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Energy Intake Over Two Days is Unaffected by Acute Spring Interval Exercise Despite Increased Appetite and Energy Expenditure

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A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science
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ENERGY INTAKE OVER TWO DAYS IS UNAFFECTED BY
ACUTE SPRINT INTERVAL EXERCISE DESPITE INCREASED
APPETITE AND ENERGY EXPENDITURE

(Thesis format: Integrated Article)

by

Kristine Beaulieu

Graduate Program in Kinesiology

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

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Abstract

A cumulative effect of increased post-exercise oxygen consumption ($\dot{V}O_2$), lipid oxidation and/or reduced energy intake (EI) may explain the fat loss associated with sprint interval exercise (SIE) training. The purpose of this study was to assess the effects of SIE on EI, appetite ratings and peptides, $\dot{V}O_2$, and substrate oxidation (RER). Eight men completed 2 treatments, consisting of consecutive 10-h trials: SIE/recovery (SIEx), and no exercise control (NoEx). Immediate post-exercise suppression of appetite and increase in peptide YY were observed during SIEx. Despite this, overall treatment appetite was greater with SIEx but this did not affect total EI. RER was lower ~6 h post-exercise and total $\dot{V}O_2$ was greater during SIEx vs NoEx. Compared with no exercise control, SIE increases daily energy expenditure but energy intake is unaffected at least acutely. SIE-induced increases in both energy expenditure and lipid oxidation may explain the fat loss associated with this training modality.

KEYWORDS: exercise-induced anorexia, fat loss, glucagon-like peptide 1, insulin, pancreatic polypeptide, peptide YY

Acknowledgements

I must give high, respectful gratitude to my advisor, Dr. Lemon, for accepting me into his laboratory, and for being so supportive and patient throughout my masters. From the beginning, he provided guidance and assistance whenever needed. His trust in me to take the lead and to make the right decisions for my project prepared me well for my doctoral studies.

I have learned a lot in the Exercise Nutrition Research Laboratory, with many challenging yet valuable experiences. Not only was my academic and research training very fulfilling, working with such a great team made my thesis feasible and enjoyable. I am so grateful that Dylan and Kole helped me develop a quality comprehensive project, were there to give me advice throughout and provided me with so much help. I enjoyed getting to know everyone in the lab and I will cherish those numerous hours spent talking about science and ranting about everything else, those countless days collecting data or trying to fix the computer, and all the good times we've had.

This work would not have been possible without the sponsorship of Subway Fresh Catering, Superstore and PepsiCo. I'm also grateful for my scholarships and awards from Gatorade Sports Science Institute, Canadian Institutes of Health Research and Ontario Graduate Scholarship. Thank you all for your support.

Finally, I would also like to thank my family and Adam for allowing me freedom to pursue my ambitions, and for always being patient, loving, encouraging and supportive. I will forever be grateful that you never stood in my way. I hope I have made you proud. Now I move on to the next chapter of my life! Let the adventure continue...

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List of Abbreviations

AUC	Area Under the Curve
CV	Coefficient of Variation
EPOC	Excess Post-Exercise Oxygen Consumption
GLP-1	Glucagon-Like Peptide-1
ICC	Intraclass Correlation Coefficient
MDC	Minimal Detectable Change
NoEx	No Exercise Control (Treatment 2)
NoEx1	No Exercise Control Day 1
NoEx2	No Exercise Control Day 2
PP	Pancreatic Polypeptide
PYY	Peptide YY
RER	Respiratory Exchange Ratio
SIE	Sprint Interval Exercise
SIEx	SIE and Recovery (Treatment 1)
SIEx1	SIE Day
SIEx2	Recovery Day
$\dot{V}O_2$	Oxygen Consumption
$\dot{V}O_{2max}$	Maximal Oxygen Consumption

Chapter 1

1 Introduction

Low volume, high intensity interval training has generated considerable recent interest in both the general population and clinical communities because it elicits similar metabolic and cardiovascular adaptations as traditional endurance training but with a much reduced time commitment (Boutcher, 2011; Gibala, Little, MacDonald, & Hawley, 2012). Unlike many exercise programs prescribed to induce fat loss but producing less than expected results (Boutcher & Dunn, 2009; Caudwell, Hopkins, King, Stubbs, & Blundell, 2009), research from our laboratory suggests 6 weeks of sprint interval exercise (SIE) can lead to significant reductions in fat mass (MacPherson, Hazell, Olver, Paterson, & Lemon, 2011). Further, these results are consistent with previous studies that observed fat loss with various intermittent exercise training (Boutcher, 2011; Gillen, Percival, Ludzki, Tarnopolsky, & Gibala, 2013; Heydari, Freund, & Boutcher, 2012). Some studies even demonstrated greater fat loss with interval training than with continuous aerobic exercise (MacPherson et al., 2011; Trapp et al., 2008; Tremblay et al., 1994).

The fat loss associated with very high intensity interval training may be a cumulative effect of metabolic and dietary adaptations to exercise that promote a negative energy status. While some metabolic, energy intake and appetite measures have been reported with acute sessions of various interval exercise protocols (Deighton, Barry, Connon, & Stensel, 2013; Deighton, Karra, Batterham, & Stensel, 2013; Hazell, Olver, Hamilton, & Lemon, 2012; Sim, Wallman, Fairchild, & Guelfi, 2013), these studies concentrated on the immediate effects so little is known about the protracted effects occurring after a SIE session. Consequently, the present study assessed the effects of SIE on energy intake, subjective appetite, appetite-related peptides, oxygen consumption, and whole-body substrate oxidation over 48 h.

1.1 Sprint Interval Exercise (SIE)

SIE is characterized by repeated bouts of “all-out” exercise (~30-60 s) separated by brief recovery periods (3-4 min). In a single session of SIE, the actual exercise time may range from 2-4 min, compared to 30-60 min in a session of continuous endurance exercise. Despite this large time difference, 2-6 weeks of SIE (3 sessions/wk) produces similar improvements in maximal oxygen consumption ($\dot{V}O_2$), power output and time trial performance as those observed with traditional endurance training (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Burgomaster et al., 2008; Cicioni-Kolsky, Lorenzen, Williams, & Kemp, 2013; Hazell, Macpherson, Gravelle, & Lemon, 2010; MacDougall et al., 1998; MacPherson et al., 2011; Whyte, Gill, & Cathcart, 2010). Additionally, muscle glycolytic and oxidative metabolism markers such as hexokinase, phosphofructokinase, citrate synthase, pyruvate dehydrogenase, β -hydroxyacyl-CoA dehydrogenase (β -HAD) and cytochrome oxidase all increase with SIE training (Burgomaster et al., 2005; Burgomaster, Heigenhauser, & Gibala, 2006; Burgomaster et al., 2008; Gibala et al., 2006). SIE training has also been associated with significant improvements in insulin sensitivity and glucose tolerance (Babraj et al., 2009; Richards et al., 2010; Sandvei et al., 2012; Trapp et al., 2008; Whyte et al., 2010).

When matched for energy expenditures, post-exercise $\dot{V}O_2$ tends to be greater after high intensity vs low-intensity exercise (Warren, Howden, Williams, Fell, & Johnson, 2009; Yoshioka et al., 2001). Similar increases in $\dot{V}O_2$ are shown following SIE and other short supramaximal exercise intervals (Bahr, Gronnerod, & Sejersted, 1992; Chan & Burns, 2013; Hazell et al., 2012; Laforgia, Withers, Shipp, & Gore, 1997; Williams et al., 2013). In addition to increases in post-exercise $\dot{V}O_2$, high intensity exercise has been associated with increased circulating plasma free fatty acids and glycerol, increased post-exercise rates of fat oxidation and lower respiratory exchange ratio (RER) (Chan & Burns, 2013; Hazell et al., 2012; Phelain, Reinke, Harris, & Melby, 1997; Warren et al., 2009; Yoshioka et al., 2001). The elevated plasma glycerol concentrations observed during and following interval exercise are indicative of increased fatty acid oxidation from adipose tissue and/or intramuscular triglycerides (Greer, McLean, & Graham, 1998; McCartney et al., 1986; Trapp, Chisholm, & Boutcher, 2007). Moreover, following 2-6 weeks of

high intensity or sprint interval training, the maximal activity of β -HAD, an enzyme involved in lipid oxidation, was increased significantly, and the content of the protein responsible for greater lipid entry into the muscle, plasma membrane fatty acid-binding protein, was also increased (Burgomaster et al., 2008; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007). Thus, SIE training leads to chronic metabolic adaptations favouring fat oxidation. Additionally, RER remained unchanged in healthy weight men 24 h following acute SIE (Hazell et al., 2012), whereas RER was lower and resting fat oxidation rates were greater in overweight/obese men 24 h after both acute and 2 weeks of SIE (Whyte et al., 2010; Whyte, Ferguson, Wilson, Scott, & Gill, 2013). However, following 2 weeks of SIE, the RER and fat oxidation rates responses were attenuated by 72 h, suggesting a last exercise bout effect (Whyte et al., 2010). Despite leading to significant increases in post-exercise $\dot{V}O_2$ and fat oxidation, the 24-h energy expenditure associated with SIE cannot explain the observed fat losses with SIE training (Hazell et al., 2012). Perhaps chronic increases in post-exercise $\dot{V}O_2$ and/or enhanced lipid oxidation with training may explain this outcome or, alternatively, a reduction in energy intake promoted by alterations in appetite could be responsible.

1.2 Quantifying Appetite and Energy Intake

Appetite can be categorized into hunger, satiation and satiety. These sensations are influenced by physiological, psychological and environmental cues (Blundell et al., 2010; Mattes, Hollis, Hayes, & Stunkard, 2005). Hunger is a state where a person seeks food and initiates eating (Weingarten, 1985). During a meal, as hunger is reduced, feelings of satiation increase, affecting meal size, duration and termination. Satiety is the state after eating that governs the time until hunger returns and when the next meal is eaten (Blundell & MacDiarmid, 1997). These appetite sensations have been monitored previously in studies using 100-point visual analog scales through ratings of hunger, fullness, satiety and motivation to eat (Flint, Raben, Blundell, & Astrup, 2000).

The physiological regulation of appetite is a complex process involving the entry of nutrients in the gut, distension of the stomach, release of hormones from the gut, and the subsequent activation of mechano- and chemoreceptors that act on the hypothalamus

(Havel, 2001). On a meal-to-meal basis, food intake is controlled by a variety of peptides including, but not limited to, appetite stimulating ghrelin, and appetite suppressing amylin, apolipoprotein A-IV, cholecystokinin, enterostatin, glucagon-like peptide 1, oxyntomodulin, pancreatic polypeptide, and peptide YY (Huda, Wilding, & Pinkney, 2006; Murphy & Bloom, 2004). In addition to episodic peptides, tonic adiposity signals, such as insulin and leptin, also have an effect on reducing hunger (Schwartz, Woods, Porte, Seeley, & Baskin, 2000). These peptides, and the mechanisms underlying their secretion, are complex and have generated substantial interest relative to their impact on energy intake, fat loss and obesity.

Quantifying energy intake can be challenging because, as mentioned, eating behaviour is influenced by many factors. Some methods, such as food frequency questionnaires, 24-h recalls and food records have limited accuracy, may be biased and tend to underreport habitual energy intake (Bingham, 1987). In non-habitual but well controlled experimental settings, *ad libitum* buffet-type meals have produced higher reproducibility (Allirot et al., 2012; Arvaniti, Richard, & Tremblay, 2000; Gregersen et al., 2008; Nair et al., 2009). However, the *ad libitum* nature of these meals and variety of food items included may lead to overconsumption (Allirot et al., 2012; Arvaniti et al., 2000; Norton, Anderson, & Hetherington, 2006). Nonetheless, both food records and buffet-type meals have been used to measure energy intake in exercise studies.

1.3 Exercise-Induced Changes in Appetite and Energy Intake

Although the effects of SIE training on metabolism and exercise performance have been well studied in recent years, the effects of this exercise modality on appetite control and energy intake remain largely uninvestigated. With respect to subjective appetite ratings, a significant suppression of hunger has been observed immediately following high intensity exercise ($>60\% \dot{V}O_{2\max}$), often referred to as exercise-induced anorexia (Blundell & King, 1999). However, this effect seems to be short-lived (i.e. 15-30 minutes) and typically does not affect subsequent post-exercise energy intake (Imbeault, Saint-Pierre,

Almeras, & Tremblay, 1997; King, Burley, & Blundell, 1994; King & Blundell, 1995; King, Snell, Smith, & Blundell, 1996; Kissileff, Pi-Sunyer, Segal, Meltzer, & Foelsch, 1990; Thompson, Wolfe, & Eikelboom, 1988; Westerterp-Plantenga, Verwegen, Ijedema, Wijckmans, & Saris, 1997). For example, moderate to high intensity aerobic or strength exercise up to 90 min had no effect on post-exercise energy intake with single or repeated buffet-type meals over the course of experimental trials ranging from 2-8 h, in healthy weight individuals (Balaguera-Cortes, Wallman, Fairchild, & Guelfi, 2011; Erdmann, Tahbaz, Lippl, Wagenpfeil, & Schusdziarra, 2007; Hagobian et al., 2013; Imbeault et al., 1997; Kelly, Guelfi, Wallman, & Fairchild, 2012; King, Wasse, Broom, & Stensel, 2010; King, Wasse, Evans et al., 2011; King, Wasse, & Stensel, 2011; Larson-Meyer et al., 2012) or in overweight/obese women (Unick et al., 2010). Even when subjective hunger is reduced and eating is delayed shortly after an exercise bout, energy intake remains similar over the following 8 to 9 h (King, Miyashita, Wasse, & Stensel, 2010; King, Wasse, & Stensel, 2013) and up to 48 h (King et al., 1994).

In contrast, there is evidence suggesting energy intake can be increased or decreased following exercise. On one hand, compared to rest, energy intake was significantly increased after aerobic and strength exercise ranging from 35 min to 2 h at intensities up to 70% $\dot{V}O_{2max}$ (Erdmann et al., 2007; Laan, Leidy, Lim, & Campbell, 2010; Martins, Morgan, Bloom, & Robertson, 2007; Pomerleau, Imbeault, Parker, & Doucet, 2004). On the other hand, compared to rest, a decrease in post-exercise energy intake has been observed following 1-2 h of cycling at up to 60% $\dot{V}O_{2max}$ in both healthy weight and obese young men (Ueda et al., 2009b; Westerterp-Plantenga et al., 1997). Further, when comparing energy intake between exercise intensities, Kissileff et al. (1990) reported a lower energy intake following 40 min of cycling at 90 W compared to 30 W in healthy weight, but not in obese women, whereas no differences were observed between 30 min of cycling at 50% and 70% $\dot{V}O_{2max}$, or at 50 W and 100 W in healthy weight individuals (Erdmann et al., 2007; Ueda, Yoshikawa, Katsura, Usui, & Fujimoto, 2009). Therefore, acute post-exercise energy intake appears to vary depending on the intensity of exercise, as well as the sex and body mass of the participant.

In a meta-analysis of 29 studies, Schubert and colleagues concluded that acute aerobic and strength exercise ranging from 36-81% $\dot{V}O_{2max}$ does not impact energy intake in post-exercise meals but they did not include maximal intensity or high intensity interval exercise in their analysis (Schubert, Desbrow, Sabapathy, & Leveritt, 2013). Only 2 studies have examined the energy intake response to low volume, high intensity interval exercise or SIE. Energy intake was decreased following 30 min of high intensity intervals (60-s at 100% $\dot{V}O_{2peak}$ with 240 s active recovery at 50% $\dot{V}O_{2peak}$) and very high intensity intervals (15 s at 170% $\dot{V}O_{2peak}$ with 60 s active recovery at 32% $\dot{V}O_{2peak}$) in overweight men (Sim et al., 2013), but energy intake was unaffected following six 30-s Wingate tests in healthy weight men (Deighton, Barry et al., 2013). Interestingly, in overweight men, Sim et al. (2013) found lower energy intake the day following very high intensity interval exercise, indicated by food records. Unfortunately, none of these studies assessed energy intake prior to exercise, which might have been reduced in anticipation of the very intense exercise to be performed. Therefore, there is potential for SIE to affect not only post-exercise energy intake on the exercise day and the following recovery day, but pre-exercise energy intake as well.

1.4 Exercise-Induced Changes in Appetite-Related Peptides

Investigations into the effects of exercise on appetite and energy intake suggest that the peptides involved in appetite control may be potential mechanisms responsible for these responses. For example, in a recent meta-analysis, Schubert and colleagues reviewed 20 studies suggesting the anorectic response following acute aerobic and strength exercise might be promoted by lower concentrations of appetite stimulating ghrelin, and greater concentrations of appetite suppressing glucagon-like peptide-1 (GLP-1), pancreatic polypeptide (PP) and peptide YY (PYY) (Schubert, Sabapathy, Leveritt, & Desbrow, 2013).

