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# Excess Postexercise Oxygen Consumption and Interval Training

Shinichi Asano

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EXCESS POSTEXERCISE OXYGEN CONSUMPTION AND INTERVAL TRAINING

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Marshall University

In partial fulfillment of the  
Requirements for the degree of  
Master of Science  
in Exercise Science  
in the Division of Exercise Science, Sport and Recreation

by

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EXCESS POSTEXERCISE OXYGEN CONSUMPTION AND INTERVAL TRAINING

Abstract

The effect of exercise intensity on excess postexercise oxygen consumption (EPOC) was determined in 18 to 30 year old apparently healthy individuals. Subjects participated in 3 different exercise sessions; aerobic exercise, interval exercise (IE) and high intensity interval exercise (HIIE), on separate days. EPOC was measured one hour after each exercise while subjects were in supine position. ANOVA with repeated measurements was used to assess differences. The mean values ( $\pm$  SEM) for EPOC of aerobic exercise, IE and HIIE were 2.106( $\pm$  0.219), 2.846 ( $\pm$  0.309) and 4.969 ( $\pm$  0.522) l·hour<sup>-1</sup>. There was no significant difference ( $p < 0.05$ ) between mean EPOC of aerobic exercise and IE, however, a significant difference ( $p < 0.05$ ) was found in mean EPOC of HIIE when it was compared with both aerobic exercise and IE. These data suggest that exercise intensity has a significant effect on EPOC.

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## Chapter 1

### INTRODUCTION

The law of thermodynamics explains the general application of changing caloric intake and expenditure to affect weight loss, or weight gain in the dieting individual. However, typical weight control and exercise regimes focus on prolonged submaximal exercise prescriptions that are specific for substrate utilization during the exercise session. More specifically, some personal trainers and exercise enthusiasts devote an inordinate amount of time and energy to make sure the overwhelming majority of substrate utilization is fat. This seems appropriate to them because fat is utilized as the main fuel; however, when post exercise energy expenditure is considered, the concept of a fat utilization program may not be the most efficient way to promote weight loss.

Typical aerobic fat burning programs may have only a small effect on weight control when observed from a holistic perspective. It may be that emphasis should not be placed on the exercise itself, but on post exercise energy expenditure. High intensity exercise requires more energy during the activity, and also it may induce greater energy expenditure during postexercise recovery. Thus, for weight reduction purposes, it may be more effective to choose an exercise program that induces additional energy expenditure after the exercise.

### Hypothesis

It was hypothesized that:

1. There will be greater total energy expenditure in high intensity interval exercise (HIIE) compared to typical aerobic exercise during exercise bout.

2. High intensity interval exercise will induce the greatest excess post exercise oxygen consumption (EPOC).

#### Null Hypothesis

1. There will be no significant difference in EPOC among 3 different exercise sessions.

#### Purpose of the Study

Aerobic exercise is widely believed to be the most effective exercise prescription for weight reduction, and therefore, exercise enthusiasts commonly use aerobic exercise for their weight reduction programs (Bouchard, Despres, & Tremblay, 1993). However, this does not follow the thermodynamic concept of weight reduction. A great deal of time is spent on exercise because the focus is on substrate utilization and little else. Carbohydrate is the main fuel during high intensity exercise; it requires higher energy expenditure and also creates a greater EPOC. Focusing on higher energy expenditure and greater EPOC may be more important than staying in the fat burning zone during exercise. This extended period of increased metabolism contributes additional energy expenditure that seems to have important implications for individuals using exercise as a means of body weight reduction. Moreover, this type of exercise solves the time consumption issue, because it takes much less time to achieve a specific demand of energy expenditure. Therefore, the purpose of this study is to determine if high intensity interval training results in a higher total energy expenditure compared to aerobic exercise, and to determine the type of exercise that induces the highest EPOC.

## Significance of the Study

According to the data from the National Center for Health Statistics by the Center for Disease Control (CDC), the average height of men and women increased one inch from 1960 to 2002, while average weight increased 25 pounds and 24 pounds in men and women respectively. (“Americans Slightly Taller, Much Heavier Than 40 Years Ago”, 2005). Since weight is increasing proportionally faster than height in the general population, understanding the principles of weight reduction is crucial. Moreover, weight control is also important in the athletic and medical fields. Weight reduction leads to improved athletic performance, reduced risk of cardiovascular or metabolic disease, and increased self-esteem.

