

## AN OVERVIEW OF TRAINING METHODS THAT PROMOTE THE HIGHEST LIPID OXIDATION DURING AND AFTER A SINGLE EXERCISE SESSION

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### ABSTRACT

*Given that physical activity is the most effective way to increase lipid oxidation, its effects are influenced by several factors. The goal of this review was to identify the most effective methods that facilitate the highest lipid oxidation during and after a single exercise session. For this purpose, the available scientific literature was examined using PubMed, Web of Science, Google Scholar and Cochrane Library databases up to June 2013 with the following keywords: excess post exercise oxygen consumption, exercise fatty acid, energy expenditure exercise and interval training. From the identified 48,583 potentially relevant references, 172 of them met all the required criteria. It was found out that prolonged (> 30 min) moderate intensity (55 – 70 %  $VO_{2max}$ ) exercise such as walking, jogging or cycling is the most effective way to increase lipid oxidation during and after a single exercise session. Low-volume high-intensity interval exercise is supposed to be as effective as traditional exercise with continuous endurance, with the main effect on lipid oxidation after the session and similar long-term metabolic adaptations. However, more research is still needed to compare the effects of regular resistance exercise with traditional endurance and high-intensity interval exercise. Finally, nutrition is also a significant factor since food rich in fat and low in carbohydrates promotes greater lipid oxidation.*

**Keywords:** endurance exercise, interval exercise, training, nutrition, fatty acid, triacylglycerol.

## PREGLED VADBENIH METOD, KI SPODBUDIJO NAJVEČJO OKSIDACIJO LIPIDOV MED IN PO POSAMEZNI VADBENI ENOTI

### IZVLEČEK

*Glede na to da je gibalna oz. telesna aktivnost najbolj učinkovit način povečanja oksidacije maščob, pa na njihovo porabo vpliva še vrsta drugih dejavnikov. Cilj tega preglednega članka je bil ugotoviti vplive in najbolj učinkovite metode za spodbujanje oksidacije maščobnih kislin med vadbeno enoto in po njej. Pregledali smo razpoložljive članke, ki so bili objavljeni do junija 2013 v sledečih bazah: PubMed, Web of Science, Google scholar in Cochrane Library. Pri iskanju smo uporabili naslednje ključne besede: poraba kisika po vadbi, poraba maščobnih kislin med telesno aktivnostjo, poraba energije med telesno aktivnostjo in intervalni trening. Tako smo dobili 48.583 člankov, od katerih je našim kriterijem ustrezalo 172 člankov. Ugotovili smo, da je dolgotrajna telesna aktivnost srednje intenzivnosti, kot sta hoja in lahkoten tek, najbolj učinkovit način za povečevanje oksidacije maščobnih kislin. Visokointenzivna intervalna vadba je bolj učinkovita s stališča porabe energije, vendar pri enaki porabi maščobnih kislin. Potrebni bo še nekaj nadaljnjih raziskav, ki bodo primerjale učinke redne vadbe za moč ter redne klasične aerobne in intervalne vadbe za razvoj vzdržljivosti. K večji oksidaciji maščobnih kislin pomembno vpliva tudi prehrana: hrana, bogata z maščobami in z nizko vsebnostjo ogljikovih hidratov, najbolj vzpodbuja oksidacijo maščobnih kislin.*

**Ključne besede:** vadba vzdržljivosti, intervalna vadba, trening, prehrana, maščobne kisline, triglicerol

### INTRODUCTION

Two of the greatest health threats in modern lifestyle are imbalanced nutrition and a sedentary lifestyle. Excessive diet and an increased sedentary lifestyle lead to obesity and metabolic syndrome which are both associated with numerous comorbidities (Poirier et al., 2006). An excessive carbohydrate intake, especially fructose, leads to an increase in body weight, visceral adipose tissue, muscle fat, as well as liver fat; furthermore, sugar enhances lipogenesis and the production of uric acid including an increase in plasma triacylglycerols (TG) concentrations (Bray, 2013). Lipids are implicated in the pathogenesis of several common human diseases, including: metabolic syndrome, cardiovascular disease and type 2 diabetes (Kiens, 2006). Enhanced lipid oxidation might be beneficial for counteracting lipid accumulation. Physical activity (PA) is the most effective way to increase lipid oxidation, due to the fact that it increases the metabolic rate (Kiens, Alsted, & Jeppesen, 2011). During PA the body has a higher energy demand. The body provides energy for PA converting chemical energy

to mechanical energy and heat. This chemical energy is derived from macronutrients (carbohydrates, proteins and lipids) in the body and converted to adenosine triphosphate (ATP) energy molecules, which are used for work. Depending on the intensity and the duration of PA, different metabolic pathways provide energy: ATP-phosphocreatine (PCr) system, anaerobic glycolysis and aerobic glycolysis with carbohydrate, protein and lipid oxidation (Frayn, 2010; McArdle, Katch, & Katch, 2010). Normally, during prolonged low- to moderate- PA intensities (33 – 65 % of maximal oxygen consumption –  $\dot{V}O_{2max}$ ), lipids metabolised at the highest rate, reaching their maximum at  $\sim 64 \pm 4$  %  $\dot{V}O_{2max}$ , whereas, at higher intensities, carbohydrates become the primary energy source (Achten, Gleeson, & Jeukendrup, 2002; Pérez-Martin et al., 2001). The type of energy source used depends on several factors, not just intensity, but also duration and the type of PA (Antonutto & di Prampero, 1995), gender, aerobic fitness, nutrition (Kiens, 2006), substrate availability within skeletal muscles (Jeppesen & Kiens, 2012; Kiens et al., 2011), amount of fatty tissue and the neuro-hormonal influence on the oxidation (Astrup et al., 1992; Mittendorfer, Fields, & Klein, 2004). However, during PA, the lipid used for energy turnover is derived from different sources (Kiens, 2006; Kiens et al., 2011): blood fatty acids bound to albumin, fatty acids liberated from the hydrolysis of circulating TG and fatty acids from lipolysis of TG located in lipid droplets in skeletal muscles – muscle TG.

Given that endurance PA is the most effective way to increase lipid oxidation (Kiens et al., 2011), its effects are influenced by several factors, and because different types of interval training and resistance exercise are used and promoted in sport and kinesiology practice, it will be the goal of this review to identify what are the influences and which are the most effective methods for facilitating the highest lipid oxidation during and after a single workout.

