in the gender, body composition, and training level of individuals have all been shown to influence the mobilization or oxidation of fat. These differences are addressed lastly as they represent widely researched aspects of exercise physiology, and thus are perhaps easier to assess from physiological and practical standpoints in the elucidation of fat-loss strategies.

Exercise duration and intensity. The duration of an exercise session may be a simpler, and perhaps less important issue, than the intensity. If a particular intensity and energy cost of exercise is desired, then the duration is essentially a function of that particular intensity and energy cost. The typical basal concentration of plasma NEFA after a mixed meal is 0.3 mmol/L, but it can increase 5- to 6-fold during very prolonged exercise (Saltin & Astrand, 1993). It should also be noted that a substantial portion of energy production during prolonged light or moderate exercise comes from fat oxidation (Gollnick, 1985; Romijn et al., 1993). The ideal situation would then be to achieve a steady state during the exercise bout, such that the duration can be as long as is necessary to produce a given volume of work. Furthermore, other research has found that an increased post-exercise rate of NEFA utilization is dependent upon the duration, as well

as the intensity, of exercise (Bahr et al., 1991). A negative lipid balance therefore appears to be favoured by exercise of longer duration.

The implications of exercise intensity on fat mobilization and oxidation require closer consideration. In reference to the literature discussed herein, an intensity below 40% VO_2max is typically considered low-intensity; approximately 40 – 70% VO_2max represents moderate-intensity; and above 70% VO_2max is typically considered high-intensity. The increased energy requirement of sustained moderate-intensity exercise induces an increase in lipolysis with a several (4 to 10) fold increase in FA mobilization (Ahlborg et al., 1974), under which conditions the uptake of NEFA is increased. High-intensity exercise, however, results in a decreased FA mobilization and utilization in trained subjects (Romijn et al., 1993), although it has been argued whether the suppressed rate of fat oxidation observed at high intensity is dependent on this decrease in plasma NEFA availability (Jones et al., 1980; Romijn et al., 1995) or on mitochondrial uptake of FA (Sidossis et al., 1997; van Loon et al., 2001). It is also unclear whether lactate accumulation during high-intensity exercise inhibits lipolysis (Boyd et al., 1974) or not (Trudeau et al., 1999). Furthermore, there is evidence that muscle triglycerides contribute significantly to oxidative energy demand at moderate-intensity exercise (i.e. ~ 40 - 65% VO_2max) (Deriaz et al., 2001; Romijn et al., 1993), which may partially explain the higher rate of fat oxidation at such intensity. These findings strongly suggest that moderate-intensity exercise is most favourable for eliciting a substantial short-term increase in fat oxidation, although a slight compromise between duration and intensity may have to be made on an individual basis in order to maximize the rate of fat oxidation in the post-exercise period. It may also be necessary for very unfit individuals to begin exercising at a lower intensity, despite the possibility of not maximizing exercise-induced fat oxidation.

Training. While the duration and intensity of an exercise bout can be controlled in order to meet the goals of the individual, certain characteristics of the exercising individual cannot be controlled, yet are influential on fat metabolism. Variations in fat metabolism between individuals can be attributed to training level, gender, and body composition. For example, although highly trained male athletes and untrained healthy men exhibit similar rates of lipolysis and NEFA uptake from plasma during low-intensity exercise (Klein et al., 1994), endurance-trained appear to oxidize more fat than untrained at various absolute or relative intensities, perhaps because of greater oxidative potential due to increased mitochondrial and enzymatic content (Costill et al., 1979; Holloszy & Coyle, 1984; Molé et al., 1971), and/or because of increased lipolysis from muscle triglyceride (Coggan et al., 2000; Hurley et al., 1986; Klein et al., 1994). Furthermore, endurance-trained individuals have been shown to exhibit a decrease in adipose tissue lipolysis for fat oxidation at the same absolute intensity in favour of increased intramuscular triglyceride utilization (Phillips et al., 1996). Obviously, increased fat oxidation in trained individuals would be a boon for maximizing an acute negative lipid balance, although it may be difficult for obese individuals to achieve such a trained state.

Gender. The mobilization and oxidation of FA should not be assumed to respond identically to exercise in both genders. Women appear to exhibit a slightly greater reliance on fat oxidation during exercise (Horton et al., 1998; Melanson et al., 2002), and possibly greater still after exercise (Melanson et al., 2002), but not under resting pre-exercise conditions (Horton et al., 1998). Furthermore, trained women also exhibit greater fat utilization than do men (Friedlander et al., 1999; Tarnopolsky et al., 1990), whereas FA mobilization and uptake is greater in endurance-trained men (Friedlander et al., 1999), an effect which may be linked to differences in insulin and epinephrine concentrations (Tarnopolsky et al., 1990). There also

appear to be luteal-phase variations in substrate oxidation during the menstrual cycle (Wenz et al., 1997). Therefore, as fat oxidation appears to vary between genders during exercise, perhaps more favourably for women, future research may advance gender-based strategies for maximizing fat metabolism.

Obesity. Finally, it may not be surprising to find evidence of a compromised lipolytic response to catecholamine secretion in obese persons. Specifically, the mobilization of FA in obese individuals, compared to lean individuals, has been observed to increase less in response to epinephrine (Horowitz & Klein, 2000; Jensen et al., 1989), as well as to exercising (Kanaley et al., 1993) and starvation conditions (Wolfe et al., 1987). However, fat oxidation in obese persons appears to be unaffected during exercise (Kanaley et al., 1993), and appears to be elevated in post-absorptive obese individuals (Groop et al., 1992). The apparently paradoxical relationship between suppressed lipolytic response and normal rates of fat oxidation may be partly explained by observances of normal or elevated baseline lipolytic rates, while sympathetic responsiveness may be blunted in obese individuals (van Baak, 2001). Furthermore, although lipolysis is normally sensitive to insulin action in these individuals (Groop et al., 1992), it is unclear how the concomitant ingestion of food (i.e. pre-exercise or post-exercise) affects lipid balance in these individuals. Further research is required in this area, as it may be necessary to develop more specific exercise strategies for maximizing fat metabolism in obese participants.

Conclusion

The literature addresses many factors that influence fat metabolism in humans under post-absorptive, exercising, or post-prandial conditions. These factors, while not all fully understood, should be carefully considered in order to devise exercise and dietary strategies that strive to achieve and maximize an acute negative lipid balance. A general understanding of

catecholamines, insulin, and lipase action reveals how the stimulatory action of catecholamines (e.g. during starvation or exercise) acts reciprocally to the inhibitory action of insulin (e.g. after food intake) in regulating the contribution of NEFA to energy demand. This hormonal regulation, in turn, is responsible for variances in fat mobilization and oxidation rates under post-absorptive, exercising, post-exercise, and post-prandial conditions, and may explain variations in fat metabolism when these conditions are attained conjointly. Furthermore, the influences of the type of exercise (e.g. low- or high-intensity), as well as the characteristics of the exercising individual (e.g. trained state, gender, adiposity), on fat metabolism deserve consideration when attempting to maximize fat mobilization and oxidation.

Although it would appear that certain factors discussed herein could be manipulated in order to produce a state of acute negative lipid balance, the ideal combination of strategies for maximizing fat metabolism in order to decrease adiposity has yet to be established. One particular and potential strategy that remains irresolute pertains to the timing of meal ingestion around the exercise period, particularly when this factor, as well as the meal's GI, can influence fat mobilization and oxidation. The literature presents strong evidence of suppressed fat metabolism when a high-carbohydrate meal is ingested either before or after an exercise session. However, in determining which of these scenarios produces the greater total fat oxidation, the entire exercise period should be evaluated, from pre-exercise, throughout post-exercise, and on to the ingestion of the individual's next meal. Furthermore, in order to achieve a long-term negative lipid balance, the daily modulation of these factors must also allow for an increased energy expenditure induced by exercise that is not compensated by subsequent food intake. Ultimately, research on this topic could contribute to the body of knowledge that individuals draw upon for decreasing adiposity.

CHAPTER III

METHODOLOGY

Methods used for the data collection process in the present study are detailed in article format within the Methodology section of the article entitled Acute Effects of Exercise Timing and Breakfast Meal Glycemic Index on Exercise-induced Fat Oxidation. To include them here as well would be redundant for the reader, and it was therefore deemed more appropriate to describe the methodology within the article itself.

PART TWO: RESULTS OF THE STUDY AND DISCUSSION

CHAPTER IV

RESULTS AND DISCUSSION OF THE EXPERIMENT IN ARTICLE FORMAT

This chapter presents the results of the present study, as well as the discussion and analysis of the findings. These are presented in article-style format within the article entitled Acute Effects of Exercise Timing and Breakfast Meal Glycemic Index on Exercise-induced Fat Oxidation, for eventual submittal of the findings as a manuscript to a peer-reviewed journal. The results of the study are presented in the Results section of said article, followed by the Discussion of the findings.

ABSTRACT: To examine the acute effects of exercise timing and meal glycemic index (GI) on fat oxidation and glycemic response, five apparently healthy young men participated in four randomly ordered morning trials during which measurements were made at rest, during exercise, and for two hours post-exercise. A factorial design [exercise timing (pre-prandial, post-prandial) x meal GI (low-GI, high-GI)] was used for repeated measures of energy expenditure and whole-body fat oxidation, as well as of plasma glucose and insulin levels after an overnight fast. Subjects were required to perform 400 kcal of moderate treadmill exercise either before consuming a 400 kcal low-GI (ELG) or high-GI (EHG) oatmeal breakfast, or after consuming the low-GI (LGE) or high-GI (HGE) meal. Exercising fat oxidation was significantly greater during ELG and EHG (14.7 ± 1.4 and 14.8 ± 3.2 g, respectively) than during LGE and HGE (8.9 ± 3.1 and 9.8 ± 2.7 g, respectively) ($p < .001$), as was total fat oxidation beyond rest and (ELG: 21.3 ± 3.7 g; EHG: 20.2 ± 5.9 g; LGE: 18.1 ± 6.0 g; HGE: 17.1 ± 3.4 g) ($p < .05$), although energy expenditure was unaffected by experimental conditions. No significant effect of meal GI on fat oxidation was observed and, unexpectedly, the glycemic response was not significantly different across experimental conditions. Total whole-body fat oxidation for the entire morning period is therefore greatest when exercise is performed in the post-absorptive state, a strategy that could help maximize acute exercise-induced fat oxidation.

Key words: fat oxidation, exercise timing, breakfast, glycemic index, glucose, insulin

Introduction

An important dimension of weight control is lipid balance (Tremblay, 1995), which indicates the sum of all metabolic activity resulting in either the esterification (i.e. storage) or oxidation (i.e. utilization) of ingested or synthesized fatty acids (FA). A negative lipid balance is achieved under conditions when more fat is oxidized than is ingested from the diet during negative energy balance (Shah & Garg, 1996). Exercise is commonly used to elicit a negative energy balance, and a substantial portion of energy production during prolonged light or moderate exercise in particular comes from fat oxidation (Gollnick, 1985; Romijn et al., 1993). The increased utilization of lipid fuel induced by such exercise is largely the result of increased fat mobilization from lipid stores, which in turn results in an increased extraction of non-esterified fatty acids (NEFA) from the blood by working muscles (Groop et al., 1991). Fat is also the main oxidative substrate in energy-balanced individuals after an overnight fast (Acheson et al., 1984). After a carbohydrate-rich meal (Acheson et al.), however, or during physiological hyperinsulinemia (Kelley et al., 1990), glucose becomes the principal oxidative substrate, and fat oxidation is inhibited at the level of long chain FA entry into mitochondria, as demonstrated by Sidossis and colleagues (1996). Furthermore, the ingestion of high-glycemic index (GI) carbohydrates produces a sharper rise in blood glucose, which elicits a more pronounced insulin response (Walton & Rhodes, 1997), and thus a sharper rise in carbohydrate uptake and oxidation with a concomitant decrease in fat oxidation.

There is strong evidence supporting the notion that fat oxidation is inhibited during exercise following carbohydrate ingestion (Bergman & Brooks, 1999; Coyle et al., 1997; Horowitz et al., 1997). There is also evidence that post-exercise substrate compensation can negate the increases in EE and fat oxidation normally observed after prolonged physical activity

of moderate intensity (Dionne et al., 1999). However, relatively little research has compared pre-prandial and post-prandial exercise substrate oxidation. Welle (1984) compared exercise RQ before and after a high-carbohydrate meal, and demonstrated that post-prandial exercise RQ was higher than pre-prandial exercise RQ. Furthermore, post-prandial exercise has been shown to elicit greater EE in the first three hours post-exercise than pre-prandial exercise (Davis et al., 1992), which may indicate a greater reliance on lipid fuel following post-prandial exercise. Wu and colleagues (2003) showed that fat oxidation during exercise was greater after a low-GI meal than after a high-GI meal. However, the effects of the GI of a pre-/post-exercise meal on exercise and post-exercise substrate oxidation have not been compared. Therefore, it was the purpose of this study to examine acute exercise-induced variations in fat oxidation (i.e. during exercise and post-exercise) when a low- or high-GI breakfast meal was ingested before or after a morning exercise session. In addition, acute variations in plasma levels of both glucose and insulin hormone were examined across these conditions. It was hypothesized that total fat oxidation observed across the exercise and post-exercise periods (i.e. until the lunch meal) would be greatest in the presence of a low-GI, pre-exercise meal, owing to a greater post-exercise fat oxidation. It was also hypothesized that the pre-exercise, low-GI meal conditions would elicit the lowest levels of plasma glucose and insulin hormone during the post-exercise period relative to resting levels.

Methodology

Subjects. Seven apparently healthy, moderately active young men were recruited among eligible volunteers who responded to recruitment posters affixed across the University of Ottawa campus. Of these participants, five completed all experimental trials. Volunteers were excluded if they smoked, or if they presented a history of cardiovascular disease, diabetes or other

metabolic disorder, or any other major health problems. Participants must have maintained a stable body weight for six months before the study, as well as currently presenting a normal body weight (i.e. body mass index of < 25 kg/m^2, and a waist girth circumference of < 100 cm, WHO, 1998). The latter criteria were verified during the initial visit to the laboratory for the first screening test. During initial contact, volunteers were screened with a Physical Activity Readiness Questionnaire, as well as with appropriate medical and dietary questionnaires. Informed consent was received from each volunteer before beginning the study.

Screening Visit 1 – DEXA and VO_2max. Upon approval from the University of Ottawa and Montfort Hospital Ethics Committees, all subjects took part in two screening visits before beginning the experimental trials. All tests were performed at the Behavioural and Metabolic Research Unit, at Montfort Hospital in Ottawa, and were performed by a qualified fitness tester and support staff. Body composition and maximal aerobic capacity (VO_2max) were measured during the first screening visit. Subjects were instructed to have a light breakfast (e.g. toast, juice) approximately 2 hours before the visit, and to arrive at the facilities around 0830, prepared for exercise. Subjects were also instructed to avoid caffeine and nicotine the morning of the visit, and to avoid exercise 24 hours prior. Anthropometric measurements of height, weight, and waist girth were made using ACSM (2001) guidelines. Body composition was assessed by dual energy X-ray absorptiometry (DEXA) using a GE-LUNAR Prodigy module (GE Medical Systems, Madison, WI) interfaced with a computer. Coefficient of variation for the DEXA module was 1.8% ($R = 0.99$). Following the assessment of body composition, resting heart rate and blood pressure were recorded, and then maximal aerobic power (VO_2max) was assessed using a continuous, incremental treadmill protocol, the "Memorial Hospital" protocol modified for active individuals, as described by Ellestad (2003). The protocol involved gradual increases in work

rate by increasing speed at the end of the early 2-minute stages (3 minutes for the first stage), while the starting incline of 10% is increased to 15% for the latter stages. Heart rate was noted at the end each stage of the test using a heart rate monitor (Polar Electro Inc., Woodbury, NY), and blood pressure was measured as often as could be discerned during the final minute of each stage using a standard sphygmomanometer and stethoscope. Breath-by-breath samples of expired air were collected through a mouthpiece, and measurements of VO_2 and non-protein RQ were made automatically using a Vmax 229 series metabolic cart (SensorMedics Corporation, Yorba Linda, CA) interfaced with a computer. Coefficient of variation for the Vmax metabolic cart was not available at the time of data collection. Inspired/expired air volumes as measured by the metabolic cart were calibrated against a 3-litre calibration syringe (SensorMedics Corporation, Yorba Linda, CA), and inspired/expired gas concentrations were calibrated against 16% O_2/4% CO_2/balance N_2 and 26% O_2/balance N_2 reference gasses (Puritan Medical Products, Inc., Overland Park, KS). Test instructions and safety measures including familiarization with the treadmill (TRUE ZTX 850, True Fitness Technology, Inc., O'Fallon, MO) were explained before the test. After an adequate warm-up, subjects performed the test protocol, and the test was ended when at least two of the following criteria were met: VO_2 reached a plateau or decreased, despite an increase in intensity (i.e. increase of < 2.0 ml/kg/min); RQ reached 1.1; the subject's heart rate reached the age-predicted maximum (i.e. 220 – age); the subject's blood pressure dropped despite an increase in intensity; the subject demonstrated signs of incapacity of maintaining the effort; the subject wished to stop the test. Rate of perceived exertion was measured using a Borg-type scale (Borg, 1982). Once the test was completed, subjects were allowed an adequate cool-down, after which heart rate and blood pressure were monitored during recovery. Maximum aerobic power was expressed as peak 30-second average VO_2 attained.