Unfortunately at this time, some businesses are taking advantage of the demand for weight loss. Some companies advertise magic pills that are supposed to increase fat metabolism without scientific evidence of effectiveness or safety (Saper, Eisengerg, & Phillips, 2004). Additionally, fit looking people on television and in advertisements recommend aerobics indiscriminately to every population, which only serves to perpetuate the fat burning exercise myth. However, specific exercise programs should be tailored to specific populations if they are to be beneficial to the individual.

This study is significant because high intensity interval exercise may have two superior benefits that allow healthy individuals to work at a higher intensity than normally would not be sustainable, and provide a high percentage of anaerobic metabolisms which is believed to cause a higher magnitude of EPOC. These factors may allow us to achieve higher total energy expenditure from exercise and exercise induced physiological reactions. Literature in regard to the effects of high intensity interval

exercise on EPOC of a particularly specific number of energy expenditure from a single exercise bout only exists in limited availability. Thus, this study will evaluate the idea that high intensity exercise is a better program than aerobic exercise for weight reduction demands.

### Limitations

The study was limited by the following factors:

1. Although subjects have exercise experience, exercising while connected to the metabolic cart may lead to apprehension.
2. A small sample size was selected from one local area.
3. Even though exercise is prohibited for 24 hours before the exercise sessions, there is a limit of controlling pre exercise condition to exact same situation. This may affect measurements of EPOC.
4. Fluctuation of body weight may affect resting metabolic rate.
5. During the post exercise oxygen consumption, subjects may not be able to relax completely.
6. Subjects may fall asleep which may cause abnormal ventilation or movement during the post exercise oxygen consumption assessment.
7. Environmental conditions including temperature, humidity, ambient noise, and psychological condition may affect the result of the study.

### Definitions

For consistency of interpretation, the following terms are defined:

*Basal metabolic rate.* Requirement of a minimum level of energy to sustain vital functions in the waking state (McArdle, Katch, & Katch, 2001, pg. 188).

*Diet-induced thermogenesis.* Increased energy metabolism from food consumption (McArdle et al., 2001, pg. 192).

*Direct calorimetry.* The measurement of heat production as an indication of metabolic rate (Powers & Howley, 2001, pg. 105).

*Energy substrates.* Molecules that provide starting materials for bioenergetic reactions, including phosphagens (adenosine triphosphate (ATP) and creatine phosphate), glucose, glycogen, lactate, free fatty acids, and amino acids (Baechle & Earle, 2000, pg. 83).

*Excess postexercise oxygen consumption (EPOC).* Oxygen uptake above resting values used to restore the body to the pre-exercise condition.

*Indirect calorimetry.* Estimation of metabolic rate via the measurement of oxygen consumption (Powers & Howley, 2001, pg. 105).

*Interval training.* Performance of repeated exercise bouts, with brief recovery periods in between.

*Lactate threshold (LT).* The highest oxygen consumption or exercise intensity with less than a 1.0 mM per liter increase in blood lactate concentration above the preexercise level (McArdle et al., 2001, pg. 291).

*Maximal oxygen consumption ( $\dot{V}O_2$  max).* The point in which oxygen consumption plateaus or increases only slightly with additional increase in exercise intensity (McArdle et al., 2001, pg. 162).

*Onset of blood lactate accumulation (OBLA).* Blood lactate shows a systematic increase equal to 4.0 mM (McArdle et al., 2001, pg. 291).

*Oxygen deficit.* The difference between the total oxygen actually consumed during exercise and the total that would have been consumed had steady-rate aerobic metabolism been reached from the start (McArdle et al., 2001, pg. 161).

*Oxygen debt.* Oxygen uptake above the resting state following exercise based on lactic acid theory.

*Respiratory compensation.* The second line of defense against pH shift during exercise for metabolic acidosis (Powers & Howley, 2001, pg. 218).

*Respiratory exchange ratio (RER, R).* The ratio of carbon dioxide produced to the oxygen consumption ( $VCO_2/VO_2$ ) (McArdle et al., 2001, pg. 185).

*Resting metabolic rate (RMR).* The sum of metabolic processes of the active cell mass required to maintain normal regulatory balance and body functions at rest (McArdle et al., 2001, pg. 188).

*Total daily energy expenditure.* Energy expenditure includes resting metabolic rate, which consists of basal and sleeping conditions plus the added metabolic cost of arousal, thermogenic effect of the food, and energy expenditure during physical activity and recovery (McArdle et al., 2001, pg. 188).

*Ventilatory threshold.* The point where pulmonary ventilation increases disproportionately with oxygen consumption during grade exercise (McArdle et al, 2001, pg. 291).