## METHODS

For the purposes of this review, the available articles in PubMed, Web of Science, Google Scholar and Cochrane Library were analysed up to June 2013. The following keywords were used for searching: excess post exercise oxygen consumption, exercise fatty acid, energy expenditure exercise and interval training. This search strategy identified 48,583 potentially relevant references, while only 172 articles met all the search topic requirements.

## LIPID OXIDATION DURING ENDURANCE PHYSICAL ACTIVITY

The factors influencing lipid oxidation during endurance physical activity are: intensity, duration and type of PA, food intake, amount of fatty tissue, gender and physical fitness.

### Effect of Intensity on Lipid Oxidation during Activity

PA can be divided into low-, moderate-, and high- intensity activities. In literature, boundaries between these levels are not strictly defined and they range from up to 55 %  $VO_{2max}$  for low-intensity, from 55 % to 70 %  $VO_{2max}$  for moderate-intensity, and exceeding 75 %  $VO_{2max}$  for high-intensity PA. During low and moderate-intensity endurance PA, the predominant energy sources are lipids (Pillard et al., 2010). Lipid oxidation during high-intensity PA is lower than during moderate-intensity (Jeppesen & Kiens, 2012). At the beginning of the previous century, it was discovered that by measuring the respiratory exchange ratio (RER), which is the ratio between eliminated  $CO_2$  and utilized  $O_2$  during respiration, it is possible to determine also the type of energy source used (Scott, 2005). If RER is around 1, the body uses carbohydrates, while if it is between 0.7 and 0.8, the body uses more lipids (Walsch, 2003) for energy transfer. By measuring RER, it was discovered that lipid oxidation increases from rest to an intensity level of 65 %  $VO_{2max}$ . Lipid oxidation during endurance PA that lasts from 60 to 90 min increases by 5 to 10 times (Krogh & Lindhard, 1920, in Jeppesen & Kiens, 2012; Cristensen & Hansen, 1939, in Jeppesen & Kiens, 2012). Further studies using RER measurements and monitoring of isotopes have confirmed the highest oxidation of lipids during moderate-intensity endurance PA, namely, at the intensity level of 65 %  $VO_{2max}$ , compared to low-intensity (25 %  $VO_{2max}$ ) and high-intensity (85 %  $VO_{2max}$ ) (Romijn et al., 1993). With the increased intensity of PA, lipid oxidation decreases. During 30 minutes of cycling, for example, at the intensity level of 75%, lipid oxidation is lower than at the intensity level of 55 %  $VO_{2max}$  (van Loon et al., 2001). Even though, it has been known for almost a century that a total amount of oxidised fatty acids during high-intensity PA is lower than during moderate-intensity PA, the mechanisms behind this are still not clear. The limiting factor of fatty acid oxidation during the transition from rest to low or moderate-intensity PA is most probably the transport of fatty acid through the sarcolemma (Bonen, Luiken, Arumugam, Glatz, & Tandon, 2000; Jeppesen et al., 2011). When considering transition from moderate to high intensity PA, some earlier authors argue that oxidation of plasma fatty acids, lipoprotein derived TGs and muscular TGs probably decrease due to unavailability of free carnitine and / or decrease in the intracellular pH, but not as a consequence of a decrease in plasma free fatty acids availability (van Loon, Greenhaff, Constantin Teodosiu, Saris, & Wagenmakers, 2001). Modern studies prefer the notion that the availability of free carnitine in blood is the sole factor limiting the transport of fatty acids to the mitochondria in the muscle (Roepstorff et al., 2005; Kiens et al., 2011; Jeppesen & Kiens, 2012). When considering lipid oxidation during endurance PA, moderate intensity is shown as the most effective from the point of highest lipid oxidation during PA.

### Effects of Duration and Type of Activity on Lipid Oxidation during Activity

The type of energy source used during PA is not determined only by intensity level (as described above), but also by duration and type of PA. Phosphagens (adenosine triphosphate and creatine phosphate) are used during the first 5 to 10 seconds of maximum PA, followed by (rapid) anaerobic glycolysis, if maximum effort continues beyond 10 seconds (obviously the intensity decreases). At this stage, glycolysis maintains the energy transfer up to 2 or 3 minutes, with the glycogen in the muscle as the main energy source (McArdle et al., 2010). When intense exercise continues beyond several minutes, aerobic metabolism provides nearly all of the energy transfer. At this point, fat becomes the primary energy fuel and lipid oxidation the primary process of energy transfer for exercise and recovery when high-intensity, long-duration exercise depletes glycogen (McArdle et al., 2010) or when carbohydrates sparing and promotion of fat utilization occur as a training adaptation (Åstrand, Rodahl, Dahl, & Strømme, 2003; Fox & Mathews, 1981).

There are various lipids sources. During moderate-intensity PA, less than a half of lipids is derived from upper-body subcutaneous adipose tissue, a quarter comes from muscular TGs, and the rest is distributed among fatty acids from lower-body subcutaneous adipose tissue, intra-abdominal adipose tissue, plasma TGs and other (Horowitz, 2003). If PA is so intense or long that glycogen stores are exhausted, the energy derived from glycogen breakdown is supplemented by the breakdown of proteins (Kisner & Colby, 2007). Nonetheless, the total energy expenditure of jogging exceeds that of walking (Wilkin, Cheryl, & Haddock, 2012), fractional lipid oxidation remains at comparable levels, since the respiratory exchange ratio does not demonstrate a discernible pattern between walking and running (Stamford, 1975), meaning that carbohydrate and lipid oxidation increase simultaneously. At same relative intensity, expressed in per cent of  $VO_{2max}$ , lipid oxidation is significantly higher while running compared to cycling (Capostagno & Bosch, 2010), also, more energy is being utilised (Zeni, Hoffman, & Clifford, 1996; Kravitz, Robergs, Heyward, Wagner, & Powers, 1997). When considering lipid oxidation, moderate intensity activity has to last at least 20 minutes to begin utilising lipids. The upper limit is dependable on individual body's glycogen storage capacity, to avoid undesirable protein depletion. Jogging and walking are shown to be more effective at lipid oxidation than cycling.