Screening Visit 2 – FATmax. A second screening visit was scheduled approximately a week after the first, whereby subjects performed a maximal fat utilization test (FATmax), which was adapted for treadmill based on the protocol described by Achten et al. (2002). The starting intensity and stage increments in power were set at 95 W and 35 W, respectively (Achten et al.). The corresponding treadmill speeds were calculated trigonometrically and algebraically, based on each subject's body weight, and based on a percentage of incline of 10% for the duration of the test. The percent incline of 10% was chosen in order to elicit a feasible work progression similar to that of the maximal aerobic power test. Subjects were instructed to fast overnight, as well as to follow the other pre-test instructions for the first screening visit, and to arrive at the facilities around 0830, prepared for exercise. After recording their body weight, the test was performed in the same manner as the aerobic power test, except that the test was not preceded by a warm-up, and the stages were of four minutes in duration instead of three (i.e. between the 3- and 5-minute stages assessed by Achten et al.) in order to ensure that VO_2 reached a plateau during each stage. The criteria for ending the FATmax test were similar to those for the VO_2max test, except that the FATmax test was terminated once RQ reached 1.0, and/or once VO_2 reached 85% of maximum aerobic power, at which point the contribution of fat to substrate mix oxidized becomes negligible (Achten et al.). All other aspects of the FATmax test were identical to the VO_2max test. The rate of fat oxidation, expressed as grams per minute, was calculated for each minute using stoichiometric equations described by Frayn (1983), and maximal fat utilization was expressed as the highest end-of-stage 2-minute average of fat oxidation (Achten et al.). VO_2 at which maximal fat utilization occurred was also recorded. It should be noted that in cases where two "peaks" were observed on the plot of fat oxidation over time, the second "peak" was taken as the experimental FATmax value, such that all subjects exercised at a moderate intensity

of at least 40% VO_2max, rather than having some exercise at a comparatively very low intensity. At the end of the second screening visit, a 3-day pre-experimental control diet was explained. Each subject was required to follow this diet, which was designed to maintain both energy balance and glycogen stores, for three days before each experimental trial. The macronutrient composition of the diet, based on the Good Health Eating Guide (a food exchange system), was set to 55% carbohydrate, 15% protein, and 30% fat. The caloric content of the diet was calculated to match REE, where the latter was calculated using the revised Harris-Benedict equation described by Mifflin et al. (1990). The format for the dietary intake was expressed by the intake of a required number of food portions or "equivalents", whereby the calculated macronutrient and caloric intake was divided across the major food groups in such a way that subjects were required to consume a certain number of equivalents for each food group. Lists of various foods that constitute one equivalent were listed in the dietary pamphlet (Appendix E) that was given to each subject before each experimental trial. Subjects were also instructed to abstain from physical activity for 48 hours before each experimental trial.

Experimental Design. In a crossover experimental design, subjects underwent four randomly ordered experimental trials ("half-days"), one for each set of experimental conditions. A period of one to two weeks was given between each of the four half-days in order to allow for a sufficient metabolic washout (i.e. to eliminate possible carryover effects of experimental diet/exercise), as well as for sufficient blood replenishment. Each half-day lasted an entire morning, from 0730 to approximately 1315. All measurements were performed during the same hours of the day in order to avoid circadian variances in metabolism. Subjects were instructed to arrive at the laboratory about one week after the second screening visit at 0720 following an overnight fast, and to arrive prepared for exercise. Upon arrival, each subject's body weight was

noted, as was resting heart rate and blood pressure. Subjects were then instructed to lie supine on a hospital bed while an FEP Polymer intravenous catheter (Becton Dickinson Infusion Therapy Systems, Inc., Sandy, UT) was introduced into an ante-cubital vein of the non-dominant arm. The catheter was connected to a three-way stopcock (T-Connector Extension Set, Baxter Healthcare Corporation, Deerfield, IL), and the line was kept patent with 0.9% NaCl injection solution (Baxter Corporation, Toronto, ON) for further blood sampling. All blood samples were obtained by a qualified nurse. Two 10 ml blood draws, one each for plasma and serum separations, were drawn for the first sample at 0730, and immediately refrigerated until separation.

Resting metabolic rate. Subjects were then instructed to remain supine and to rest quietly for approximately 20 minutes before the resting metabolic rate (RMR) measurement. At 0800, RMR was then determined by measurement of expired gas for 30 minutes using a Deltatrac II metabolic cart (SensorMedics Corporation, Yorba Linda, CA) equipped with a ventilated hood. Coefficient of variation for the Deltatrac II metabolic cart was 2.3% ($R = 0.98$). The metabolic cart was calibrated against 95% O_2/5% CO_2 reference gas (SensorMedics Corporation, Yorba Linda, CA) at the beginning of each half-day. The first and last 5 minutes of measurement were discarded, and the values of VO_2 and VCO_2 for the middle 20 minutes were averaged for the calculation of the rate of REE, whole-body non-protein RQ, and fat oxidation as described above. At 0830, another blood sample was drawn.

Experimental conditions – breakfast and exercise. Subjects were assigned to each of four randomly ordered half-days that represented the experimental conditions. For two of the half-days, subjects were required to perform a bout of continuous, moderate-intensity exercise on a treadmill after consuming either a low-GI (LGE conditions) or high-GI (HGE conditions)

breakfast meal. For the other two half-days, subjects were required to exercise before consuming either of the same low-GI (ELG conditions) or high-GI (EHG conditions) meals. On the LGE and HGE half-days, breakfast was served immediately after the blood sample at 0830, whereas exercise began immediately after the 0830 blood sample on the ELG and EHG half-days. For all four experimental conditions, breakfast and exercise were counterbalanced in such a way that the total time required for both remained constant for all four half-days (see Figure 3).

The low-GI breakfast meal consisted of large flake porridge oats (Quaker Oats Company of Canada, Ltd., Peterborough, ON), dietary fructose (Flora Distributors, Ltd., Burnaby, BC), and unsweetened apple juice (Rougemont Mellow, A. Lassonde, Inc., Rougemont, QC), while the high-GI meal consisted of one-minute porridge oats (Quaker Oats Company of Canada, Ltd., Peterborough, ON), dietary sucrose (Lantic Sugar, Ltd., Montreal, QC), and orange-flavoured Gatorade beverage (QTG Canada, Inc, Peterborough, ON). Calculation of the composition of each meal is summarized in Table 2. The two isocaloric meals each contained the same quantity of dietary carbohydrate (LG: 80.0 g; HG: 80.3 g), while the quantity of each ingredient was determined in such a way as to minimize the GI of the low-GI meal, and to maximize the GI of the high-GI meal. Furthermore, the quantity of porridge in either meal was adjusted to be as equal as possible in order to minimize any difference in gastric emptying time. The GI of each meal, as indicated in Table 2, was calculated based on the weighted food GI (i.e. food GI multiplied by the proportion of total meal carbohydrate) of each food ingredient (Wolever & Jenkins, 1986). The GI of each food ingredient was rated against a white bread standard, values for which were taken from Foster-Powell and colleagues (2002). Food ingredients were measured out with an electronic scale (Scout Pro SP2001, Ohaus Corporation, Pine Brook, NJ) accurate to 0.1 g, and the porridge oats were cooked according to the package instructions, but

without the addition of salt or sugar. Subjects were instructed to consume the entire breakfast meal within 15 minutes (i.e. 0830 to 0845 for LGE and HGE, 0930 to 0945 for ELG and EHG). Subjects were offered water ad libitum during the meal, as well as during the rest of the half-day. If for any reason a subject was unable to consume the entire meal, then the results of that half-day were removed from the data analysis. It should be noted that the caloric content of the breakfast meals was maintained at 400 kcal, which was equivalent to the volume of the exercise bout. On the LGE and HGE half-days, subjects were then instructed to rest from 0845 to 0930, by either studying or reading quietly, and they were required to move about only for using the washroom. At 0930, a blood sample was drawn immediately before the start of exercise. On the ELG and EHG half-days, subjects were instructed to rest in a similar manner from 0945 to 1030, at which time another blood sample was drawn.

Exercise began at 0930 on the LGE and HGE half-days, and at 0830 on the ELG and EHG half-days. In order to represent free-living conditions, and in order to maintain energy balance throughout the half-day between energy intake from breakfast and energy expenditure from exercise, exercise volume was set at 400 kcal to match the 400 kcal caloric content of the experimental meal. The intensity of exercise was set at FATmax, which represented the work rate at which the rate of fat oxidation was maximal, as determined during the second screening visit. The results of both screening tests were used to determine the duration of the exercise bout for a volume of 400 kcal during the experimental trials. Using the Weir equation (Weir, 1949), the VO_2 and RQ values corresponding with the intensity at which maximal fat utilization was determined were used to predict the duration of exercise necessary for an expenditure of 400 kcal at this intensity (i.e. at the speed at which FATmax occurred, with 10% incline). This work rate was chosen to represent a typical free-living aerobic exercise bout for the purpose of maximizing

the oxidation of lipid fuel, the inhibition of which occurs at higher exercise intensities (Achten et al., 2002; Romijn et al., 1993), and was also chosen to maximize any effects of carbohydrate ingestion on exercising fat oxidation for the study factor of exercise timing. The same exercising instructions as for the screening visits were given before the start of exercise. A three-minute warm-up was given before the start of every exercise bout, and a three-minute cool-down was always given at the end of exercise, and these were always performed at an intensity corresponding to the first stage of the VO_2max protocol (i.e. 1.7 mph, 10% incline) in order to maintain equal EE for every bout.

Subjects began exercising at the pre-determined (i.e. FATmax) intensity, while heart rate was monitored during exercise, and they were instructed to minimize both talking and superfluous movements during the bout. Measurement of expired gas was performed in order to monitor the energy cost of exercise, using the same equipment described for the screening visits. Five-minute measurements of expired gas were performed after 10 min, and again after 25 min of exercise. Per-minute values of VO_2 and VCO_2 during these two five-minute measurements were used to determine the rate of EE, calculated using the Weir equation (Weir, 1949), as well as to determine both non-protein RQ and the rate of fat oxidation as described above. Per-minute EE was summed for the five minutes of measurement, and this sum was then multiplied by three for an approximation of total EE during each 15-minute period. This procedure was repeated for the next 15 minutes of exercise. It should be noted that if VO_2 values during the first five-minute measurement were more than 0.1 L/min over or under the VO_2 value calculated for FATmax, then the treadmill speed was adjusted accordingly before the next 15 minutes of exercise. If VO_2 was within 0.1 L/min of the FATmax value, then the corresponding treadmill speed was used during all subsequent trials for that subject. Furthermore, another blood sample was drawn at the

predicted halfway mark of the exercise bout, thus drawn at approximately 0955 during the LGE and HGE half-days, and at approximately 0855 during the ELG and EHG half-days. For the remainder of the exercise bout, the rate of EE was calculated using the mean of the VO_2 and VCO_2 values measured for minutes 28 through 30. Total EE determined for the first 30 minutes was subtracted from 400 kcal, and the difference was divided by the mean rate of EE for minutes 28 – 30, thus giving the amount of time in minutes required for a total exercising EE of 400 kcal. Exercising EE and time were determined in this way in order to avoid having subjects breathe through the mouthpiece for the entire duration of exercise, thus avoiding undue stress to the subjects. If a subject was unable to complete the exercise bout for any reason, that half-day was removed from the data analysis.

Upon completion of exercise, subjects were instructed to cool down for three minutes, as described above. At approximately 1020, subjects were instructed to take a shower, and were instructed to maintain both the temperature of the water and the duration of the shower constant in an attempt to maintain thermoneutrality (i.e. for all trials, water temperature was maintained warm for ambient conditions, and shower duration was limited to approximately 5 minutes). The catheter was properly shunted and covered by the nurse before the shower. Once the shower was taken, another blood sample was drawn at 1030 during the LGE and HGE half-days and at 0930 during the ELG and EHG half-days.

Post-prandial/post-exercise resting period. Once the breakfast and exercise periods were over, subjects were instructed to rest from 1030 until 1230, under the same conditions as after breakfast, and during which time two further blood samples were drawn every hour (i.e. at 1130 and 1230). As per the counter-balanced timelines of the half-days (see Figure 3), the post-exercise resting period began at 1030 for all experimental conditions. Two measurements of

RMR were also performed during this resting period at 1100 – 1130, and at 1200 – 1230, in the same manner as described above. REE, RQ, and fat oxidation for each 30-minute measurement were determined using the same equations described for exercise calculations, and were then multiplied by 2 to obtain an average of metabolic rate and fat oxidation for each 1-hour of the resting period. After the final blood sample was drawn at 1230, the catheter was removed by the nurse, and subjects were instructed to sit in the kitchen for the lunch buffet meal.

Buffet-style lunch meal. At approximately 1240, once the catheter was removed, subjects were offered a buffet-style lunch meal adapted from the protocol described by Arvaniti, Richard, and Tremblay (2000) (Appendix G). The purpose of offering this meal was to determine how the subsequent intake of a large lunch meal affected energy and lipid balance following the experimental conditions. All foods in the meal were weighed along with packaging and/or serving plates. Subjects were instructed to consume as much as was desired among a variety of foods within 30 minutes. If subjects desired more of a particular food item, another similar portion was prepared and weighed accordingly. Before leaving the laboratory, subjects were given another copy of the control diet pamphlet and the same pre-testing instructions. Finally, the food items of the meal were again weighed, and the ingested quantity of each item was recorded. Energy and macronutrient intake of the lunch meal were later analyzed using computer software (Nutrifiq version 0.99, FCAN97).

Blood Sampling and Analysis. For each blood sample drawn, two 10 ml vials of blood were obtained: one for serum determinations of insulin, and one for plasma determinations of glucose. Thus, a total volume of approximately 100 – 150 ml of blood was drawn during each half-day. The timeframe of blood sampling for each experimental condition is illustrated in Figure 3. Immediately following the experimental trial, all refrigerated blood samples were

centrifuged, and sub-samples of serum and plasma were prepared for analysis. After immediately storing sub-samples at -80^0C, plasma glucose concentrations were later assayed for each sample time using spectrophotometric analysis after conversion of glucose to glucose-6-phosphate by hexokinase. Laboratory-grade reagents (Sigma-Aldrich Canada Ltd., Oakville, ON; Fisher Scientific Limited, Nepean, ON) were used for preparing the hexokinase reaction, and after a 30-minute incubation of prepared samples at room temperature, spectrophotometric analysis of NADH concentrations was performed using a Synergy HT Series Multi-Detection Reader (Bio-Tek Instruments, Inc., Highland Park, Winooski, VT), with absorbance readings at a wavelength of 340 nm. Serum sub-samples were drawn at the end of each half-day after refrigeration and centrifugation, and were immediately sent to the Montfort Hospital Biochemistry laboratory for assay of insulin levels at each sample time. It should be also noted that coefficients of variation for blood measurement techniques were not available at the time the study was conducted.

Statistical Analysis. All statistical analyses were performed using Statistical Product and Service Solutions software, version 12.0 (SPSS Inc., Chicago, IL). In order to assess the effects of exercise timing and meal GI on EE, RQ, whole-body fat oxidation, and plasma glucose levels, a two-way factorial analysis of variance [exercise timing (pre- and post-exercise meal) x meal GI (low-GI and high-GI)] was performed for repeated measures of EE and RQ (three measurements), of whole-body fat oxidation (four measurements), and of blood glucose levels (six measurements). Bonferonni's adjustment was applied for multiple comparisons. For all statistical tests, alpha was set at $p = .05$ to determine the significance of any observed differences among within-subject factors, as well as among pairwise comparisons of levels of the main effects. Data are presented as $M \pm SD$, unless otherwise noted.

Results

Experimental conditions. Descriptive statistics for the subjects who participated in this study ($N = 5$) are summarized in Table 1. Body weight remained stable across conditions (LGE: 75.0 ± 7.7 kg; HGE: 75.1 ± 8.3 kg; ELG: 75.3 ± 7.6 kg; EHG: 75.6 ± 7.7 kg, $p = .01$). Subjects' mean maximal rate of fat oxidation during exercise, expressed as a percentage of VO_2max, was 54.6 ± 8.6%, as measured during the second screening visit by the FATmax protocol adapted from Achten et al. (2002). As summarized in Table 2, the experimental exercise bout was carefully controlled during the trials. The energy cost of exercise, when calculated by multiplying the actual time of exercise by the mean energy cost determined with Weir's equation (1949), was closely matched to 400 kcal (expenditure compensation for caloric intake of breakfast meal) for all experimental conditions, and although one subject completed the LGE exercise bout at an intensity that was slightly above FATmax, neither the time to complete the exercise bout, nor the percent VO_2max at which subjects exercise were significantly different across conditions. Elaboration of the experimental breakfast meals (low-GI and high-GI) was also carefully controlled, as both meals were isocaloric (400 kcal), and both contained the same amount of dietary carbohydrate (80.0 g for low-GI, 80.3 g for high-GI) (see Appendix G for summary of GI calculations). Finally, it should be noted that despite the stringent controls for extraneous variables in this experiment, data for some dependent variables were not normally distributed.

EE and RQ. Data for EE and RQ across experimental conditions are summarized in Table 3. Resting EE and RQ were not significantly different between experimental conditions. As expected, an effect of time on EE and RQ was observed during each experimental condition ($p < .05$). Although exercising EE was the same across conditions, a significant effect of exercise

timing was observed for exercising RQ. During the pre-exercise meal conditions (LGE and HGE), exercising RQ was significantly higher than during the post-exercise meal conditions (ELG and EHG) ($p < .001$). Unexpectedly, no significant effect of meal GI was observed for exercise RQ, however. During the first hour of the post-exercise period (1030 – 1130), no significant effect of meal GI on either EE or RQ was observed. There was a tendency for EE and RQ to be higher during this time under post-exercise meal conditions (ELG and EHG), but the effect was not significant. During the second hour post-exercise (1130 – 1230), however, RQ was found to be near-significantly higher during ELG and EHG than during LGE and HGE conditions ($p = .051$), but no significant effect of meal GI was observed. Furthermore, EE in the second hour post-exercise was not affected by either exercise timing or meal GI. Therefore, meal GI had no effect on either EE or RQ at any time during the half-days.

Fat oxidation. Resting fat oxidation measured in the post-absorptive state was not different among experimental conditions (LGE: 4.8 ± 0.4 kcal; HGE: 4.2 ± 0.8 kcal; ELG: 4.2 ± 0.8 kcal; EHG: 3.8 ± 0.8 kcal). Absolute fat oxidation during exercise and post-exercise periods is illustrated in Figure 4 for all experimental conditions. Because resting fat oxidation was not different across experimental conditions (one-way analysis of variance, $p < .05$), it was deemed unnecessary to calculate incremental fat oxidation. It should be noted that fat oxidation measurements made during the first and second hours of the post-exercise period were of 20 minutes in duration, but because these measurements were made under stable conditions (i.e. subjects were at rest), values were extrapolated to one hour, and are presented as such in Figure 4. Values presented in this figure for exercise-induced fat oxidation, on the other hand, are for actual exercising time, rather than extrapolated to one hour, as fat oxidation during exercise may increase as moderate-intensity exercise is prolonged. On the basis of a consistent exercising EE

of 400 kcal for all experimental conditions, subjects oxidized significantly more fat during exercise under ELG and EHG conditions (14.7 ± 1.4 and 14.8 ± 3.2 g, respectively) than under LGE and HGE conditions (8.9 ± 3.1 and 9.8 ± 2.7 g, respectively) ($p < .001$), whereas no significant effect of meal GI was observed across experimental conditions. Furthermore, no significant interaction effect of exercise timing and meal GI was observed across experimental conditions.