### Assumptions

It was assumed that subjects filled out the screening sheet about their health and exercise history honestly and to their best knowledge. Also, it was assumed that each subject followed the overnight fasting condition instructions and avoided strenuous

exercise before the each test. Therefore, all three pre-exercise conditions were equivalent. In addition, subjects were not on any type of medication or supplement that might affect heart rate (HR) and metabolism for example, thyroid medication, ephedrine, and so on. During each RMR and EPOC assessment, subjects were fully relaxed each time. Additionally, physical and psychological stresses were maintained equally so that the comparison of EPOC was made by based on the different exercise intensities.

## Chapter 2

### REVIEW OF LITERATURE

This literature review will describe the concept of weight reduction. Secondly, this review will focus on the physiological responses induced by high intensity interval exercise.

#### Resting Metabolic Rate

The first law of thermodynamics is defined “energy cannot be created or destroyed but, instead, transforms from one to another without being depleted” (McArdle et al., 2001, p.116). When applying this system to human energy balance, energy intake represents food the individual consumes, and the energy expenditure which includes RMR, daily activity, dietary thermodynamic effect and recovery from exercise. RMR represents the amount of energy that the human body requires to maintain involuntary physiological functions including respiration, cardiac output, body temperature regulation, and other functions of the sympathetic nervous system. RMR accounts for 60 to 75% of total daily energy expenditure, while the other roughly 25 to 40% may be composed of agents that add to the total daily energy expenditure such as the thermic effect of food, and thermic effect of physical activity.

Many methods are commonly used to calculate energy expenditure including measuring the actual heat production of the human body, which gives the most accurate energy expenditure. However this method involves greater cost, effort, and nuisance. Indirect calorimetry also measures heat production from a known amount of gas exchange associated with oxidation of substrate and provides a less expensive and more convenient alternative. In addition, the values of difference in direct and indirect

calorimetry remain minute, even though direct calorimetry is considered the gold standard. Therefore, the indirect calorimetry method is applicable in some specific instances. The number of calories expended to burn one liter of oxygen depends on the ratio of carbohydrate to fat utilized. 5.0 kcal per one liter of oxygen is accepted as a universal value, understanding that the actual caloric expenditure may vary slightly. Therefore, measuring oxygen consumption is a function of energy expenditure and substrate utilization in a variety of situations from daily activity through exercise.

When measuring RMR, conditions are strictly regulated to avoid factors that affect RMR including time of the day, prior exercise, food consumption, room temperature, and stress. A study showed that RMR was approximately 100 kcal higher when RMR measurement was conducted in the afternoon after 4 hour of fasting compared to the measurement in the morning after 12 hours of fasting (Haugen, Melanson, Tran, Kearney, & Hill, 2003). Thus, the time of the day is usually set in the morning, eliminating possible additional energy expenditure from thermic effect of food and physical activity. In addition to these external factors, there also internal factors that affect result of RMR. Although several physiological factors such as thyroid hormone, and catecholamine may play a role, the volume of free fat mass (FFM) contributes the major factor in increasing RMR. Simply put, the more muscle tissue you have, the more calories you burn. Because of this, females generally have a lower RMR due to lower FFM; however, there is no gender difference when FFM is considered per unit of cross sectional area. Since RMR accounts for the main total daily energy expenditure, increasing or maintaining RMR is important in order to achieve a negative energy balance.

According to the law of thermodynamics, caloric restriction is necessary for weight reduction; however, caloric restriction itself causes not only decreased energy intake but also a decline of RMR that negatively affects long term weight reduction purposes. One study (Lennon, Nagle, Stratman, Shargo, & Dennis, 1985) has shown that a self-selected aerobic exercise group and a prescribed exercise group with diet restrictions increased their RMR, while a restricted diet only group decreased their RMR. In order to prevent RMR from decreasing, which may result in positive energy balance, exercise is crucial for individuals who are on a restricted diet, and even participating in a low intensity activity has a positive effect (Lennon et al., 1985).

#### Thermodynamic Effect of Food

Though individuals may differ, the dietary thermodynamic effect generally makes up about 5 to 10% of the total energy an individual consumes (Wardlaw, 2003). Specifically, this extra energy is utilized to digest, absorb, metabolize nutrients and contribute to the activation of sympathetic neural activity known as dietary induced thermogenesis. Efficient dietary thermogenesis allows people to store less food as fat, instead they metabolize food effectively.