### Effects of Food Ingestion on Lipid Oxidation during Activity

The ingested food has also an effect on lipid oxidation as muscles adjust the levels of muscle enzymes depending on the food ingested (Spriet, 2011). In addition, most liver enzymes, which regulate glycolysis and fatty acid oxidation, are also regulated by nutrient availability (Rui, 2014). A 7-week diet, rich in fats, increases muscles' ability to metabolized lipids, by increasing activity of enzymes and binding proteins, involved in the process of fatty acid oxidation in the muscle (Helge & Kiens, 1997; Kiens,

2006). When untrained men were consuming fat rich diet for three days leading up to a moderate-intensity PA (cycling at an intensity of 70 %  $VO_{2max}$ ), lipid oxidation was significantly higher, compared to when they consumed a diet rich in carbohydrates (Christensen & Hansen, 1939, in Kiens et al., 2011). In the study performed by Galbo, Holst, and Christensen (1979), seven untrained men ran on a treadmill at 70 %  $VO_{2max}$  intensity until exhaustion. Four days prior to the PA they were consuming carbohydrate and fat rich diets respectively. When they consumed fats, their glucagon, epinephrine, cortisol and growth hormone levels rose significantly compared to the carbohydrate diet. Food consumption in the days leading up to the PA affected not only their bodies' energy stores, but also their hormone reaction, directly affecting their oxidation during PA. Food rich in carbohydrates, on the other hand, promotes oxidation of glucose, production of lactate and inhibits oxidation of lipids (Galbo et al., 1979). A more recent study by Helge found once again a significantly greater lipid oxidation in the group that consumed fat rich diet (Helge, Richter, & Kiens, 1996; Helge, Wulff, & Kiens, 1998). Measuring exhaustion time parameters at the same absolute workload showed that endurance performance was enhanced similarly after both two and four weeks of adaptation to training and a fat-rich or a carbohydrate-rich diet (Helge et al., 1998). Helge, Watt, Richter, Rennie, and Kiens (2001) also demonstrated that circulating TG made a significant contribution to fuel utilization during endurance training after adaptation to a fat-rich diet. The increased lipid oxidation observed after training (cycling 60 to 75 minutes per session at 65 to 85 %  $VO_{2max}$ , four times per week, seven weeks) and fat diet adaptation originated from both a higher plasma fatty acid oxidation and utilization of circulating TG. In contrast, the carbohydrate sparing observed after fat diet adaptation was due to muscle glycogen sparing and not to a diminished plasma glucose uptake (Helge et al., 2001). A longer-term adaptation to a fat-rich diet also leads to measurable changes in the capacity to recruit, transport and oxidize lipids (Helge & Kiens, 1997).

Another physiological process that increases lipid oxidation is starvation (Cahill, 2006). In the fasted state, fatty acids are oxidized mainly in the mitochondria to generate energy supply as well as ketone bodies (Rui, 2014). Free fatty acid levels were increased approximately 9-fold after 60 h of fasting in healthy male subjects, leading to elevated muscular TG levels and decreased muscular insulin sensitivity (Hoeks et al., 2010). Despite an increase in whole-body lipid oxidation, Hoeks et al. (2010) observed a reduction in mitochondrial capacity. Van Proeyen, Szlufcik, Nielens, Ramaekers, and Hespel (2011) investigated the effect of endurance training in the fasted state vs. training in the fed state on muscle oxidation and substrate utilization. They found out that moderate-intensity endurance training on an empty stomach significantly stimulates muscle cells to increase lipid oxidation and facilitate utilizing muscular TGs, compared to the group that consumed carbohydrates prior to, and during PA (untrained men, cycling 60 to 90 minutes daily for 6 weeks at intensity level of 70 %  $VO_{2max}$ ) (Van Proeyen et al., 2011). Availability of endogenous fuels in muscles affects lipid oxidation in muscles as well as on the level of entire body. The amount of muscular TGs in muscles before the activity also influences lipid oxidation, in addition to intensity, duration and type of activity as well as gender, fitness level and ingested food (Kiens, 2006). Mode-

rately trained boys showed 2.5 times local lipid oxidation in their muscles when they consumed fat rich diet, compared to carbohydrate rich diet (which replenished glycogen stores in muscles), at same workload (60 minutes of cycling at 65 %  $VO_{2max}$  intensity). Since it is spent for acetyl-carnitine formation, free carnitine availability significantly decreases, reducing lipid oxidation capability during endurance PA (Roepstorff et al., 2005). When muscle glycogen stores are low, a two times higher concentration of fatty acids can be observed in the plasma, as well as a significantly increased concentration of enzymes and binding proteins that participate in lipid oxidation (Wojtaszewski et al., 2003). It has been shown that glycogen stores are inversely proportional to the levels of free carnitine. When glycogen stores in muscles are high, there is very little free carnitine in the cells; this limits fatty acid transport into mitochondria, which consequently limits lipid oxidation in the muscles (Jeppesen & Kiens, 2012). The intake of carnitine dietary supplement increased ability to metabolise lipids during low-intensity endurance PA (untrained healthy men cycled for 30 minutes at 50 %  $VO_{2max}$  intensity), a 44 % decrease in Lactate concentration in the blood during high-intensity PA (30 minutes of cycling at 80 %  $VO_{2max}$  intensity) was also observed (Wall et al., 2011). This may indicate increased lipid oxidation while taking carnitine dietary supplement at higher intensities as well (Jeppesen & Kiens, 2012). When considering lipid oxidation, after adaptation to a high-fat diet due to an increased uptake of lipids originating from the bloodstream and only a minor extent to an increased muscle TG utilization, greater lipid oxidation is observed during PA. The intake of carnitine dietary supplement might also stimulate lipid oxidation. Training on an empty stomach stimulates muscle cells to increase lipid oxidation and facilitate utilizing muscular TGs.