Post-exercise fat oxidation values were not significantly different across conditions during the first hour of the post-exercise period (LGE: 4.6 ± 1.8 g; HGE: 3.4 ± 1.1 g; ELG: 3.4 ± 1.6 g; EHG: 3.0 ± 2.2 g). There was also no significant effect of meal GI on fat oxidation during this time. During the second hour of the post-exercise period, however, subjects oxidized a near-significantly greater amount of fat under LGE and HGE conditions (4.5 ± 1.6 and 3.9 ± 1.0 g, respectively) than under ELG and EHG conditions (3.1 ± 1.4 and 2.4 ± 1.4 g, respectively) ($p = .057$), although again there was no effect of meal GI, nor any interaction effect of exercise timing and meal GI on fat oxidation. These observances also hold true when fat oxidation for the two hours post-exercise are added together (LGE: 9.1 ± 3.3 g; HGE: 7.3 ± 1.8 g; ELG: 6.6 ± 2.7 g; EHG: 5.4 ± 3.3 g) ($p = .067$), thus indicating a tendency for greater post-exercise fat oxidation when exercise is preceded by meal ingestion. Finally, total fat oxidation measured during the half-days (Exercise + Post-exercise 1 + Post-exercise 2) was significantly greater under ELG and EHG conditions (21.3 ± 3.7 and 20.2 ± 5.9 g, respectively) than under LGE and HGE conditions (18.1 ± 6.0 and 17.1 ± 3.4 g, respectively) ($p < .05$). Nevertheless, there was no significant effect of meal GI on total fat oxidation, nor any interaction effect of exercise timing and meal GI. Therefore, meal GI, as for EE and RQ, had no effect on fat oxidation throughout the half-days, whereas exercise timing was a significant affecter of substrate oxidation, particularly as whole-

body fat oxidation was generally greater when exercise was performed in the post-absorptive state compared with the post-prandial state.

Plasma glucose and insulin. To further investigate the effects of exercise timing and meal GI on substrate metabolism, hourly plasma glucose and insulin levels were determined for each half-day. Figure 5 illustrates the variations in plasma glucose and insulin across time under LGE and HGE conditions (Panel A), and under ELG and EHG conditions (Panel B). It should be noted that the mid-exercise measurement has been omitted from both the figure and the analysis, as the chronological sample time for this measurement differs by one hour between pre- and post-exercise meal conditions, although exercising insulin levels were significantly higher during HGE ($p < .05$). Analysis revealed few important significant differences in plasma glucose and insulin levels across experimental conditions. A significant effect of meal-GI on plasma glucose was observed at only one time point, whereas exercise timing had no significant effect. No trends were observed between experimental conditions. As for plasma insulin levels, an expected significant effect of exercise timing was observed after meal ingestion: levels were higher for LGE and HGE at 0930 than for ELG and EHG (108.8 ± 36.5, 214.0 ± 161.4, 27.8 ± 13.6, and 33.8 ± 7.8 pmol/L, respectively), and were lower for LGE and HGE at 1030 (35.3 ± 9.7, 41.6 ± 20.1, 107.2 ± 36.6, and 238.6 ± 142.7 pmol/L, respectively) ($p < .05$). Insulin levels were also significantly higher for ELG and EHG than for LGE and HGE at 1130 (32.8 ± 13.4, 46.4 ± 31.1, 17.8 ± 6.6, and 16.2 ± 5.4 pmol/L, respectively), and at 1230 (18.4 ± 9.8, 26.8 ± 11.9, 12.8 ± 1.3, and 15.3 ± 6.6 pmol/L, respectively) ($p < .05$). However, as for glucose levels, no significant trends were observed between experimental conditions, and between-subject variance for glucose and insulin levels was high.

Buffet-style lunch meal. Data for caloric and macronutrient intake from the buffet-style lunch meal that was offered to subjects at the end of each half-day are presented in Table 4. No significant effects of either exercise timing or meal GI on either caloric or macronutrient intake were observed. Interestingly, for all experimental conditions, fat intake at lunch was nearly 3-fold greater than total fat oxidation as measured across half-days, and a large mean caloric intake was observed across experimental conditions (approximately 1500 – 1800 kcal).

Discussion

Investigations of the effects of exercise timing have previously reported a higher exercise RQ (Welle, 1984) and a lower post-exercise RQ (Matsuo and Suzuki, 1999) when exercise is preceded by meal ingestion, a finding related to pre-exercise carbohydrate ingestion (Bergman & Brooks, 1999; Coyle et al., 1997; Horowitz et al., 1997). The GI of the pre-exercise meal also affects fat oxidation during exercise (Wee et al., 1999; Wu et al., 2003). However, the implications of both exercise timing and meal GI on exercise-induced fat oxidation, as well as on the glycemic response to meal ingestion, are unclear. The present study was therefore undertaken in order to investigate the acute effects of exercise timing and meal GI on whole-body fat oxidation, along with plasma glucose and insulin levels, during both exercise and the post-exercise period in young men. The main hypothesis of this study postulated that total fat oxidation across the entire morning period would be greatest when exercise was preceded by a low-GI breakfast meal, as elevated post-exercise fat oxidation was presumed to make the most important contribution to total exercise-induced fat oxidation under post-prandial exercise conditions. It was also hypothesized that these conditions would elicit the lowest plasma glucose and insulin levels relative to resting. Experimental conditions were rigorously respected, as reflected by the subjects' adherence to the pre-experimental diet, and by the stability of subjects'

body weight across experimental conditions. The important findings of this study were 3-fold. First, RQ was lower and fat oxidation higher during pre-prandial exercise (i.e. ELG and EHG conditions) than during post-prandial exercise (i.e. LGE and HGE conditions). Second, total fat oxidation as measured across the half-days was greater during ELG and EHG than during LGE and HGE conditions, despite a tendency towards elevated post-exercise fat oxidation during LGE and HGE. Finally, meal GI had no effect on either EE or substrate oxidation, nor on plasma glucose and insulin levels across all experimental conditions, despite a tendency of higher glucose levels at the end of LGE and ELG half-days.

Substrate oxidation response to exercise timing – during exercise. There has been considerable investigation in the last decade into the effects of meal ingestion on substrate oxidation measured during (Bergman & Brooks, 1999; Coyle et al., 1997; Horowitz et al., 1997; Welle, 1984; Wee et al., 1999; Wu et al., 2003), and after (Calles-Escandon et al., 1991; Davis et al., 1992; Dionne et al., 1999; Matsuo and Suzuki, 1999) moderate-intensity exercise, particularly where variations in whole-body fat oxidation are concerned. The present study is the first known to have quantified fat oxidation during and after exercise in response to ingestion of either a pre- or post-exercise carbohydrate meal. It was found that subjects oxidized more fat, as well as exhibited a lower RQ, during exercise performed in the fasted state, rather than in the post-prandial state. This is in agreement with previous findings that pre-prandial exercise, compared to post-prandial exercise, elicited a lower RQ (Welle) and higher fat oxidation (Wu et al.) during exercise. The lower exercising fat oxidation observed during LGE and HGE in the present study is also supported by the results of investigations that demonstrated how exercising fat metabolism is inhibited by pre-exercise carbohydrate ingestion (Bergman & Brooks, Coyle et al., Horowitz et al.), an effect of inhibited fat oxidation at the mitochondrial level (Coyle et al.),

as well as of insulin-mediated suppression of lipolysis (Horowitz et al.). Thus, the present findings support the notion that fat utilization is greater during pre-prandial exercise than during post-prandial exercise. Furthermore, in contrast to the results of Calles-Escandon and colleagues, the pattern of substrate utilization during exercise in this study was clearly affected by prior meal ingestion, as evidenced by differences in RQ and fat oxidation, although the intensity of exercise in the former study was higher than that in the present, the latter being specifically set at subjects' maximal rate of exercising fat oxidation (70% VO_2max vs. ~ 52% VO_2max, respectively). Carbohydrate contributes relatively more to energy output, and fat less, at higher intensities (Romijn et al., 1993), a factor which may explain the resistance to change in exercising substrate utilization after a carbohydrate meal reported by Calles-Escandon and colleagues. In light of this, the FATmax intensity was chosen for the present study not only to represent a free-living bout of moderate exercise, but also to maximize the effect of carbohydrate ingestion on exercising fat oxidation. Indeed, Achten and Jeukendrup (2003) showed that FATmax is decreased when glucose is ingested before exercise, which further supports the notion that fat oxidation is greater during pre-prandial exercise than during post-prandial exercise. The findings of this study therefore agree with the bulk of the literature regarding the inhibitory effects of a pre-exercise meal on exercising fat oxidation.

Substrate oxidation response to exercise timing – post-exercise. Although exercising fat oxidation across conditions was a key variable in the present study, one of the primary purposes was to compare total fat oxidation across experimental conditions. Total fat oxidation in this study was described as the amount of fat oxidized during exercise plus that oxidized during the two hours following the exercise/meal period. Two assumptions were made for this variable: first, as whole-body indirect calorimetry methods permitting constant measurement of substrate

metabolism were unavailable for this study, fat oxidation in the transition times between measurements during each half-day was treated as negligible; second, *de novo* lipogenesis was not occurring in the resting time following the breakfast meal. Thus, it was found in this study that total fat oxidation was significantly greater by almost 18% under ELG and EHG conditions than under LGE and HGE conditions. This effect was observed in spite of a tendency for greater total post-exercise fat oxidation during LGE and HGE half-days, suggesting that the exercise period has more impact than the post-exercise period on acute, post-absorptive fat metabolism in energy-balanced individuals. Specifically, the higher rate of fat metabolism during 40 minutes of pre-prandial exercise appears to outweigh the higher rate of fat metabolism during two hours of rest after 40 minutes of post-prandial exercise. This is likely due to a higher lipolytic rate and/or a lower rate of NEFA reesterification (Wolfe et al., 1990) in post-absorptive exercise, compared to an insulin-mediated inhibition of lipolysis (Horowitz et al., 1997) and/or of mitochondrial NEFA uptake (Coyle et al., 1997) in post-prandial exercise.

Although this finding is in sharp contrast to the exercise timing hypothesis postulated in this study, a lack of previous research on the effects of meal and exercise timing on fat oxidation throughout both exercise and post-exercise periods has until now made it unclear which conditions promote the greatest acute exercise-induced fat oxidation. It should be noted that the tendency for greater post-exercise fat oxidation observed under LGE and HGE conditions was not significant, thus differing from previous findings of post-exercise fat oxidation attenuated by a pre-exercise meal (Calles-Escandon et al., 1991). It is possible that the requirement for subjects in this study to remain in a state of energy balance, rather than negative energy balance, for 3 days prior to the experimental trials may partially explain the attenuated post-exercise fat oxidation observed under ELG and EHG conditions, a factor that has be shown to affect post-

exercise fat oxidation following a post-exercise meal (Tittelbach et al., 2000). Furthermore, the lower post-exercise fat oxidation observed under the latter conditions may also be explained by insulin-mediated inhibition of NEFA uptake by mitochondria (Sidossis et al., 1996). Nevertheless, although post-exercise fat oxidation was near-significantly greater during LGE and HGE half-days, it remains unclear how total fat oxidation would be affected by prolonging the resting period after post-prandial exercise until the next meal is ingested. Moreover, in light of evidence by Dionne and colleagues (1999) who showed that post-exercise RQ is unchanged in the 24-hour period after moderate exercise due to a compensatory post-exercise meal, it remains unclear if and how the effects of any exercise timing strategy carry over throughout the rest of the day to affect lipid balance in the longer term.

Glycemic and substrate oxidation responses to meal GI. The high degree of between-subject variability between experimental conditions has resulted in a data set that is not normally distributed, which very likely results from having only five subjects participate in the study. Ingestion of a high-GI carbohydrate meal, compared to a low-GI meal, produces a sharper rise in blood glucose, which elicits a more pronounced insulin response (Walton & Rhodes, 1997), and thus a sharper rise in carbohydrate uptake and oxidation. The effects of meal GI are also evident during subsequent exercise (Wu et al., 2003), as well as after prior exercise (Tittelbach et al., 2000), although the glycemic responses observed during these two time periods were oppositely affected by low- and high-GI meals. It should be noted here that subjects in the former study were healthy, lean, young men, similar to the present study, while the latter study involved healthy, obese men and women. Comparisons between these two studies and the present one in terms of exercise timing and meal GI effects, however, cannot be made at this time from the present data due to the high degree of between-subject variability in the data set, despite stringent

control of the contents of each experimental meal. Both the high- and low-GI meals were consumed at similar time points (i.e. at 0830 or 0930), such that a 1-hour difference alone in ingestion time would not affect the glycemic response (Wolever, 2003), notwithstanding the inclusion of an exercise bout. As summarized in Appendix G, both meals contained the same amount of carbohydrate, were isocaloric and similar in appearance and content, and their compositions were adjusted to produce as great a difference as possible between each meal's GI (low-GI: 48.3; high-GI: 103.3). In other words, every attempt was made to control all other aspects of the meals in order to maximize within-subject variance between low- and high-GI conditions.

The small sample size of this study resulting from five out of seven subjects completing all four half-days may explain the high degree of between-subject variability observed for plasma glucose levels, while technical difficulties with the hospital-based protocol for serum insulin analysis, as well as very limited funds and time constraints, prevented further testing from being accomplished. The small sample size may have also compounded within-subject variability in the glycemic response to carbohydrate ingestion that can be expected from individual day-to-day variations (Wolever, 1990), thus masking any effect of meal GI. It is also possible that a meal with a high-carbohydrate content could elicit a pronounced insulin response even if the GI is low (i.e. a meal with a high glycemic load), which may also mask the effect of meal GI. In this way, both the size and the FQ of the meal would have more impact than the GI. However, in the study by Wu and colleagues, a meal GI effect was observed where mean carbohydrate load ingested by subjects was approximately 150.0 g, whereas the load in the present study was 80.0 g without evidence of any meal GI effect. Finally, the 2-hour measurement period of post-exercise/post-prandial metabolism may have been too short to

observe important changes in glycemia due to hyperinsulinemia induced by the high-GI meal. Nutrient absorption from the gastrointestinal tract begins to decline 2 – 4 hours after a high-GI meal, but the effects of elevated insulin persist, inducing a hypoglycemic state (Ludwig, 2002). Therefore, no meaningful conclusions as to the effects of meal GI on glycemic response can be made at this time, perhaps not until a larger data set has been obtained.

Fat oxidation during exercise following a high-GI meal has been shown to be lower compared with a low-GI meal (Wee et al.; Wu et al., 2003). Alternately, fat oxidation in the post-exercise period following a high-fructose (i.e. low-GI) meal has been shown to be lower compared with a high-glucose (i.e. high-GI) meal (Tittelbach et al., 2000). Each of these studies has compared fat oxidation in response to meal GI during only two of the four experimental conditions as elaborated in the present study (i.e. LGE and HGE in Wee et al. and Wu et al.; ELG and EHG in Tittelbach et al.). Thus, no known study has compared both exercise timing and meal GI on the level of total exercise-induced fat oxidation in the short-term. However, because of the issues detailed above regarding a lack of effect of meal GI on glycemic response, no meaningful conclusions as to the effects of meal GI on total exercise-induced fat oxidation can be drawn at this time.

Glycemic response to exercise timing. No known study has previously examined the glycemic response between exercise/carbohydrate meal timing conditions. In the present study, apparently unchanged plasma glucose levels between pre-prandial and post-prandial exercise conditions are somewhat surprising, considering the significant effect of exercise timing on exercise-induced fat oxidation, particularly as glucose levels were expected to be different at 0930 and 1030. The expected difference between pre-prandial and post-prandial exercise conditions would be the result of the different meal ingestion times. It could be reasoned that

blood glucose levels were maintained by increased hepatic glycogenolysis during exercise, and by increased hepatic glucose uptake post-prandially, independently of meal GI. This reasoning may also be applied to the plasma insulin responses to these stimuli. Insulin levels were indeed different between exercise timing conditions at meal ingestion times, as was expected, and were also higher in the post-exercise period during the pre-prandial exercise conditions. A clearer picture regarding the effects of exercise/meal timing on the glycemic response could be provided if the experimental conditions in the present study included a meal-only group for comparison of the outcomes.

Ad libitum lunch intake. The purpose of the buffet-style lunch meal at the end of each half-day was not-only to maintain free-living conditions by taking into consideration the lunch meal likely to follow morning exercise, but also to evaluate the potential compensation by food intake for the caloric and/or lipid deficit elicited by exercise. However, subjects overcompensated for the morning exercise bout with a caloric and fat intake that was largely in excess of exercise-induced energy expenditure and fat oxidation. It may simply be that young men demonstrate no restraint towards food intake after exercise, although this is merely speculative, as dietary restraint was not evaluated during this study. Another possible explanation is that subjects may normally consume larger breakfast meals than were offered during this study, such that the breakfast meals in this study would increase hunger at lunchtime beyond normal. Thus, a relatively greater food intake at lunch may have masked any effects of the study factors.

In summary, morning exercise-induced fat oxidation in this study was shown to be greater when exercise was performed in the post-absorptive state, compared to the post-prandial state. This effect applies to the entire morning period until the ingestion of lunch, and, despite the

lack of effect of meal GI, appears to be largely the result of a greater whole-body fat oxidation during pre-prandial exercise being more important than the greater post-exercise fat oxidation elicited by exercising after breakfast ingestion.

PART THREE: CONCLUSIONS AND RECOMMENDATIONS

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The main findings of the present study suggest that a short-term strategy for exercise timing around breakfast intake can be employed by young men for the purpose of maximizing acute exercise-induced fat oxidation. As total whole-body fat oxidation was shown to be greater during exercise in the fasted state, individuals wishing to maximize their efforts for exercise-induced weight control could benefit from these findings if prolonged moderate-intensity exercise were regularly performed soon after waking in the morning, before breakfast is taken. The effects of such a strategy appear to be independent of the glycemic index of the breakfast meal. Furthermore, these findings apply only to the morning period before lunch.