1.4.1 Ghrelin

Ghrelin is involved in growth hormone release, appetite regulation, and exerts both gastric and cardiovascular effects (Kojima & Kangawa, 2005). When infused intravenously, ghrelin enhances hunger and food intake (Wren et al., 2001), stimulates

gastric acid secretion and motility (Masuda et al., 2000), and decreases blood pressure without affecting heart rate (Nagaya et al., 2001). Typically, plasma ghrelin concentrations decrease with increasing body mass, and rise in cases of energy deficit, such as following fat loss (Cummings et al., 2002; Martins, Kulseng, King, Holst, & Blundell, 2010; Scheid, De Souza, Leidy, & Williams, 2011). Ghrelin is secreted by the stomach and gut 1-2 h before meals, and after eating, reaches its lowest point within 1 h (Cummings et al., 2001). Acylated ghrelin represents 10% of total ghrelin circulating in the blood and is the only form of ghrelin able to cross the blood-brain barrier to exert its appetite-stimulating effects (Kojima & Kangawa, 2005; Mackelvie et al., 2007). Evidence suggests exercise affects acylated and des-acylated ghrelin differently, making total ghrelin an inadequate measure in exercise interventions (Mackelvie et al., 2007). For example, total ghrelin was unaltered or increased following 20-60 min of moderate to maximal intensity continuous aerobic exercise (Dall et al., 2002; Erdmann et al., 2007; Jurimae, Jurimae, & Purge, 2007; Kraemer et al., 2004; Larson-Meyer et al., 2012; Russel, Willis, Ravussin, & Larson-Meyer, 2009), whereas several studies found a suppression of acylated ghrelin following various exercise modalities (intermittent, cycling, swimming, running, strength, rope skipping) at intensities ranging from moderate to repeated Wingate tests (Balaguera-Cortes et al., 2011; Becker et al., 2012; Broom, Stensel, Bishop, Burns, & Miyashita, 2007; Broom, Batterham, King, & Stensel, 2009; Deighton, Barry et al., 2013; Kawano et al., 2013; King, Miyashita, et al., 2010; King, Wasse, & Stensel, 2011; King, Wasse, Ewens et al., 2011; Sim et al., 2013; Wasse, Sunderland, King, Miyashita, & Stensel, 2013). In contrast, some studies found acylated ghrelin did not change following 40-120 min of continuous aerobic exercise up to 70% $\dot{V}O_{2peak}$ (Hagobian et al., 2013; Kelly et al., 2012; King, Wasse et al., 2010; Larson-Meyer et al., 2012; Unick et al., 2010) and tended to increase following 60 min of running at 70% $\dot{V}O_{2max}$ in endurance trained women (Larson-Meyer et al., 2012). These studies suggest circulating acylated ghrelin is suppressed by exercise in most cases, but the response may be affected by the exercise modality and duration, as well as sex and training status of the participant.

1.4.2 Glucagon-like Peptide-1

Glucagon-like peptide-1 (GLP-1) suppresses appetite and reduces food intake when administered intravenously (Flint, Raben, Astrup, & Holst, 1998). It also inhibits both gastric secretions and gastric emptying (Naslund et al., 1999; Tolessa, Gutniak, Holst, Efendic, & Hellstrom, 1998). It is secreted from the gut after carbohydrate or mixed meal ingestion, falling 30-150 min after eating and helps stimulate insulin secretion following glucose intake (Elliott et al., 1993). Compared to rest, some studies found no change in GLP-1 following 40 min of brisk walking in overweight/obese women (Unick et al., 2010) and with 30 min of intermittent rope skipping or cycling at 65% $\dot{V}O_{2max}$ in men (Kawano et al., 2013), despite hunger being suppressed in both cases. However, increased GLP-1 has been reported during and following 30-60 min of intermittent or continuous exercise at 50-70 % $\dot{V}O_{2max}$ (Larson-Meyer et al., 2012; Martins, Morgan et al., 2007; Ueda et al., 2009a; Ueda et al., 2009b). In most of these studies, the elevation in GLP-1 was maintained up to 30-60 min post-exercise (Martins, Morgan et al., 2007; Ueda et al., 2009a; Ueda et al., 2009b). Moreover, when comparing 30 min of exercise at 50% and 75% $\dot{V}O_{2max}$, no differences in the amplitude of the GLP-1 response were observed (Ueda et al., 2009a). Consequently, elevated GLP-1 following exercise may contribute to the physiological mechanisms behind its appetite suppressive effect.

1.4.3 Insulin

Insulin is secreted into the blood from the pancreatic beta cells basally in proportion to fat mass and within 15 min of carbohydrate intake, particularly glucose (Bagdade, Bierman, & Porte, 1967; Polonsky, Given, & Van Cauter, 1988). In the short term, insulin is responsible for glucose uptake into muscle and fat via GLUT4 transporters, and is involved in substrate metabolism by increasing the synthesis of triglycerides, glycogen and protein, and by decreasing their breakdown (James, Brown, Navarro, & Pilch, 1988; Saltiel & Kahn, 2001). During aerobic exercise at 70% $\dot{V}O_{2max}$ ranging from 45-90 min, insulin concentrations remained similar to rest (Balaguera-Cortes et al., 2011; Broom et al., 2009; Hagobian et al., 2013; King, Miyashita et al., 2010), whereas, insulin was lower following 3 h of cycling at 40% $\dot{V}O_{2max}$ (Hilsted et al., 1980) or a $\dot{V}O_{2max}$ test (Sliwowski,

Lorens, Konturek, Bielanski, & Zoladz, 2001). The insulin response following strength exercise is also variable. One study showed elevated insulin compared to rest after 45 min of exercise (Balaguera-Cortes et al., 2011), while another found no difference after 90 min (Broom et al., 2009). In the long term, insulin is involved in the control of energy homeostasis, acting centrally to reduce energy intake (Schwartz et al., 2003). This was first observed following 3-6 wk of central infusion in baboons, where body mass was subsequently reduced (Woods, Lotter, McKay, & Porte, 1979). Consequently, insulin concentrations are greater in states of positive energy status to promote appetite suppression, even when energy expenditure is increased by a bout of exercise (Chin-Chance, Polonsky, & Schoeller, 2000; Hagobian & Braun, 2006; van Aggel-Leijssen, van Baak, Tenenbaum, Campfield, & Saris, 1999). In contrast, in states of negative energy status such as following fat loss, fasting insulin concentrations decrease, in conjunction with leptin, promoting food intake and mass gain and thus, protecting body adiposity (Cummings et al., 2002; Martins et al., 2010; Schwartz et al., 2003). While lower circulating insulin promotes fat oxidation acutely and food intake chronically, its response to SIE remains to be elucidated and could provide some indication of the mechanisms involved in the fat loss following SIE training.

1.4.4 Pancreatic Polypeptide

Pancreatic polypeptide (PP) inhibits pancreatic secretions, gastric acid secretion and gastric emptying (Hazelwood, 1993; Schmidt et al., 2005), and has been shown to reduce both appetite and food intake at a buffet meal following intravenous infusion (Batterham et al., 2003). It is secreted from the endocrine pancreas following meal consumption in proportion to energy content, peaking between 15 and 30 min postprandial and remaining elevated for up to 6 hours (Adrian et al., 1976; Track, McLeod, & Mee, 1980). The release of PP is also stimulated by gastric distension, observed following water intake (Christofides et al., 1979), and by other gut hormones such as ghrelin (Arosio et al., 2003). There have been some conflicting observations regarding the effects of exercise on plasma PP concentrations. Some studies found an increase during and immediately following a $\dot{V}O_{2\max}$ test or 60-180 min of moderate intensity exercise (Gingerich, Hickson, Hagberg, & Winder, 1979; Greenberg, Marliss, & Zinman, 1986; Hilsted et al.,

1980; Holmqvist et al., 1986; Martins, Morgan et al., 2007; Sliwowski et al., 2001; Sullivan, Champion, Christofides, Adrian, & Bloom, 1984), whereas others showed no increase in PP immediately after 45 min of aerobic or strength exercise (Balaguera-Cortes et al., 2011; Kelly et al., 2012). Nonetheless, in three of those studies, PP concentrations were greater than rest for 15-60 min following exercise (Balaguera-Cortes et al., 2011; Holmqvist et al., 1986; Martins, Morgan et al., 2007). This elevation in PP, in addition to increased GLP-1, could contribute to exercise-induced anorexia.

1.4.5 Peptide YY

Peptide YY (PYY) promotes satiety by inhibiting gastric secretions (Adrian, Savage et al., 1985) and by delaying gastric emptying (Allen et al., 1984). This peptide is secreted from the gut within 30 min of nutrient intake proportionally to the energy and fat content of the meal (Adrian, Ferri et al., 1985). It exists in two forms: PYY₁₋₃₆ and PYY₃₋₃₆; the latter being the main circulating form after a meal (Grandt et al., 1994). During intravenous infusion, PYY₃₋₃₆ suppresses appetite and reduces energy intake at a buffet meal (Batterham et al., 2002), and increases energy expenditure and fat oxidation (Sloth, Holst, Flint, Gregersen, & Astrup, 2007). Both total PYY (PYY₁₋₃₆ and PYY₃₋₃₆) and PYY₃₋₃₆ were elevated during and shortly following moderate to high intensity aerobic exercise (Broom et al., 2009; Kawano et al., 2013; King, Wasse, Ewans et al., 2011; Larson-Meyer et al., 2012; Russel et al., 2009; Ueda et al., 2009a; Ueda et al., 2009b), but not strength exercise (Balaguera-Cortes et al., 2011; Broom et al., 2009) compared to rest. However, Hagobian and colleagues (2013) found no difference in PYY₃₋₃₆ compared to rest after 80 min of cycling at 70% $\dot{V}O_{2peak}$ in both men and women; although PYY₃₋₃₆ was greater post-exercise compared to baseline in women. Unlike GLP-1, the elevation in PYY₃₋₃₆ does not seem to be maintained post-exercise. Moreover, Ueda and colleagues (2009a) observed a greater increase in PYY₃₋₃₆ with 30 min of cycling at 75% than 50% $\dot{V}O_{2max}$, in contrast to GLP-1, suggesting different mechanisms behind the increase in GLP-1 and PYY₃₋₃₆ following exercise. However, at much greater exercise intensities such as with high intensity interval exercise ranging from 85-170% $\dot{V}O_{2max}$ and SIE, total PYY and PYY₃₋₃₆ tended to increase, but failed to achieve significance (Deighton, Barry et al., 2013; Deighton, Karra et al., 2013; Sim et al., 2013). These studies suggest PYY

may play an immediate role in the suppression of appetite, which could be magnified with increasing exercise intensities. Additionally, the rise in postprandial PYY tends to be greater following exercise compared to rest, suggesting exercise may potentiate PYY secretion after food intake (Broom et al., 2009; Cheng, Bushnell, Cannon, & Kern, 2009; King, Wasse, Ewens et al., 2011).

1.5 Summary, Objectives, and Hypotheses

A cumulative effect of increased post-exercise oxygen consumption, lipid oxidation and/or reduced energy intake may explain the observed fat loss with SIE training.

Typically, a SIE day is followed by a recovery (rest) day. Any dietary compensation or prolonged metabolic effects occurring the day following SIE, and the reproducibility of energy status and appetite measures on consecutive days remain to be elucidated.

Therefore, the objectives of this study were to 1) determine the effects of SIE on energy intake, subjective appetite ratings, appetite-related peptides, oxygen consumption, and substrate oxidation over 2 consecutive days; and 2) assess the reliability of these measures over consecutive non-exercise days. It was hypothesized that 1) SIE would increase PP, PYY and GLP-1, resulting in appetite suppression and lower energy intake; 2) SIE would increase energy expenditure and decrease energy intake, leading to a lower energy status; and 3) the intraclass correlation coefficient for energy status and appetite measures would be greater than 0.8.

Chapter 2

2 Energy Intake Over Two Days is Unaffected by Acute Sprint Interval Exercise Despite Increased Appetite And Energy Expenditure

2.1 Statement of Contribution

This manuscript will be submitted for publications soon with authorship as follows, Kristine Beaulieu (KB), T. Dylan Olver (TDO), Kolten C. Abbott (KCA), and Peter W.R. Lemon (PWRL). The authors' responsibilities were as follows– KB, TDO, and PWRL designed the study; KB, KCA, and TDO conducted the research; KB analyzed the data; KB wrote the manuscript; and KB and PWRL had primary responsibility for final content. All authors read, edited and approved the final manuscript. None of the authors had a conflict of interest.

2.2 Introduction

Many exercise programs are designed to induce fat loss, but these interventions often produce variable and less than expected results (Boutcher & Dunn, 2009; Caudwell et al., 2009). However, several studies have observed significant fat loss with various protocols of high intensity interval training (Boutcher, 2011; Gillen et al., 2013; Heydari et al., 2012; MacPherson et al., 2011). Some studies even showed greater fat loss with interval exercise than with continuous aerobic exercise (MacPherson et al., 2011; Trapp et al., 2008; Tremblay et al., 1994). In particular, low volume sprint interval exercise (SIE) training has generated interest in both the general population and clinical communities, eliciting similar metabolic and cardiovascular adaptations as traditional endurance training, but requiring a much reduced time commitment (Gibala et al., 2012).

Several factors may be involved in the fat loss associated with SIE. First, SIE results in elevated post-exercise oxygen consumption ($\dot{V}O_2$) (Chan & Burns, 2013; Hazell et al., 2012; Williams et al., 2013). However, the fat loss associated with 6 weeks of SIE

training (3 sessions/wk) cannot be explained fully by the 24-h energy expenditure of acute SIE (Hazell et al., 2012; MacPherson et al., 2011). Second, high intensity exercise also leads to elevated circulating plasma free fatty acids, glycerol, and catecholamines, increased rates of fat oxidation and lower respiratory exchange ratio (RER) during recovery (Chan & Burns, 2013; Hazell et al., 2012; Warren et al., 2009; Williams et al., 2013; Yoshioka et al., 2001). Third, subjective hunger is suppressed and eating is delayed shortly after a bout of intense exercise (Blundell, Stubbs, Hughes, Whybrow, & King, 2003; King et al., 2013) which is not surprising given that exercise affects appetite-related peptides such as ghrelin, glucagon-like peptide-1 (GLP-1), pancreatic polypeptide (PP), and peptide YY (PYY) (Schubert, Sabapathy et al, 2013). Moreover, energy intake was lower following acute very high intensity interval exercise compared to rest in overweight men (Sim et al., 2013), but not following SIE in healthy weight men (Deighton, Barry et al., 2013).

Thus, the observed fat loss with SIE training may be explained by a cumulative effect of increased post-exercise oxygen consumption ($\dot{V}O_2$), increased lipid oxidation, and/or alterations in appetite regulation and energy intake; however, these proposed mechanisms have never been investigated together. Furthermore, in typical training, a SIE day is followed by a recovery (rest) day. Any dietary compensation or prolonged metabolic effects occurring the day following SIE, and the reproducibility of energy status and appetite measures on consecutive days remains to be elucidated. Therefore, the objectives of this study were to 1) determine the effects of SIE on energy intake, subjective appetite ratings, appetite-related peptides, oxygen consumption, and substrate oxidation over 2 consecutive days; and 2) assess the reliability of these measures over consecutive non-exercise days. It was hypothesized that 1) SIE would increase PP, PYY and GLP-1, resulting in appetite suppression and lower energy intake; 2) SIE would increase energy expenditure and decrease energy intake, leading to a lower energy status; and 3) the intraclass correlation coefficient for energy status and appetite measures would be greater than 0.8.

2.3 Methods

2.3.1 Participants

Ten men volunteered to participate in the study but only eight started and completed the treatments due to scheduling conflicts. The characteristics of the eight participants are as follows: age 25 ± 3 y, height 179.4 ± 7.5 cm, mass 79.6 ± 9.7 kg, body fat $13.3 \pm 5.8\%$, maximal oxygen consumption 58.1 ± 5.4 ml•kg⁻¹•min⁻¹; mean \pm SD. They were recruited by poster advertisement and email. They were screened and excluded from the study if they smoked, had gained or lost more than 4.5 kg during the six months prior to the study, were engaged in any structured exercise training program or varsity team, or had health issues or food allergies that could preclude them from participating (Appendix A). This study was approved by the Health Sciences Research Ethics Board at Western University (Appendix B).

All procedures and risks were explained to the participants and they all provided written, informed consent prior to the start of the study (Appendix C). To avoid any alterations in eating behaviour and any influences on the measurements of energy intake, participants were not informed about the energy intake portion of the study until all data were collected. At that point, this part of the study was explained to them and consent to utilize these data was obtained (Appendix D).

2.3.2 Preliminary Visit

Approximately 1 wk prior to the start of the study, participants were oriented to sprinting on the manual treadmill and completed a food-rating questionnaire to assure acceptability of the food items served during the test days (Appendix E). Body composition was determined by densitometry using air displacement plethysmography (BodPod®; Life Measurements, Concord, CA) and the Siri equation (Siri, 1961) as described previously (Noreen & Lemon, 2006). Maximal oxygen consumption ($\dot{V}O_{2\max}$) was assessed with an incremental treadmill running test. After a 5-min warm-up, participants ran at 7 mph and the grade was increased from 0% by 2% every minute until volitional exhaustion. Heart rate was monitored throughout the test (Polar WearLink®+ transmitter belt; Polar Electro

Inc, Lake Success, NY). $\dot{V}O_2$ was measured using an online breath-by-breath gas collection system (Vmax; Sensor Medics, Yorba Linda, CA) routinely used in our laboratory (Hazell et al., 2010; Hazell et al., 2012; MacPherson et al., 2011).

2.3.3 Experimental Protocol

Participants completed two experimental treatments SIE and recovery (SIEx) and no exercise control (NoEx). Each consisted of a 34-h period including 2 consecutive 10-h test days in the laboratory, separated by 14 h: SIEx1 and SIEx2, and NoEx1 and NoEx2. The treatments were separated by at least 1 wk, and their order was systematically counterbalanced (i.e. participant 1, NoEx – SIEx; participant 2, SIEx – NoEx, etc.) to avoid an order effect. The participants were advised to keep their diet and physical activity constant throughout the entire study period. For 24 h prior to either experimental treatment, they refrained from exercise, did not consume caffeine or alcohol, recorded and ate the same food items, and slept the same number of hours. On test days, the participants arrived at the laboratory at 08:00 following a 12-h fast (no food or drink except water), and with limited activity (drove or took the bus, used the elevator in the building). Between 08:00-18:00, repeated measurements of $\dot{V}O_2$, energy intake, subjective appetite ratings, and appetite-related peptides were taken (**Figure 1**). Details are outlined below. At 18:00 on the first test day, participants went home with a snack bag, remained sedentary, and returned the following morning for the second test day. During all test days, participants remained in the laboratory and were allowed to read or write quietly between measures.

The SIE session consisted of a 5-min warm up at a comfortable running pace followed by 4, 30-s “all out” sprints on a manually driven treadmill (Desmo Pro; Woodway®, Waukesha, WI), each separated by 4 min of recovery (total time ~20 min). Running speed and ratings of perceived exertion (Noble, Borg, Jacobs, Ceci, & Kaiser, 1983) were recorded during and after each interval, respectively.