### **Effects of Adipose Tissue Amounts on Lipid Oxidation during Activity**

The amount of fat tissue also affects the capability of lipid oxidation. Chronic imbalances between energy intake and oxidation ultimately result in excess intracellular lipid accumulation, both at the whole body level and in individual organs or tissues. Obese and overweight people have a higher concentration of plasma fatty acids, most likely due to increased fatty acid release from an expanded fat mass; providing a link between obesity and ectopic lipid accumulation (Savage, Petersen, & Schulman, 2007). “Sedentary overweight subjects, compared to controls at the same exercise intensities, exhibit an alteration of the balance of substrate oxidation, reflected by lower rates of lipid oxidation and a shift of quantitative parameters to lower intensities” (Pérez-Martin et al., 2001). The amount of fatty acids increases as a reaction to low-intensity PA (untrained men cycling for 90 minutes at 50 %  $VO_{2max}$  intensity). The increase in fatty acids oxidation during exercise is  $\approx$  50 % lower in obese and  $\approx$  35 % lower in overweight people when compared to lean controls (Scheen, Pirnay, Luyckx, & Lefebvre, 1983; Mittendorfer et al., 2004). The obese have four times higher transport of fatty acids through sarcolemma (Bonen et al., 2004) and fatty acids penetrate into muscles, where they accumulate as muscular TGs (Goodpaster, Theriault, Watkins, & Kelley, 2000).

The accumulation of muscular TGs in muscles may result in insulin resistance and the development of diabetes (Kelley & Goodpaster, 2001). The rate of total fat oxidation, assessed by indirect calorimetry during the last 30 minutes of exercise, was not significantly different between groups: lean, overweight and obese (Mittendorfer et al., 2004). That is why comparable RER levels were measured in overweight and lean untrained men (30 minutes cycling at anaerobic threshold) (Wong & Harber, 2006). Fat oxidation provided ~30 % of total energy requirements during the exercise in lean, overweight, and obese men. However, the source of fatty acids used as fuel during the exercise varied between groups. In lean subjects about one-half of the fatty acids oxidized during the exercise, having derived from systemic plasma fatty acids and the other half from non-systemic fatty acids. The relative contribution of systemic plasma fatty acids to total fat oxidation decreased and the relative contribution of non-systemic fatty acid to total fat oxidation increased with increasing adiposity. Presumably, the predominant source of non-systemic fatty acids was the fatty acids that were released during lipolysis of intramuscular TG (Mittendorfer et al., 2004).

“Lipid accumulation in skeletal muscle and liver may be a result of increased delivery / synthesis of fatty acids to / in these tissues in those conditions in which energy intake exceeds adipose tissue storage capacity (as seen in obesity and lipodystrophy), or a consequence of either acquired or inherited mitochondrial dysfunction” (Savage et al., 2007). Obese people have a lower expression of the adipose tissue hormone adiponectin (Civitarese et al., 2006), which is one of the hormones (along with leptin and FGF21) that enhance mitochondrial proliferation in white adipose tissue and whole-body energy expenditure associated with the lipid burning in beige adipocytes (Unger, Scherer, & Holland, 2013). In addition to lower hormonal response to endurance PA, ineffective lipid oxidation can be observed with obese people. They have smaller mitochondria and / or impaired oxidation process within the mitochondria thus, being handicapped in their effort to lose fat (Colberg, Simoneau, Thaete, & Kelly, 1995; Borer, 2008). It is possible that “hyperinsulinemia” in overweight and obese compared with lean men contributed also to the blunted lipolytic response to exercise. Although the relative decrease in plasma insulin concentration was similar in lean, obese and overweight, the absolute plasma insulin concentrations were greater during exercise in overweight and obese than in lean subjects. It is also likely that the attenuated lipolytic response to exercise in overweight and obese men was caused by a blunted increase in epinephrine secretion and a concomitant reduction in adipose tissue lipolytic response to circulating catecholamines (Mittendorfer et al., 2004). “Adipocyte dysfunction due to either obesity or lipodystrophy is associated with excessive and untimely delivery of fatty acids to the liver and skeletal muscle and probably contributes to insulin resistance in both organs by altering the balance between fatty acids uptake / synthesis and the disposal leading to increases in intracellular lipid content.” Also, an increasingly sedentary lifestyle and the associated relative increase in fat mass almost certainly contribute to aging induced insulin resistance. Muscle insulin resistance and accumulation of muscular TG precede the development of hepatic insulin resistance and type 2 diabetes (Savage et al., 2007). Exercise (insulin resistant boys; 45 minutes on elliptical

trainer) resulted in a greater than threefold increase in postprandial net muscle glycogen synthesis, reflecting improved muscle insulin responsiveness, and a  $\approx 40\%$  reduction in net hepatic TG synthesis. The changes in the whole body energy storage were accompanied by a  $\approx 30\%$  decrease in hepatic lipogenesis and were independent of changes in fasting or postprandial plasma glucose and insulin concentrations (Rabøl, Petersen, Dufour, Flannery, & Schulman, 2011). Exercise, as well as weight loss, reduced insulin resistance (Houmar et al., 2002). When considering lipid oxidation, it seems an inverse relationship between adiposity and the lipolytic response to exercise. The limited availability of systemic plasma fatty acids as a fuel in overweight and obese men was associated with a compensatory increase in the oxidation of non-systemic fatty acids.

### **Effects of Gender and Fitness Level on Lipid Oxidation during Activity**

Women utilise more of lipids than men at the same relative activity intensity level (Kiens, 2006), Lipid oxidation remains the same during luteal and follicular phase of the menstrual cycle (Matsuo, Saitoh, & Suzuki, 1999). Due to adaptation mechanisms, regular endurance PA increases lipid utilisation during PA (Costill, Fink, Getchell, Ivy, & Witzmann, 1979; Koivisto, Hendler, Nadel, & Felig, 1982; Gollnick, 1977; Short & Sedlock, 1997; Jeppesen & Kiens, 2012). An increased lipid oxidation occurs due to increased capillary density (Kiens, Essen-Gustavsson, Christensen, & Saltin, 1993), increased lipid binding protein activity, an increase of fatty acid oxidation controlling enzymes (Kiens & Lithell, 1993; Alsted et al., 2009; Jeppesen et al., 2012), reduced insulin secretion and increased activation of adiponectin hormone in the adipose tissue (Civitaresi et al., 2006). These adaptations to regular endurance PA increase the ability of mobilisation, transportation and oxidation of lipids in people with aerobic stamina (Henriksson, 1977; Kiens et al., 1993; Jeppesen et al., 2012). When considering lipid oxidation, women utilise more lipids than men, and after adaptation to training, greater lipid oxidation (recruit, transport and oxidize lipids) is observed during PA.