The thermogenesis of food is not only influenced by the body, but also from the kinds of food that we eat. It is beyond the focus of this study to mention the thermodynamics of specific nutrients; however, it should be noted briefly since consumers are flooded with a variety of diets. Generally, carbohydrates and protein have a higher thermodynamic effect than fat has because they require more energy to metabolize to specific form, specifically, protein has the highest thermic effect, burning off about 25 to 30% of the calories consumed, while about 6 to 8% of the calories from

carbohydrates is burned off and thermic effect of fat is less than 3%. (Karst, Steiniger, Noack, & Steglich, 1984). Out of the many popular diets today, a variety of them manipulate the ratio of these macronutrients which can have an effect on weight loss, partially due to the thermic effect of feeding.

#### Thermic Effect of Physical Activity

The last factor that has an effect on total daily energy expenditure is daily activity and exercise. The benefit of using exercise combined with caloric restriction as opposed to caloric restriction alone prevents loss of lean body mass when weight loss occurs. In the long term perspective, maintaining FFM gives an advantage over losing FFM due to the relationship between the amount of FFM and RMR. It may be that physically active individuals appear lean because extra calories are required to match the imposed demand of increasing or maintaining FFM. For instance, oxygen consumption is elevated during exercise, promoting more energy expenditure to be included in total daily energy expenditure. Oscai, Spirakis, Wolff, and Beck (1972) showed exercise with food restriction induced fewer and smaller fat cells in rats. Therefore, exercise is recommended for weight control for both energy balance and biomechanical aspects.

A negative relationship exists between exercise intensity and fat utilization during exercise. Almost all energy expenditure comes from fat at 25% of  $VO_2$  max, however, the energy turnover is small, and thus absolute energy expenditure from fat seems to be the highest between 50%-70%  $VO_2$  max (Jeukendrup, Saris, & Wagenmakers, 1998). Therefore, low-moderate intensity aerobic exercise has been preferably selected for weight control purposes. However, weight change is solely dependent on the balance of energy, despite the origin of the energy. Studies have shown

no change in subject's body weight or anthropometric characteristics when they consumed either a low fat diet or a high fat diet containing the same amount of calories (Leibel, Hirsh, Appel, & Checani, 1992; Hirsch, 1995). The general population often overlooks the isocaloric principle of the law of thermodynamics as evidenced by the prevalence of quackish advertisements, unscientific popular exercise and diet myths.

On the other hand, Tremblay, Despres, Leblanc, Craig, Stephens, and Bouchard (1990) analyzed a large sample size of data (male 1257, female 1366) which showed that people who participate in vigorous physical activity have less subcutaneous fat than people who don't participate in such activities. Interestingly, they also found the effect of exercise intensity on body fat and its distribution was not due to the energy cost of the activity, but from other components of energy balance. Tremblay, Simoneau, and Bouchard (1994) completed another study that revealed high intensity intermittent exercise or supramaximal exercise to be the most optimal for fat loss. Subjects participated in either an endurance training program for 20 weeks or a high-intensity intermittent training program for 15 weeks. The mean estimated energy expenditure of high intensity intermittent training was less than half of endurance training. Nonetheless, they found that high intensity intermittent training induces a greater loss of subcutaneous fat. Specifically, the decrease in six-site subcutaneous skinfolds tended to be greater in the high intensity intermittent training group than the endurance training group, regardless of the dramatically lower energy cost of training.

From these studies (Tremblay et al., 1990; Tremblay et al., 1994), it is clear that exercise intensity plays a role in weight control with diet restriction. One of the most plausible reasons for these results might be a longer and higher recovery  $O_2$  uptake

known as EPOC not usually considered in the energy cost of exercise. If manipulation of exercise intensity, duration, and type produces an altered effect on the post physiological reaction, the selection of efficient exercise may lead to a desirable outcome. In addition, high intensity exercise may suppress appetite. This phenomenon has been explained by an animal study in which rat food intake was decreased immediately after enforced exercise (Stevenson, Box, Feleki, & Beaton, 1966). Although this study did not specify the mechanism of this phenomenon, it mentioned adrenaline secretions from exercise as one possible factor of appetite suppression.

These findings suggest that the intensity of exercise may be the key for weight control, not substrate utilization during the exercise bout. Participating in a high intensity activity results in higher energy expenditure due not only to the energy requirement from the activity itself, but also the energy demand from recovery with possible appetite suppression. The magnitude of EPOC may be an important implication for an individual who uses exercise as a part of their weight reduction remedy.