Although the plasma glucose variations were apparently not different between trials in this study, it is possible that blood glucose levels were simply well maintained by hormonal control in response to the exercise and food stimuli presented during the half-days. Plasma insulin levels also showed little difference between meal GI conditions. Both of these findings are in disagreement with the existing literature. However, technical difficulties encountered with the hospital-based protocol for serum insulin analysis, as well as very limited funds and time constraints prevent meaningful conclusions from being drawn at this time, and further data collection is to be expected in light of a small cohort of subjects who completed all experimental trials. Nevertheless, the identification of a set of conditions that can acutely maximize exercise-induced fat oxidation is the most important finding of this study.

Recommendations and Future Perspectives

In light of the unchanged glycemic responses to meal ingestion during this study, it would make for a stronger comparison of the effects of meal GI on substrate oxidation if a meal-only half-day was included among the experimental conditions in this study. In this way, it could be more reasonably determined whether the glycemic responses to meals of different GI are a function of exercise timing.

Furthermore, it would be of great interest to examine the effects of exercise timing and meal GI on the metabolic outcomes throughout an entire day. In this way, the GI of an entire diet could be designated, and using whole-body indirect calorimetry methods, the effects of exercise timing could be evaluated over the whole day, possibly by the timing of exercise around later meals during the day.

PART FOUR: CONTRIBUTION OF COLLABORATORS

CHAPTER VI

STATEMENT OF CONTRIBUTION OF COLLABORATORS

The collaborators involved in the conception and writing of this thesis (P.B., E.D., and P.I.) contributed as follows: P.B., E.D., and P.I. collaborated to write the published Review of Literature; P.B. was the primary author of this review. P.B. and E.D. were involved in the elaboration of the experimental design. P.B. collected all the data. P.B. and E.D. interpreted the findings. Finally, P.B. wrote the submitted thesis article. None of the collaborators had any financial motivation in the conception or writing of this thesis.

PART FIVE: REFERENCES AND APPENDICES

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Table 1

Descriptive Characteristics of Subjects

Variable	Value
Age (y)	24.1 ± 2.2 (21.8 – 27.8)
Body weight (kg)	75.3 ± 9.1 (63.8 – 86.6)
Height (m)	1.77 ± 0.06 (1.70 – 1.87)
BMI (kg/m^2)	24.0 ± 1.8 (22.2 – 26.4)
Body fat (%)	20.4 ± 6.0 (15.5 – 30.7)
VO_2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	54.5 ± 5.8 (44.9 – 60.1)
FATmax (% VO_2max)	54.6 ± 8.6 (46.0 – 69.0)

Note. All values are *M* ± *SD*; range in parentheses. BMI = body mass index; VO_2max = maximal oxygen uptake; FATmax = maximal rate of fat oxidation, expressed as percentage of VO_2max, as described in Achten et al. (2002).

Table 2

Energy Expenditure and Duration of the Experimental Exercise Session

Conditions	Duration (min)	EE	% VO_2max
LGE[a]	37.6 ± 5.7	400.0 ± 0.1	54.0 ± 11.4
HGE	38.4 ± 3.3	399.1 ± 1.1	51.6 ± 6.1
ELG	39.1 ± 2.9	399.9 ± 1.6	51.5 ± 6.2
EHG	39.6 ± 2.2	400.1 ± 0.3	50.8 ± 5.7

Note. All values are $M \pm SD$; exercise volume was set at 400 kcal; intensity was set at subjects' FATmax (maximal rate of fat oxidation, determined during second screening test); duration of exercise was calculated using Weir's equation (1949). EE = energy expenditure; % VO_2max = exercise intensity expressed as percentage of maximal oxygen consumption, the latter which was determined during the first screening test. EE, Duration, and % VO_2max were not significantly different across Conditions.

[a]One subject completed the exercise bout at an intensity slightly above FATmax, thus accounting for the lower mean Duration and for the higher mean % VO_2max.

Table 3

Energy Expenditure and RQ Across Experimental Conditions

	Rest		Exercise	Post-exercise 1[a]		Post-exercise 2[b]	
	EE	RQ	RQ	EE	RQ	EE	RQ
Conditions	(kcal/h)			(kcal/h)		(kcal/h)	
LGE	67.5 ± 5.8	0.79 ± 0.02	0.94 ± 0.02_a	73.5 ± 9.1	0.82 ± 0.06	75.3 ± 7.6	0.83 ± 0.05_a
HGE	68.4 ± 4.3	0.82 ± 0.04	0.93 ± 0.02_a	72.2 ± 8.4	0.86 ± 0.05	73.1 ± 6.6	0.84 ± 0.04_a
ELG	68.5 ± 4.6	0.82 ± 0.03	0.89 ± 0.01_b	73.4 ± 2.8	0.87 ± 0.06	72.8 ± 4.2	0.88 ± 0.05_b
EHG	71.9 ± 13.3	0.84 ± 0.04	0.90 ± 0.02_b	79.3 ± 8.1	0.89 ± 0.08	76.6 ± 10.1	0.90 ± 0.05_b

Note. All values are $M \pm SD$. Means in the Exercise RQ column that do not share a subscript are significantly different ($p < .001$) for within-subjects contrasts [two-way factorial repeated measures ANOVA (exercise timing x meal GI)]. Means in the Post-exercise 2 RQ column that do not share a subscript are near-significantly different ($p = .051$) in the same ANOVA model. EE = energy expenditure; RQ = respiratory quotient.

[a]Post-exercise 1 refers to measurements made after exercise and meal ingestion, from 1100 – 1130.

[b]Post-exercise 2 refers to measurements made after exercise and meal ingestion, from 1200 – 1230.

Table 4

Caloric and macronutrient intake of buffet-style lunch meals

Conditions	EI (kcal)	CHO intake (g)	Lipid Intake (g)	Protein Intake (g)
LGE	1693.1 ± 500.6	189.4 ± 79.1	66.7 ± 21.5	89.4 ± 13.5
HGE	1587.6 ± 449.2	181.6 ± 49.1	63.1 ± 22.7	79.3 ± 28.3
ELG	1481.9 ± 304.3	175.1 ± 41.1	57.6 ± 18.2	70.6 ± 21.2
EHG	1810.1 ± 785.9	198.8 ± 86.6	70.6 ± 40.6	99.3 ± 38.5

Note. All values are $M \pm SD$. EI = energy intake; CHO = carbohydrate. No significant effects of either exercise timing or meal GI on either caloric or macronutrient intake were observed.

FIGURE CAPTION

Figure 1. Interrelationships between the major hormones and enzymes involved in fatty acid (FA) turnover. Epinephrine-activated hormone-sensitive lipase (HSL) hydrolyzes triglyceride (TG) into FA, which are transported in the blood bound to albumin. Lipoprotein lipase (LPL), stimulated by insulin, hydrolyzes FA from TG in plasmatic lipoprotein complexes, after which FA are reesterified in the adipocyte. During fasting and exercise, epinephrine inhibits insulin action, which suppresses reesterification, and increases non-esterified FA (NEFA) availability, whereas insulin inhibits HSL and promotes reesterification post-prandially.

Figure 1. Interrelationships between the major hormones and enzymes involved in FA (fatty acid) turnover.

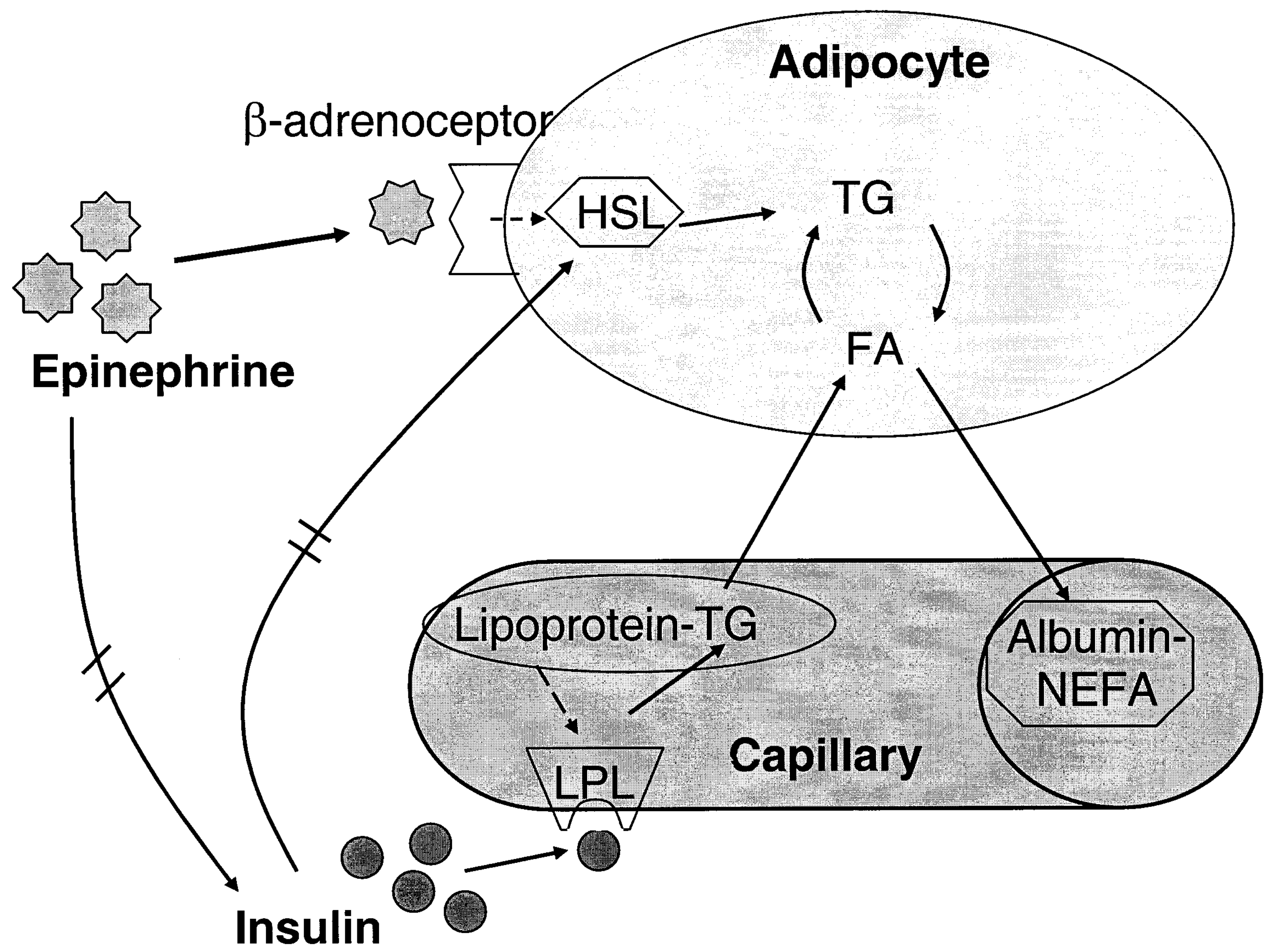


FIGURE CAPTION

Figure 2. Hypothetical model summarizing the various acute influences of fasting, exercise, and food ingestion on lipolysis (TG to FA) and fat oxidation (FA to ATP). The sign convention is used to illustrate the effect – either an increase (+) or a decrease (-) – of each stimulus on lipolysis and/or fat oxidation. Large arrows indicate a direct or primary effect; small arrows indicate an indirect or secondary effect; the question mark indicates a possible effect (i.e. discordant or lacking findings). It remains unclear as to the overall effects of eliciting one particular combination of stimuli over another.

Figure 2. Hypothetical model summarizing the various acute influences of fasting, exercise, and food ingestion on lipolysis and fat oxidation.

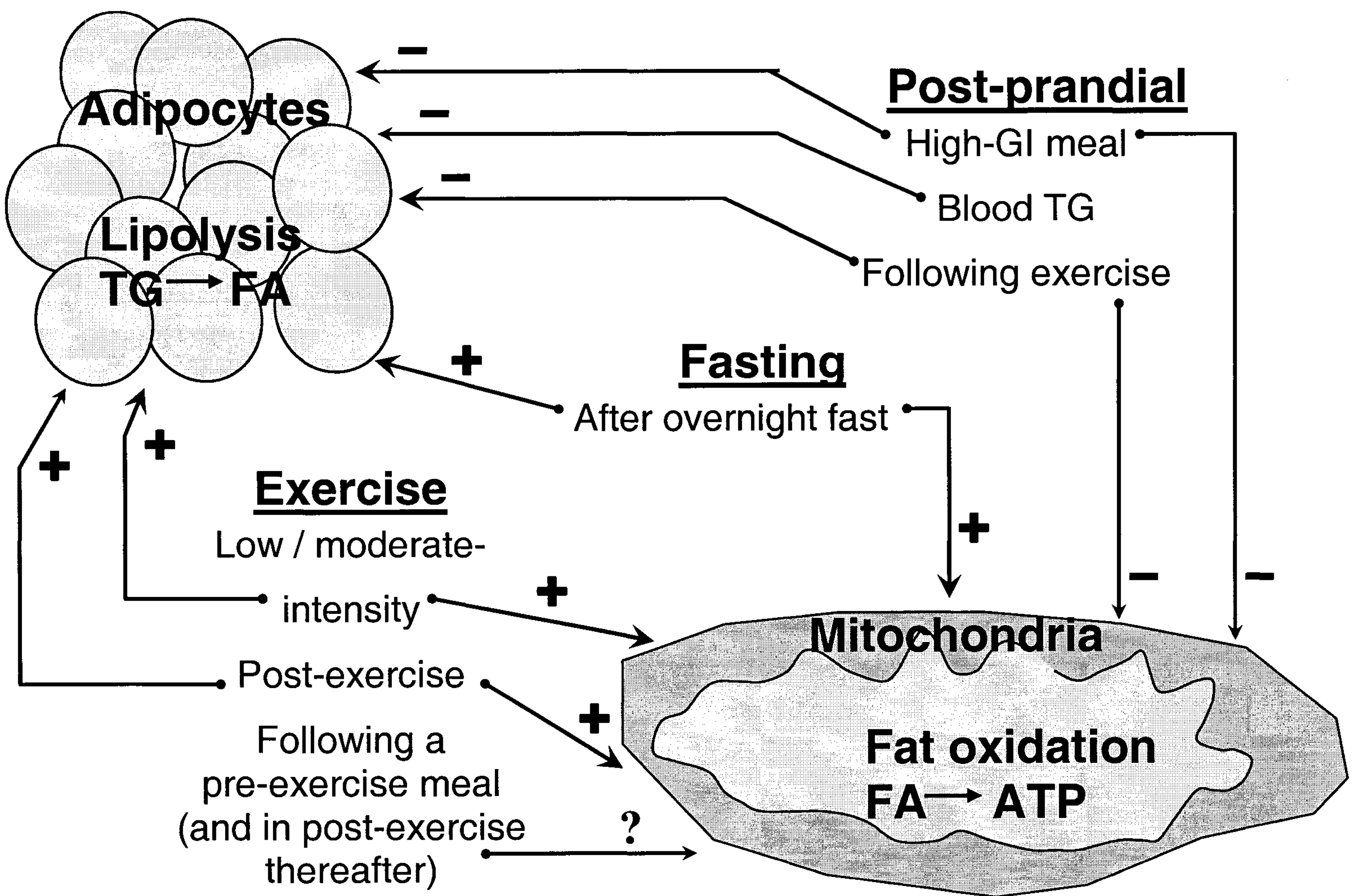


FIGURE CAPTION

Figure 3. Illustration of experimental design for comparison of high- and low-glycemic index pre-exercise meals on fat oxidation across meal, exercise, and resting periods, as well as for comparison of high- and low-glycemic index post-exercise meals on fat oxidation across these periods. Time frame of blood sampling is incorporated into each of the pre-exercise and post-exercise illustrations.

Figure 3. Illustration of experimental design.

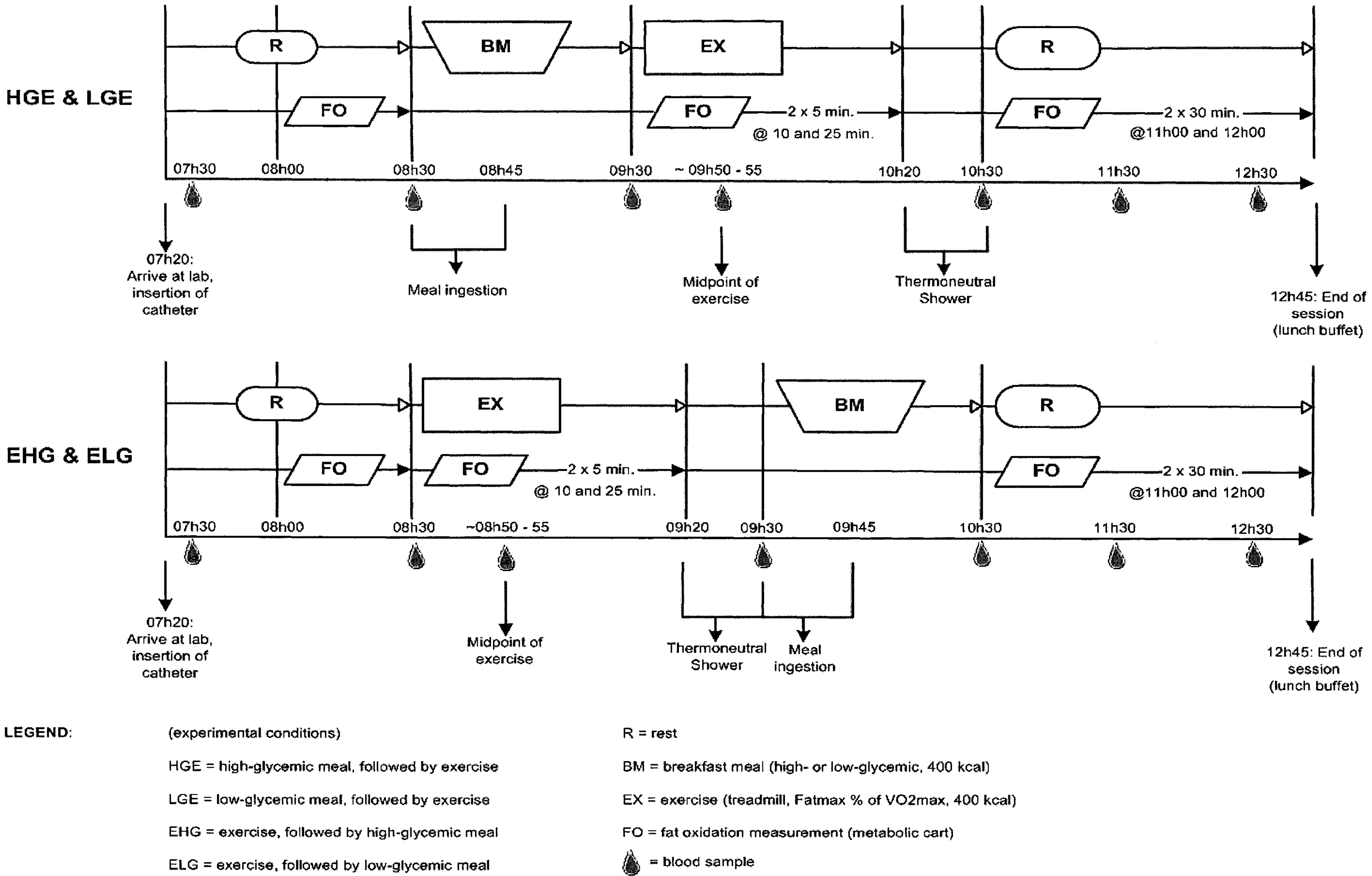


FIGURE CAPTION

Figure 4. Mean total fat oxidation (± *SE*) determined across pre-exercise (LGE: low-GI meal; HGE: high-GI) and post-exercise meal conditions (ELG: low-GI meal; EHG: high-GI). $N = 5$. Fat oxidation was calculated using stoichiometric equations described by Frayn (1983). Resting values were not different across experimental conditions (one-way ANOVA, $p < .05$), and as such were omitted from the figure. Bar segments represent the three non-prandial time periods beyond resting values: during exercise; one hour after experimental exercise/breakfast; and two hours after experimental exercise/breakfast. Fat oxidation values for Post-exercise 1 and Post-exercise 2 periods were extrapolated to 1-hour from 20-minute measurements, whereas Exercise values shown are for actual exercising time (see explanation in text). The main effects of the model were assessed with a two-way factorial ANOVA for repeated measures (exercise timing x meal GI). Bar segments sharing a letter are significantly different from those that do not (as well for entire bars). Exercising and total fat oxidation for ELG and EHG were significantly higher than LGE and HGE ($p < .05$).