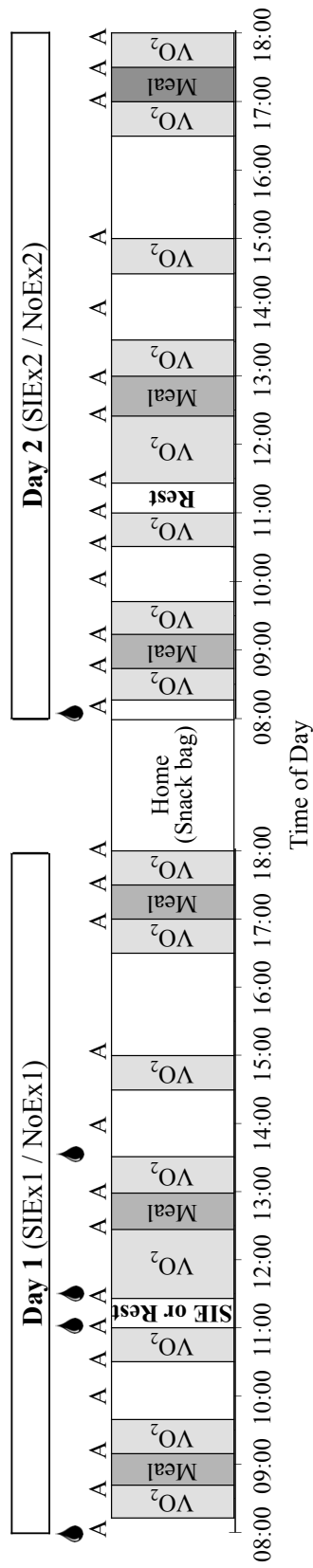


Figure 1: Experimental protocol. Day 1 (on the left) consisted exercise (SIEx1) or no exercise control (NoEx1) and Day 2 (on the right) recovery from exercise (SIEx2) or no exercise control day 2 (NoEx2); ●, blood sample; A, appetite ratings; SIE, sprint interval exercise; $\dot{V}O_2$, oxygen consumption

2.3.4 Energy Intake

Energy intake was assessed with 3 *ad libitum* buffet-type meals (breakfast ~10.5 MJ, lunch ~14.6 MJ, and dinner ~24.3 MJ), found to be reproducible previously (Allirot et al., 2012; Arvaniti et al., 2000; Nair et al., 2009). At every meal, oranges, apples, bananas, yogurt, chocolate milk, juice boxes, and sports drinks were offered. Additional food items for breakfast included bread, peanut butter, jam, and margarine; for lunch, a platter of submarine sandwiches with baby carrots and granola bars; and for dinner, spaghetti, meat sauce, bread, margarine and granola bars. Meals were consumed in a quiet room in the laboratory free of distractions and social interactions. Participants were instructed to eat as little or as much as they wanted. After consumption of the food, any uneaten items were weighed and subtracted from the total provided to obtain net intake. At the end of first day, participants selected snack items (fruit, yogurt, bars, juice, etc) to take home for the night and were instructed to bring back any leftovers. The Canadian Nutrient File (Health Canada, 2010) and food labels were used to analyze the energy and macronutrient content of all items consumed at the laboratory and in the food records. Total energy intake was calculated by adding the energy intake from the 3 meals of the 2 test days and the overnight snack.

2.3.5 Appetite Ratings

Appetite was evaluated by subjective feelings of hunger, fullness, satiety and motivation to eat (Flint et al., 2000) reported on 100-point online visual analog scales at 14 time points (Figure 1) via 4 questions: How hungry do you feel? [I am not hungry at all (0)...I have never been more hungry (100)]; How satisfied do you feel? [I am completely empty (0)...I cannot eat another bite (100)]; How full do you feel? [Not at all full (0)...Totally full (100)]; and How much do you think you can eat? [Nothing at all (0)...A lot (100)]. See Appendix F for website.

2.3.6 Gas Collections

$\dot{V}O_2$ was measured at 8 time points (Figure 1) using the same gas collection system as described above. During measures, participants lay supine with a silicon facemask (8940 Series; Hans Rudolph Inc, Kansas, MO) positioned over the nose and mouth, and were instructed to remain motionless and to avoid falling asleep. For the first 30-min measure, the first 15 min of collection was discarded, and the breath-by-breath data over the last 15 min were averaged and expressed as $L O_2 \cdot \text{min}^{-1}$. For the remaining 30-min measures, the entire collection was averaged. During the 60-min measure post-exercise during SIEx1 (or rest during NoEx1) the data were averaged over four 15-min periods in order to document the post-exercise $\dot{V}O_2$ curve whereas the entire 60-min collection was averaged for SIEx2 and NoEx2, as both were at rest. Total $\dot{V}O_2$ was determined with the trapezoid method by plotting $\dot{V}O_2$ at the mid point of each time point and calculating area under the curve (AUC) to obtain total L of oxygen consumed.

The respiratory exchange ratio (RER) was used to estimate whole-body substrate oxidation (assuming minimal and similar protein oxidation between treatments) and rates of fat oxidation ($g \cdot \text{min}^{-1}$) were calculated as follows: $1.695 \cdot \dot{V}O_2 (L \cdot \text{min}^{-1}) - 1.701 \cdot \dot{V}CO_2 (L \cdot \text{min}^{-1})$ (Holloway et al., 2006). Energy expenditure was calculated with the energy equivalent of oxygen using total $\dot{V}O_2$ and average RER (Peronnet & Massicotte, 1991). Energy status (commonly referred to as energy balance) was determined by subtracting energy expenditure from energy intake.

2.3.7 Biochemical Analysis

Blood samples were collected by venipuncture in the forearm at 4 time points during day 1, and at 1 time point during day 2 (see Figure 1). Blood was drawn into 8.5 mL vacutainers treated with EDTA and a proprietary inhibitor cocktail to prevent degradation of GLP-1 (BD P800 Blood Collection System, Franklin Lakes, NJ). Vacutainers were immediately put on ice, and within 30 min of blood collection, were centrifuged for 20 min at 1300 g at 4°C (Allegra 21R; Beckman Coulter, Mississauga, ON). Plasma obtained was aliquoted and stored at -70°C for later analysis of active GLP-1, insulin, PP,

and total PYY in duplicate using a commercially available multiplex kit (Milliplex Human Gut Hormone Panel, EMD Millipore Corp, Billerica, MA). The assay was read on a Bio-Plex 200 system (Bio-Rad Laboratories, Hercules, CA). Intra- and inter-assay coefficients of variation were 7% and 10% for GLP-1, 3% and 6% for insulin, 4% and 7% for PP, and 2% and 11% for PYY, respectively.

2.3.8 Statistics

Data are presented as mean \pm SD and were analyzed using SigmaPlot Version 12.5 (Systat Software Inc, San Jose, CA) and SPSS Version 20 (IBM SPSS Statistics, Somers, NY). The trapezoid method was also used to calculate the AUC for appetite ratings and 24-h appetite-related peptides. Student's *t*-test for paired samples was used to establish differences between AUC for $\dot{V}O_2$, appetite ratings, and appetite-related peptides, as well as total energy intake, energy expenditure, and energy status. Two-way repeated-measures ANOVA were used to determine treatment differences for $\dot{V}O_2$, RER, appetite ratings, meal energy intake and peptide concentrations. When necessary, Tukey *post hoc* testing was performed. Intra-subject reliability was determined using intraclass correlation coefficient (ICC). An ICC ≥ 0.8 was considered to indicate excellent agreement, ICC ≥ 0.7 -0.79 good agreement, and ICC ≥ 0.6 -0.69 moderate agreement (Choi et al., 2000; Nair et al., 2009). The within-subject coefficient of variation (CV), indicative of the relative variability of the method, was calculated as follows (Gregersen et al., 2008):

$$CV = \{\sqrt{[\sum(x_1 - x_2)^2 / (2n)]}\} / x_{\text{mean}}$$

Minimal detectable change at the 95% level of confidence (MDC) was calculated using the following formula:

$$MDC = [SD \times \sqrt{(1-ICC)}] \times \sqrt{2} \times 1.96$$

Relationships between variables were determined with Pearson's correlation coefficient. Significance was defined as $P \leq 0.05$.

2.4 Results

2.4.1 Experimental Treatments

Body mass remained stable between experimental treatments (SIEx: 79.7 ± 10.0 vs NoEx: 79.4 ± 10.0 kg; $P = 0.32$), which were separated by 6.6 ± 4.4 wk (range: 1-14). Further, there were no differences in energy intake and macronutrient distribution (all $P \geq 0.18$) 24 h prior to both treatments, as indicated by the 24-h food records. Mean energy intake was $8,468 \pm 2,344$ kJ (32.0 \pm 7.4% fat, 43.6 \pm 11.1% carbohydrate and 25.2 \pm 8.6% protein). Average speed achieved during each sprint bout was 11.2 \pm 0.8 (bout 1), 10.4 \pm 0.8 (bout 2), 9.2 \pm 1.1 (bout 3), and 8.4 \pm 1.4 (bout 4) km \cdot h⁻¹ with ratings of perceived exertion for each bout on a scale of 1-10 being 8 \pm 2, 9 \pm 1, 10 \pm 1 and 10 \pm 1. Four of the participants vomited during SIE and 1 participant could only complete 3 bouts. All participants felt lightheaded and exhausted after the exercise, and most felt nauseous.

2.4.2 Energy Intake

No significant treatment, test day, and treatment \times test day interaction effects were present for energy intake at breakfast, lunch, and dinner (all $P \geq 0.37$; **Table 1**). Energy intake of the 3 meals plus overnight snack during SIEx1 and NoEx1 was significantly greater than the energy intake indicated by the 24-h food records ($P < 0.001$). There was a tendency towards a main test day effect for dinner, where energy intake during day 1 may have been greater than day 2 (dinner day 1: $5,260 \pm 1,481$ vs dinner day 2: $4,593 \pm 1,084$ kJ; $P = 0.12$). There were no treatment differences in the energy content of the overnight snack (SIEx: $1,661 \pm 610$ vs NoEx: $1,661 \pm 764$ kJ; $P = 0.99$). Similarly, there were no significant differences between treatments for total energy intake (SIEx: 24.6 ± 4.8 vs NoEx: 24.9 ± 5.4 MJ; $P = 0.76$). Mean daily macronutrient distribution was 65 \pm 5% carbohydrate, 17 \pm 2% protein and 20 \pm 4% fat and daily water intake was 1.47 \pm 0.72 for SIEx and 1.80 \pm 1.71 L for NoEx ($P = 0.47$). The macronutrient distribution of the test meals were significantly different than those indicated by the 24-h food records ($P < 0.001$).

Table 1: Energy intake (kJ) for each meal during SIEx and NoEx

Meal	Breakfast	Lunch	Dinner
SIEx1	3,461 ± 1,113	3,052 ± 647	5,191 ± 1,737
SIEx2	3,372 ± 1,069	3,396 ± 857	4,543 ± 966
NoEx1	3,477 ± 1,867	3,152 ± 569	5,330 ± 1,294
NoEx2	3,443 ± 1,567	3,120 ± 844	4,643 ± 1,257

SIEx1: SIE day; SIEx2: recovery day; NoEx1: No exercise day 1;
NoEx2: No exercise day 2

2.4.3 Appetite Ratings

A significant effect of treatment, time and treatment × time interaction (all $P \leq 0.04$) was present for ratings of motivation to eat. There was also a significant effect of time and treatment × time interaction ($P \leq 0.03$) but no effect of treatment ($P \geq 0.47$) for ratings of hunger, fullness, and satiety. Additionally, there was a significant difference in AUC between treatments for ratings of hunger ($P = 0.003$) and motivation to eat ($P = 0.02$) but not for ratings of fullness ($P = 0.46$) and satiety ($P = 0.90$) (**Figure 2**).

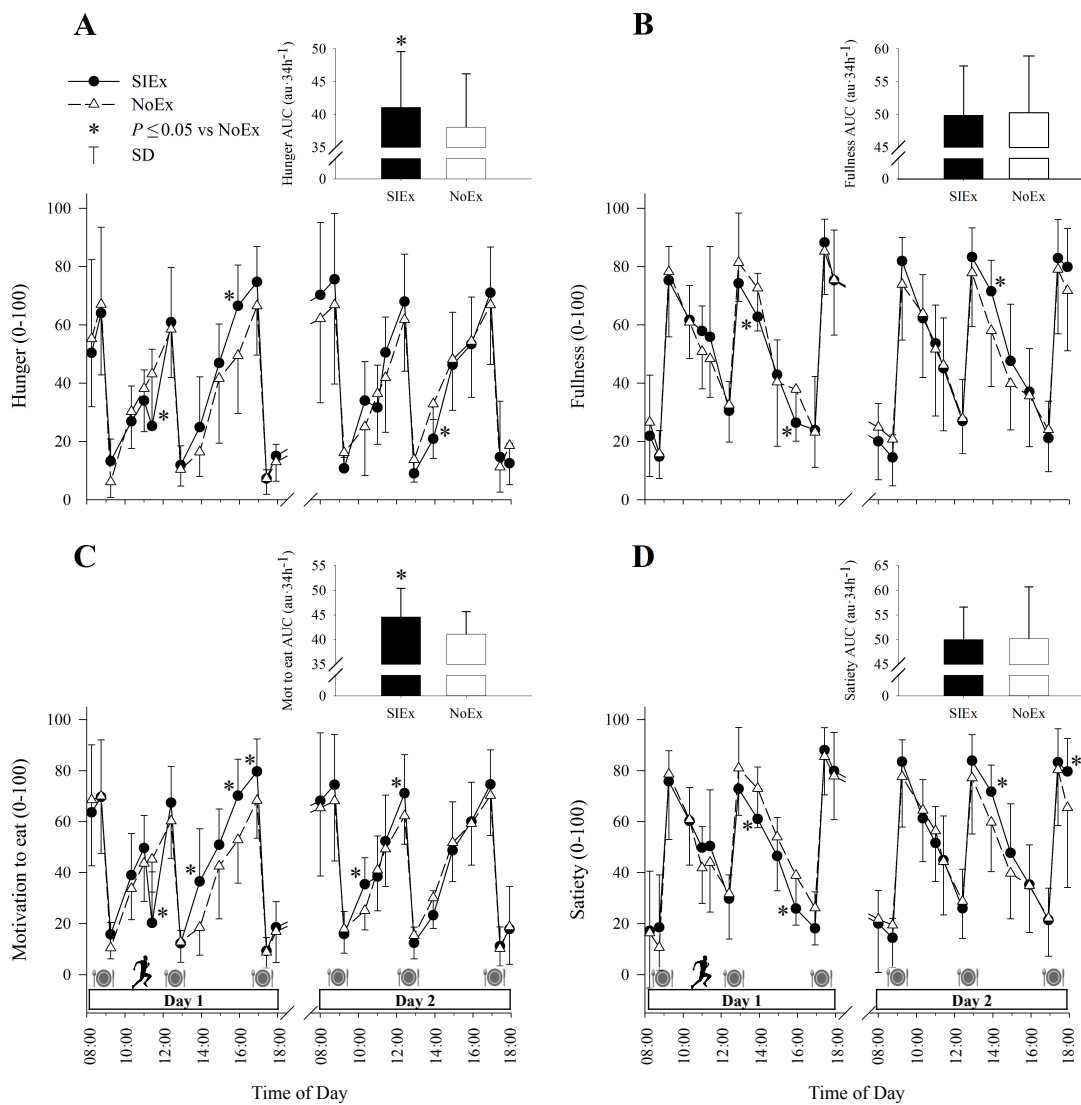


Figure 2: Ratings and area under the curve (AUC) for hunger (A), fullness (B), motivation to eat (C) and satiety (D) during SIEx and NoEx.

2.4.4 Appetite-Related Peptides

There were no significant differences in fasting hormone concentrations between treatments and test days. Two participants were excluded from the GLP-1 analysis due to out of assay range or non-detectable values. A significant effect of treatment, time and treatment \times time interaction (all $P = 0.01$) was present for PYY, where concentrations were significantly greater at 3.4 h during SIEx vs NoEx (**Table 2**). There was only a significant main effect of time (all $P < 0.001$) for PP, GLP-1 and insulin. AUC was greater for PYY during SIEx compared to NoEx ($P = 0.08$), but there were no AUC differences between treatments for PP, GLP-1, and insulin (all $P \geq 0.72$).

Table 2: Plasma GLP-1, insulin, PP, and PYY concentrations during SIEx and NoEx

Peptide (pg·mL ⁻¹)	Treatment	08:00 (Fasting)	11:00 (Pre-SIE)	11:20 (Post-SIE)	13:30 (Post-Meal)	08:00 (day 2) (Fasting)	AUC (pg·mL ⁻¹ ·24h)
GLP-1†	SIEx	13.5 ± 8.7	14.1 ± 6.6	14.6 ± 7.6	30.4 ± 13.4	15.3 ± 7.3	29,212 ± 7,837
	NoEx	15.2 ± 7.5	17.9 ± 10.5	22.6 ± 9.0	25.9 ± 12.5	13.4 ± 5.1	29,865 ± 10,987
Insulin	SIEx	128.3 ± 41.6	447.4 ± 153.4	368.7 ± 119.0	674.6 ± 390.3	155.5 ± 38.8	605,016 ± 259,350
	NoEx	110.2 ± 38.6	406.7 ± 209.6	409.8 ± 286.7	766.1 ± 239.4	131.1 ± 47.7	628,244 ± 184,701
PP	SIEx	11.9 ± 4.6	55.2 ± 35.9	109.6 ± 50.2	142.9 ± 105.5	17.3 ± 13.4	112,829 ± 73,575
	NoEx	13.2 ± 5.5	61.3 ± 40.8	74.8 ± 55.5	156.0 ± 128.8	14.7 ± 7.6	117,582 ± 84,861
PYY	SIEx	47.2 ± 14.1	48.0 ± 18.4	130.5 ± 106.8*	56.5 ± 20.2	42.2 ± 31.9	77,621 ± 17,614
	NoEx	38.0 ± 20.9	39.9 ± 21.5	38.1 ± 11.4	52.4 ± 10.3	39.1 ± 15.6	64,399 ± 14,672

† n = 6; *P<0.001 vs NoEx; AUC: area under the curve

2.4.5 Oxygen Consumption and Energy Expenditure

$\dot{V}O_2$ during SIE was estimated using an average of approximately $18 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ determined previously in our laboratory and converted in $\text{L}\cdot\text{min}^{-1}$ using the current study's participants body mass (Hazell et al., 2012). There was a 2-5 min lag between the end of exercise and the measurement of $\dot{V}O_2$ due to feelings of lightheadedness and nausea. A significant effect for treatment, time, and treatment \times time interaction (all $P \leq 0.03$) was present, where $\dot{V}O_2$ was significantly greater during SIEx at 11:28, 11:35, 11:43, 11:50 and 13:15 (**Figure 3**). Also, $\dot{V}O_2$ tended to be greater at 17:45 during SIEx ($P = 0.12$).