### **LIPID OXIDATION AFTER CONTINUOUS ACTIVITY**

Following PA, body immediately requires oxygen to regenerate its oxygen energy supplies (in myoglobin and haemoglobin), synthesis of phosphagens, removal of lactate from blood, increased need for oxygen in tissues due to increased temperature, an increase in catecholamine concentration and increased heart and respiratory muscles activity (Børsheim & Bahr, 2003). The period following the PA is known as the “excess post-exercise oxygen consumption” phase (EPOC) (Scott, 1997). Increased lipid oxidation and decreased carbohydrate oxidation can be observed during most of the EPOC period (Maehlum, Grandmontagne, Newsholme, & Sejersted, 1986; Bahr, Ingnes, Vaage, Sejersted, & Newsholme, 1987; Bahr, Hansson, & Sejersted, 1990; Chad & Quigley, 1991; Short & Sedlock, 1997), thus, it was imperative to further study the literature on

how it describes the EPOC amount and duration, and how lipids are utilised after PA. Amount and duration of EPOC are influenced by intensity and duration of PA (Sedlock, Fissinger, & Melby, 1989), the method of carrying out the PA (Almuzaini, Potteiger, & Green, 1998), the menstrual cycle (Matsuo et al., 1999), aerobic fitness level (Sedlock, Lee, Flynn, Park, & Kamimori, 2010) and amount of fatty tissue (Wong & Harber, 2006). Gender per se (Lamont, Romito, & Rossi, 2010), the amount of active muscles (Sedlock, 1991b) and nutrition (Bahr & Sejersted, 1991b) show no effect on EPOC.

### **Effect of Intensity and Duration of Activity on Lipid Oxidation after Activity**

Intensity of PA is exponentially related to the EPOC level (Brehm & Gutin, 1986; Sedlock et al., 1989; Gore & Withers, 1990; Bahr & Sejersted, 1991a; Frey, Byrnes, & Mazzeo, 1993; Smith and Naughton, 1993; Hardman, 2001; Børsheim & Bahr, 2003), the duration of PA on the other hand is in linear relation to the EPOC level (Bahr et al., 1987; Chad & Wenger, 1988; Sedlock et al., 1989; Gore & Withers, 1990; Smith & Mc Naughton, 1993). Lipid oxidation following low-intensity long-term PA can be more than three times higher than during resting state (within three hours following 120 minutes of PA at 51 %  $VO_{2max}$  intensity) (Bahr et al., 1990), low-intensity and / or short-term PA, however, does not lead to longer EPOC (Børsheim & Bahr, 2003). When EPOC length was studied at constant intensity level and various durations of moderate-intensity PA (cycling at 70 %  $VO_{2max}$  intensity), it was discovered that EPOC after 30, 45 and 60 minutes of moderate-intensity PA lasted  $\approx$  2 hours,  $\approx$  3 hours and  $\approx$  7 hours, respectively (Chad & Wenger, 1988), after 80 minutes of cycling up to 12 hours (Maehlum et al., 1986; Bahr et al., 1987). When EPOC length was studied at constant duration (80 minutes of cycling) and various intensity levels of moderate-intensity PA (29 %, 50 % and 75 %  $VO_{2max}$ ), it was discovered that EPOC lasted respectively for  $\approx$  20 minutes,  $\approx$  3 hours and  $\approx$  10 hours after the activity; respective EPOC amounts were  $\approx$  10 kcal,  $\approx$  30 kcal and  $\approx$  150 kcal (Bahr & Sejersted, 1991b). EPOC amount studies on trained participants, 8 hours after a 20-, 50-, and 80-minute run at of 70%  $VO_{2max}$  intensity, showed the values of  $\approx$  25 kcal,  $\approx$  50 kcal and  $\approx$  75 kcal, respectively (Gore & Withers, 1990). Studying effect of intensity and duration on amount and duration of EPOC in trained men, showed that short term high-intensity PA brought about significantly higher EPOC ( $\approx$  30 kcal) than a low-intensity PA, regardless of its duration ( $\approx$  15 kcal). A short-term low-intensity PA resulted in significantly shorter EPOC ( $\approx$  20 minutes), compared to a long-term low-intensity PA ( $\approx$  30 minutes) and a short-term high-intensity PA ( $\approx$  35 minutes) (Sedlock et al., 1989).

### **Effect of Dividing Activity into Sets and Amount of Working Muscles on Lipid Oxidation after Activity**

Studying whether dividing PA into sets has any effect, it was found that the EPOC amount after 2 sets separated by 6 hours of rest (15 minutes of cycling at 70 %  $VO_{2max}$  intensity) was significantly greater than if same PA was done in a single set (30 minutes of cycling at 70 %  $VO_{2max}$  intensity) (Almuzaini et al., 1998). Similar findings were reached in an earlier study where the EPOC amount after two sets (25 minutes of running at 70 %  $VO_{2max}$  intensity) was significantly greater than if the PA was done in a single set (50 minutes of running at 70 %  $VO_{2max}$  intensity) (Kaminsky, Padjen, & LaHam-Saeger, 1990).

EPOC amount and duration following PA for upper limbs (20 minutes, at 60 %  $VO_{2max}$  intensity on upper limb cycle ergometer) and PA for lower limbs (20 minutes, 60 %  $VO_{2max}$  intensity on lower limb cycle ergometer) are virtually the same (23 minutes and  $\approx 9$  kcal for the upper limbs and 24 minutes and  $\approx 10$  kcal for the lower limbs). It can be concluded that the amount of active muscles as well as absolute  $VO_{2max}$  value do not affect the amount and the duration of EPOC (Sedlock, 1991b).