Figure 4. Mean total fat oxidation across experimental conditions

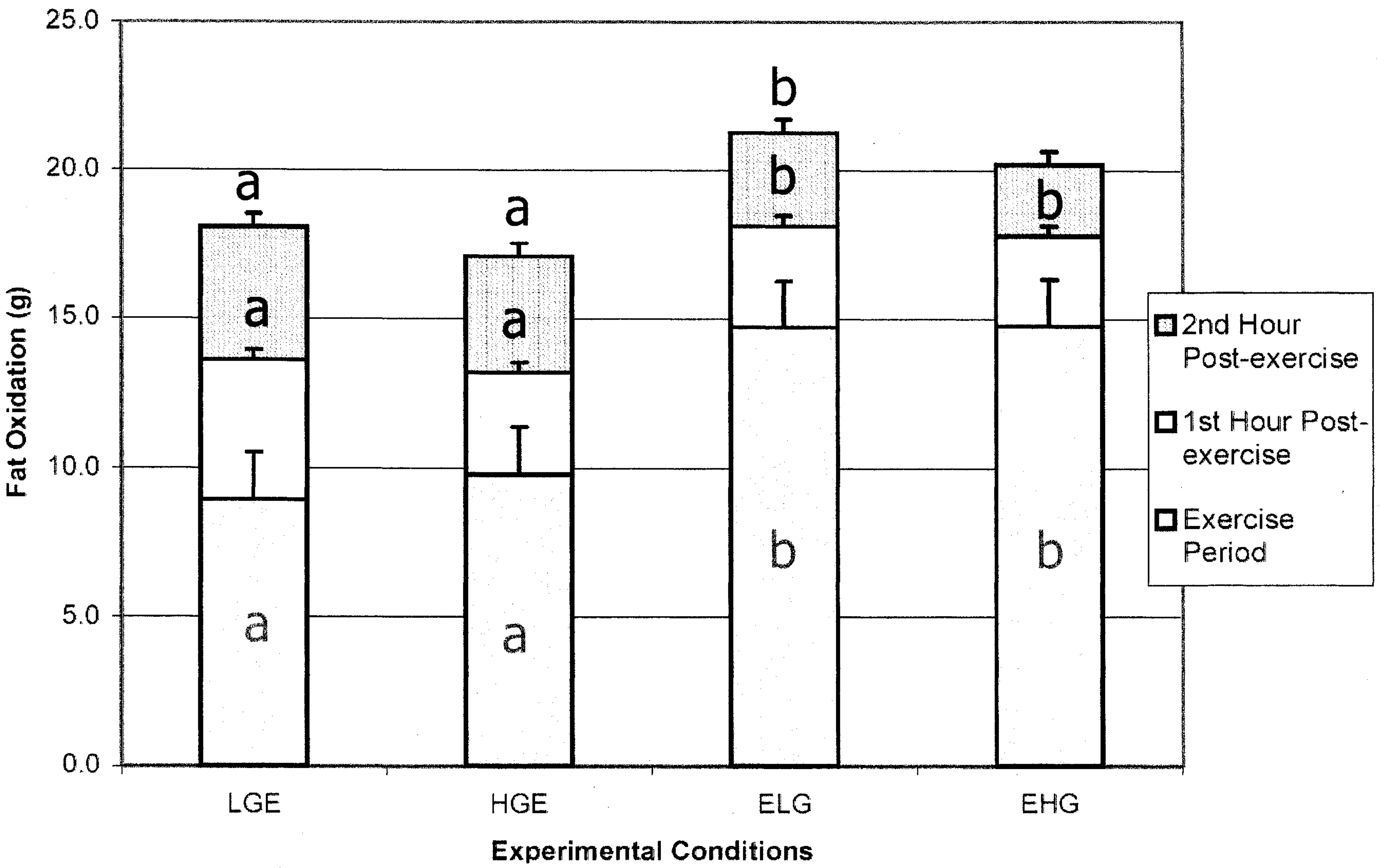
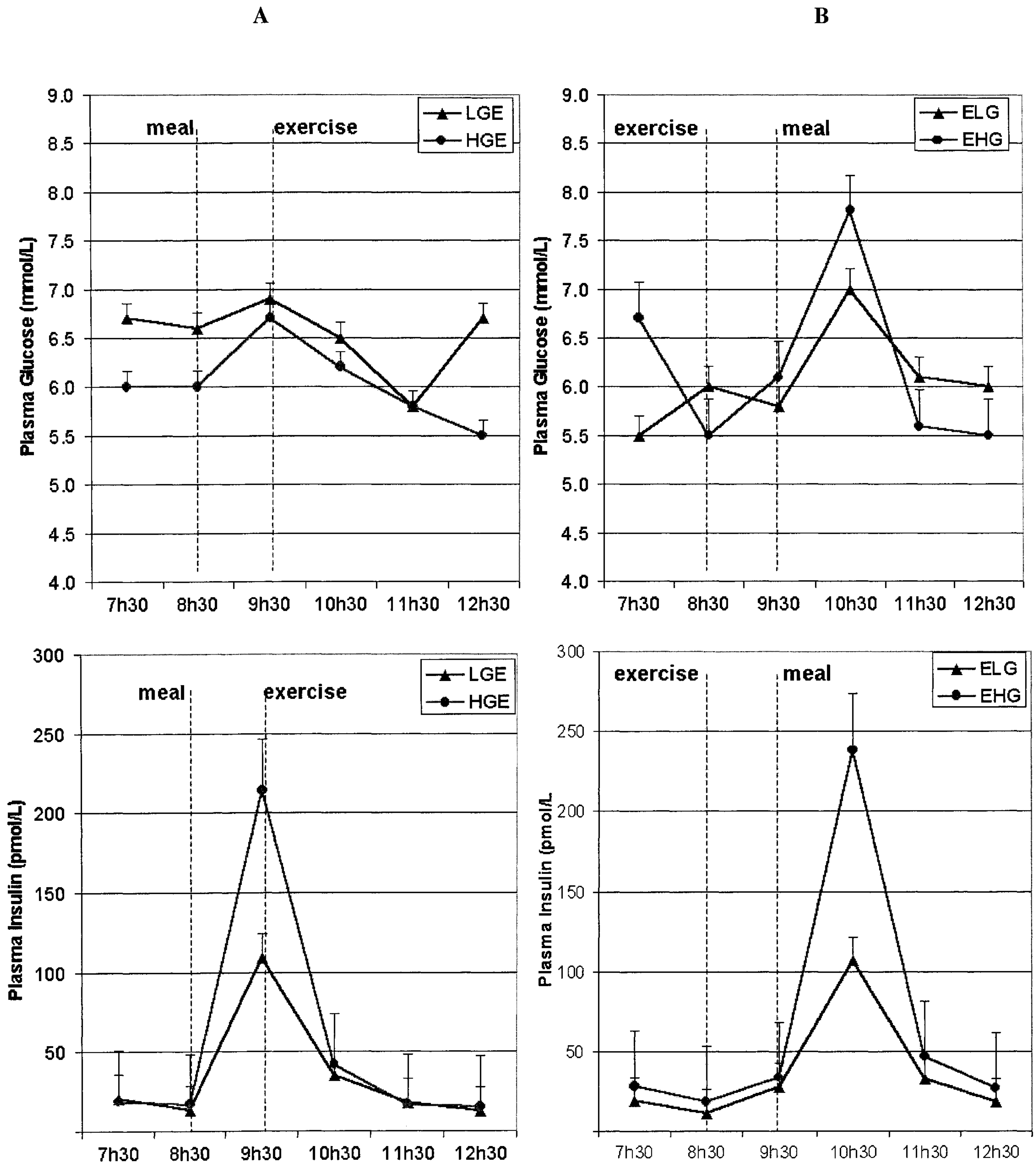


FIGURE CAPTION

Figure 5. Mean (± *SE*) plasma glucose and insulin levels across time for experimental conditions. $N = 5$. Panel A shows the variations in glucose and insulin levels across pre-exercise meal conditions, for both low- and high-glycemic index meals (LGE and HGE). Panel B shows glucose and insulin variations across post-exercise meal conditions with the same meals (ELG and EHG). The mid-exercise sample time has been omitted from the plots (see explanation in text). The main effects of the model were assessed with a two-way factorial ANOVA for repeated measures (exercise timing x meal GI). A significant effect of meal GI on glucose was observed only at 1230, whereas a significant effect of exercise timing on insulin was observed only at 1130 and 1230.

Figure 5. Plasma glucose and insulin levels across experimental conditions.



APPENDICES

APPENDIX A

Ethics Board Approval Letter

October 30, 2003

Dr. Eric Doucet
School of Human Kinetics
University of Ottawa
125 Université
Room 353
Ottawa, ON K1N 6N5

Mr. Patrick Bennard
406-125 Stewart
Ottawa, ON K1N 6J3

Object: **The effects of pre and postexercise meals of varying glycemic index on morning exercise-induced fat utilization in obese men (our file: H 10-03-03)**

Dear Researchers,

You will find enclosed the Health Sciences and Sciences Research Ethics Board Certification for your research project.

Please note that it is the responsibility of the researchers to:

a) Notify the ethics office of any changes in the research project; and
b) Fill out an annual status report to be sent to the Protocol officer for ethics in research. Such report can be found on the ethics web site at:
http://www.uottawa.ca/services/research/rge/rebs/download/rapport_annuel_projets_anglais.doc

A copy of this approval will be sent to Research Services, if necessary.

If you have any questions, you can contact me either at extension 5387 or extension 1783.

Sincerely yours,

Rita D'Alessandro
Protocol Officer for Ethics in Research
For Daniel Lagarec
Chair of the Health Sciences and Sciences REB

HEALTH SCIENCES AND SCIENCES RESEARCH ETHICS BOARD

CERTIFICATE OF ETHICAL APPROVAL

This is to certify that the University of Ottawa Health Sciences and Sciences Research Ethics Board has examined the application for ethical approval for the research project **The effects of pre and postexercise meals of varying glycemic index on morning exercise-induced fat utilization in obese men (our file: H 10-03-03)** submitted by Patrick Bennard and supervised by Eric Doucet, both of the School of Human Kinetics, Faculty of Health Sciences. The Board found that this research project met appropriate ethical standards as outlined in the Tri-Council Policy Statement and in the Procedures of the University of Ottawa Research Ethics Boards, and accordingly gave it a Category 1a (approval). This certification is valid for one year from the date indicated below.

____
Rita D'Alessandro
Protocol Officer for Ethics in Research,
For the Chairperson of the Health
Sciences and Sciences REB
Daniel Lagarec

October 30, 2003
Date

APPENDIX B

Recruitment Poster (English and French)

APPENDIX C

Telephone Questionnaire (English and French)

Telephone questionnaire for inclusion in the study entitled:

ACUTE EFFECTS OF EXERCISE TIMING AND BREAKFAST MEAL GLYCEMIC INDEX ON MORNING EXERCISE-INDUCED FAT OXIDATION

1) What is your age? __________

2) What is your body weight? __________

3) What is your height? __________

4) Do you smoke? Yes ☐ No ☐

5) Has your body weight been stable (± 2 kg) for at least the past 6 months?
Yes ☐ No ☐

6) Are you sedentary? Yes ☐ No ☐
If not, how many minutes of physical activity do you perform per week?
____

7) Do you take any medications? Yes ☐ No ☐

If yes, which ones?
____
____
____
____
____

8) Do you have diabetes? Yes ☐ No ☐

9) Do you have any heart problems? Yes ☐ No ☐

10) Do you have high blood pressure? Yes ☐ No ☐

11) Do you have asthma or any other respiratory problems? Yes ☐ No ☐

12) Has your physician ever said that you have a thyroid gland problem?Yes ☐ No ☐

13) Do you have any other health problems that were not mentioned in this questionnaire?
Yes ☐ No ☐

Questionnaire téléphonique d'inclusion de l'étude intitulée:

"ACUTE EFFECTS OF EXERCISE TIMING AND BREAKFAST MEAL GLYCEMIC INDEX ON MORNING EXERCISE-INDUCED FAT OXIDATION"

1) Quel est votre âge? __________

2) Quel est votre poids corporel? __________

2) Quelle est votre taille? __________

4) Êtes-vous fumeur? Oui ▯ Non ▯

5) Avez-vous un poids stable (± 2 kg) depuis au moins 6 mois? Oui ▯ Non ▯

6) Êtes-vous sédentaire? Oui ▯ Non ▯
Si non, combien de minutes d'activité physique continue pratiquez-vous par semaine?

____

7) Prenez-vous des médicaments? Oui ▯ Non ▯

Si oui, lesquels?

____
____
____
____
____

8) Souffrez-vous de diabètes? Oui ▯ Non ▯

9) Souffrez-vous de problèmes cardiaques? Oui ▯ Non ▯

10) Souffrez-vous d'hypertension? Oui ▯ Non ▯

11) Souffrez-vous d'asthme ou d'autres problèmes respiratoires? Oui ▯ Non ▯

12) Est-ce que votre médecin vous a déjà dit que vous aviez des problèmes de glandes thyroïdes? Oui ▯ Non ▯

13) Souffrez-vous de tout autre problème de santé qui n'a pas été mentionné dans le présent questionnaire? Oui ▯ Non ▯

APPENDIX D

Medical and Dietary Questionnaires (English and French)

MEDICAL AND NUTRITIONAL HISTORY

PARTICIPANT HISTORY:	YES	NO
Have you participated in a research protocol before?	&	&
Where? ____________________		
Are you presently participating in a research protocol?	&	&

FAMILY HISTORY	YES	NO	If « yes », specify : Code : 1- father 2- mother 3- siblings 4- grandparents
Diabetes	&	&	____________________ ____________________
Heart Disease	&	&	____________________ ____________________
Other	&	&	____________________ ____________________

MEDICAL/SURGICAL HISTORY: NONE &

(If this condition persists, please describe it below)

CONDITION	YEAR

MEDICAL AND NUTRITIONAL HISTORY cont...

MEDICATIONS:

Enumerate all medications that the participant has taken over the course of the past month:

NONE &

NAME (GENERIC ONLY)	TOTAL DAILY DOSE (MG/DAY)	DATE BEGAN (D/M/A)	DATE ENDED* (D/M/A)	INDICATION
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________
________	________	___/___/___	___/___/___	________

* If medication is ongoing, leave the DATE ENDED space blank.

MEDICAL AND NUTRITIONAL HISTORY cont...

TOBACCO CONSUMPTION:

		SMOKER	QUIT SMOKING	NEVER SMOKED
A.	Cigarettes	&	&	&
B.	Cigars	&	&	&
C.	Pipes	&	&	&

At what age did you begin? ____years How long? ____years

A) How many cigarettes, cigars and/or pipes do you smoke daily (during the past year)? _____/day

B) How old were you when you quit smoking? ______ years

C) How long has it been since you quit smoking? ____ years

D) How many times have you tried to quit? _________

PHYSICAL ACTIVITY HISTORY:

Do you do exercise (moderate physical activity "more than 60% of VO2max") for 30 continuous minutes or more during the week? **YES** &**NO** &

If "YES", what type of exercise? ____

If "YES", how many times per week? ________________ /week

TYPE OF DIET (CHOOSE ONLY ONE):

Without restriction	&	Reduced fat and cholesterol	&	Reduced sodium	&
I watch what I eat	&	Diabetic	&	Vegetarian	&
Reduced calories	&				

BODY WEIGHT HISTORY BEFORE THE STUDY:

Have you ever followed a weight reduction program (loss of weight ≥ 4 kg or 10 lbs)? **YES** &**NO** &

If "YES", weight before the first program: _____________ kg/lbs

Your current age: _____________ years

If "YES", how many programs have you followed? ____________________

MEDICAL AND NUTRITIONAL HISTORY cont...