## EPOC

### *Traditional Considerations*

Measurement of  $O_2$  consumption indicates aerobic ATP production, thus measurement of  $O_2$  consumption during exercise can provide information about aerobic metabolism. However, not all metabolic pathways require  $O_2$  to produce energy, and it is a mixture of three metabolic pathways that are responsible for energy turnover. For instance,  $O_2$  uptake does not reach the steady state immediately in the beginning of an exercise. Instead, the anaerobic energy system substitutes a delay known as oxygen deficit until the steady state has been achieved. Traditionally, recovery  $O_2$  uptake, termed

O<sub>2</sub> debt by the British physiologist A. V. Hill was considered a compensation of the oxygen deficit that represents the lack of O<sub>2</sub> uptake until steady state was achieved. Specifically, Hill theorized that recovery O<sub>2</sub> uptake represented as rapid decline immediately after exercise corresponds to the replenishment of muscle glycogen from the lactic acid produced by anaerobic metabolism, while slow decline reveals the oxidation of the remaining lactic acid for other tissues.

Later, Margaria transformed and modified Hill's O<sub>2</sub> debt theory into the alactacid oxygen debt phase and the lactacid oxygen debt phase in which the alactacid phase does not involve lactate, while the lactacid phase is related to lactate metabolism. More specifically, the rapid part of O<sub>2</sub> debt serves to resynthesize stored ATP, phosphocreatine (PC) and to replace O<sub>2</sub> in the tissue, while the slow part of O<sub>2</sub> debt represents gluconeogenesis (Gaesser & Brooks, 1984). Therefore, the two parts of O<sub>2</sub> debt depend on several divergent physiological reactions that evolved from Hill's original theory.

As more research techniques became available, the O<sub>2</sub> debt theory remains a controversial topic, and the term may be considered as a misnomer because of the concept of the human body replenishing borrowed oxygen. This theory may overestimate the involvement of anaerobic metabolism during exercise by measuring oxygen debt. Oxygen deficit is not equal to oxygen debt; therefore, the method utilized in studies to evaluate the degree of aerobic and anaerobic metabolism may provide more accurate information of energy turnover during exercise (Tabata, Irisawa, Kouzaki, Nishimura, Ogita, & Miyachi, 1997). According to the oxygen debt theory, approximately 75 to 90% of the lactate produced during exercise is converted to muscle glycogen while 10 to 25%

of lactic acid is oxidized in other tissue. However, recent studies state that the primary fate of lactate after the exercise is oxidation (Gaesser & Brooks, 1984). Replenishment of glycogen is mainly caused by food consumed not lactate produced during exercise.

Biochemically, it can not be declared that lactic acid is the only reason for recovery  $O_2$  uptake even though the connection between recovery  $O_2$  and blood lactate exists. A study showed that  $O_2$  consumption was significantly increased for 4 h after exercise, however, blood lactate and plasma norepinephrine concentrations were significantly increased for only 2 h (Bahr, Gronnerod, & Sejersted, 1992). The blood lactate was removed in less than 60 minutes while recovery  $O_2$  uptake was elevated for more than 12 hours (Bahr, 1992), suggesting that blood lactate was dissociated from recovery  $O_2$  uptake at some point. The main point of this argument was whether or not the elevated  $O_2$  uptake was caused mainly by lactic acid and if so, how much of that part is caused by lactic acid. There seem to be several other biochemical factors that may cause the elevated  $O_2$  uptake including hormonal activity, body temperature regulation, and sympathetic nervous system activity. Thus, the present consideration of recovery  $O_2$  uptake is related to physiological processes including not only the lactate specific issues, but also other physiological reactions that return the human body to homeostasis. EPOC is a better term than  $O_2$  debt since recent studies have shown that lactic acid is not the only consideration in recovery oxygen uptake. Also, recent studies have abandoned the financial term concept of the relationship between borrowed  $O_2$  and paying back  $O_2$ . Although oxygen debt theory may be rejected in the future, a new theory has not yet been completed. Therefore, the basis of traditional oxygen debt theory is still accepted.