Total 24-h $\dot{V}O_2$ over day 1 was significantly greater during SIEx than NoEx ($P = 0.01$; Figure 3), while there were no treatment differences in 10-h $\dot{V}O_2$ during day 2 ($P = 0.85$). The estimated $\dot{V}O_2$ during SIE was $29 \pm 3 \text{ L}$ and the excess post-exercise $\dot{V}O_2$ (EPOC; SIEx $\dot{V}O_2 - \text{NoEx } \dot{V}O_2$) over the following 21 h (until 08:00 on day 2) was $60 \pm 23 \text{ L}$ ($n = 6$, as 2 outliers were discarded in the EPOC analysis due to negative values obtained following the subtraction).

The calculated energy expenditure during SIE was $611 \pm 73 \text{ kJ}$ and EPOC was $1,250 \pm 492 \text{ kJ}$, resulting in a SIE-induced energy expenditure of $1,850 \text{ kJ}$. Energy status over 24 h (energy intake-energy expenditure) was significantly lower during SIEx1 ($5.0 \pm 2.2 \text{ MJ}$) compared to NoEx1 ($6.7 \pm 3.0 \text{ MJ}$; $P = 0.04$). Energy status over 34 h was also lower during SIEx ($13.2 \pm 4.1 \text{ MJ}$) vs NoEx ($14.9 \pm 4.2 \text{ MJ}$), but this did not achieve significance ($P = 0.15$).

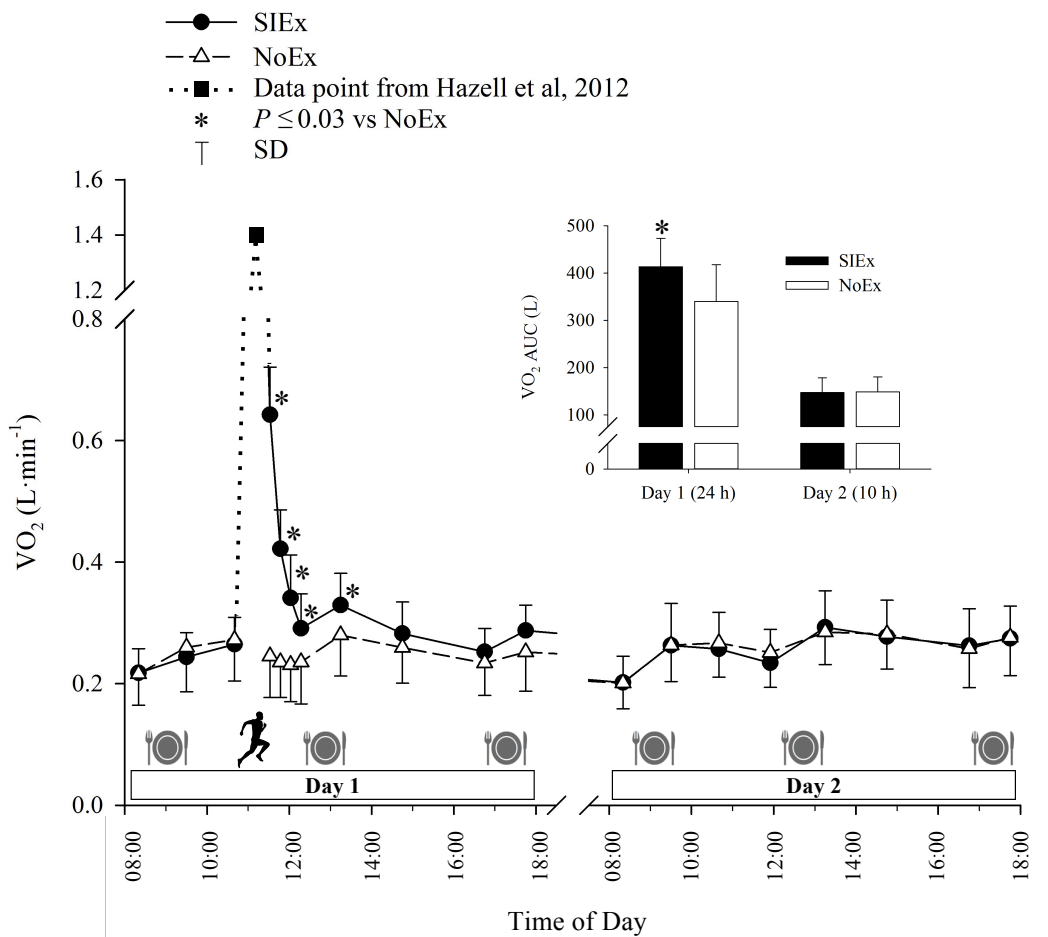


Figure 3: Oxygen consumption ($\dot{V}O_2$) and area under the curve (AUC) during SIEx and NoEx

2.4.6 Substrate Oxidation

RER during SIE was estimated using an average of 1.2 determined previously in our laboratory (Hazell et al., 2012). There was a significant effect for treatment, time, and treatment \times time interaction (all $P \leq 0.01$) for RER, where RER was greater than control at 11:28 and lower at 11:35, 11:43, 11:50 and 16:45 (**Figure 4**). At 13:15, RER tended to be lower than control ($P = 0.08$). Post-exercise rates of fat oxidation were calculated following the return of RER to physiological levels. An effect of time and treatment \times time interaction (all $P < 0.001$) was present, indicating significantly greater fat oxidation rates during SIE at 13:15 (0.097 ± 0.044 vs NoEx: 0.063 ± 0.023 $\text{g}\cdot\text{min}^{-1}$; $P < 0.001$) and 16:45 (0.095 ± 0.018 vs NoEx: 0.057 ± 0.021 $\text{g}\cdot\text{min}^{-1}$; $P < 0.001$).

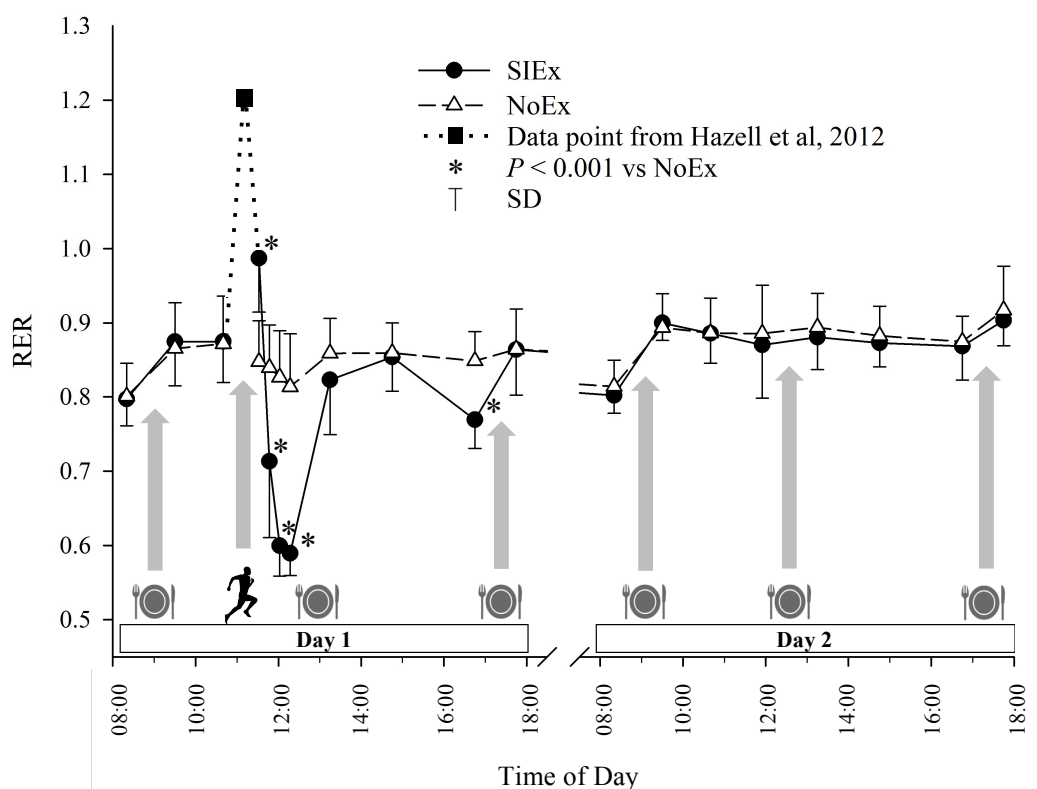


Figure 4: Respiratory exchange ratio (RER) during SIEx and NoEx

2.4.7 Reliability

As shown in **Table 3**, reliability on consecutive non-exercise days was excellent for $\dot{V}O_2$, fullness and satiety, good for hunger, moderate for 10-h energy intake and motivation to eat, and low for RER. There were no significant differences in day-to-day measures of energy status and appetite, except for RER, which was greater on NoEx2 vs NoEx1 ($P < 0.01$).

Table 3: Reproducibility of energy status and appetite measures over consecutive non-exercise days (NoEx1 and NoEx2)¹

Measure	$\dot{V}O_2$ (L•10h⁻¹)	RER	EI (MJ•10h⁻¹)	Hunger (au•10h⁻¹)	Fullness (au•10h⁻¹)	Satiety (au•10h⁻¹)	Motivation (au•10h⁻¹)
NoEx1	142 ± 34	0.84 ± 0.04*	11.96 ± 3.34	36.4 ± 10.3	51.6 ± 9.0	51.9 ± 11.9	39.5 ± 7.3
NoEx2	149 ± 31	0.88 ± 0.04	11.29 ± 2.23	40.5 ± 10.2	49.0 ± 12.0	49.1 ± 12.9	42.6 ± 6.9
ICC ²	0.91	0.59	0.73	0.71	0.86	0.89	0.63
CV ³ (%)	6.6	3.3	12.6	14.7	7.8	8.3	10.9
MDC ⁴	26.7	0.07	4.81	15.4	9.3	11.0	12.3

¹Mean ± SD; ²Intraclass Correlation Coefficient; ³Coefficient of Variation; ⁴Minimal Detectable Change;

* $P < 0.01$ vs NoEx2; RER: Respiratory Exchange Ratio; EI: Energy Intake

2.4.8 Correlations

Ratings of hunger and motivation to eat were significantly correlated ($r = 0.92$; $P < 0.0001$) as well as ratings of fullness and satiety ($r = 0.93$; $P < 0.0001$). Further, ratings of hunger were negatively correlated to fullness ($r = -0.81$; $P < 0.0001$). Also, there were significant correlations ($P \leq 0.01$) between ratings of hunger and insulin ($r = -0.37$), hunger and PP ($r = -0.48$), fullness and insulin ($r = 0.60$), fullness and PP ($r = 0.59$), satiety and insulin ($r = 0.52$), satiety and PP ($r = 0.55$), motivation to eat and GLP-1 ($r = -0.32$), motivation to eat and insulin ($r = -0.44$), and motivation to eat and PP ($r = -0.49$).

Meal energy intake was significantly correlated ($P < 0.0001$) to pre-meal ratings of hunger ($r = 0.48$) and motivation to eat ($r = 0.45$), and post-meal ratings of fullness ($r = 0.55$), and satiety ($r = 0.54$). Additionally, there was a significant correlation ($P < 0.0001$) between meal energy intake and the pre- to post-meal difference in ratings of hunger ($r = 0.51$), fullness ($r = 0.50$), satiety ($r = 0.44$) and motivation to eat ($r = 0.51$). There were no significant correlations between energy intake and pre- or post-meal concentrations of the appetite-related peptides. Meal energy intake was significantly, albeit weakly, correlated to pre-meal $\dot{V}O_2$ ($r = 0.35$; $P < 0.0001$) and post-meal $\dot{V}O_2$ ($r = 0.28$; $P = 0.005$). Finally, there was a significant correlation between 34-h energy intake and expenditure ($r = 0.59$; $P < 0.02$).

2.5 Discussion

The objectives of this study were to determine the effects of acute SIE on several measures of energy status, appetite, and substrate oxidation over 2 consecutive days in an attempt to assess the mechanisms responsible for the fat loss associated with SIE training. In addition, the reliability of these measures over 2 consecutive non-exercise days was assessed.

SIE induced immediate and short-lived (<1 h) post-exercise suppression of appetite and increase in PYY. However, AUC for hunger and motivation to eat were greater during SIEx, without affecting total energy intake. Total $\dot{V}O_2$ was greater during SIEx, inducing

a significantly lower energy status suggesting that this could account for at least part of fat loss observed with SIE training. The ICC of our measures ranged from 0.59-0.92.

2.5.1 Energy Intake

Despite the occurrence of exercise-induced anorexia and increase in PYY immediately after SIE, we observed no significant changes in energy intake 1 h later. Recent studies on SIE (six 30-s Wingate tests) and high intensity interval exercise (ten 4-min cycling bouts at 85-90% $\dot{V}O_{2max}$) in young healthy men also found no impact on energy intake (Deighton, Barry et al., 2013; Deighton, Karra et al., 2013). In contrast, Sim et al. (2013) observed a lower energy intake in overweight sedentary men following two distinct 30-min interval protocols consisting of 60-s intervals at 100% $\dot{V}O_{2peak}$ and 15-s intervals at 170% $\dot{V}O_{2peak}$ compared to rest. Additionally, food records indicated that 48-h energy intake was lower in the 15-s interval protocol compared to rest and continuous moderate intensity exercise (Sim et al., 2013).

Furthermore, the individuals who vomited ate approximately 630 kJ less at lunch than those who did not, so nausea may be a contributing factor. However, without impeding sprint capacity, feelings of nausea and vomiting are commonly experienced in the first session of SIE, and tend to subside as training progresses. Moreover, in the present study, we offered an *ad libitum* instead of a standardized breakfast to assess pre-exercise energy intake, as there was evidence for a decrease in energy intake in anticipation of the intense exercise to be performed (Westerterp-Plantenga et al., 1997). No changes in intake were observed before SIE; but the unfamiliarity of the exercise and/or the nature of the buffet-type meal likely influenced the eating behaviour of the participants to overconsume because the food was free. The participants, especially those who vomited, may have eaten less before the exercise if they had been given a few practice sessions prior to the study. Some support for this idea was observed following the study, as one of the participants who had performed SIE previously and vomited during exercise still ate a larger than normal breakfast, and most participants reported they would eat a very light meal at least 2 h, or not eat at all, prior to future SIE. Further, an overconsumption was probable as energy intake of the test meals was significantly greater than the food

records. In addition, the differences in macronutrient distribution between the test meals and the participant's usual diet may have influenced food intake. However, these comparisons may be incorrect due to the frequent underreporting seen in food records (Bingham, 1987). This reinforces the challenges in recording energy intake without bias in an experimental setting (see below).

2.5.2 Appetite Ratings

The observed immediate suppression of hunger and motivation to eat following SIE supports the literature on the effects of high intensity exercise on appetite (Bilski, Teleglow, Zahradnik-Bilska, Dembinski, & Warzecha, 2009). However, this exercise-induced anorexia was short-lived with SIE, as ratings did not differ from control values at 1 h post-exercise, and in contrast to our hypothesis, both hunger and motivation to eat increased in the hours following SIE, leading to greater 34-h AUC. These results corroborate a recent study investigating the appetite response to SIE, where a post-exercise suppression of appetite was followed by an AUC for hunger and prospective food consumption that tended to be greatest with SIE compared to continuous endurance exercise. By itself, this provides conflicting evidence regarding greater fat loss with SIE vs endurance exercise (Deighton, Barry et al., 2013). However, it remains unknown if this increase in appetite following SIE is unique to acute SIE or if it leads to increases in food intake with SIE training. While energy intake was not affected in this study, the significant correlations observed between meal energy intake and pre-meal/post-meal appetite ratings provide evidence of the validity of repeated subjective appetite ratings over several meals and days.

2.5.3 Appetite-Related Peptides

Prior to our study, we hypothesized that SIE would increase PP, PYY and GLP-1, as observed in several studies reviewed in a recent meta-analysis on various modalities and intensities of exercise (Schubert, Sabapathy et al., 2013). However, only PYY achieved a significant increase immediately after SIE. We are the first to show this, as previously observed increases in PYY and PYY₃₋₃₆ with SIE or high intensity interval exercise failed

to achieve significance compared to rest (Deighton, Barry et al., 2013; Deighton, Karra et al., 2013; Sim et al., 2013). Although all our participants had an elevation in PYY pre- to post-exercise, the greatest increases came from 2 individuals who vomited during and after the exercise sessions (~300 and 500%; average 170%). The increase in PYY coincided with the decrease in appetite, however these were not correlated significantly throughout the trials. While we measured total PYY instead of the more potent form PYY₃₋₃₆, both are highly correlated ($r = 0.98$) (Tsilchorozidou, Batterham, & Conway, 2008); thus, we suspect the changes we observed in total PYY reflect those of PYY₃₋₃₆. Following SIE, PP increased in all but one participant and like PYY, the highest increases were from 2 individuals who vomited (~230 and 330%; average ~100%). These results may provide evidence of a sympathetic nervous system stimulation of PYY and PP release (Zhang et al., 1993), corresponding with feelings of nausea and vomiting, observed with maximal intensity exercise and SIE previously (Holmqvist et al., 1986; Williams et al., 2013). The changes observed in appetite-related peptides with exercise and their contribution to appetite, food intake and fat loss are still in their infancy. Consequently, until the mechanisms of action of these peptides, individual variability and impact of exercise are better understood, subjective appetite ratings may provide better overall indicators of appetite because presumably they reflect the sum of all these responses.