& None **TYPE OF PROGRAM**

Name of program	Age (year)	Duration (days/weeks/months)	Weight lost (kg or lbs)	Weight regained (kg or lbs)	Duration of regain (days/weeks/months)
& Weight Watchers	___	___	___.___	___.___	___
& Scarsdel	___	___	___.___	___.___	___
& Nutri-bars	___	___	___.___	___.___	___
& Diuretics	___	___	___.___	___.___	___
& Laxative	___	___	___.___	___.___	___
& Pills	___	___	___.___	___.___	___
& Protein shake	___	___	___.___	___.___	___
& Surgical intervention	___	___	___.___	___.___	___
& Montignac	___	___	___.___	___.___	___
& Others (specify)	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___
___	___	___	___.___	___.___	___

HISTOIRE MÉDICALE ET NUTRIONNELLE

ANTÉCÉDENTS DU PARTICIPANT :	**OUI**	**NON**
Est-ce que vous avez déjà participé à un protocole de recherche?	❐	❐
Où? ______________________________		
Est-ce que vous participé à une étude présentement?	❐	❐

ANTÉCÉDENTS FAMILIAUX	**OUI**	**NON**	**Si " oui " : spécifier** Code : 1-père 2- mère 3- fratries 4- grand parents
Diabètes	❐	❐	______________________________ ______________________________
Maladies du coeur	❐	❐	______________________________ ______________________________
Autres	❐	❐	______________________________ ______________________________

ANTÉCÉDENTS MÉDICAUX/CHIRURGICAUX: **AUCUN** ❐
(Si cette condition persiste, veuillez la décrire ci-dessous)

CONDITION	**ANNÉE**
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________
______________________________	__________

HISTOIRE MÉDICALE ET NUTRIONNELLE suite...

MÉDICAMENTS:

Énumérer tous les médicaments que le participant a pris au cours du dernier mois: **AUCUNE ☐**

NOM (GÉNÉRIQUE SEULEMENT)	DOSE JOURNALIÈRE TOTALE (MG/JOUR)	DATE DE DÉBUT (J/M/A)	DATE DE FIN* (J/M/A)	INDICATION
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	
		__/__/__	__/__/__	

* Si le médicament est en cours, laisser la case DATE DE FIN vacante.

HISTOIRE MÉDICALE ET NUTRIONNELLE suite...

TABAGISME:

		FUMEURS	ARRÊTÉ DE FUMER	JAMAIS FUMÉ
A.	Cigarettes	❒	❒	❒
B.	Cigares	❒	❒	❒
C.	Pipes	❒	❒	❒

À quel âge avez vous débuté? ____ans Durée? ____années

A) Combien de cigarettes, cigares ou/et pipes fumiez-vous (pendant la dernière année)? _____/jour

B) Quel âge aviez-vous lorsque vous avez arrêté de fumer? _______ ans

C) Depuis combien de temps avez-vous arrêté de fumer? _____ ans

D) Combien de fois avez-vous essayé d'arrêter? __________

ANTÉCÉDENTS D'ACTIVITÉ PHYSIQUE:

Est-ce que vous faites de l'exercice (activité physique modérée " plus de 60 % de la VO_2max ") pendant plus de 30 minutes continues ou plus par semaine? **OUI** ❒ **NON** ❒

Si "OUI", quel type d'exercice? ____

Si "OUI", combine de fois par semaine? __________________ /semaine

TYPE DE RÉGIME (CHOISIR UN SEUL):

Sans restriction	❒	Réduit en gras et en cholestérol	❒	Réduit en sodium	❒
Je soigne mon alimentation	❒				
Réduit en calories	❒	Diabétique	❒	Végétarien	❒

HISTOIRE DE POIDS CORPOREL AVANT L'ÉTUDE:

Avez-vous déjà suivi un régime amaigrissant (perte de poids ≥ 4 kg ou 10 livres)? **OUI** ❒ **NON** ❒

Si "OUI", poids avant le premier régime: _______________ kg

Votre âge à ce moment: _______________ ans

Si "OUI", combien de régimes avez-vous suivi? ________________________

HISTOIRE MÉDICALE ET NUTRIONNELLE suite...

❐ Aucun	TYPE DE RÉGIME				
Nom du régime	**Âge (année)**	**Durée** (jours/semaines/mois)	**Poids perdu (kg ou lbs)**	**Poids regagné (kg ou lbs)**	**Durée du regain** (jours/semaines/mois)
❐ Weight Watcher	___	___	___.__	___.__	___
❐ Scarsdel	___	___	___.__	___.__	___
❐ Nutri-bars	___	___	___.__	___.__	___
❐ Diurétiques	___	___	___.__	___.__	___
❐ Laxatif	___	___	___.__	___.__	___
❐ Pillules	___	___	___.__	___.__	___
❐ Protéines liquides	___	___	___.__	___.__	___
❐ Intervention chirurgicale	___	___	___.__	___.__	___
❐ Montignac	___	___	___.__	___.__	___
❐ Autres, spécifier	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___
___	___	___	___.__	___.__	___

APPENDIX E

Consent Form (English and French)

Université d'Ottawa • University of Ottawa

Faculté des sciences de la santé
École des sciences de l'activité physique

Faculty of Health Sciences
School of Human Kinetics

CONSENT FORM

ACUTE EFFECTS OF EXERCISE TIMING AND BREAKFAST MEAL GLYCEMIC INDEX ON MORNING EXERCISE-INDUCED FAT OXIDATION

Graduate student:
Patrick Bennard (B.Sc.)
Supervisor:
Éric Doucet (Ph.D.)
School of Human Kinetics
Faculty of Health Sciences, University of Ottawa

1. INVITATION TO PARTICIPATE

I, ______________________________, am invited to participate in the above mentioned research protocol. The researchers conducting this study are: Patrick Bennard, B.Sc. and the supervisor Eric Doucet, Ph.D., of the School of Human Kinetics.

2. PURPOSE OF THE RESEARCH

The aim of this study is to examine the variation of fat utilization when normal-weight men ingest a low- or high-glycemic index meal either before or after a morning session of physical activity.

3. BACKGROUND

During this study, the effects on fat oxidation of a breakfast with different types of carbohydrates will be assessed when consumed early in the morning either before or after a moderate exercise session. Given the fact that the balance between the intake and the output of fat is crucial for weight maintenance and that no clear recommendation yet exists regarding the timing of exercise and meal composition that maximize fat oxidation, this study will yield important information. Indeed, the results of this research program will allow the researchers to determine the type of intervention modality that permits an increased utilization of fats.

4. DESCRIPTION OF THE PROPOSAL

Initial contact and s*creening visits*: After a brief screening interview by telephone, I will be required to come to the laboratory for a first brief visit (*initial contact*) so that the study and consent form can be explained to me (one to two hours). I can then bring the consent form home, along with accompanying questionnaires, so that further reading and discussion with family members are made possible. After having signed the consent form, I will then take part in two s*creening visits* at the research center during which some preliminary measurements will be made. These visits will each take approximately two to three hours.

Date (d/m/y):__________________ Participant's initials:______________

SCREENING VISIT 1

A. Weight, height, and body composition (9h30-10h30)

For the first *screening visit,* I will be required to arrive at the laboratory at around 8h30. My height and body weight will be measured. Afterwards, my body composition (percent body fat and percent lean mass) will be determined by means of a method called dual energy X-ray absorptiometry (DEXA). For this measurement, I will have to lie on an examination table, fully clothed, while a low-intensity X-ray scans my entire body. This measurement takes on average 20 minutes. This measurement is routinely performed and is not associated with any great risk to health, and it will be made by qualified technicians.

B. Maximal aerobic power - VO2max (10h30-12h00)

After an adequate warm-up, and after the installation of security measures such as a blood pressure cuff and electrodes for obtaining an electrocardiographic tracing during exercise, I will be invited to get on a treadmill. This measurement consists of evaluating maximal aerobic capacity during an exercise to exhaustion. This measurement is used as an indicator of cardiorespiratory health. The test will be performed on the treadmill, and will be conducted by a certified kinesiologist. The intensity of exercise will be increased every 2 minutes until I reach exhaustion. This test is on average of 10 to 15 minutes in duration. The kinesiologist will end the test at any moment if such is my desire. I will have to breathe through a mouthpiece (similar to a diving mouthpiece) while my nose is clipped for the duration of this test. The total duration of the test (preparation, warm-up, the measurement, and cool-down) is usually less than one hour. The risks associated with this measurement include dyspnoea (shortness of breath), extreme fatigue, and muscle soreness. Although the probability is low, irregular heart beats and even heart attacks may occur during this type of effort. In order to prevent these situations, heart rate and blood pressure will be continuously monitored by the kinesiologist throughout the test. The equipment necessary for intervention in emergencies will be on hand during the measurement. Although this test is very intense, the occurrence of an emergency is extremely rare.

SCREENING VISIT 2

A. Maximal lipid utilization – FATmax (9h30-11h00)

In order to determine the intensity of exercise at which lipid utilization is maximal, a second exercise test on the treadmill will be conducted. While similar to the maximal aerobic capacity test described in the preceding section, this test differs by the duration of the stages which are 4 minutes instead of 3, and by the fact that it will not be carried on until exhaustion (until about 85% of maximal intensity). It is important to note that the same security measures will be in place during this measurement. At the end of this second *screening visit,* I will be given instructions regarding experimental sessions including: i) detailed instructions regarding pre-experimental dietary recommendations to which I must adhere. These recommendations will include an energy-balanced dietary regime incorporating 55% carbohydrates, 30% lipids, and 15% proteins. This regime must be followed for 3 days before each experimental session (thus, followed 4 times in total); ii) abstenance of all physical activities during the 48 hours prior to each experimental session; iii) tentative scheduling of each experimental session (*half-days 1 to 4* described in detail in the following sections).

HALF-DAYS 1 to 4

If I accept to take part in this study, I will undergo four (4) experimental sessions (*half-days*) which are each about 6 hours in duration. During these 4 sessions, the arrival time at the laboratory will be at 7h20. The 4 sessions will proceed as follows:

Date (d/m/y):________________ Participant's initials: ____________

Sessions 1 and 2- 7h30: Insertion of a catheter; 8h00: Measurement of basal metabolism; 8h30: Breakfast (*session 1* = high glycemic index and *session 2* = low glycemic index); 9h30: Exercise session with measurement of exercise energy expenditure; 10h20: Shower; 10h30-12h30: Measurement of energy expenditure and blood samples; 12h35: A buffet-style meal will be served.
Sessions 3 and 4 will be identical to *sessions 1 and 2*, with the exception that the exercise will take place at 8h30, followed by the shower at 9h20, and the breakfast at 9h30 (*session 3* = high glycemic index and *session 4* = low glycemic index). A detailed description of the methods used is presented is the following sections.

Resting metabolic rate and catheter insertion (7h30-8h30)
Shortly after my arrival, a catheter (a plastic tube) will be placed in a vein in my arm so that blood samples may be collected throughout the entire morning. An initial blood sample will be drawn. The total quantity of blood to be drawn during the Session will equal about 100-150 ml (4-5 ounces), which is less than a blood donation. After a 20-minute rest, the measurement of resting metabolic rate will be performed. This measurement is done to establish resting energy expenditure based on the consumption of oxygen and the production of carbon dioxide. I will be required to rest on a bed while a transparent plastic hood will be placed over my head for the duration of the test. I will then be required to breathe normally in this system for a period of about 30 minutes. This measurement poses no risk, and it is important to note that this equipment is configured to ensure that adequate supplies of fresh air circulate through the plastic hood. Another blood sample will be drawn at 8h30.

Breakfast meal (8h30-9h30 during Sessions 1-2, and 9h30-10h30 during Sessions 3-4)
I will be required to consume a low-glycemic index breakfast meal (*sessions 1 and 3*) that consists of large-flake porridge oats with fructose sweetener, and apple juice; or a high-glycemic index breakfast (*sessions 2 and 4*) that consists of one-minute instant porridge oats with sucrose sweetener, and Gatorade beverage. During all of the experimental sessions, I will be required to consume the entire meal within 15 minutes.

Exercise sessions (9h30-10h20 during Sessions 1-2, and 8h30-9h20 during Sessions 3-4)
Following an adequate warm-up, I will be required to take part in an exercise session of moderate intensity (this will be determined during the *FATmax* measurement). During this measurement, I will be required to walk on a treadmill at a speed and incline corresponding to the predetermined intensity. The intensity of the exercise session will be constant, and the duration of the session will be about 35-40 minutes. Furthermore, samples of expired air will be taken for 5 minutes at each 15-minute interval (thus, 2 times during the exercise), using the same equipment as the one used during the *maximal aerobic power* measurement. Blood samples will also be drawn before the start, at the midpoint, and at the end of the exercise bout. A cool-down of 5 minutes will follow the exercise session. It is important to note that I can decide to terminate the exercise session if I so desire. This measurement poses little risk, and it must be noted that a qualified kinesiologist as well as a nurse will be present at all times during the measurement.

Shower (10h20-10h30 during Sessions 1-2, and 9h20-9h30 during Sessions 3-4)
I will have access to showers at the research centre, and must take a shower after the exercise. In order to minimize any influence of water temperature on resting metabolism, I will be required to maintain the temperature of the water between 35-40^{0}C with the aid of a thermometer that will be provided for me.

Date (d/m/y):_________________ Participant's initials:______________

Resting period (10h30-12h30)
During the four experimental sessions, I will be required to undergo a resting period between 10h30 and 12h30. During this period, blood samples will be drawn at intervals of 60 minutes, that is, at 10h30, 11h30, and 12h30. Furthermore, resting metabolic rate measurements will be performed (as described above) at intervals of 60 minutes for a period of 30 minutes per sample, that is, at 11h00 and at 12h00. The catheter will then be withdrawn at 12h30. There is no risk associated with this measurement.

"Buffet"-style lunch meal (12h45-13h15)
At 12h45, a meal comprising a wide variety of foods will be served to me. I will be asked to eat as much food as I desire. My caloric intake and the composition of ingested food will be calculated from the quantities of ingested foods. Appetite will also be evaluated immediately before and after the lunch.

13h15 - 13h30 – END OF HALF-DAYS 1 to 4

In summary, there is a commitment to one (1) brief *initial contact* (1-2 h), two (2) *screening visits* (2-3 h), and four (4) *half-days* (about 6 h each). The experimental sessions will be separated by a minimum of two weeks, and they will be randomly assigned. It must be noted that the experimental days involving blood sampling will be separated by a period of one week.

5. RISKS/DISCOMFORTS
The risks associated with this project are few and very low. The testing procedures will be explained to me in detail by the staff before my participation in any aspect of the study. The measurement of body composition (DEXA) presents few risks. It should nevertheless be noted that this apparatus exposes me to minimal radiation (0.02-0.05 mRem, which is less than the equivalent of exposure to one day of sunlight). Furthermore, this measurement will only be performed at the beginning of the study, thus minimizing the risk associated to repeated exposure to radiation from this procedure. Blood sampling methods also present few risks. A total of approximately 100-150 ml of blood will be drawn for sampling, which is less than a blood donation. However, a small hematoma (bruising or discoloration from where blood is drawn) can occur for a few days following the sampling. Since the catheter must be worn for several hours during the experimental days (the *half-days*), I may experience a certain discomfort. It should be noted that the risk of infections, of phlebitis (inflammation of the vein), and of vaso-vagal shock (loss of consciousness) are small under such conditions, but they remain a possibility nevertheless. Exercise testing is a common procedure with minimal risks. Nevertheless, it should be noted that these tests will be monitored by a kinesiologist trained in cardio-pulmonary resuscitation and exercise testing, and physicians trained in emergency treatment of cardiac emergencies with appropriate equipment will be nearby. The persons responsible will stop the exercise if they suspect occurrences of fainting, dizziness, chest pain, irregular heartbeats, or a heart attack. The incidence of a heart attack is extremely rare, however, with one death per 10,000 tests in people with no history of heart disease. Blood pressure, heart rate, and breathing will be monitored closely and constantly by the kinesiologist during this procedure.

6. BENEFITS
My participation in this study will offer me the opportunity to receive body composition and maximal aerobic power tests, which offer good indices of overall health. I will also obtain information as to my blood pressure and other metabolic information pertaining to the study. Certain notions and practical concepts will be conveyed in regards to healthy eating and to the practice of physical activity.

Date (d/m/y):__________________ Participant's initials: ______________

Université d'Ottawa • University of Ottawa

Faculté des sciences de la santé
École des sciences de l'activité physique

Faculty of Health Sciences
School of Human Kinetics

FORMULAIRE DE CONSENTEMENT

"ACUTE EFFECTS OF EXERCISE TIMING AND BREAKFAST MEAL GLYCEMIC INDEX ON MORNING EXERCISE-INDUCED FAT OXIDATION"

Étudiant gradué :

Patrick Bennard (B.Sc.)

Superviseur :

Éric Doucet (Ph.D.)

École des sciences de l'activité physique
Faculté des sciences de la santé, Université d'Ottawa

1. VOTRE PARTICIPATION

Je, ______________________________, suis intéressé à collaborer à cette recherche menée par Patrick Bennard, étudiant gradué, ainsi que par Dr Éric Doucet de l'École des sciences de l'activité physique à l'Université d'Ottawa.

2. OBJECTIF DE L'ÉTUDE

Cette recherche vise à examiner les changements d'utilisation de gras chez des hommes de poids normal suite à l'ingestion d'un repas à indice glycémique élevé ou faible soit avant ou après une séance d'activité physique.

3. BUT DE L'ÉTUDE

À l'aide de cette recherche, les chercheurs visent à établir les modalités qui favorisent la plus grande augmentation d'utilisation de graisses. Lors de cette étude, des données de prise alimentaire, de dépense calorique et de composition corporelle seront récoltées pendant les sessions expérimentales. Il est espéré que les résultats de cette recherche permettront de mieux comprendre les effets de l'exercice et de la modification de l'indice glycémique sur l'utilisation des lipides et le contrôle du poids. Enfin, ceci permettra d'établir des recommandations plus précises relatives à l'apport alimentaire et le niveau d'activité physique permettant un meilleur contrôle du poids corporel.

4. DESCRIPTION DE L'ÉTUDE

Premier contact et visites préliminaires

Je devrai me présenter au laboratoire pour une brève visite (*premier contact*) lors de laquelle l'étude et le formulaire de consentement me seront expliqués. Par la suite, vous pourrez emporter le formulaire de consentement à la maison afin de le lire plus attentivement et d'en discuter avec les membres de ma famille. Après avoir signé le formulaire de consentement, je devrai prendre part à deux v*isites préliminaires* au centre de recherche lors desquelles des mesures seront effectuées. Ces visites au laboratoire seront d'une durée d'environ deux à trois heures chacune.