### **Effects of Gender on Lipid Oxidation after Activity**

When comparing low-intensity PA (50 %  $VO_{2max}$ , 500 kcal) and high-intensity PA (75 %  $VO_{2max}$ , 500kcal) in women, three hours after a high-intensity PA ( $\approx 40$  kcal) EPOC amount was greater than after low-intensity PA ( $\approx 20$  kcal). During and after a low-intensity PA, more lipids were used ( $\approx 37$  g) than during and after a high-intensity PA ( $\approx 28$  g). This difference, however, was not statistically significant ( $p = 0.07$ ) (Phelain, Reinke, Harris, & Melby, 1997). The study has two obvious shortcomings: lipid oxidation following a high-intensity PA was still increased, indicating EPOC after high-intensity PA lasts longer than three hours, which in turn indicates that the EPOC period was not measured in its entirety, and, secondly, the effect of menstrual cycle was not taken into account. EPOC and lipid oxidation after PA (60 minutes of cycling at 60 %  $VO_{2max}$  intensity) are significantly greater in luteal than in follicular phase of the menstrual cycle (Matsuo et al., 1999). Not controlling the menstrual cycle could be the reason why no statistically significant differences were found in the amount and duration of EPOC (women cycling at various intensities) (Sedlock, 1991a). Normalising absolute EPOC value to lean mass results in disappearing gender differences (Tahara et al., 2008; Lamont et al., 2010).

### Effects of Amount of Fatty Tissue, Food Ingestion and Fitness Level on Lipid Oxidation after Activity

EPOC in lean people lasts longer and is greater by one third than in overweight and obese people. Overweight and obese people have significantly higher RER value, which indicates that during the regeneration phase they use less lipids and that PA has a reduced effect on lipid oxidation increase during rest than in lean people (Wong & Harber, 2006). Similar conclusions were drawn in an earlier study; it was found that during rest neither low-intensity PA (40 %  $VO_{2max}$ ) nor moderate-intensity PA (70 %  $VO_{2max}$ ) resulted in fatty acid oxidation increase in obese people (van Baak, 1999). Ingesting food prior to moderate-intensity PA had no effect on the EPOC amount and duration (Bahr & Sejersted, 1991b).

Regular PA results in increased recovery effectiveness following PA (Hagberg, Hickson, Ehsani, & Holloszy, 1980): blood lactate level decreases, as does rectal temperature and hormonal response (Sedlock et al., 2010), thus reducing the EPOC duration (Frey et al., 1993; Sedlock, 1994; Short & Sedlock, 1997). The amount of EPOC when comparing relative values is identical (Kaminsky et al., 1986; Sedlock, 1994; Short & Sedlock, 1997; Børsheim & Bahr, 2003; Sedlock et al., 2010). If we compare absolute values, EPOC amount following a regular PA is significantly reduced (Sedlock et al., 2010), while the ability to metabolise fatty acids increases (Kaminsky, Knowlton, Perkins, & Hetzler, 1986; Short & Sedlock, 1997; Børsheim & Bahr, 2003; Gill et al., 2006; Ferreira et al. 2011).

Energy expenditure was significantly higher during rest even up to 48 hours after moderate-intensity PA (60 minutes running at 70 to 75 %  $VO_{2max}$  intensity) (Jamurtas et al., 2004). In spite of all this, we must be aware that EPOC represents only up to 10 % of energy expended during PA (LeCheminant et al., 2008) or, depending on PA duration (from 20 to 80 minutes) and intensity (30, 50 or 70 %  $VO_{2max}$ ) only 1 to 8.9 % of energy expended during PA (Gore & Withers, 1990). When considering lipid oxidation in recovery phase after PA, the increased lipid oxidation can be observed during most of the EPOC period. Intensity of PA is exponentially related to the EPOC level, the duration of PA on the other hand is in linear relation to the EPOC. Dividing PA into sets causes greater EPOC than if the PA is in a single set. EPOC in lean people lasts longer and is greater. Normalising absolute EPOC value to lean mass results in disappearing gender differences, however, lipid oxidation after PA is significantly greater in luteal than in follicular phase of the menstrual cycle. If we compare absolute values, EPOC amount following regular PA is significantly reduced when comparing relative values is identical, while the ability to metabolise lipids increases. The amount of active muscles as well as ingested food prior to PA has no effect on the amount and duration of EPOC.

## INTERVAL TRAINING AND LIPID OXIDATION

During a high intensity PA, oxidation of plasma fatty acids, muscle and plasma TGs are lower than during a moderate intensity PA, despite greater energy expenditure (van Loon et al., 2001; Jeppesen & Kiens, 2012). Plasma glucose and muscle glycogen utilization is directly proportional to PA intensity (Romijn et al., 1993; van Loon et al., 2001), while the mechanism balancing lipid oxidation has yet to be explained (Jeppesen et al., 2011). “Carnitine could act as an acceptor of acetyl groups from acetyl-CoA, by forming acetylcarnitine, a reaction catalyzed by the mitochondrial enzyme carnitine acetyltransferase, when acetyl-CoA is generated faster than utilized by the Krebs cycle” (Jeppesen & Kiens, 2012). With increasing exercise intensities, muscle acetylcarnitine content is increased concomitantly with a decrease in the free carnitine content (Jeppesen & Kiens, 2012). On the other hand, a low muscle content of free carnitine is supposed to lead to a diminished supply of the long chain fatty acid CoA to  $\beta$ -oxidation, limiting long chain fatty acid oxidation during high intensity exercise. Thus, an increased availability of pyruvate, acetyl-CoA formation, and ‘binding’ of the free carnitine during high intensity exercise also provide a potential mechanism, whereby fatty acid oxidation is down-regulated” (Jeppesen & Kiens, 2012). A comparison was made between lipid oxidation in untrained boys (aged 8 to 12), during and two hours after PA (30 minutes of cycling at intensity that maximises lipid oxidation determined for individual boy) and during and two hours after PA to which high-intensity short-term burst was added (cycling at intensity that utilized maximum lipids and every two minutes four seconds of maximum intensity). It was found that the amount of lipids metabolised and the EPOC amount were the same, for the same work performed (Crisp, Fournier, Licari, Braham, & Guelfi, 2012). The same EPOC amount and the duration was found in adult men as well following interval PA (three minutes of cycling at 30 %  $VO_{2max}$  intensity and two minutes at 90 %  $VO_{2max}$  intensity, seven repetitions) and continuous moderate-intensity PA ( $\approx$  30 minutes of cycling at 65 %  $VO_{2max}$  intensity) for the same work performed. RER values were significantly lower in the 2-hour period following interval PA (McGarvey, Jones, & Petersen, 2005). Absolute values of metabolised fatty acids during and following the exercise were, unfortunately, not measured, so we still do not know whether lipid oxidation during and following a continuous PA is lower, equal or higher than during and after an interval PA for the same work performed. However, Hazell, Olver, Hamilton, and Lemon (2012) have recently demonstrated a similar total  $VO_2$  over 24 hours after sprint (high-intensity) interval exercise (four times 30-second maximal cycling at resistance of 10 % body mass with four-minutes rests) and continuous endurance exercise (30 minutes cycling at  $\sim$  70 %  $VO_{2max}$ ) session (SIE =  $498.0 \pm 29.4$  L; CEE =  $500.2 \pm 49.2$  L; CTRL =  $400.2 \pm 44.6$  L), indicating that the significant body-fat losses observed previously with sprint interval trainings are partially due to increases in oxidation post exercise.