2.5.4 Oxygen Consumption and Energy Expenditure

$\dot{V}O_2$ remained elevated significantly over the first 2 h post-exercise, much longer than the 30-45 min observed in recent SIE studies (Chan & Burns, 2013; Williams et al., 2013). The tendency for an elevated $\dot{V}O_2$ at 17:45 (6.75 h post-exercise) corroborates the prolonged elevation in $\dot{V}O_2$ up to 8 h following SIE or similar supramaximal exercise documented previously (Hazell et al., 2012; Laforgia et al., 1997). Further, the 2 outliers with negative calculated EPOC were eliminated due to the consistently elevated resting $\dot{V}O_2$, likely due to movement or restlessness during the measures. Nonetheless, the EPOC greatly contributed to the exercise-induced energy expenditure. While we estimated $\dot{V}O_2$ during SIE from a previous study with different participants (Hazell et al., 2012), the consistent responses to SIE suggests the current study's participants of comparable

exercise capacity and training history would respond with similar $\dot{V}O_2$ values. Therefore, despite not having a significant effect on energy intake, acute SIE led to a 24-h energy status of approximately 1,750 kJ lower than rest, mediated by the exercise-induced energy expenditure. This provides a partial explanation for the fat loss induced with SIE training, suggesting adaptations over a training program likely account for the remaining decrease in energy status.

2.5.5 Substrate Oxidation

The rapid decrease in RER in the first hour post-exercise seen in this study is in agreement with past SIE studies (Chan & Burns, 2013; Hazell et al., 2012; Williams et al., 2013), indicative of the transient increase in CO_2 retention linked with lactate buffering during exercise. This retention, assessed by blood bicarbonate concentrations and bicarbonate tracers, tends to subside within 30-60 min following submaximal exercise (Henderson et al., 2007; Phelain et al., 1997). Therefore, the lower RER and greater rates of fat oxidation observed at 13:15 (1.5 h post-exercise and immediately post-prandial) and 16:45 (6 h post-exercise and 4 h post-prandial) appear to represent increased fat utilization, assuming any contribution from protein was minimal and consistent. Although Laforgia et al. (1997) demonstrated lower RER 8 h post-exercise with supramaximal interval running (20×1 -min at $105\% \dot{V}O_{2max}$), we are the first to demonstrate a significant elevation in fat oxidation 6 h after SIE, which is an important distinction because our exercise protocol was much shorter and more intense. This provides evidence for the fat loss induced by this exercise modality, possibly driven by training adaptations promoting further fat oxidation. The increase in fat oxidation following SIE could be mediated by an elevation in catecholamines due to sympathetic activation lingering into recovery (Greer et al., 1998; Williams et al., 2013). Another possible mechanism behind elevated post-exercise fat oxidation could be a sparing of carbohydrate oxidation to facilitate muscle glycogen repletion (Kiens & Richter, 1998; Malatesta, Werlen, Bulfaro, Cheneviere, & Borrani, 2009). Interestingly, a significant glycogen depletion approximating 40% has been documented following four 30-s maximal intensity cycling bouts (McCartney et al., 1986). These authors also observed muscle glycogen decreased only during the first 2 bouts and glycerol concentration

increased linearly over the 4 bouts, suggesting a greater reliance on triglycerides for ATP regeneration. Thus, greater utilization of fat during and following exercise likely plays a role in SIE-induced fat loss.

2.5.6 Acute SIE vs SIE training

As mentioned previously, in the hours following SIE, there are increases in post-exercise $\dot{V}O_2$, lower RER and elevations in rates of fat oxidation, all consistent with the fat loss previously associated with this training modality. We did not observe a greater $\dot{V}O_2$ the morning following acute SIE, in agreement with previous studies, suggesting any metabolic effect does not extend past 24 h (Hazell et al., 2012; MacPherson et al., 2011; Whyte et al., 2010). Moreover, our study corroborates our earlier results where RER remained unchanged the morning following acute SIE in healthy weight men (Hazell et al., 2012). However, in overweight and obese men, responses may differ because RER was lower and rates of fat oxidation were still greater 24 h following both one session and 2 weeks of SIE (Whyte et al., 2010; Whyte et al., 2013). Regardless, metabolic adaptations over the course of a SIE training program need to be determined as they may elevate fat utilization even more, both during and following exercise.

In contrast to the favourable effects on metabolism, we and others (Deighton, Barry et al., 2013) have shown that acute SIE does not affect energy intake and may even increase in appetite later in the day in healthy men; however, this was not seen in overweight men, where energy intake was decreased over 48 h (Sim et al., 2013). While no studies have been conducted on the dietary and appetite adaptations during 6 weeks of SIE, there is some information about other types of exercise programs. Interestingly, after a 6-wk moderate intensity exercise program (30-45 min, 4 sessions/wk), sedentary healthy weight individuals consumed significantly less at a buffet-meal following a high-energy milkshake (~2500 kJ) compared to a low-energy milkshake (~1000 kJ) of similar properties, indicating improvements in appetite control (Martins, Truby, & Morgan, 2007). In addition to its potential impact on eating behaviour, SIE training may also lead to anticipatory pre-exercise dietary adjustments to minimize feelings of discomfort.

Further, because of its relatively low energy expenditure, SIE training may not induce compensatory increases in energy intake seen with long-term continuous aerobic exercise training (Blundell et al., 2003; Caudwell et al., 2009). In terms of subjective appetite, the fat loss associated with SIE training suggests the increase in appetite following acute SIE is reduced over time and does not affect energy intake, but this remains to be determined.

2.5.7 Reliability

We achieved an ICC greater than 0.8 in most of our measures taken over consecutive days: $\dot{V}O_2$, fullness and satiety were all between 0.8 and 0.94; 10-h energy intake, hunger and motivation to eat, between 0.63 and 0.79; and RER, 0.59. ICC in the range of 0.82 to 0.97 and CV between 8.9 to 14.5% have been documented previously for energy intake over single meals separated by approximately 1 wk (Allirot et al., 2012; Arvaniti et al., 2000; Gregersen et al., 2008; Laan et al., 2010; Nair et al., 2009). Our CV for energy intake of 12.6% is within the range of previous studies. Likely, the ICC of 0.73 we obtained combining 3 meals over 2 days falls below that range because of our lower sample size ($n = 8$ vs ~ 15) and our low reproducibility of energy intake at lunch and dinner (data not shown). This may be an indicator of the day-to-day and meal-to-meal variability in food consumption and could contribute to the lack of significant differences in energy intake seen in most acute exercise studies (Bilski et al., 2009; Melzer, Kayser, Saris, & Pichard, 2005; Schubert, Desbrow et al., 2013). With respect to appetite ratings, our CV between 8 and 15% are within the range of those documented previously (Flint et al., 2000).

The significantly greater RER for NoEx2 compared to NoEx1 impacted reproducibility negatively; however, this result does not affect the treatment comparison between SIEx1 and NoEx1. The consumption of high carbohydrate meals leads to significant increases in RER, indicative of greater carbohydrate oxidation (Westerterp, 1993). Therefore, the greater RER on NoEx2 likely reflected the cumulative effect of the higher carbohydrate test meals we provided relative to the participants' usual diet ($\sim 65\%$ vs $\sim 40\%$ energy intake). These results reflect the importance of controlling for diet composition over the days prior to testing and provide evidence of the limitation of free selection buffet-type

meals in the assessment of substrate oxidation, although previous reports have not shown this consistently (Arvaniti et al., 2000).

2.5.8 Limitations

This study was conducted in a controlled laboratory setting where buffet-type meals were provided during test days and participants remained sedentary in the laboratory between measures. Thus, the considerable 24-h positive energy status of ~6 MJ reflected a greater food consumption than normal (perhaps in part because the food was good, available, and free) and a lower energy expenditure than usual (due to being restricted to the lab setting). This is likely not representative of typical SIE and may have influenced the metabolic and hormonal responses observed. Moreover, the challenge encountered in all studies that measure energy intake following exercise is doing so accurately. Food records, a classic method to monitor habitual dietary intake, often underreport energy intake in the range of 20 to 50% that can reach up to 90% (Bingham, 1987), while buffet meals in experimental settings can lead to overconsumption (Allirot et al., 2012; Arvaniti et al., 2000). Consequently, practical and unbiased new approaches are necessary to assess the possible adaptations in energy intake occurring during SIE or any type of exercise training.

Chapter 3

3 Summary, Future Directions and Conclusion

In the current study, we observed that acute SIE suppresses appetite briefly, but subsequently results in greater feelings of hunger and motivation to eat. Despite its effects on appetite and PYY, SIE did not affect total energy intake. We also determined that the reliability of measures for energy intake, subjective appetite ratings, appetite-related peptides, oxygen consumption, and substrate oxidation over 2 consecutive no exercise days is acceptable.

The observations of the current study were for a single bout of SIE. Further studies are needed to illustrate the metabolic, dietary and hormonal adaptations occurring throughout training and their contribution towards the fat loss associated with SIE. The acylated ghrelin and blood glucose response to SIE would be interesting to pursue. Measuring post-exercise $\dot{V}O_2$ and RER responses on a weekly basis and assessing energy intake on each sprint day over the course of 4-6 weeks of training could illustrate those adaptations. In addition, methods to measure habitual energy intake accurately in actual living conditions need to be developed and validated in order to remove the confounding variable of the buffet-meal in a laboratory setting. Recently, Alliot et al. (2012) documented good reproducibility for several measures of food consumption in an experimental restaurant fitted with hidden video cameras designed to replicate a natural eating environment. Although this experimental setting may not be achievable in all studies, an adaptation of this method in university cafeterias or research kitchens may provide a feasible alternative.

We conclude that following acute SIE, energy expenditure is increased but energy intake remains similar, suggesting that increases in energy expenditure and/or alterations in fat oxidation most likely explain the fat loss associated with this training modality. However, this assumes the response to acute SIE reflects the response to SIE training, which may not be the case.

References

- Adrian, T. E., Bloom, S. R., Bryant, M. G., Polak, J. M., Heitz, P. H., & Barnes, A. J. (1976). Distribution and release of human pancreatic polypeptide. *Gut*, *17*(12), 940-944.
- Adrian, T. E., Ferri, G. L., Bacarese-Hamilton, A. J., Fuesl, H. S., Polak, J. M., & Bloom, S. R. (1985). Human distribution and release of a putative new gut hormone, peptide YY. *Gastroenterology*, *89*(5), 1070-1077.
- Adrian, T. E., Savage, A. P., Sagor, G. R., Allen, J. M., Bacarese-Hamilton, A. J., Tatemoto, K., . . . Bloom, S. R. (1985). Effect of peptide YY on gastric, pancreatic, and biliary function in humans. *Gastroenterology*, *89*(3), 494-499.
- Allen, J. M., Fitzpatrick, M. L., Yeats, J. C., Darcy, K., Adrian, T. E., & Bloom, S. R. (1984). Effects of peptide YY and neuropeptide Y on gastric emptying in man. *Digestion*, *30*(4), 255-262.
- Allirot, X., Saulais, L., Disse, E., Roth, H., Cazal, C., & Laville, M. (2012). Validation of a buffet meal design in an experimental restaurant. *Appetite*, *58*(3), 889-897. doi: 10.1016/j.appet.2012.02.011
- Arosio, M., Ronchi, C. L., Gebbia, C., Cappiello, V., Beck-Peccoz, P., & Peracchi, M. (2003). Stimulatory effects of ghrelin on circulating somatostatin and pancreatic polypeptide levels. *The Journal of Clinical Endocrinology and Metabolism*, *88*(2), 701-704.
- Arvaniti, K., Richard, D., & Tremblay, A. (2000). Reproducibility of energy and macronutrient intake and related substrate oxidation rates in a buffet-type meal. *The British Journal of Nutrition*, *83*(5), 489-495.
- Babraj, J. A., Vollaard, N. B., Keast, C., Guppy, F. M., Cottrell, G., & Timmons, J. A. (2009). Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. *BMC Endocrine Disorders*, *9*, 3-6823-9-3. doi: 10.1186/1472-6823-9-3
- Bagdade, J. D., Bierman, E. L., & Porte, D., Jr. (1967). The significance of basal insulin levels in the evaluation of the insulin response to glucose in diabetic and nondiabetic subjects. *The Journal of Clinical Investigation*, *46*(10), 1549-1557. doi: 10.1172/JCI105646
- Bahr, R., Gronnerod, O., & Sejersted, O. M. (1992). Effect of supramaximal exercise on excess postexercise O₂ consumption. *Medicine and Science in Sports and Exercise*, *24*(1), 66-71.

- Balaguera-Cortes, L., Wallman, K. E., Fairchild, T. J., & Guelfi, K. J. (2011). Energy intake and appetite-related hormones following acute aerobic and resistance exercise. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, 36(6), 958-966. doi: 10.1139/h11-121
- Batterham, R. L., Cowley, M. A., Small, C. J., Herzog, H., Cohen, M. A., Dakin, C. L., . . . Bloom, S. R. (2002). Gut hormone PYY(3-36) physiologically inhibits food intake. *Nature*, 418(6898), 650-654. doi: 10.1038/nature02666
- Batterham, R. L., Le Roux, C. W., Cohen, M. A., Park, A. J., Ellis, S. M., Patterson, M., . . . Bloom, S. R. (2003). Pancreatic polypeptide reduces appetite and food intake in humans. *The Journal of Clinical Endocrinology and Metabolism*, 88(8), 3989-3992.
- Becker, G. F., Macedo, R. C., Cunha Gdos, S., Martins, J. B., Laitano, O., & Reischak-Oliveira, A. (2012). Combined effects of aerobic exercise and high-carbohydrate meal on plasma acylated ghrelin and levels of hunger. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, 37(1), 184-192. doi: 10.1139/h11-149
- Bilski, J., Teleglow, A., Zahradnik-Bilska, J., Dembinski, A., & Warzecha, Z. (2009). Effects of exercise on appetite and food intake regulation. *Medicina Sportiva*, 13(2), 82-94.
- Bingham, S. A. (1987). The dietary assessment of individuals; methods, accuracy, new techniques and recommendations. *Nutrition Abstracts and Reviews (Series A)*, 57(10), 705-742.
- Blundell, J. E., de Graaf, C., Hulshof, T., Jebb, S., Livingstone, B., Luch, A., . . . Westerterp, M. (2010). Appetite control: Methodological aspects of the evaluation of foods. *Obesity Reviews*, 11(3), 251-270. doi: 10.1111/j.1467-789X.2010.00714.x
- Blundell, J. E., & King, N. A. (1999). Physical activity and regulation of food intake: Current evidence. *Medicine and Science in Sports and Exercise*, 31(11 Suppl), S573-83.
- Blundell, J. E., & MacDiarmid, J. I. (1997). Fat as a risk factor for overconsumption: Satiation, satiety, and patterns of eating. *Journal of the American Dietetic Association*, 97(7 Suppl), S63-9.
- Blundell, J. E., Stubbs, R. J., Hughes, D. A., Whybrow, S., & King, N. A. (2003). Cross talk between physical activity and appetite control: Does physical activity stimulate appetite? *The Proceedings of the Nutrition Society*, 62(3), 651-661. doi: 10.1079/PNS2003286
- Boutcher, S. H. (2011). High-intensity intermittent exercise and fat loss. *Journal of Obesity*, 2011, 868305. doi: 10.1155/2011/868305

- Boutcher, S. H., & Dunn, S. L. (2009). Factors that may impede the weight loss response to exercise-based interventions. *Obesity Reviews*, *10*(6), 671-680. doi: 10.1111/j.1467-789X.2009.00621.x
- Broom, D. R., Batterham, R. L., King, J. A., & Stensel, D. J. (2009). Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin, and peptide YY in healthy males. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, *296*(1), R29-35. doi: 10.1152/ajpregu.90706.2008
- Broom, D. R., Stensel, D. J., Bishop, N. C., Burns, S. F., & Miyashita, M. (2007). Exercise-induced suppression of acylated ghrelin in humans. *Journal of Applied Physiology*, *102*(6), 2165-2171. doi: 10.1152/jappphysiol.00759.2006
- Burgomaster, K. A., Heigenhauser, G. J., & Gibala, M. J. (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *Journal of Applied Physiology*, *100*(6), 2041-2047. doi: 10.1152/jappphysiol.01220.2005
- Burgomaster, K. A., Howarth, K. R., Phillips, S. M., Rakobowchuk, M., MacDonald, M. J., McGee, S. L., & Gibala, M. J. (2008). Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *The Journal of Physiology*, *586*(1), 151-160. doi: 10.1113/jphysiol.2007.142109
- Burgomaster, K. A., Hughes, S. C., Heigenhauser, G. J., Bradwell, S. N., & Gibala, M. J. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology*, *98*(6), 1985-1990. doi: 10.1152/jappphysiol.01095.2004
- Caudwell, P., Hopkins, M., King, N. A., Stubbs, R. J., & Blundell, J. E. (2009). Exercise alone is not enough: Weight loss also needs a healthy (mediterranean) diet? *Public Health Nutrition*, *12*(9A), 1663-1666. doi: 10.1017/S1368980009990528
- Chan, H. H., & Burns, S. F. (2013). Oxygen consumption, substrate oxidation, and blood pressure following sprint interval exercise. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, *38*(2), 182-187. doi: 10.1139/apnm-2012-0136
- Cheng, M. H., Bushnell, D., Cannon, D. T., & Kern, M. (2009). Appetite regulation via exercise prior or subsequent to high-fat meal consumption. *Appetite*, *52*(1), 193-198. doi: 10.1016/j.appet.2008.09.015
- Chin-Chance, C., Polonsky, K. S., & Schoeller, D. A. (2000). Twenty-four-hour leptin levels respond to cumulative short-term energy imbalance and predict subsequent intake. *The Journal of Clinical Endocrinology and Metabolism*, *85*(8), 2685-2691.