Date (j/m/a):____________________ Initiales du participant: ______________

VISITE PRÉLIMINAIRE 1

A. Masse, taille et composition corporelle (8h30-9h30)

Lors de la première *visite préliminaire*, je devrai me présenter au laboratoire vers 8h30. Ma taille et mon poids corporel seront mesurés. Par la suite, ma composition corporelle (pourcentage de masse grasse et pourcentage de masse maigre) sera déterminée à l'aide de la méthode du «dual energy X-ray absorptiometry» (DEXA). Lors de cette mesure, je devrai me coucher vêtu normalement sur une table d'examen pendant qu'un rayon x de faible intensité parcourra tout mon corps. Cette mesure est d'une durée moyenne d'environ 20 minutes. Cette mesure est effectuée de façon routinière en recherche et n'est pas associée à un risque élevé pour la santé, et elle sera effectuée par des techniciens qualifiés.

B. Puissance aérobie maximale - VO2max (9h30-11h00)

Après un échauffement adéquat et l'installation de mesures de sécurité tels un brassard de tension artérielle et des électrodes pour obtenir un tracé électrocardiographique pendant l'effort, je serai invité à monter sur un tapis roulant. Cette mesure consiste à évaluer ma capacité aérobie maximale lors d'un exercice jusqu'à l'épuisement. On utilise cette mesure comme indicateur de ma santé cardiorespiratoire. Ce test sera effectué sur le tapis roulant, et sera mené par un kinésiologue certifié. L'intensité de l'exercice sera augmentée à toutes les 2 minutes jusqu'à ce que j'atteigne l'épuisement. Ce test est d'une durée moyenne de 10 à 15 minutes. Le kinésiologue mettra fin au test à tout moment si tel est mon désir. La consommation d'oxygène sera mesurée par l'entremise d'un embout de caoutchouc (semblable à un embout de plongée) dans lequel je devrai respirer pendant toute la durée du test. La durée totale du test (préparation, échauffement, mesure, et retour au calme) est d'habitude moins qu'une heure. Les risques normalement associés à cette mesure sont : dyspnée (difficulté à respirer), fatigue extrême et douleurs musculaires. Quoi que peu probable, des arythmies cardiaques et même des infarctus peuvent survenir lors de ce genre d'épreuves. Afin de prévenir ces situations, la fréquence cardiaque et la tension artérielle seront inspectées avec minutie par le kinésiologue tout au long du test. Le matériel nécessaire pour intervenir en situation d'urgence sera disponible durant cette mesure. En dépit du fait que cette épreuve est très intense, il est peu probable qu'une situation d'urgence se produira.

VISITE PRÉLIMINAIRE 2

Utilisation maximale des lipides – «FATmax» (9h30-11h00)

Afin de déterminer l'intensité d'exercice à laquelle l'utilisation de lipides est maximale, un deuxième test à l'effort sur tapis roulant sera effectué. Bien que semblable au test de capacité aérobie maximale décrit à la section précédente, ce test diffère par la durée de ces paliers qui s'élèvent à 4 minutes, plutôt qu'à 3, et par le fait qu'il ne se poursuivra pas jusqu'à l'épuisement (jusqu'à environ 85 % de l'intensité maximale). Il est important de noter que les mêmes mesures de sécurité seront appliquées lors du test. À la fin de cette deuxième *visite préliminaire*, je recevrai des instructions détaillées quant aux sessions expérimentales auxquelles j'aurai accepté de me soumettre. Ces recommandations comprendront : i) un régime alimentaire à bilan énergétique balancé incorporant 55% de glucides, 30% de lipides et 15% de protéines. Ce régime devra être respecté pendant 3 jours avant chacune des sessions expérimentales (respecté 4 fois au total); ii) l'abstenence de toute activité physique pendant les 48 heures avant chaque session expérimentale; iii) un horaire provisoire de chaque session expérimentale (*demi-journées 1 à 4* décrites en détail dans les prochaines sections).

Date (j/m/a):________________ Initiales du participant:______________

DEMI-JOURNÉES 1 à 4

Si j'accepte de participer à cette étude, je devrai me soumettre à quatre (4) sessions expérimentales (*demi-journées*) qui seront d'une durée d'environ 6 heures chacune. Lors de ces 4 sessions, l'arrivée au laboratoire se fera à 7h20. Les 4 sessions se dérouleront comme suit :

Sessions 1 et 2- 7h30: Insertion d'un cathéter; 8h00: Mesure du métabolisme basal; 8h30: Petit déjeuner (*session 1* = indice glycémique élevé et *session 2* = indice glycémique faible); 9h30: Session d'exercice avec mesure de dépense énergétique à l'effort; 10h20: Douche; 10h30-12h30: Mesure de dépense énergétique et prélèvements sanguins; 12h35: Un repas de type buffet sera servi.

Sessions 3 et 4 seront identiques aux *sessions 1 et* 2, à l'exception du fait que l'exercice prendra place à 8h30, et sera suivi de la douche à 9h20, et du petit déjeuner à 9h30 (*session 3* = indice glycémique élevé et *session 4* = indice glycémique faible). Une description détaillée des méthodes qui seront utilisées est présentée dans les sections qui suivent.

Mesure de métabolisme de repos et insertion du cathéter (7h30-8h30)

Peu après mon arrivée, un cathéter (un tube de plastique) sera installé dans une veine de mon bras afin de recueillir les échantillons sanguins pendant toute la matinée. Un échantillon initial sera ainsi prélevé. La quantité totale de sang qui sera prélevé sera donc équivalente à environ 100-150ml (4-5 onces), ce qui est moins qu'une donation de sang. Après avoir reposé pendant 20 minutes, la mesure de métabolisme de repos sera effectuée. Cette mesure vise à établir la dépense énergétique de repos en se basant sur la consommation d'oxygène et la production de bioxyde de carbone. Je devrai me coucher sur un lit pendant qu'un casque de plastique transparent sera placé au-dessus de ma tête, dans lequel je devrai respirer pendant une période d'environ 30 minutes. Il est également important de souligner que cet appareil assure un apport adéquat en air frais lors de la mesure, ce qui réduit considérablement l'inconfort y associé. Cette mesure ne comporte aucun risque. Un autre échantillon sanguin sera ensuite prélevé à 8h30.

Repas petit déjeuner (8h30-9h30 lors des Sessions 1-2 et de 9h30-10h30 lors des Sessions 3-4)

Je devrai consommer un petit déjeuner à indice glycémique faible (*sessions 1 et 3*) qui comprendra du gruau à flocons d'avoine gros, et du jus de pomme; ou un déjeuner à indice glycémique élevé (*sessions 2 et 4*) qui comprendra du gruau à flocons d'avoine instants, et du boisson « Gatorade ». Lors de toutes les sessions expérimentales, je devrai consommer tout le repas en 15 minutes.

Sessions d'exercices (9h30-10h20 lors des Sessions 1-2 et de 8h30-9h20 lors des Sessions 3-4)

Suite à un échauffement adéquat, je devrai prendre part à une session d'exercice d'intensité modérée (celle-ci sera déterminée lors de la mesure du *FATmax*). Lors de cette mesure, je devrai marcher sur un tapis roulant à une vitesse et à une pente correspondant à l'intensité prédéterminée. L'intensité de la session d'exercice sera constante et la session sera d'une durée d'environ 35-40 minutes. De plus, des échantillons d'air expiré seront prélevés pendant 5 minutes à toutes les périodes de 15 minutes (donc, 2 fois pendant l'exercice), au moyen des mêmes équipements tels qu'utilisés durant la mesure de *puissance aérobie maximale*. Des échantillons sanguins seront aussi prélevés avant le début, au point milieu, et à la fin de l'exercice. Un retour au calme de 5 minutes suivra la session d'exercice. Il est à noter que je peux décider de mettre fin à la session d'exercice si je le désire. Cette mesure comporte peu de risque, et il est à noter qu'un kinésiologue qualifié ainsi qu'une infirmière seront présents en tout temps lors de la mesure.

Date (j/m/a):__________________ Initiales du participant: ______________

Douche (10h20-10h30 lors des Sessions 1-2 et de 9h20-9h30 lors des Sessions 3-4)
J'aurai accès aux douches du centre de recherche et je devrai prendre une douche suite à l'exercice. Afin de minimiser toute influence sur le métabolisme de repos engendrée par la température de l'eau, je devrai maintenir la température de l'eau entre 35-40^{0}C à l'aide d'un thermomètre qui me sera fourni.

Période de repos (10h30-12h30)
Lors des quatre sessions expérimentales, je devrai me soumettre à une période de repos entre 10h30 et 12h30. Lors de cette période, des échantillons sanguins seront prélevés à intervalles de 60 minutes, soit à 10h30, 11h30, et 12h30. De plus, des mesures du métabolisme au repos seront effectuées (telles que décrites ci-haut) à intervalles de 60 minutes pendant une période de 30 minutes par échantillon, soit à 11h00 et à 12h00. Le cathéter sera ensuite retiré à 12h30. Il n'y a pas de risques associés à cette mesure.

Repas de type « buffet » (12h45-13h15)
À 12h45, un repas comprenant une grande variété d'aliments me sera servi. Je serai demandé de manger autant de nourriture que désiré. Mon apport calorique et la composition de nourriture ingérée seront calculés à partir des quantités de nourritures ingérées. L'appétit sera aussi évalué immédiatement avant et après le dîner.

13h15 - 13h30 – FIN DES DEMI-JOURNÉES 1 à 4

En résumé, il y a un engagement à un (1) *premier contact* bref (1-2 h), deux (2) *visites préliminaires* (2-3 h), et quatre (4) *demi-journées* (environ 6 h chacune). Les sessions expérimentales seront séparées par un minimum de deux semaines, et elles seront assignées de façon aléatoire.

5. RISQUES PRÉVISIBLES
Les risques associés à la participation à cette étude sont peu nombreux et très faibles. Les procédures de mesure me seront expliquées de façon détaillée par les chercheurs avant que je participe à quelconque aspect de l'étude. Les diverses mesures de graisse corporelle (DEXA) présentent peu de risques. Il importe toutefois de souligner que cet appareil m'exposera à un minimum de radiation (0.02-0.05 mRem, ce qui est moins que l'équivalent d'une journée exposé au soleil). De plus, puisque la mesure sera seulement effectuée au début de l'étude, ceci réduit considérablement les effets qui peuvent être associés à l'exposition répétée à cet appareil. Les prélèvements sanguins présentent également peu de risques. Un total d'environ 100-150 ml de sang sera prélevé, lequel est moins qu'une donation sanguine. Cependant, un léger hématome local (contusion ou décoloration à l'endroit où le sang est tiré) pourrait se manifester pendant quelques jours suite aux prélèvements. Puisque le cathéter devra être porté pendant quelques heures durant les journées expérimentales (les *demi-journées*), je pourrais ressentir un certain inconfort. Il est à noter que les risques d'infections, de phlébites (inflammation de la veine) et de chocs vaso-vagals (perte de conscience) sont faibles dans de telles conditions, mais ils demeurent toutefois une possibilité. Les tests d'effort à l'exercice sont des procédures communes dont les risques sont minimes. Toutefois, il est important de souligner que ces tests seront supervisés par un kinésiologue entraîné pour la réanimation cardio-pulmonaire et les tests d'exercice, et des médecins entraînés pour le traitement d'urgences cardiaques avec l'équipement approprié seront tout près. Les responsables mettront fin à l'exercice dans les cas où ils soupçonneront la présence d'étourdissements, des douleurs à la poitrine,

Date (j/m/a):________________ Initiales du participant:____________

d'arythmies cardiaques ou d'infarctus. L'incidence d'infarctus est rare, cependant, avec un décès par 10,000 tests chez des individus n'ayant aucune histoire de maladie du cœur. Ma tension artérielle, ma fréquence cardiaque, et ma respiration seront constamment suivis de très près par le kinésiologue lors de cette procédure.

6. AVANTAGES

Ma participation à cette étude me permettra de recevoir des mesures de composition corporelle et de puissance aérobie maximale. J'obtiendrai aussi de l'information quant à ma pression sanguine, mes niveaux d'acides gras libres et autre information métabolique pertinente à l'étude. Certaines notions et conseils pratiques seront véhiculés quant à l'alimentation saine et à la pratique d'activité physique.

7. COMPENSATION MONÉTAIRE

Les tests auxquels j'aurai accepté de me soumettre sont gratuits. Il en va de même pour le stationnement au centre de recherche. Je serai remboursé pour les frais de déplacements en accord avec la politique de l'Université d'Ottawa et je recevrai également une compensation financière de $100.00 en parties de $25.00 lorsque chaque session expérimentale sera complétée. Si je ne complète pas l'étude, je serai compensé au *prorata* pour le nombre de tests complétés.

8. CONFIDENTIALITÉ ET ANONYMAT

Afin de garantir ma confidentialité et mon anonymat, toutes les précautions et mesures nécessaires seront suivies afin d'assurer que mes résultats et mon information personnelle seront gardés sous la confidentialité la plus sévère.

- Mon nom n'apparaîtra dans aucuns rapports. Un code numérique sera utilisé pour m'identifier dans tous les documents de recherche.
- Si les résultats sont utilisés pour des analyses subséquentes, seul mon code numérique apparaîtra sur les documents de recherche.
- Tous les matériaux et l'information auxquels me pourraient être associés ne seront pas disponible au publique, et seront gardés dans la confidentialité la plus sévère, sauf pour les cas requis par la loi.
- Les données recueillies seront gardées dans un dossier sous clé dans une pièce à accès limité. De plus, les documents sur ordinateur seront protégés par un mot de passe. Les données seront détruites cinq ans suivant leur publication.

9. PARTICIPATION VOLONTAIRE

- Ma participation à cette étude est entièrement volontaire. À tout moment lors de cette étude, mes intérêts vont prévaloir sur les objectifs de l'étude.
- Je serai averti de nouvelles trouvailles qui pourrait influencer ma décision à prendre part dans la présente étude.

10. DROITS DES PARTICIPANTS

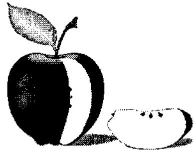
Les chercheurs garantissent que :

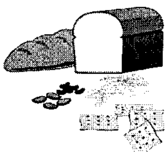
- Je peux me retirer du projet à tout moment.
- La confidentialité de mon information recueillie ainsi que mon anonymat seront rigoureusement protégés comme indiqué plus haut.

Date (j/m/a):________________ Initiales du participant: ____________

APPENDIX F

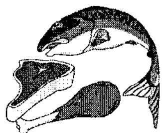
Control Diet Pamphlet (English and French)





FOOD

EQUIVALENTS



✓ ***The following pages describe food quantities which correspond to equivalents in each of the food groups.***

<u>Your personalized plan :</u>

✓ ***You will find at the beginning of each food group a number of equivalents that you must consume everyday to attain your goal.***

Notes

<u>* You must strictly follow this plan for 3 days prior to each experimental session (i.e. full morning sessions – Visits 3 to 6).</u>

To assist you ...
in eating healthy !

Equivalents recommended per day :

_____ ☑ Grain products : □□□□□□□□□□□□□□□
_____ ☑ Vegetables : □□□□□□□□□□□□□□□
_____ ☑ Fruits : □□□□□□□□□□□□□□□
_____ ☑ Dairy products : □□□□□□□□□□□□□□□
_____ ☑ Meat and substitutes : □□□□□□□□□□□□□□□
_____ ☑ Fats : □□□□□□□□□□□□□□□

Tip *: After each meal, fill in the number of squares that represent the number of equivalents that you consumed, and compare your daily profile with your personal food plan.*

Ex : Breakfast : 175ml cereal ☑ Grain products : ■□□□□□□□□□□□□
125 ml milk ☑ Dairy products : ■□□□□□□□□□□□□
1 apple ☑ Fruits : ■□□□□□□□□□□□□

Equivalents	Date : ________	Date : ________
Grain pr. :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□
Vegetables :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□
Fruits :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□
Dairy prod. :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□
Meats :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□
Fats :	□□□□□□□□□□□□□□□	□□□□□□□□□□□□□□□

Equivalents	Date : ________
Grain pr. :	□□□□□□□□□□□□□□□
Vegetables :	□□□□□□□□□□□□□□□
Fruits :	□□□□□□□□□□□□□□□
Dairy prod. :	□□□□□□□□□□□□□□□
Meats :	□□□□□□□□□□□□□□□
Fats :	□□□□□□□□□□□□□□□

Vegetables □ *equivalents/day*

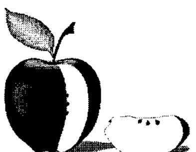
One equivalent corresponds to :

- Juice : tomato, vegetable, carrot,....................125 ml (1/2 cup)
- Winter squash, mixed vegetables, green peas, parsnips ....75 ml (1/3 cup)
- Artichoke hearts, artichokes..........................125 ml (1/2 moyen)
- Beats, carrots, Brussel sprouts, pumpkin puree, onions, leaks, peas, asparagus, brocoli, cabbage, cauliflower, summer squash, zucchini, endives, spinach (cooked), chives, yellow or green beans, canned tomatoes, turnips.........................125 ml (1/2 cup)
- Diced eggplant..250 ml (1 cup)

Vegetables with high % of water

- Celery, mushrooms, cucumbers, fiddle head greens, raw spinach, bean sprouts, alfalfa sprouts, radishes, lettuce, peppers, tomatoes.................500 ml (2 cups)

Fruits □ *equivalents/day*



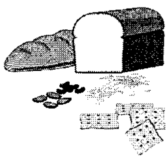
One equivalent corresponds to :

- Apricots, kiwis dates, figs, clementines, prunes.......................................2 fruits
- Stewed fruit (sauces), salads, juices and canned fruit (unsweetened).......................125 ml (1/2 cup)
- Bananas, mangoes, kakis, grapefruit, papayas...................................1/2 fruit
- Nectarines, oranges, peaches, pears, apples, tangerines..............................1 fruit
- Cantaloup..1/4 fruit
- Honeydew..1/8 fruit
- Strawberries, currants, raspberries, water melon, honeydew..............................250 ml (1 cup)
- Rhubarb...500 ml (2 cup)
- **N.B.** For each 1 tsp of table sugar consumed, substract 1 Fruit eq.
- For each 125 ml (1/2 cup) of regular cola, substract 1 Fruit eq.