Comparing the two hours following the high-intensity short-term burst of activity (four repetitions of 30-second intervals of sprinting on a cycle ergometer and 4.5 minutes of rest) to resting, a 75 % lipid oxidation increase was found (Chan & Burns,

2013). The EPOC duration and the amount following the interval PA depends on a number of high-intensity short-term burst (Bahr, Grønnerød, & Sejersted, 1992) and work to rest ratio (Gosselin, Kozłowski Devinney-Boymel, & Hambridge, 2012). After two minutes of cycling at 108 %  $VO_{2max}$  intensity, EPOC amount is  $\approx$  30 kcal and lasts 30 minutes; after 2 series with a 3-minute break, EPOC amount is  $\approx$  35 kcal and lasts 60 minutes; after 3 sets with two 3-minute breaks EPOC amount is  $\approx$  80 kcal and lasts 4 hours (Bahr et al., 1992). The highest energy expenditure and the lowest blood lactate values are found when using 30 seconds of moderate-intensity PA and 30 seconds of high-intensity short-term activity (Gosselin et al., 2012; Zuniga et al., 2011). In a recent study (Kelly, King, Goerlach, & Nimmo, 2013), two commonly used high-intensity short-term activity protocols (10 times 1 minute of high-intensity cycling followed by a one-minute rest and 10 times 4-minute high-intensity cycling followed by a 2-minute rest), supposedly suitable for untrained people and people suffering from chronic diseases, were compared. It was discovered that during the 60 minutes after interval exercises, RER was lower in both protocols. But during the slow phase of EPOC period (from 1.25 to 9.25 hours) there were no significant differences between RER values or energy expenditure. They conclude that the effect on post-exercise metabolic rate was transient and relatively minor (Kelly et al., 2013).

Interval training causes both central (cardiovascular) and peripheral (skeletal muscle) body adaptations (Gibala, Little, MacDonald, & Hawley, 2012). High-intensity short-term training (10 x 2 minutes 105 %  $VO_{2max}$  with 2 minutes breaks) increased the anaerobic threshold significantly more than endurance training (50 %  $VO_{2max}$ , 55 minutes or 70 %  $VO_{2max}$ , 35 minutes) in the same time period (Poole & Gaesser, 1985). Modern studies have come to similar conclusions. Just two-week high-intensity intermittent training (7 sessions every second day, each session consisted of 10 4-min cycling bouts at 90 %  $VO_{2max}$  separated by 2 minutes of rest), increased post-training whole body lipid oxidation during the 60 minutes of cycling at 60 %  $VO_{2max}$  in moderate trained women (menstrual cycle was not monitored) (Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007). Studies show that low-volume high-intensity interval training increases a body's capability of lipid oxidation just as effectively as higher-volume moderate-intensity endurance training (Gibala et al., 2006; Gibala & McGee, 2008). However, considering time, interval training is a more time efficient strategy than continuous training (Burgomaster et al., 2008). Interval training is safe for healthy people as well as people "at risk" (Shiraev & Barclay, 2012). Studies on obese and metabolic syndrome patients report a similar or a greater effect of high-intensity interval training compared to endurance training on life quality, cardio-metabolic risk factors, aerobic fitness and cardio-vascular function, as well as blood glucose reduction, insulin signaling in adipose tissue and skeletal muscles, lowering the mean arterial blood pressure, and finally, on adipose tissue lipogenesis reduction, body weight and adipose tissue reduction (Earnest, 2008; Guiraud et al., 2012; Hansen, Dendale, van Loon, & Meeuseen, 2010; Kessler, Sisson, & Short, 2012; Tjønnå et al., 2008). In practice, this translates to similar or greater effect, for at least 60 % less time spent doing high-intensity interval training compared to endurance training. This is a very significant aspect considering

that “a lack of time” is the most commonly cited obstacle for participating in regular PA (Stutts, 2002; Kimm et al., 2006). When considering lipid oxidation, interval training is at least as effective as endurance training, with less time invested. Some people also find it more interesting.

## RESISTANCE TRAINING AND LIPID OXIDATION

Muscles do not utilize lipids during resistance exercises (Melby, Scholl, Edwards, & Bullough, 1993). Resistance exercises are first ensued by anaerobic regeneration, the so-called “delayed glycolysis”, a glycolytic process at the start of regeneration. It is significantly faster and more effective at phosphagens re-synthesis than at aerobic processes. It ensures re-synthesis of depleted phosphagens, up to the levels allowing oxidative processes to allow sufficient regeneration (Margarita et al., 1933, in di Prampero & Feretti, 1999). An increase in lipid oxidation has been found during EPOC period 30 to 120 minutes after finishing a resistance exercise (Binzen, Swan, & Manore, 2001). Intensity has been identified as a significant factor in EPOC amount following resistance exercise (Thornton & Potteiger, 2002) as well as the speed of execution (Scott, 2012) and the size of active muscle groups, a slight dependency on breaks between sets was also observed (Farinatti & Castinheiras, 2011; Haltom et al., 1999). No dependency was found towards the order in which the resistance exercise is carried out (Da Silva, Brentano, & Krueel, 2010), or the type of muscle contraction (Scott, 2012). During the first two hours following resistance exercise (2 sets, 8 repetitions at 85 % 8 RM), the EPOC amount was significantly higher than after the activities for strength endurance (2 sets, 15 repetitions 45 % 8 RM) at equal work carried out. No difference in RER was found following the resistance exercise or the activities for strength endurance (Thornton & Potteiger, 2002). Absolute lipid oxidation values were not measured, so we cannot conclude whether lipid oxidation after resistance exercise differs from those following activities for strength endurance. EPOC amount is dependent on execution speed of resistance exercises; it is significantly greater during slower execution (4s lift / 1s release or 1s lift / 4s release) in comparison to faster execution (1.5 second lift / 1.5 second release) (Scott, 2012). The EPOC amount following the resistance exercise of larger muscle groups is significantly greater than following resistance exercise of smaller muscle groups at equal repetitions and unequal work performed. Duration of rest between sets (1 or 3 minutes) had no effect on EPOC in larger muscle groups (Farinatti & Castinheiras, 2011). A shorter break between sets (20 seconds; 2 circular exercises for the upper and lower part, 20 repetitions 75 % 20 RM) resulted in significantly greater EPOC amount compared to same resistance exercise with a 1-minute break (Haltom et al., 1999). EPOC amount during the first 2 hours following resistance exercise was  $\approx 30$  kcal (Binzen et al., 2001). Part of these calories is used for delayed glycolysis and part for lipid oxidation.