- Choi, J. S., Wexner, S. D., Nam, Y. S., Mavrantonis, C., Salum, M. R., Yamaguchi, T., . . . Yu, C. F. (2000). Intraobserver and interobserver measurements of the anorectal angle and perineal descent in defecography. *Diseases of the Colon and Rectum*, *43*(8), 1121-1126.
- Christofides, N. D., Sarson, D. L., Albuquerque, R. H., Adrian, T. E., Ghatei, M. A., Modlin, I. M., & Bloom, S. R. (1979). Release of gastrointestinal hormones following an oral water load. *Experientia*, *35*(11), 1521-1523.
- Cicioni-Kolsky, D., Lorenzen, C., Williams, M. D., & Kemp, J. G. (2013). Endurance and sprint benefits of high-intensity and supramaximal interval training. *European Journal of Sport Science*, *13*(3), 304-311. doi: 10.1080/17461391.2011.606844
- Cummings, D. E., Purnell, J. Q., Frayo, R. S., Schmidova, K., Wisse, B. E., & Weigle, D. S. (2001). A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes*, *50*(8), 1714-1719.
- Cummings, D. E., Weigle, D. S., Frayo, R. S., Breen, P. A., Ma, M. K., Dellinger, E. P., & Purnell, J. Q. (2002). Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *The New England Journal of Medicine*, *346*(21), 1623-1630. doi: 10.1056/NEJMoa012908
- Dall, R., Kanaley, J., Hansen, T. K., Moller, N., Christiansen, J. S., Hosoda, H., . . . Jorgensen, J. O. (2002). Plasma ghrelin levels during exercise in healthy subjects and in growth hormone-deficient patients. *European Journal of Endocrinology*, *147*(1), 65-70.
- Deighton, K., Barry, R., Connon, C. E., & Stensel, D. J. (2013). Appetite, gut hormone and energy intake responses to low volume sprint interval and traditional endurance exercise. *European Journal of Applied Physiology*, *113*(5), 1147-1156. doi: 10.1007/s00421-012-2535-1
- Deighton, K., Karra, E., Batterham, R. L., & Stensel, D. J. (2013). Appetite, energy intake, and PYY3-36 responses to energy-matched continuous exercise and submaximal high-intensity exercise. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, *38*(9), 947-952. doi: 10.1139/apnm-2012-0484
- Elliott, R. M., Morgan, L. M., Tredger, J. A., Deacon, S., Wright, J., & Marks, V. (1993). Glucagon-like peptide-1 (7-36)amide and glucose-dependent insulinotropic polypeptide secretion in response to nutrient ingestion in man: Acute post-prandial and 24-h secretion patterns. *The Journal of Endocrinology*, *138*(1), 159-166.
- Erdmann, J., Tahbaz, R., Lippl, F., Wagenpfeil, S., & Schusdziarra, V. (2007). Plasma ghrelin levels during exercise - effects of intensity and duration. *Regulatory Peptides*, *143*(1-3), 127-135. doi: 10.1016/j.regpep.2007.05.002

- Flint, A., Raben, A., Astrup, A., & Holst, J. J. (1998). Glucagon-like peptide 1 promotes satiety and suppresses energy intake in humans. *The Journal of Clinical Investigation*, *101*(3), 515-520. doi: 10.1172/JCI990
- Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity and Related Metabolic Disorders*, *24*(1), 38-48.
- Gibala, M. J., Little, J. P., MacDonald, M. J., & Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *The Journal of Physiology*, *590*(Pt 5), 1077-1084. doi: 10.1113/jphysiol.2011.224725
- Gibala, M. J., Little, J. P., van Essen, M., Wilkin, G. P., Burgomaster, K. A., Safdar, A., . . . Tarnopolsky, M. A. (2006). Short-term sprint interval versus traditional endurance training: Similar initial adaptations in human skeletal muscle and exercise performance. *The Journal of Physiology*, *575*(Pt 3), 901-911. doi: 10.1113/jphysiol.2006.112094
- Gillen, J. B., Percival, M. E., Ludzki, A., Tarnopolsky, M. A., & Gibala, M. J. (2013). Interval training in the fed or fasted state improves body composition and muscle oxidative capacity in overweight women. *Obesity*, doi: 10.1002/oby.20379; 10.1002/oby.20379
- Gingerich, R. L., Hickson, R. C., Hagberg, J. M., & Winder, W. W. (1979). Effect of endurance exercise training on plasma pancreatic polypeptide concentration during exercise. *Metabolism: Clinical and Experimental*, *28*(12), 1179-1182.
- Grandt, D., Schimiczek, M., Beglinger, C., Layer, P., Goebell, H., Eysselein, V. E., & Reeve, J. R., Jr. (1994). Two molecular forms of peptide YY (PYY) are abundant in human blood: Characterization of a radioimmunoassay recognizing PYY 1-36 and PYY 3-36. *Regulatory Peptides*, *51*(2), 151-159.
- Greenberg, G. R., Marliss, E. B., & Zinman, B. (1986). Effect of exercise on the pancreatic polypeptide response to food in man. *Hormone and Metabolic Research = Hormon- Und Stoffwechselforschung = Hormones Et Metabolisme*, *18*(3), 194-196. doi: 10.1055/s-2007-1012268
- Greer, F., McLean, C., & Graham, T. E. (1998). Caffeine, performance, and metabolism during repeated wingate exercise tests. *Journal of Applied Physiology*, *85*(4), 1502-1508.
- Gregersen, N. T., Flint, A., Bitz, C., Blundell, J. E., Raben, A., & Astrup, A. (2008). Reproducibility and power of ad libitum energy intake assessed by repeated single meals. *The American Journal of Clinical Nutrition*, *87*(5), 1277-1281.

- Hagobian, T. A., & Braun, B. (2006). Interactions between energy surplus and short-term exercise on glucose and insulin responses in healthy people with induced, mild insulin insensitivity. *Metabolism: Clinical and Experimental*, 55(3), 402-408. doi: 10.1016/j.metabol.2005.09.017
- Hagobian, T. A., Yamashiro, M., Hinkel-Lipsker, J., Streder, K., Evero, N., & Hackney, T. (2013). Effects of acute exercise on appetite hormones and ad libitum energy intake in men and women. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, 38(1), 66-72. doi: 10.1139/apnm-2012-0104
- Havel, P. J. (2001). Peripheral signals conveying metabolic information to the brain: Short-term and long-term regulation of food intake and energy homeostasis. *Exp.Biol.Med.*, 226(11), 963-977.
- Hazell, T. J., Macpherson, R. E., Gravelle, B. M., & Lemon, P. W. (2010). 10 or 30-S sprint interval training bouts enhance both aerobic and anaerobic performance. *European Journal of Applied Physiology*, 110(1), 153-160. doi: 10.1007/s00421-010-1474-y; 10.1007/s00421-010-1474-y
- Hazell, T. J., Olver, T. D., Hamilton, C. D., & Lemon, P. W. (2012). Two minutes of sprint-interval exercise elicits 24-hr oxygen consumption similar to that of 30 min of continuous endurance exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, 22(4), 276-283.
- Hazelwood, R. L. (1993). The pancreatic polypeptide (PP-fold) family: Gastrointestinal, vascular, and feeding behavioral implications. *Proceedings of the Society for Experimental Biology and Medicine*, 202(1), 44-63.
- Health Canada. (2010). Canadian nutrient file. Retrieved from <http://webprod3.hc-sc.gc.ca/cnf-fce/index-eng.jsp>
- Henderson, G. C., Fattor, J. A., Horning, M. A., Faghihnia, N., Luke-Zeitoun, M., & Brooks, G. A. (2007). Retention of intravenously infused [13C]bicarbonate is transiently increased during recovery from hard exercise. *Journal of Applied Physiology*, 103(5), 1604-1612. doi: 10.1152/jappphysiol.00309.2007
- Heydari, M., Freund, J., & Boutcher, S. H. (2012). The effect of high-intensity intermittent exercise on body composition of overweight young males. *Journal of Obesity*, 2012, 480467. doi: 10.1155/2012/480467
- Hilsted, J., Galbo, H., Sonne, B., Schwartz, T., Fahrenkrug, J., de Muckadell, O. B., . . . Tronier, B. (1980). Gastroenteropancreatic hormonal changes during exercise. *The American Journal of Physiology*, 239(3), G136-40.

- Holloway, G. P., Bezaire, V., Heigenhauser, G. J., Tandon, N. N., Glatz, J. F., Luiken, J. J., . . . Spriet, L. L. (2006). Mitochondrial long chain fatty acid oxidation, fatty acid translocase/CD36 content and carnitine palmitoyltransferase I activity in human skeletal muscle during aerobic exercise. *The Journal of Physiology*, *571*(Pt 1), 201-210. doi: 10.1113/jphysiol.2005.102178
- Holmqvist, N., Secher, N. H., Sander-Jensen, K., Knigge, U., Warberg, J., & Schwartz, T. (1986). Sympathoadrenal and parasympathetic responses to exercise. *Journal of Sports Sciences*, *4*(2), 123-128. doi: 10.1080/02640418608732108
- Huda, M. S., Wilding, J. P., & Pinkney, J. H. (2006). Gut peptides and the regulation of appetite. *Obesity Reviews*, *7*(2), 163-182. doi: 10.1111/j.1467-789X.2006.00245.x
- Imbeault, P., Saint-Pierre, S., Almeras, N., & Tremblay, A. (1997). Acute effects of exercise on energy intake and feeding behaviour. *The British Journal of Nutrition*, *77*(4), 511-521.
- James, D. E., Brown, R., Navarro, J., & Pilch, P. F. (1988). Insulin-regulatable tissues express a unique insulin-sensitive glucose transport protein. *Nature*, *333*(6169), 183-185. doi: 10.1038/333183a0
- Jurimae, J., Jurimae, T., & Purge, P. (2007). Plasma ghrelin is altered after maximal exercise in elite male rowers. *Exp.Biol.Med.*, *232*(7), 904-909.
- Kawano, H., Mineta, M., Asaka, M., Miyashita, M., Numao, S., Gando, Y., . . . Higuchi, M. (2013). Effects of different modes of exercise on appetite and appetite-regulating hormones. *Appetite*, *66*, 26-33. doi: 10.1016/j.appet.2013.01.017
- Kelly, P. J., Guelfi, K. J., Wallman, K. E., & Fairchild, T. J. (2012). Mild dehydration does not reduce postexercise appetite or energy intake. *Medicine and Science in Sports and Exercise*, *44*(3), 516-524. doi: 10.1249/MSS.0b013e318231c176
- Kiens, B., & Richter, E. A. (1998). Utilization of skeletal muscle triacylglycerol during postexercise recovery in humans. *The American Journal of Physiology*, *275*(2 Pt 1), E332-7.
- King, J. A., Miyashita, M., Wasse, L. K., & Stensel, D. J. (2010). Influence of prolonged treadmill running on appetite, energy intake and circulating concentrations of acylated ghrelin. *Appetite*, *54*(3), 492-498. doi: 10.1016/j.appet.2010.02.002
- King, J. A., Wasse, L. K., Broom, D. R., & Stensel, D. J. (2010). Influence of brisk walking on appetite, energy intake, and plasma acylated ghrelin. *Medicine and Science in Sports and Exercise*, *42*(3), 485-492. doi: 10.1249/MSS.0b013e3181ba10c4

- King, J. A., Wasse, L. K., Ewens, J., Crystallis, K., Emmanuel, J., Batterham, R. L., & Stensel, D. J. (2011). Differential acylated ghrelin, peptide YY3-36, appetite, and food intake responses to equivalent energy deficits created by exercise and food restriction. *The Journal of Clinical Endocrinology and Metabolism*, *96*(4), 1114-1121. doi: 10.1210/jc.2010-2735
- King, J. A., Wasse, L. K., & Stensel, D. J. (2011). The acute effects of swimming on appetite, food intake, and plasma acylated ghrelin. *Journal of Obesity*, *2011*, 10.1155/2011/351628.
- King, J. A., Wasse, L. K., & Stensel, D. J. (2013). Acute exercise increases feeding latency in healthy normal weight young males but does not alter energy intake. *Appetite*, *61*(1), 45-51. doi: 10.1016/j.appet.2012.10.018; 10.1016/j.appet.2012.10.018
- King, N. A., & Blundell, J. E. (1995). High-fat foods overcome the energy expenditure induced by high-intensity cycling or running. *Eur.J.Clin.Nutr.*, *49*(2), 114-123.
- King, N. A., Burley, V. J., & Blundell, J. E. (1994). Exercise-induced suppression of appetite: Effects on food intake and implications for energy balance. *Eur.J.Clin.Nutr.*, *48*(10), 715-724.
- King, N. A., Lluch, A., Stubbs, R. J., & Blundell, J. E. (1997). High dose exercise does not increase hunger or energy intake in free living males. *Eur.J.Clin.Nutr.*, *51*(7), 478-483.
- King, N. A., Snell, L., Smith, R. D., & Blundell, J. E. (1996). Effects of short-term exercise on appetite responses in unrestrained females. *Eur.J.Clin.Nutr.*, *50*(10), 663-667.
- Kissileff, H. R., Pi-Sunyer, F. X., Segal, K., Meltzer, S., & Foelsch, P. A. (1990). Acute effects of exercise on food intake in obese and nonobese women. *The American Journal of Clinical Nutrition*, *52*(2), 240-245.
- Kojima, M., & Kangawa, K. (2005). Ghrelin: Structure and function. *Physiological Reviews*, *85*(2), 495-522. doi: 10.1152/physrev.00012.2004
- Kraemer, R. R., Durand, R. J., Acevedo, E. O., Johnson, L. G., Kraemer, G. R., Hebert, E. P., & Castracane, V. D. (2004). Rigorous running increases growth hormone and insulin-like growth factor-I without altering ghrelin. *Exp.Biol.Med.*, *229*(3), 240-246.
- Laan, D. J., Leidy, H. J., Lim, E., & Campbell, W. W. (2010). Effects and reproducibility of aerobic and resistance exercise on appetite and energy intake in young, physically active adults. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, *35*(6), 842-847. doi: 10.1139/H10-072

- Laforgia, J., Withers, R. T., Shipp, N. J., & Gore, C. J. (1997). Comparison of energy expenditure elevations after submaximal and supramaximal running. *Journal of Applied Physiology*, 82(2), 661-666.
- Larson-Meyer, D. E., Palm, S., Bansal, A., Austin, K. J., Hart, A. M., & Alexander, B. M. (2012). Influence of running and walking on hormonal regulators of appetite in women. *Journal of Obesity*, 2012(730409) doi: 10.1155/2012/730409
- MacDougall, J. D., Hicks, A. L., MacDonald, J. R., McKelvie, R. S., Green, H. J., & Smith, K. M. (1998). Muscle performance and enzymatic adaptations to sprint interval training. *Journal of Applied Physiology*, 84(6), 2138-2142.
- Mackelvie, K. J., Meneilly, G. S., Elahi, D., Wong, A. C., Barr, S. I., & Chanoine, J. P. (2007). Regulation of appetite in lean and obese adolescents after exercise: Role of acylated and desacyl ghrelin. *The Journal of Clinical Endocrinology and Metabolism*, 92(2), 648-654. doi: 10.1210/jc.2006-1028
- MacPherson, R. E., Hazell, T. J., Olver, T. D., Paterson, D. H., & Lemon, P. W. (2011). Run sprint interval training improves aerobic performance but not maximal cardiac output. *Medicine and Science in Sports and Exercise*, 43(1), 115-122. doi: 10.1249/MSS.0b013e3181e5eacd
- Malatesta, D., Werlen, C., Bulfaro, S., Cheneviere, X., & Borrani, F. (2009). Effect of high-intensity interval exercise on lipid oxidation during postexercise recovery. *Medicine and Science in Sports and Exercise*, 41(2), 364-374. doi: 10.1249/MSS.0b013e3181857edo
- Martins, C., Kulseng, B., King, N. A., Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *The Journal of Clinical Endocrinology and Metabolism*, 95(4), 1609-1616. doi: 10.1210/jc.2009-2082
- Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *The Journal of Endocrinology*, 193(2), 251-258. doi: 10.1677/JOE-06-0030
- Martins, C., Truby, H., & Morgan, L. M. (2007). Short-term appetite control in response to a 6-week exercise programme in sedentary volunteers. *The British Journal of Nutrition*, 98(4), 834-842. doi: 10.1017/S000711450774922X
- Masuda, Y., Tanaka, T., Inomata, N., Ohnuma, N., Tanaka, S., Itoh, Z., . . . Kangawa, K. (2000). Ghrelin stimulates gastric acid secretion and motility in rats. *Biochemical and Biophysical Research Communications*, 276(3), 905-908. doi: 10.1006/bbrc.2000.3568

- Mattes, R. D., Hollis, J., Hayes, D., & Stunkard, A. J. (2005). Appetite: Measurement and manipulation misgivings. *Journal of the American Dietetic Association, 105*(5 Suppl 1), S87-97. doi: 10.1016/j.jada.2005.02.029
- McCartney, N., Spriet, L. L., Heigenhauser, G. J., Kowalchuk, J. M., Sutton, J. R., & Jones, N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology, 60*(4), 1164-1169.
- Melzer, K., Kayser, B., Saris, W. H., & Pichard, C. (2005). Effects of physical activity on food intake. *Clinical Nutrition, 24*(6), 885-895. doi: 10.1016/j.clnu.2005.06.003
- Murphy, K. G., & Bloom, S. R. (2004). Gut hormones in the control of appetite. *Experimental Physiology, 89*(5), 507-516. doi: 10.1113/expphysiol.2004.027789
- Nagaya, N., Kojima, M., Uematsu, M., Yamagishi, M., Hosoda, H., Oya, H., . . . Kangawa, K. (2001). Hemodynamic and hormonal effects of human ghrelin in healthy volunteers. *American Journal of Physiology.Regulatory, Integrative and Comparative Physiology, 280*(5), R1483-7.
- Nair, N. S., Brennan, I. M., Little, T. J., Gentilcore, D., Hausken, T., Jones, K. L., . . . Feinle-Bisset, C. (2009). Reproducibility of energy intake, gastric emptying, blood glucose, plasma insulin and cholecystokinin responses in healthy young males. *The British Journal of Nutrition, 101*(7), 1094-1102. doi: 10.1017/S0007114508042372
- Naslund, E., Bogefors, J., Skogar, S., Gryback, P., Jacobsson, H., Holst, J. J., & Hellstrom, P. M. (1999). GLP-1 slows solid gastric emptying and inhibits insulin, glucagon, and PYY release in humans. *The American Journal of Physiology, 277*(3 Pt 2), R910-6.
- Noble, B. J., Borg, G. A., Jacobs, I., Ceci, R., & Kaiser, P. (1983). A category-ratio perceived exertion scale: Relationship to blood and muscle lactates and heart rate. *Medicine and Science in Sports and Exercise, 15*(6), 523-528.
- Noreen, E. E., & Lemon, P. W. (2006). Reliability of air displacement plethysmography in a large, heterogeneous sample. *Medicine and Science in Sports and Exercise, 38*(8), 1505-1509. doi: 10.1249/01.mss.0000228950.60097.01
- Norton, G. N., Anderson, A. S., & Hetherington, M. M. (2006). Volume and variety: Relative effects on food intake. *Physiology & Behavior, 87*(4), 714-722. doi: 10.1016/j.physbeh.2006.01.010
- Peronnet, F., & Massicotte, D. (1991). Table of nonprotein respiratory quotient: An update. *Canadian Journal of Sport Sciences = Journal Canadien Des Sciences Du Sport, 16*(1), 23-29.