### *Methodological Issues*

Even though EPOC is minute, it may play in substantial role in making a potentially crucial difference in overall energy expenditure; therefore, precise controls of pre/during and post exercise condition are necessary to obtain valid data. Also, several methodological differences in measuring EPOC exist that may explain the presence of a variety of results. Some studies (Hagberg, Mullin, & Nagle, 1980; Kaminisky, Kanter, Lesmes, & Laham-Saeger, 1987; Sedlock, Fissinger, & Melby, 1989) use the net  $VO_2$  calculated by gross  $VO_2$  and pre-exercise resting value, while others (Bahr, Ingnes, Vaage, Sejersted, & Newsholme, 1987; Bahr, & Sejersted, 1991; Maehlum, Gradmontagne, Newsholme, & Sejersted, 1986) utilize a control experiment that measures  $VO_2$  while subjects are at rest. Moreover, Quinn, Vroman, and Kertzer, (1994) used control  $VO_2$  measurement to make sure pre-exercise baseline values of other exercise sessions were not significantly different from the control value. On top of that, there are differences in obtaining baseline data. Quinn et al. (1994) utilized a 15 minute baseline after 30 minutes of bed rest, while other studies (Sedlock et al., 1989; Maehlum et al., 1986) used one hour of baseline data; in addition, subjects were transported to the laboratory by car to avoid any physical activity in several studies. (Bahr et al., 1987; Bahr, & Sejersted, 1991; Maehlum et al., 1986) There also may be a slight difference between data collected with subjects in the supine position and in the seated position.

Because of the methodological issues mentioned above, several studies failed to show the relationship between EPOC and exercise intensity or duration. For example, there was no difference in the magnitude of EPOC between 30 minutes of continuous exercise at 70% of  $VO_2$  max and 15 one minute exercise bouts at 100% of  $VO_2$  max

with 1 minute of rest between each bout (Parmenter, Manore, & Daniels, 2001). On analysis the data, this study rejected the theory that high intensity intermittent exercise produces greater EPOC than moderate continuous exercise. However this idea is not logical for several reasons. First, moderate continuous exercise did not induce EPOC in more than 30 minutes, whereas high intensity exercise of EPOC was elevated until 60 minutes. However magnitude of 2 hour EPOC was not different between them ( $6.1 \pm 4.1$  L,  $7.5 \pm 2.8$  L). Secondly, statistically there was no difference, but a difference exists between them. High standard deviation in moderate continuous exercise explains the inconsistency of data.

A study by Bahr, and Sejersted (1991) included actual measurement of the thermodynamic response of food when feeding subjects during recovery. EPOC during recovery is hard to measure due to intra/inter-individual variability of the thermodynamics of feeding. In order to gain proper EPOC results from a study including thermodynamic effect of feeding, they pointed out the following three possible mistakes of studies that failed to document any interaction of food and exercise on energy expenditure:

1. The intensity and duration of exercise need to be sufficient.
2. Meal size also needs to be adequate.
3. Highly trained individuals who have developed energy-sparing mechanisms need to be eliminated.

These differences mentioned above, in conjunction with the difficulties in controlling prior and post exercise conditions, make the comparison of results difficult.

## *Mechanisms*

### *Rapid phase EPOC*

Several physiological factors contribute to recovery  $O_2$  uptake; however, some of them still are unknown. Commonly, respiratory rate, HR, ATP and CP restoration, lactate removal and temperature regulation are well defined as mechanisms of rapid EPOC (Børsheim, & Bahr, 2003). The changes of ventilation and HR after the submaximal exercise are similarly affected and both measurements decrease to resting values within one hour (Bahr, 1992). In addition, blood lactate levels also returned to resting value within one hour, which was considered as main idea of the traditional oxygen debt theory.

Rectal temperatures also return to resting values in less than one hour following 80 minutes of exercise at 75% of  $VO_2$  max in non-athletic but physically active individuals (Bahr & Sejersted, 1991). Quinn et al. (1994) also confirmed that the core temperature elevation from three different durations (20, 40, 60 minutes) of exercise at the same intensity for well trained women exists for one hour. Evidently this temperature regulation increases recovery metabolism.

Even in the rapid phase of recovery, the relative contribution of each of these factors may be difficult to define, because so many factors are involved in and influenced by variables such as intensity, duration, type, environment, subject's training level, and so on. Since one hour seems to be the criteria for the diminishment of several physiological reactions, Børsheim and Bahr (2003) defined the rapid phase of EPOC in their review article as lasting only within one hour.

### *Slow phase of EPOC*

Compared to the rapid portion of EPOC, several factors have been associated with the recovery  $O_2$  uptake. However, the contribution of each factor remains unknown.