Grain products

equivalents/day

One equivalent corresponds to :



- Biscuits, dry crackers (all kinds)..................2 units
- Soda crackers, Melba..............................5 units
- Breakfast cereals (warm or ready to serve, little or no sugar).............................30 g (3/4 cup)
- Rice, couscous, pasta, mashed potatoes, corn (cooked)....................125 ml (1/2 cup)
- Popcorn (no butter)......................................750 ml (3 cups)
- Hamburger or hot dog buns, bagel English muffin, Keiser, pita......................1/2 unit
- Sliced bread, small salad bread....................1 unit
- Baguette, 1½ slices.....................................4 units
- Pasta soup..250 ml (1 cup)
- Pea soup..125 ml (1/2 cup)
- ____________________
- ____________________
- ____________________

Dairy Products

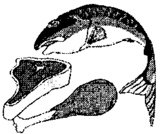
equivalents/day

One equivalent corresponds to :

- Milk, 2%, 1% or skimmed.......................250 ml
- Milk, 3.25%*......................................250 ml
- Yogurt, < 2% m.f..................................175 ml
- Unsweetened evaporated milk (Carnation).....100 ml
- Ice cream..250 ml
- Powdered milk.....................................60 ml
- ____________________
- ____________________
- ____________________

* Substract 1 Fat from your fat equivalents

Meat & substitutes



equivalents/day

One equivalent corresponds to :

- Cheese (skimmed) (7 to 18% m.f.).......30 g (1 ounce)
- Regular cheese (> 20% m.f.)*.............30 g (1 ounce)
- Cottage cheese, 1%, 2% m.f. or ricotta (skimmed)........................60 ml (1/4 cup)
- Meat, poultry, fish..............................30 g (1 ounce)
- Shrimp..5 big, 18 small
- Egg..1 unit
- Peanut butter...............................15 ml (1 tbsp)
- Legumes✞....................................125 ml (1/2 cup)
- Tofu...125 ml (1/2 cup)
- ____________________

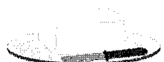
* Substract 2 Fats from your fat equivalents

✞ Substract 1 Bread from your bread equivalents

Fats

equivalents/day

One equivalent corresponds to (5 g of fat products) :



- Butter, margarine, mayonnaise, oil...5 ml (1 tea sp.)
- Cream cheese, light margarine, light mayonnaise, light salad dressing, vinaigrette.............................15 ml (1 table sp.)
- Avocado...1/8 unit
- Olives...8 units
- Nuts and grains.................................15 ml (1 table sp.)
- ____________________

Unlimited consumption!!

- Seasonning
- Bouillons and Consommés
- Diet soft drinks
- Tea, coffee, herbal tea
- Soya sauce, Worcestershire
- Dry or prepared mustard, Ketchup, Vinegar
- Mayonnaise and dressing ULTRA LOW IN FAT

URCM, Pavillion B
Hôpital Montfort
Ottawa, ON

projet FOGIE

« Plan alimentaire »

<u>*Étudiant gradué*</u>

Patrick Bennard

Tel :746-4621 ext. 6029

Code S : ______________________________

Date : ________________________________

ÉQUIVALENTS ALIMENTAIRES

- ✓ ***Les pages suivantes présentent les quantités d'aliments correspondant à l'équivalent pour chaque groupe d'aliments.***

Votre prescription personnalisée :

- ✓ ***Vous trouverez au début de chaque groupe alimentaire le nombre d'équivalents que vous devez consommer chaque jour pour atteindre vos objectifs***

Notes

* Vous devez suivre ce plan de façon stricte pendant 3 jours avant chaque session expérimentale (i.e. sessions durant le matin entier – visites 3 à 6).

Pour vous aider ...
à manger sainement !

Équivalents recommandés par jour :

_____ ✓ Produits céréaliers : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
_____ ✓ Légumes : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
_____ ✓ Fruits : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
_____ ✓ Produits laitiers : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
_____ ✓ Viande et substituts : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
_____ ✓ Gras : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Consigne **:** À chaque repas, remplir le nombre de carreaux correspondant au nombre d'équivalents consommés et comparer votre profil quotidien avec celui de votre plan alimentaire personnalisé.

Ex : Déjeuner : 175 ml céréales ✓ Produits céréaliers : ☐☐☐☐☐☐☐☐☐☐☐☐☐
125 ml lait ✓ Produits laitiers : ☐☐☐☐☐☐☐☐☐☐☐☐☐
1 pomme ✓ Fruits : ☐☐☐☐☐☐☐☐☐☐☐☐☐

Équivalents	Date : ____________	Date : ____________
Pr.Céréaliers	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Légumes :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Fruits :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Pr. Laitiers :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Viandes :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Gras :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Équivalents	Date : ____________
Pr.Céréaliers	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Légumes :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Fruits :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Pr. Laitiers :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Viandes :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
Gras :	☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Légumes ☐ *équivalents/jour*

Un équivalent correspond à :

- Jus : tomates, légumes, carottes,..........125 ml (1/2 tasse)
- Courges d'hiver, macédoine de Légumes, pois verts, panais.................75 ml (1/3 tasse)
- Cœurs d'artichauts, artichaut...............125 ml (1/2 moyen)
- Betteraves, carottes, choux de Bruxelles, citrouille (purée), oignons, poireaux, pois, asperges, brocoli, choux, chou-fleur, courges d'été, courgettes, endives, épinards (cuits), haricots jaunes ou verts, tomates en conserve, navet..125 ml (1/2 tasse)
- Aubergines en dés...........................250 ml (1 tasse)

Légumes à % élevé en eau

- Céleri, champignons, concombre, crosses de fougères, épinards (crus), germes de haricots, germes de luzerne, radis, laitue, poivrons, tomates..............500 ml (2 tasses)

Fruits ☐ *équivalents/jour*

Un équivalent correspond à :

- Abricots, kiwis, dattes, figues, clémentines, pruneaux......................2 fruits
- Compotes, salades ou jus de fruits et fruits en conserve (non-sucrés)............125 ml (1/2 tasse)
- Banane, mangue, kaki, pamplemousse, papaye.............................1/2 fruit
- Nectarine, orange, pêche, poire, pomme, tangerine...................1 fruit
- Cantaloup.....................................1/4 fruit
- Melon miel....................................1/8 fruit
- Fraises, groseilles, framboises, melon d'eau, melon miel..................250 ml (1 tasse)
- Rhubarbe......................................500 ml (2 tasses)
- **N.B.** Pour chaque c. à thé de sucre raffiné consommée, soustraire 1 éq. de Fruit
- Pour chaque 125 ml (1/2 tasse) de cola régulier, soustraire 1 éq. de Fruit

Produits céréaliers

☐ *équivalents/jour*

Un équivalent correspond à :

- Biscottes, biscuits secs (tout genre)...........2 unités
- Biscuits soda, Melba...........................5 unités
- Céréales à déjeuner (cuites ou prêtes à servir, peu ou non-sucrées)......................30 g (3/4 tasse)
- Riz, couscous, pâtes alimentaires, pommes de terre purée, maïs (cuits)..........125 ml (1/2 tasse)
- Maïs soufflé léger, sans beurre.................750 ml (3 tasses)
- Pain hamburger ou hot dog, bager muffin anglais, Keiser, pita.....................1/2 unité
- Pain tranché, petit pain salade..................1 unité
- Pain baguette, tranches 1 1/2pc.................4 unités
- Soupe aux pâtes..................................250 ml (1 tasse)
- Soupe aux pois...................................125 ml (1/2 tasse)
- ______________________
- ______________________
- ______________________

Produits laitiers

☐ *équivalents/jour*

Un équivalent correspond à :

- Lait 2%, 1% ou écrémé........................250 ml
- Lait 3.25%*.......................................250 ml
- Yogourt < 2% m.g.............................175 ml
- Lait concentré non-sucré (Carnation).........100 ml
- Lait glacé...250 ml
- Lait en poudre....................................60 ml
- ______________________
- ______________________
- ______________________

* Soustraire 1 Gras de vos équivalents de gras

Viandes et substituts

☐ *équivalents/jour*

Un équivalent correspond à :

- Fromage part. écrémé (7 à 18% m.g.)...30 g (1 once)
- Fromage régulier (> 20% m.g.)*.........30 g (1 once)
- Fromage cottage 1%, 2% m.g. ou ricotta part. écrémé.....................60 ml (1/4 tasse)
- Viande, volaille, poisson.................30 g (1 once)
- Crevettes...................................5 gr., 18 petites
- Oeuf...1 unité
- Beurre d'arachides.........................15 ml (1 c. à table)
- Légumineuses✞..............................125 ml (1/2 tasse)
- Tofu..125 ml (1/2 tasse)
- ______________________

* Soustraire 2 Gras de vos équivalents de gras

✞ Soustraire 1 Pain de vos équivalents de pain

Matières grasses

☐ *équivalents/jour*

Un équivalent correspond à (5 g de matières grasses) :

- Beurre, margarine, mayonnaise, huile...5 ml (1 c. à thé)
- Fromage crème, margarine légère, mayonnaise légère, sauce salade légère, vinaigrette...........................15 ml (1 c. à table)
- Avocat...1/8 d'unité
- Olives..8 olives
- Noix et graines..............................15 ml (1 c. à table)
- ______________________

Aliments à volonté!!

- Assaisonnements
- Bouillons et Consommés
- Boissons gazeuses diètes
- Thé, Café, Tisanes natures
- Sauce soya, Worcestershire
- Moutarde sèche ou préparée, Ketchup, Vinaigre
- Mayonnaise et Vinaigrette ULTRA FAIBLE EN GRAS

APPENDIX G

Summary of Glycemic Index Calculations for Experimental Breakfast Meals

Ingredient	Food GI[ab]	Portion (g)	Portion CHO (g)	Meal GI[c]	Portion (kcal)
Low-GI					
Large flake porridge oats	83.0	49.0	27.8	28.8	186.2
Unsweetened apple juice	27.0	393.0	44.2	14.9	180.1
Fructose	46.0	8.0	8.0	4.6	33.6
Meal total		450.0	80.0	48.3	399.9
High-GI					
One-minute porridge oats	94.0	50.0	28.3	33.2	190.0
Gatorade (orange)	111.0	665.0	42.0	58.0	168.0
Sucrose	97.0	10.0	10.0	12.1	42.0
Meal total		725.0	80.3	103.3	400.0

Note. "Portion" headers refer to the portions of each ingredient included in the experimental meals. GI = glycemic index; CHO = carbohydrate.

[a]GI values for meal ingredients are mean values as reported in Foster-Powell et al. (2002). [b]GI values indicated are indexed against white bread (GI = 100.0) standard. [c]Weighted food GI based on proportion of total meal CHO, as described by Wolever & Jenkins (1986).

APPENDIX H

Food Appreciation Questionnaire (English and French) and Buffet-style Meal Food List

APPRECIATION OF CERTAIN FOODS

1- Ask the participant to give his level of appreciation of each of the foods of the buffet and the other meals.

2- Specify that on the appreciation scale, number 1 represents a food that he does not like at all and that number 5 represents a food that he likes a lot.

Meats	I do not like at all				I like a lot
Sliced turkey	1	2	3	4	5
Liver pate	1	2	3	4	5
Cheeses					
Brie cheese double cream	1	2	3	4	5
Cheddar cheese	1	2	3	4	5
Cottage cheese	1	2	3	4	5
Condiments					
Salted butter	1	2	3	4	5
Mayonnaise / salade dressing	1	2	3	4	5
Italian dressing	1	2	3	4	5
Cesar dressing	1	2	3	4	5
Mustard	1	2	3	4	5
Tomato ketchup	1	2	3	4	5
Breads					
White Kaiser bread	1	2	3	4	5
Whole wheat Kaiser bread	1	2	3	4	5
Soda crackers	1	2	3	4	5
Vegetables	I do not like at all				I like a lot
Romaine lettuce	1	2	3	4	5
Raw red tomatoes	1	2	3	4	5
Raw carrots	1	2	3	4	5
Fruits					
Red apple	1	2	3	4	5
Desserts					
Chocolate Brownies bits	1	2	3	4	5
Mixed fruit yogurt	1	2	3	4	5

Chocolate chip cookies	1	2	3	4	5
Beverages					
Whole-fat milk (3.25%)					
Partially skimmed milk (2%)	1	2	3	4	5
Skimmed milk (0%)	1	2	3	4	5
Pulp-free orange juice	1	2	3	4	5
Cola	1	2	3	4	5
7-up	1	2	3	4	5
Others					
Raspberry jam	1	2	3	4	5
Peanut butter	1	2	3	4	5
Plain potato chips	1	2	3	4	5
Water	1	2	3	4	5

Comments :

____

____

____

APPRÉCIATION DE CERTAINS ALIMENTS

1- Demander au participant de donner son niveau d'appréciation de chacun des aliments contenus dans le buffet et lors des autres repas.

2- Spécifier au sujet que sur l'échelle d'appréciation, le numéro 1 représente un aliment qu'il n'aime pas du tout et que le numéro 5 représente un aliment qu'il aime beaucoup.

Viandes	Je n'aime pas du tout				J'aime beaucoup
Dinde en tranches	1	2	3	4	5
Pâté de foie	1	2	3	4	5
Fromages					
Brie double crème	1	2	3	4	5
Fromage cheddar	1	2	3	4	5
Fromage Cottage	1	2	3	4	5
Condiments					
Beurre salé	1	2	3	4	5
Mayonnaise / sauce à salade	1	2	3	4	5
Vinaigrette italienne	1	2	3	4	5
Vinaigrette césar	1	2	3	4	5
Moutarde	1	2	3	4	5
Ketchup aux tomates	1	2	3	4	5
Pains					
Pain keiser blanc	1	2	3	4	5
Pain keiser blé entier	1	2	3	4	5
Biscuits soda	1	2	3	4	5
Légumes	Je n'aime pas du tout				J'aime beaucoup
Laitue romaine	1	2	3	4	5
Tomate rouge crue	1	2	3	4	5
Carotte crue	1	2	3	4	5
Fruits					
Pomme rouge	1	2	3	4	5
Desserts					
Brownies au chocolat	1	2	3	4	5
Yogourt aux fruits brassé	1	2	3	4	5

Biscuits aux brisures de chocolat	1	2	3	4	5
Breuvages					
Lait entier (3.25%)	1	2	3	4	5
Lait partiellement écrémé (2%)	1	2	3	4	5
Lait écrémé (0%)	1	2	3	4	5
Jus orange sans pulpe	1	2	3	4	5
Cola	1	2	3	4	5
7-up	1	2	3	4	5
/Autres					
Confitures de framboises	1	2	3	4	5
Beurre d'arachides	1	2	3	4	5
Croustilles natures	1	2	3	4	5
Eau	1	2	3	4	5

Commentaires :

____

____

____

BUFFET DU MIDI – ALIMENTS CONSOMMÉS

Présenter le buffet au participant et l'inviter à manger à volonté jusqu'à satiété sans suralimentation. Le participant doit manger dans un endroit calme sans aucune source de distraction au cours d'une période de 30 minutes.

Aliments	Code Nutrifiq	Portion voulu (g)	Poids avant (g)	Poids après (g)	Poids total consommé (g)
Poitrine de dinde en tranche	50220	130			
Pâté de foie	70055	70			
Fromage brie double crème	10006	100			
Fromage cheddar	10027	100			
Fromage cottage	13015	100			
Beurre	13001	40			
Mayonnaise	45018	60			
Vinaigrette italienne	45114	60			
Vinaigrette césar	45017	60			
Moutarde	66008	30			
Ketchup	113935	40			
Pain blanc	180416	150			
Pain de blé	183075	150			
Biscuits soda	180228	100			
Feuilles de laitue	110251	60			
Tranche de tomate	113529	100			
Bébé carottes coupées	113124	150			
Quartier de pomme rouge	93003	100			
Biscuits Chips Ahoy!	180158	100			
"Brownies" au chocolat	180096	100			
Yogourt aux fruits	15120	250			
Lait écrémé (0%)	12085	1000			
Lait partiellement écrémé (2%)	10079	1000			
Lait entier (3.25%)	12077	1000			
Jus d'orange	93207	1000			
Coca-Cola	140400	355			
7-up	140145	355			
Chips nature	196411	60			
Eau	140429	1000			

Entrer les résultats dans Nutrifiq pour trouver l'énergie ainsi que les macronutriments consommés.
Est-ce que le participant a pris 30 minutes pour manger? **OUI** ☐ **NON** ☐
Si non, expliquer :

APPENDIX I

Study Release/Payment Form (English and French)

Université d'Ottawa • University of Ottawa

Faculté des sciences de la santé
École des sciences de l'activité physique

Faculty of Health Sciences
School of Human Kinetics

I ,____________________________, hereby confirm that I have participated in the study entitled ***"THE EFFECTS OF PRE- OR POST-EXERCISE MEALS OF VARYING GLYCEMIC INDEX ON MORNING EXERCISE-INDUCED FAT UTILIZATION"*** that was conducted by Patrick Bennard of the School of Human Kinetics at the University of Ottawa. For this reason, I am entitled to receive the monetary compensation of one hundred dollars as stipulated in the consent form of this study.

Researcher's signature: ___________________________ Date: ______________

Research Subject's signature: ___________________________ Date: ______________

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Ottawa (Ontario) K1N 6N5 Canada

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Université d'Ottawa • University of Ottawa

Faculté des sciences de la santé
École des sciences de l'activité physique

Faculty of Health Sciences
School of Human Kinetics

Par la présente, je,______________________________, confirme que j'ai participé à l'étude intitulée ***"LES EFFETS DE REPAS PRÉ- OU POST-EXERCICE D'INDICES GLYCÉMIQUES DIFFÉRENTS SUR L'UTILISATION DE GRAS INDUITE PAR L'EXERCICE MATINAL"*** menée par Patrick Bennard de l'École des sciences de l'activité physique à l'Université d'Ottawa. Une compensation financière de cent dollars doit donc m'être remise comme il l'est mentionné dans le formulaire de consentement de cette étude.

Signature du chercheur: ______________________________ Date: ______________

Signature du sujet du participant: ______________________________ Date: ______________

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Ottawa, Ontario K1N 6N5 Canada

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