- Phelain, J. F., Reinke, E., Harris, M. A., & Melby, C. L. (1997). Postexercise energy expenditure and substrate oxidation in young women resulting from exercise bouts of different intensity. *Journal of the American College of Nutrition*, *16*(2), 140-146.
- Polonsky, K. S., Given, B. D., & Van Cauter, E. (1988). Twenty-four-hour profiles and pulsatile patterns of insulin secretion in normal and obese subjects. *The Journal of Clinical Investigation*, *81*(2), 442-448. doi: 10.1172/JCI113339
- Pomerleau, M., Imbeault, P., Parker, T., & Doucet, E. (2004). Effects of exercise intensity on food intake and appetite in women. *The American Journal of Clinical Nutrition*, *80*(5), 1230-1236.
- Richards, J. C., Johnson, T. K., Kuzma, J. N., Lonac, M. C., Schweder, M. M., Voyles, W. F., & Bell, C. (2010). Short-term sprint interval training increases insulin sensitivity in healthy adults but does not affect the thermogenic response to beta-adrenergic stimulation. *The Journal of Physiology*, *588*(Pt 15), 2961-2972. doi: 10.1113/jphysiol.2010.189886
- Russel, R. R., Willis, K. S., Ravussin, E., & Larson-Meyer, D. E. (2009). Effects of endurance running and dietary fat on circulating ghrelin and peptide YY. *Journal of Sports Science & Medicine*, *8*(4), 574-583.
- Saltiel, A. R., & Kahn, C. R. (2001). Insulin signalling and the regulation of glucose and lipid metabolism. *Nature*, *414*(6865), 799-806. doi: 10.1038/414799a
- Sandvei, M., Jeppesen, P. B., Stoen, L., Litlekare, S., Johansen, E., Stensrud, T., . . . Jensen, J. (2012). Sprint interval running increases insulin sensitivity in young healthy subjects. *Archives of Physiology and Biochemistry*, *118*(3), 139-147. doi: 10.3109/13813455.2012.677454
- Scheid, J. L., De Souza, M. J., Leidy, H. J., & Williams, N. I. (2011). Ghrelin but not peptide YY is related to change in body weight and energy availability. *Medicine and Science in Sports and Exercise*, *43*(11), 2063-2071. doi: 10.1249/MSS.0b013e31821e52ab
- Schmidt, P. T., Naslund, E., Gryback, P., Jacobsson, H., Holst, J. J., Hilsted, L., & Hellstrom, P. M. (2005). A role for pancreatic polypeptide in the regulation of gastric emptying and short-term metabolic control. *The Journal of Clinical Endocrinology and Metabolism*, *90*(9), 5241-5246. doi: 10.1210/jc.2004-2089
- Schubert, M. M., Desbrow, B., Sabapathy, S., & Leveritt, M. (2013). Acute exercise and subsequent energy intake. A meta-analysis. *Appetite*, *63*, 92-104. doi: 10.1016/j.appet.2012.12.010; 10.1016/j.appet.2012.12.010

- Schubert, M. M., Sabapathy, S., Leveritt, M., & Desbrow, B. (2013). Acute exercise and hormones related to appetite regulation: A meta-analysis. *Sports Medicine*, doi: 10.1007/s40279-013-0120-3
- Schwartz, M. W., Woods, S. C., Porte, D., Jr, Seeley, R. J., & Baskin, D. G. (2000). Central nervous system control of food intake. *Nature*, 404(6778), 661-671. doi: 10.1038/35007534
- Schwartz, M. W., Woods, S. C., Seeley, R. J., Barsh, G. S., Baskin, D. G., & Leibel, R. L. (2003). Is the energy homeostasis system inherently biased toward weight gain? *Diabetes*, 52(2), 232-238.
- Sim, A. Y., Wallman, K. E., Fairchild, T. J., & Guelfi, K. J. (2013). High-intensity intermittent exercise attenuates ad-libitum energy intake. *International Journal of Obesity*, doi: 10.1038/ijo.2013.102
- Siri, W. E. (1961). Body composition from fluid spaces and density: Analysis of methods. In J. Brozek, & A. Henschel (Eds.), *Techniques for measuring body Composition*. (pp. 223-244). Washington (DC): National Academy of Sciences, National Research Council.
- Sliwowski, Z., Lorens, K., Konturek, S. J., Bielanski, W., & Zoladz, J. A. (2001). Leptin, gastrointestinal and stress hormones in response to exercise in fasted or fed subjects and before or after blood donation. *Journal of Physiology and Pharmacology*, 52(1), 53-70.
- Sloth, B., Holst, J. J., Flint, A., Gregersen, N. T., & Astrup, A. (2007). Effects of PYY1-36 and PYY3-36 on appetite, energy intake, energy expenditure, glucose and fat metabolism in obese and lean subjects. *American Journal of Physiology. Endocrinology and Metabolism*, 292(4), E1062-8. doi: 10.1152/ajpendo.00450.2006
- Sullivan, S. N., Champion, M. C., Christofides, N. D., Adrian, T. E., & Bloom, S. R. (1984). Gastrointestinal regulatory peptide responses in long-distance runners. *The Physician and Sportsmedicine*, 12(7), 77-82.
- Talanian, J. L., Galloway, S. D., Heigenhauser, G. J., Bonen, A., & Spriet, L. L. (2007). Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *Journal of Applied Physiology*, 102(4), 1439-1447. doi: 10.1152/jappphysiol.01098.2006
- Thompson, D. A., Wolfe, L. A., & Eikelboom, R. (1988). Acute effects of exercise intensity on appetite in young men. *Medicine and Science in Sports and Exercise*, 20(3), 222-227.

- Tolessa, T., Gutniak, M., Holst, J. J., Efendic, S., & Hellstrom, P. M. (1998). Inhibitory effect of glucagon-like peptide-1 on small bowel motility. fasting but not fed motility inhibited via nitric oxide independently of insulin and somatostatin. *The Journal of Clinical Investigation*, 102(4), 764-774. doi: 10.1172/JCI942
- Track, N. S., McLeod, R. S., & Mee, A. V. (1980). Human pancreatic polypeptide: Studies of fasting and postprandial plasma concentrations. *Canadian Journal of Physiology and Pharmacology*, 58(12), 1484-1489.
- Trapp, E. G., Chisholm, D. J., & Boutcher, S. H. (2007). Metabolic response of trained and untrained women during high-intensity intermittent cycle exercise. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, 293(6), R2370-5. doi: 10.1152/ajpregu.00780.2006
- Trapp, E. G., Chisholm, D. J., Freund, J., & Boutcher, S. H. (2008). The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. *International Journal of Obesity*, 32(4), 684-691. doi: 10.1038/sj.ijo.0803781
- Tremblay, A., Simoneau, J. A., & Bouchard, C. (1994). Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism: Clinical and Experimental*, 43(7), 814-818.
- Tsilchorozidou, T., Batterham, R. L., & Conway, G. S. (2008). Metformin increases fasting plasma peptide tyrosine tyrosine (PYY) in women with polycystic ovarian syndrome (PCOS). *Clinical Endocrinology*, 69(6), 936-942. doi: 10.1111/j.1365-2265.2008.03285.x
- Ueda, S. Y., Yoshikawa, T., Katsura, Y., Usui, T., & Fujimoto, S. (2009a). Comparable effects of moderate intensity exercise on changes in anorectic gut hormone levels and energy intake to high intensity exercise. *The Journal of Endocrinology*, 203(3), 357-364. doi: 10.1677/JOE-09-0190
- Ueda, S. Y., Yoshikawa, T., Katsura, Y., Usui, T., Nakao, H., & Fujimoto, S. (2009b). Changes in gut hormone levels and negative energy balance during aerobic exercise in obese young males. *The Journal of Endocrinology*, 201(1), 151-159. doi: 10.1677/JOE-08-0500
- Unick, J. L., Otto, A. D., Goodpaster, B. H., Helsel, D. L., Pellegrini, C. A., & Jakicic, J. M. (2010). Acute effect of walking on energy intake in overweight/obese women. *Appetite*, 55(3), 413-419. doi: 10.1016/j.appet.2010.07.012
- van Aggel-Leijssen, D. P., van Baak, M. A., Tenenbaum, R., Campfield, L. A., & Saris, W. H. (1999). Regulation of average 24h human plasma leptin level; the influence of exercise and physiological changes in energy balance. *International Journal of Obesity and Related Metabolic Disorders*, 23(2), 151-158.

- Warren, A., Howden, E. J., Williams, A. D., Fell, J. W., & Johnson, N. A. (2009). Postexercise fat oxidation: Effect of exercise duration, intensity, and modality. *International Journal of Sport Nutrition and Exercise Metabolism*, 19(6), 607-623.
- Wasse, L. K., Sunderland, C., King, J. A., Miyashita, M., & Stensel, D. J. (2013). The influence of vigorous running and cycling exercise on hunger perceptions and plasma acylated ghrelin concentrations in lean young men. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, 38(1), 1-6. doi: 10.1139/apnm-2012-0154
- Weingarten, H. P. (1985). Stimulus control of eating: Implications for a two-factor theory of hunger. *Appetite*, 6(4), 387-401.
- Westerterp, K. R. (1993). Food quotient, respiratory quotient, and energy balance. *The American Journal of Clinical Nutrition*, 57(5 Suppl), 759S-764S; discussion 764S-765S.
- Westerterp-Plantenga, M. S., Verwegen, C. R., Ijedema, M. J., Wijckmans, N. E., & Saris, W. H. (1997). Acute effects of exercise or sauna on appetite in obese and nonobese men. *Physiology & Behavior*, 62(6), 1345-1354.
- Whyte, L. J., Ferguson, C., Wilson, J., Scott, R. A., & Gill, J. M. (2013). Effects of single bout of very high-intensity exercise on metabolic health biomarkers in overweight/obese sedentary men. *Metabolism: Clinical and Experimental*, 62(2), 212-219. doi: 10.1016/j.metabol.2012.07.019
- Whyte, L. J., Gill, J. M., & Cathcart, A. J. (2010). Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism: Clinical and Experimental*, 59(10), 1421-1428. doi: 10.1016/j.metabol.2010.01.002
- Williams, C. B., Zelt, J. G., Castellani, L. N., Little, J. P., Jung, M. E., Wright, D. C., . . . Gurd, B. J. (2013). Changes in mechanisms proposed to mediate fat loss following an acute bout of high-intensity interval and endurance exercise. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, 38(12), 1236-1244. doi: 10.1139/apnm-2013-0101
- Woods, S. C., Lotter, E. C., McKay, L. D., & Porte, D., Jr. (1979). Chronic intracerebroventricular infusion of insulin reduces food intake and body weight of baboons. *Nature*, 282(5738), 503-505.
- Wren, A. M., Seal, L. J., Cohen, M. A., Brynes, A. E., Frost, G. S., Murphy, K. G., . . . Bloom, S. R. (2001). Ghrelin enhances appetite and increases food intake in humans. *The Journal of Clinical Endocrinology and Metabolism*, 86(12), 5992.

- Yoshioka, M., Doucet, E., St-Pierre, S., Almeras, N., Richard, D., Labrie, A., . . . Tremblay, A. (2001). Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness. *International Journal of Obesity and Related Metabolic Disorders*, 25(3), 332-339. doi: 10.1038/sj.ijo.0801554
- Zhang, T., Uchida, T., Gomez, G., Lluís, F., Thompson, J. C., & Greeley, G. H., Jr. (1993). Neural regulation of peptide YY secretion. *Regulatory Peptides*, 48(3), 321-328.

Appendices

Appendix A: Participant health information form

Participant Health Information Form

Participant I.D.: _____ Date: _____

Age: _____ Height: _____ Weight: _____ Smoker: **Yes / No**

Age of menarche: _____ Do you have a regular menstrual cycle? **Yes / No**

Ethnic Background: _____

Medical History (please check any and all that apply)

Family history of heart disease	<input type="checkbox"/>	Endocrine disorder	<input type="checkbox"/>
Heart murmur	<input type="checkbox"/>	Raynaud's syndrome	<input type="checkbox"/>
Phlebitis	<input type="checkbox"/>	Polycystic ovary syndrome	<input type="checkbox"/>
Other heart disorder (please specify)	<input type="checkbox"/>	Seizures	<input type="checkbox"/>
_____		Digestive/gastrointestinal problems	<input type="checkbox"/>
Family history of stroke	<input type="checkbox"/>	Asthma	<input type="checkbox"/>
Migraines	<input type="checkbox"/>	Bronchitis	<input type="checkbox"/>
Sinus problems	<input type="checkbox"/>	Other respiratory disorder (please specify)	<input type="checkbox"/>
Hypertension	<input type="checkbox"/>	_____	
Diabetes	<input type="checkbox"/>		

Have you ever fainted? **Yes / No**

If yes, under what circumstances:

Are you taking any medications? **Yes / No**

If yes, please specify: _____

Have you had any major surgeries, illnesses or injuries? **Yes / No**

If yes, please specify (include dates): _____

Do you consume alcohol or any caffeinated beverages on a regular basis? **Yes / No**

If yes, please specify the quantity: _____

Have you followed a weight loss diet in the previous 6 months? **Yes / No**

If no, has your weight been stable over the previous 6 months? **Yes / No**

Are you physically active? **Yes / No**

If yes, please specify the type, frequency, and typical duration of exercise:

How long have you been physically active?

Do you have any food allergies? **Yes / No**

If yes, please specify _____

Appendix B: Copy of ethics approval



Use of Human Participants - Ethics Approval Notice

Research Ethics

Principal Investigator: Dr. Peter Lemon
 File Number:102788
 Review Level:Delegated
 Approved Local Adult Participants:20
 Approved Local Minor Participants:0
 Protocol Title:Effects of Sprint Interval Training on Energy Intake, Appetite-Related Peptides, and Metabolism in Men and Women
 Department & Institution:Health Sciences/Kinesiology,Western University
 Sponsor:Costco Wholesale

Ethics Approval Date:January 18, 2013 Expiry Date:December 31, 2013
 Documents Reviewed & Approved & Documents Received for Information:

Document Name	Comments	Version Date
Revised Letter of Information & Consent		2013/01/09
Letter of Information	Debrief	2013/01/09
Revised Western University Protocol	Revised sponsor information, study procedures, tissue sample collection, added partial deception and revised study risks	

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/CH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced revision(s) or amendment(s) on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the University of Western Ontario Updated Approval Request Form.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

Signature _____

Ethics Officer to Contact for Further Information

Jamie Sutherland (jsutherland@uwo.ca)	Grace Kelly (grace.kelly@uwo.ca)	Shantel Walcott (swalcot@uwo.ca)
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This is an official document. Please retain the original in your files.

Appendix C: Letter of information & consent form



Title of Study: Effects of Sprint Interval Training on Metabolism and Fat Oxidation in Men and Women

Sponsored by Gatorade Sports Science Institute

Principal Investigator: Dr. Peter W.R. Lemon (PhD)

Graduate Student: Kristine Beaulieu (RD, BSc)

Exercise Nutrition Research Laboratory (Room 2235 – 3M Centre)
School of Kinesiology, Western University.

LETTER OF INFORMATION REGARDING RESEARCH

1. INVITATION TO PARTICIPATE

You are being invited to participate in research study at the Exercise Nutrition Research Laboratory (Room 2235 – 3M Centre) involving 8 weeks of sprint interval training (SIT) and the measurement of several metabolic measures because we think you could help us answer questions we have regarding this training program.

2. PURPOSE OF THE LETTER

The purpose of this letter is to provide you with information required for you to make an informed decision regarding participation in this research.

3. PURPOSE OF THIS STUDY

SIT has generated much interest in our society. This type of training produces similar adaptations as traditional endurance training, but requires a much less time. Research from our laboratory suggests SIT can stimulate significant reductions in fat mass. However, the observed fat loss is more than the predicted energy cost of the exercise training. This suggests SIT may induce long term changes in metabolism or appetite. The purpose of this research experiment is to assess metabolism and fat utilization in 10 men and 10 women over 8 weeks of sprint interval training to determine the processes involved in the fat loss associated with this training program. The funds required to conduct this study are provided by the Gatorade Sports Science Institute.