Regular resistance training, however, increases muscle mass, therefore increasing oxidation rate during rest as well as absolute lipid utilisation (Dolezal, Potteiger, Jacob-

sen, & Benedict, 2000). Significantly, increased oxidation was found even 38 hours after the resistance training (31 minutes, 10 repetitions, maximum until exhaustion, for the entire body) (Schuenke, Mikat, & McBride, 2002), and 24 hours following the resistance training (60 minutes, 70 – 75 %, 1 repetition maximum, for the entire body) (Jamurtas et al., 2004). Heden (2011) found that in overweight people, there was a  $\approx 5\%$  increase in oxidation rate up to 72 hours during rest after the resistance training, following the recommendations of the American Society for Sport Medicine (Haskell et al., 2007; Heden, 2011).

Measuring local fatty acid oxidation during resistance exercise for the individual muscle groups (Dean et al., 2000) and (Helge, Stallknecht, Richter, Galbo, & Kiens, 2007) concluded that resistance exercise caused no noticeable hormonal changes and enabled exceptionally good circulation and oxygen supply to active muscles. By monitoring isotopes, muscle biopsies, oxygen consumption and RER during unilateral extension of the knee against resistance intensity ranging from 25 % to 85 % of maximum power –  $P_{\max}$ , the total fatty acid oxidation in the thigh increased 15 times (at 25 %  $P_{\max}$  intensity) compared to rest, and remained the same regardless of the intensity. Free fatty acid oxidation in the thigh increased with resistance exercise intensity, reducing oxidation of plasma TG and muscular TG. At 100 %  $P_{\max}$  intensity fatty acid oxidation capability decreased by a third compared to 85 %  $P_{\max}$  intensity. These results contradict activities that incorporate the muscles of entire body, such as cycling, where maximum lipid utilisation is at 65 %  $VO_{2\max}$ . The reason may lie in significantly greater blood circulation in the active muscles, when we perform resistance exercise for individual muscle groups than when we perform PA that includes greater muscle mass. This “super fusion” creates muscle environment that retains free carnitine and promotes lipid oxidation processes even at 80 %  $P_{\max}$  intensity. At intensities exceeding 80 %  $P_{\max}$ , the availability of free carnitine decreases, heavily diminishing the ability of lipid oxidation (Jeppeson & Kiens, 2012).

When combining endurance PA with resistance exercise it was found that the order of PA and the resistance exercise execution had no effect on the size and the duration of EPOC (Drummond, Vehrs, Schaalje, & Parcell, 2005; Oliveira & Oliveira, 2011). The order, however, did have an effect on RER. Execution of endurance PA prior to resistance exercise resulted in significantly lower RER, resulting in higher lipid oxidation, compared to the reverse order of endurance PA following resistance exercise (Oliveira & Oliveira, 2011). Resistance exercise (3 series at 70 % 1 repetition maximum, 10 repetitions, 105-second break between series or exercises, 7 exercises for upper and lower body), moderate-intensity PA (25 minutes of running at 70 %  $VO_{2\max}$  intensity), combination of moderate-intensity PA and resistance exercise (there was a 5-minute break in between) or resistance exercise and moderate-intensity PA, resulted in the same EPOC duration ( $\approx 40$  minutes) following the session (Drummond et al., 2005). When considering lipid oxidation, the total amount of lipids utilized during and following resistance exercise is negligible compared to lipid oxidation during and following the endurance PA or interval training, but regular resistance training increases muscle mass, therefore increasing oxidation rate and lipid utilisation during rest.

## CONCLUSIONS

Prolonged (> 30 minutes) moderate intensity (55 – 70 %  $\text{VO}_2\text{max}$ ) PA, such as walking, jogging or cycling, are the most effective way to increase lipid oxidation during and after a single exercise session. Studies also suggest no difference in lipid oxidation between periods following low-volume high-intensity interval exercise and traditional continuous endurance PA. However, time benefit of low-volume, high-intensity interval exercise compared to traditional endurance PA is significant. Further research is required to confirm whether adding just a few seconds of sprints during moderate-intensity endurance PA every so often would result in the same lipid oxidation as during a continuous PA without sprint intervals.

Regularity of PA seems most important. Regular continuous and regular interval exercise or activity triggers the same adaptation mechanisms, and increases one's fatty acid mobility, transport and oxidation capability. Further research is necessary to compare the effects of regular resistance, regular endurance and regular interval training on lipid oxidation effectiveness. Both regular endurance and regular interval training increase a certain amount of muscle mass, therefore increasing resting oxidation. It may also be of interest to compare the effects of Nordic walking, which additionally activates upper limb muscles in the movements and, thus, more muscle mass, as well as resistance exercise of the entire body on the effect of lipid oxidation.

Nutrition also significantly affects lipid oxidation. Food rich in fat and low in carbohydrates encourages lipid oxidation. It is essential not to exceed body's daily energy requirements, which leads to increased body weight and obesity. Obese and overweight people are recommended to take part in regular endurance or interval training. However, from the standpoint of relieving lower limbs joints, it may be better to participate in cycling or swimming, despite lower lipid oxidation potential.

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