Although core temperature has been suggested as one of the factors that may affect both rapid and slow portion of EPOC, several studies have failed to show a major connection with core temperature during the slow portion of EPOC (Bahr et al., 1987; Frey, Byrners & Mazzeo, 1993; Maehlum et al., 1986) Bahr et al. (1987) examined the effect of exercise duration on EPOC. In this study, the rectal temperature of the subjects was elevated in all three exercise sessions (80, 40, and 20minutes). However, the temperature decreased rapidly and reached control values within 30 minutes in all exercise sessions, which may suggest that prolonged EPOC is not influenced by rectal temperature. Another study also found that EPOC was still present even though the core temperature returned to resting level after 40 minutes of low intensity exercise in trained subjects (Frey et al., 1993). Maehlum et al. (1986) examined magnitude and duration of EPOC, resulting in the elevation of rectal temperature and HR after the exercise, however, both rapidly dropped to normal values within 30 minutes. From these observations, it may be suggested that thermoregulation and HR do not influence prolonged EPOC.

Maehlum et al. (1986) also suggests that lower R values seen 24 hours after exercise indicates higher fat metabolism compared to the control group. The elevated plasma fatty acid level and plasma glycerol are consistent with R value. Bahr et al. (1987) also confirmed a similar 24 hour R value reaction after exercise. In this study, a linear decrease in R was seen when the duration of exercise was increased. A significant reduction of R was observed for 6 hours postexercise in both the feeding and fasting

exercise groups compared to control non exercise group. Since R values indicate indirect substrate utilization, these studies may suggest the elevated energy expenditure associated with increased fat metabolism can account for part of the slow phase of EPOC.

Several scientists have suggested that increased hormonal activities including insulin, cortisol, thyroid hormones, growth hormones, and catecholamine may play a role in the slow phase of EPOC (Børsheim, & Bahr, 2003; Gaesser, & Brooks, 1984; Maehlum et al., 1986) Since norepinephrine facilitates the Na/K pump that requires ATP for active transport, it may be logical to say that elevated oxygen consumption is a necessity for this energy turnover. Although Maehlum could not find a significant increase of insulin, cortisol or thyroxine in the recovery period, a transient increase in plasma cortisol was present for one hour after exercise.

Bahr (1992) found that there was an increase in plasma adrenaline and noradrenalin concentrations for 2 hours after 80 minutes of exercise at 75% of  $VO_2$  max. There are not many studies about hormonal responses during recovery; however, hormonal effects on EPOC may play a role because catecholamines are known to increase the rate of lipolysis.

#### *Factors of EPOC*

The magnitude and duration of EPOC may rely on the intensity and duration of exercise; it may also depend upon the environmental temperature or previous dietary history. In short term intense exercise, the amount of anaerobic metabolism involvement may be the key for the magnitude of EPOC. During very short term intense exercise (e.g. 5 seconds to 10 seconds); the primary metabolic pathway is ATP-PC which the replenishment of ATP and PC is considered as one of the factors in the rapid portion of

EPOC. The main metabolic pathway changes over to fast glycolysis and oxidative metabolic pathways when exercise lasts between 15 seconds to 3 minutes (Conley, 2000). As exercise intensity increases, the human body comes to depend more on anaerobic metabolism because aerobic metabolism becomes insufficient to supply energy. ATP-PC system depletes stored ATP and PC, while fast glycolysis produces lactic acid as a metabolic byproduct which disturbs homeostasis. Thus, exercise intensity is related to the replenishment of ATP/PC, and lactate metabolism. On the other hand, the energy to complete prolonged steady state exercise comes mainly from the oxidative metabolic pathway that does not induce ATP/PC depletion or lactate accumulation as much as the anaerobic metabolic pathway does.

### *Intensity*

Several studies have focused on the effect of exercise intensity on EPOC (Bahr, & Sejersted, 1991; Sedlock et al., 1989; Dooly, Reed, & Dotson, 1997). Although the EPOC was transient, the duration and magnitude of EPOC was significantly higher in high intensity exercise than low intensity exercise when energy expenditure of exercise was held constant (Dooly et al., 1997). In this study, 50% and 70% of  $VO_2$  max induced different durations of EPOC (15.2 min + 3.6, 21.8 min + 3.9) respectively. Although none of the exercises resulted in more than 35 minutes of EPOC, a study has shown that high intensity exercise induced EPOC more than twice as much as low intensity exercises when energy expenditure was held constant during exercise (Sedlock et al., 1989).