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4. INCLUSION CRITERIA

In order to be eligible to participate in this study you must be a healthy, 18 to 35 year old man or woman.

5. EXCLUSION CRITERIA

You will be excluded from this study if you:

- smoke
- are pregnant (you must advise the research team if you become pregnant during the study)
- are taking part in other research.
- have followed a weight loss diet in the previous 6 months.
- have gained or lost more than 10 pounds during the previous 6 months.
- exercise more than 2-3 days/week (excluding intramural sports, fitness classes and weight lifting), and engaged in any structured training program.
- have a history of gastrointestinal, endocrine, cardiovascular disease or diabetes.
- have an injury limiting running/sprinting.
- have any contraindications to this type of intense exercise or not deemed safe to undergo a physical activity program.
- have any allergies to the food presented in the food-rating questionnaire.

6. STUDY PROCEDURES

If you volunteer to participate in this study, we will ask you to do the following things:

1. Undergo an assessment of various metabolic measures during 5 test days (ranging from 5 to 10h), before and during the 8-week SIT training.
2. If you are chosen randomly to be part of the subsample of this study (50% of the participants, 5 women and 5 men), blood (12mL) will be drawn at four time points per test day (48mL over ~10 h) for a total of 12 samples (144mL) per participant over an 8-week period. This involves sitting comfortably in a chair with an armrest while a certified phlebotomist (someone trained to take blood) takes a small sample of blood from a vein in your arm.
3. Complete 8 weeks of sprint interval training (3 days/week = 24 sessions). Each session will consist of 3-10 intervals of 30 seconds running efforts on a manually driven treadmill, with each effort separated by 4 minutes of no exercise. Your rating of perceived exertion (on a 10-point scale) will be assessed after each exercise session.
4. Complete 3-day dietary records at weeks 0, 1, 3, 6 and 8.

Time commitment:

Familiarization session 1 h, test day 1 ~ 10 h, test day 2 ~ 10 h, test day 3 ~ 5 h, test day 4 ~ 5h, test day 5 ~ 10 h, and 20 non-test SIT sessions (3 times/wk): 30 min-1 h per day for a total of 26 visits (55 h) to the Exercise Nutrition Lab in a 5-week period. See study outline for schedule and below for details.

Study Outline:

Week	0	1		2		3		4	
Sessions	FS	1	TD 2 3x30s	4	4x30s	7	5x30s	10	6x30s
		2	3x30s	5	4x30s	8	TD 3 5x30s	11	6x30s
	TD 1	3	3x30s	6	4x30s	9	5x30s	12	6x30s
Week		5		6		7		8	
Sessions		13	7x30s	16	TD 4 8x30s	19	9x30s	22	10x30s
		14	7x30s	17	8x30s	20	9x30s	23	10x30s
		15	7x30s	18	8x30s	21	9x30s	24	TD 5 10x30s

FS = Familiarization session, TD = Test day

Testing will be conducted in the Exercise Nutrition Research Laboratory (Room 2235 – 3M Centre). Women will be tested during the beginning of the menstrual cycle to ensure hormone levels are consistent during testing. Women will be asked to keep track of their menstrual cycle so that testing will occur during the first seven days of the menstrual cycle. Whereby, the first day of menstruation marks day one of the menstrual cycle.

****For the test days, you will be asked to arrive at the laboratory at 08:00 h following a 1-day weight-maintaining diet (that will be prescribed to you) and an overnight fast (no food or drink except water after 22:00 h). You will also be instructed to refrain from exercise and from consuming caffeine or alcohol 24 h prior.**

Familiarization session: You will be asked to fill out a physical activity readiness questionnaire and a participant information form for personal and familial health history. You will also complete a food questionnaire and rate different food items according to your personal preferences to determine the composition of the meals to be provided to you. Finally, you will undergo a trial run of sprinting and a maximal oxygen uptake test (you will run at progressively greater intensities while breathing through a facemask until you feel you cannot continue) on a treadmill. You will bring home 3-day food records to complete during weeks 0, 1, 3, 6 and 8 of the study. You will record as precisely as possible every single item that you consume (this includes water, vitamins, condiments, margarine, etc.) during 2 non-consecutive weekdays and 1 weekend day.

Week 0 – Test day 1: You will report to the lab at 08:00 h and your metabolic rate will be measured for 30 minutes (breathing through a facemask).

Then, we will measure your body composition in the BodPod® (this involves sitting in a chamber for a few minutes), ask you to rate your appetite, and take a blood sample (subsample participants only).

At 08:45 h, we will serve you breakfast and you will be allowed time to read/study.

At 10:30 h, we will take your metabolic rate for another 30 minutes, your appetite ratings and a blood sample (subsample participants only).

At 11:00 h you will rest comfortably for 30 minutes.

At 11:30 h we will take your metabolic rate for another 30 minutes, your appetite ratings and a blood sample (subsample participants only).

At 12:00 h, we will serve you lunch; you will eat until you are satisfied during the 30 minutes allowed.

At 12:30 h, we will take your metabolic rate for another 30 minutes, your appetite ratings and a final blood sample (subsample participants only). You will have time to read/study.

At 16:30 h, we will take your metabolic rate for another 30 minutes and your appetite ratings.

At 17:00 h, we will serve you supper; you will eat until you are satisfied during the 30 minutes allowed.

At 17:30 h, we will take your metabolic rate for a final 30 minutes and your appetite ratings. You will be able to leave at 18:00 h.

Week 1 – Test day 2: Same procedures as test day 1, but you will perform a SIT session at 11:00h instead of resting.

Week 3 – Test day 3: You will report to the lab at 08:00 h and we will measure your body composition in the BodPod®, and ask you to rate your appetite.

At 08:45 h, we will serve you breakfast and you will be allowed time to read/study.

At 10:30 h, we will take your appetite ratings.

At 11:00 h you will perform a SIT session.

At 11:35 h we will take your appetite ratings.

At 12:05 h we will serve you lunch; you will eat until you are satisfied during the 30 minutes allowed.

At 12:35 h, we will take your appetite ratings and you will be able to leave.

Week 6 – Test day 4: Same procedures as test day 3.

Week 8 – Test day 5: Same procedures as test day 2.

Test day outline:

		Time & Measures										
		08:00	08:45	10:30	11:00	11:30	12:00	12:30	16:30	17:00	17:30	18:00
Week 0	Test day 1	MR BC AP (BS)	Standard breakfast	MR AP (BS)	Rest	MR AP (BS)	Lunch	MR AP (BS)	MR AP	Dinner	MR AP	Leave
Week 1	Test day 2	MR BC AP (BS)	Standard breakfast	MR AP (BS)	SIT 3 x 30s RPE	MR AP (BS)	Lunch	MR AP (BS)	MR AP	Dinner	MR AP	Leave

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Week 3 Test day 3	08:00	08:45	10:30	11:00	11:35	12:05	12:35				
	BC AP	Standard breakfast	AP	SIT 5 x 30s RPE	AP	Lunch	AP				
Week 6 Test day 4	08:00	08:45	10:30	11:00	11:35	12:05	12:35				
	BC AP	Standard breakfast	AP	SIT 8 x 30s RPE	AP	Lunch	AP				
Week 8 Test day 5	08:00	08:45	10:30	11:00	11:55	12:25	12:55	16:55	17:25	17:55	18:25
	MR BC AP (BS)	Standard breakfast	MR AP (BS)	SIT 10 x 30s RPE	MR AP (BS)	Lunch	MR AP (BS)	MR AP	Dinner	MR AP	Leave

MR = Metabolic Rate, BC = Body composition, AP = Appetite ratings,
BS = Blood sample (subsampling), RPE = Rating of perceived exertion

7. POSSIBLE RISK AND HARMS

This study involves strenuous exercise that may pose a risk of minor injury or discomfort. All exercise involves some health risk (primarily cardiovascular or hydration-related) and you may experience symptoms of fatigue or muscle soreness while participating in this study. The risks of cardiovascular complications are usually reduced in young, healthy individuals. This type of exercise is completed by many Western students in kinesiology classes, in intramural sports and by Mustang athletes. You will be encouraged to hydrate adequately. As for the maximal oxygen uptake test, you may experience muscle fatigue, discomfort, dizziness and/or nausea, however this is a standard test of aerobic fitness employed frequently in scientific investigations as well as in kinesiology classes and athletes' training. There are a few minor risks associated with the blood sample; the needle stick may produce momentary discomfort and possibly some residual soreness and minor bruising of the skin due to blood leaking from the vein. This discoloration may last a few days but is generally harmless. Infrequently, the procedure causes someone to faint or infections may occur when proper blood handling techniques are not used.

8. POTENTIAL BENEFITS

Your aerobic fitness will likely improve using this training protocol and/or you may lose some body fat.

9. COMPENSATION

You will not be compensated for your participation in this study. Additional costs for parking or transportation may be incurred and reimbursement will not be provided.

10. VOLUNTARY PARTICIPATION

Participation in this study is voluntary. You may refuse to participate, refuse to answer any questions or withdraw from the study at any time with no effect on your future academic or employment status. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

11. CONFIDENTIALITY

Any information that is obtained in connection with this study that can identify you will remain confidential and will be disclosed only with your permission. This information will be collected on a master list that will be kept in a password protected file with access to only the investigators in this study. All data will be collapsed before results are printed (only group averages and variability). All participants will be assigned an arbitrary number to ensure anonymity. Mean data will be stored in a password protected file for comparison with future studies. Original and unanalysed data will not be released to any other parties.

12. CONTACT FOR FURTHER INFORMATION

If you have any questions about this research project, feel free to call us (Dr. Peter Lemon or Kristine Beaulieu) for clarification. Further, if you have any questions about the conduct of this study or your rights as a research subject you may contact the Office of Research Ethics at Western University at

13. PUBLICATION

We plan to publish this study in a reputable academic journal upon the completion of the research. The information published in a journal or subsequent studies will not identify you in any way. Copies of such articles will be available upon request.

14. INFORMED CONSENT STATEMENT

Consent Form

Effects of Sprint Interval Training on Metabolism and Fat Oxidation in Men and Women

Investigators: Dr. Peter W.R. Lemon and Kristine Beaulieu, RD, BSc

I have read the accompanying "Letter of Information", have had the nature of the study explained to me and I agree to participate. All questions have been answered to my satisfaction.

If you wish to participate in future studies in the Exercise Nutrition Research Lab, the research team will collect your contact information.

I wish to be contacted for future studies in the Exercise Nutrition Research Laboratory.

Yes _____ (check mark), No _____ (check mark) Date: _____

By signing below, I agree to participate in this study.

Name of Participant (please print): _____

Signature of Participant: _____

Date: _____

Name of Person Obtaining Informed Consent: _____

Signature of Person Obtaining Informed Consent: _____

Date: _____

You will receive a copy of the consent form after it has been signed. You do not waive any legal rights by signing the consent form.

This letter is for you to keep for future references.

Sincerely,

Dr. Peter Lemon

Kristine Beaulieu, RD

Appendix D: Debriefing Letter



Title of Study: Effects of Sprint Interval Training on Metabolism and Fat Oxidation in Men and Women

DEBRIEFING LETTER

1. REAL PURPOSE

The purpose of this research experiment was not only to assess metabolism and fat utilization in men and women over 8 weeks of sprint interval training. In addition, we measured food consumption (energy intake) during all meals on the test days and analyzed blood samples for appetite-related hormones.

2. PARTIAL DISCLOSURE

In order not to influence your eating behaviours (i.e. choices of food and amount eaten), we had to withhold these study details.

3. WITHDRAWAL OF STUDY DATA

Because you did not give us your permission/consent to do so, you may withdraw the study data we collected pertaining to your food consumption and appetite-related hormones; however, we would like to use these data as they provide important information to our study. Of course, only group data will be published so that no one will be able to identify your information.

4. REB CONTACT INFORMATION

If you have any questions about the conduct of this study or your rights as a research subject you may contact the Office of Research Ethics at Western University at

5. RE-CONSENT

I have read the accompanying "Debriefing Letter", have had the nature of the study explained to me and I give permission to use the study data collected. All questions have been answered to my satisfaction.

By signing below, I give permission to use the study data collected.

Name of Participant (please print):

Signature of Participant:

_____ Date: _____

Name of Person Obtaining Informed Consent:

Signature of Person Obtaining Informed Consent:

_____ Date: _____

Sincerely,

Dr. Peter Lemon

Kristine Beaulieu, RD

Appendix E: Food-rating questionnaire

Food-rating questionnaire (Adapted from Schutz, 1965)

Participant I.D.: _____

Please rate the following foods to your personal preferences:

1 = Dislike very much

4 = Like moderately


2 = Dislike moderately

5 = Like very much

3 = Neither like nor dislike

- | | |
|--|--|
| <p>_____ Raisin Bran cereal</p> <p>_____ Mini-Wheats cereal</p> <p>_____ Honey Nut Cheerios cereal</p> <p>_____ Whole-wheat bread</p> <p>_____ White bread</p> <p>_____ Pita bread</p> <p>_____ Whole-wheat spaghetti pasta</p> <p>_____ White spaghetti pasta</p> <p>_____ Chewy chocolate chip granola bar</p> <p>_____ Nutri-Grain cereal bar</p> <p>_____ Clif bar</p> <p>_____ Rice Krispies squares</p> <p>_____ Orange Gatorade</p> <p>_____ Orange juice (no pulp)</p> <p>_____ Apple juice</p> <p>_____ Oasis juice boxes (variety)</p> <p>_____ Fruit salad</p> <p>_____ Red grapes</p> <p>_____ Green grapes</p> <p>_____ Oranges</p> <p>_____ Apples</p> <p>_____ Bananas</p> <p>_____ Baby carrots (raw)</p> <p>_____ Cucumber</p> <p>_____ Cherry tomatoes</p> <p>_____ Baby spinach (raw)</p> <p>_____ Ranch salad dressing</p> <p>_____ Caesar salad dressing</p> <p>_____ Margarine</p> <p>_____ Butter</p> | <p>_____ Mayonnaise</p> <p>_____ Miracle Whip</p> <p>_____ Mustard</p> <p>_____ Peanut butter (smooth)</p> <p>_____ Honey</p> <p>_____ Strawberry jam</p> <p>_____ Raspberry jam</p> <p>_____ Soy milk (original)</p> <p>_____ Skim milk</p> <p>_____ 1% milk</p> <p>_____ 2% milk</p> <p>_____ Chocolate milk</p> <p>_____ Cheddar cheese</p> <p>_____ Mini Babybel cheese</p> <p>_____ Non-fat vanilla yogurt</p> <p>_____ Non-fat fruit yogurt</p> <p>_____ Regular vanilla yogurt</p> <p>_____ Regular fruit yogurt</p> <p>_____ Hard-boiled eggs</p> <p>_____ Turkey deli meat</p> <p>_____ Ham deli meat</p> <p>_____ Roast beef deli meat</p> <p>_____ Canned tuna</p> <p>_____ Pre-cooked chicken breast</p> <p>_____ Plain hummus</p> <p>_____ Tomato marinara sauce</p> <p>_____ Alfredo pasta sauce</p> |
|--|--|

Appendix F: Website for appetite ratings



Appetite Ratings

Participant ID:

1. How hungry do you feel?

I am not hungry at all I have never been more hungry

2. How satisfied do you feel?

I am completely empty I cannot eat another bite

3. How full do you feel?

Not at all full Totally full

4. How much do you think you can eat?

Nothing at all A lot

Curriculum Vitae

Kristine Beaulieu

Post-Secondary Education:

Western University. MSc Kinesiology (Integrative Physiology), 2012-2013
 Université de Moncton. BSc (Nutrition) with integrated internship, 2006-2011

Credentials: Registered Dietitian

Honours and Awards:

Ontario Graduate Scholarship, 2013
 CIHR Master's Award – Nutrition and Dietetic Research, 2012-2013
 Gatorade Sports Science Institute Student Research Award, 2012

Manuscripts:

Beaulieu K, Olver TD, Abbott K, & Lemon PWR. Energy Intake Over Two Days is Unaffected by Acute Sprint Interval Exercise Despite Increased Appetite. in preparation

Olver TD, Abbott K, **Beaulieu K**, Lemon PWR, & Shoemaker JK. Vagal Activity Following Acute Sprint Interval Exercise. in preparation

Book Chapters:

Beaulieu K & Power N. Will an apple a day keep the urologist away? Dietary considerations in urology. In *The Consumer's Handbook for Urological Health: A Guidebook for Patients and their Families*. *Canadian Urological Association*. 2013, in press

Magazine Articles:

Beaulieu K, McLaughlin-Chaisson N, Ward S, Ward D, & Johnson M. Fatigue and shift word. *RCMP Gazette*. 2013, 75(3): 26-27.

McLaughlin-Chaisson N, Ward S, **Beaulieu K**, Curtis B, & Johnson M. Fatigue in EMS shift workers and countermeasures to maximize performance. *Canadian Paramedicine Magazine*. 2013, 36(5): 26-27

Conference Presentations:

Beaulieu K, Olver TD, Abbott K, & Lemon PWR. Effects of Sprint Interval Exercise on Energy Intake, Appetite-Related Peptides and Metabolism. *Ontario Exercise Physiology Conference*, January 2013

Conference Abstracts:

Beaulieu K, Olver TD, Abbott K, & Lemon PWR. Test-retest reliability of energy status and appetite is moderate to high over two consecutive days in healthy men. *Appl Physiol Nutr Metab*. 2013, 38(10): 1023

Beaulieu K, Olver TD, Abbott K, & Lemon PWR. Energy Intake Over Two Days is Unaffected by Acute Sprint Interval Exercise Despite Increased Appetite. *American College of Sport Medicine Annual Meeting 2014*, submitted

Olver TD, Abbott K, **Beaulieu K**, Lemon PWR & Shoemaker JK. Cardio-Vagal Activity Following Acute Sprint Interval Exercise. *Experimental Biology Annual Meeting 2014*, submitted