Bahr and Sejersted (1991) conducted a study that investigated the relationship between exercise intensity and EPOC. The EPOC threshold appears to be at least above the 50% of  $VO_2$  max. At 80 minutes of exercise, the least strenuous exercise intensity

(29% of  $\text{VO}_2$  max) had only a small amount of EPOC ( $1.3 \pm 0.46$ ), however, 50 and 75% of  $\text{VO}_2$  max had dramatically higher EPOC ( $5.7 \pm 1.7$ , and  $30.1 \pm 6.4$ ) respectively. In addition, this least strenuous exercise did not induce prolonged EPOC whereas significantly greater duration of EPOC ( $10.5 \pm 1.6$  hour) was observed after 80 minutes of exercise at 75% of  $\text{VO}_2$  max.

According to these studies, exercise intensity affects the magnitude of EPOC regardless of energy expenditure during exercise. Also, intensity of approximately 50% of  $\text{VO}_2$  max seems to be required to elicit significant recovery  $\text{O}_2$  uptake.

#### *Duration*

Chad and Wenger (1988) found that exercise durations of 30, 45 and 60 minutes have a significantly different effect on magnitude of EPOC. Specifically, when exercise duration was increased 1.5 and 2 times, EPOC increased by 2.35 and 5.3 fold respectively. Additionally, the longest duration of exercise resulted in over 7 hours of EPOC while 30 and 45 minutes induced approximately 2 and 3 hours of EPOC.

Bahr et al. (1987) examined the effect of exercise duration on EPOC, which resulted in  $14.4 \pm 1.2$ ,  $6.8 \pm 1.7$ , and  $5.1 \pm 1.2$  liters for 80, 40, and 20 minutes of exercise at 70% of  $\text{VO}_2$  max respectively. Although 80 minutes of exercise showed the greatest magnitude of EPOC, there was no significant difference between the 40 minute and 20 minute bouts of exercise. In a similar study, Quinn et al. (1993) tested the effect of EPOC on 60, 40, and 20 minutes of exercise at 70% of  $\text{VO}_2$  max. Although 60 minutes of exercise resulted in significantly greater 3 hour EPOC than 20 and 40 minutes of exercise at the same intensity, there was not a significant difference in 3 hour EPOC between 20

Minutes and 40 minutes of exercise. If stimulating EPOC is the primary goal of exercise, what is the point of exercising for 40 minutes if 20 minutes has the same effect?

Sedlock et al. (1989) found the manipulation of exercise duration has an effect on only the duration of EPOC. According to another study, slow phase EPOC was proportional to exercise intensity and was not altered by exercise duration (Hagberg et al, 1980).

These studies demonstrate that exercise duration has a significant effect on EPOC, particularly on the duration of EPOC. However, 60 or 80 minutes of continuous exercise may not be attractive unless participants are fervently devoted to or preoccupied with exercise and improving their performance levels. When magnitude of EPOC is considered, manipulation of exercise duration may not be as effective as manipulation of exercise intensity.

### Conclusion

Since A. V. Hill discovered elevated  $O_2$  uptake after exercise, scientists have been trying to explain EPOC. However, there has been a variety of conflicting research because the study of EPOC requires precise control of pre-post exercise conditions including proper training status of subjects, environment, exercise intensity and duration. Therefore, studies are not easily compared to other studies due to differences and inconsistencies in study design. For these reasons, there is not a definitive explanation of recovery  $O_2$  uptake.

Although the mechanism of rapid phase of EPOC is well known, the slow phase of EPOC is still not clear. At this time, several factors that have been looked at closely include the replenishment of ATP, creatine phosphate, lactate removal/oxidation,

temperature regulation, and increased cardiac output and ventilation. Nonetheless, it seems like the slow phase is not related to these factors as much as they are in the rapid phase. The mechanism of slow phase of EPOC is not well defined and may not occur more than 3 hours after exercise.

One of the reasons that slow phase EPOC is not well understood may be limitations of study design. Recovery  $O_2$  uptake may be influenced by many factors such as previous exercise, food, or activity during measurement that are difficult to control for a prolonged period of time, and the slow phase of EPOC is a miniscule amount to detect. As of now, it is suspected that hormonal activity and fat metabolism may be possible factors since lactic acid and core temperature are no longer believed to contribute to the slow phase of EPOC.

Both exercise intensity and duration influence EPOC, however, manipulating the intensity may be more beneficial for a healthy individual since less than 60 minutes of continuous exercise induces similar EPOC. From a practical view point, short term high intensity exercise may be applicable in today's time restricted society. For those reasons, high intensity exercise is the more efficient choice compared to aerobic exercise. If the focus is only substrate utilization for weight reduction purposes, it may be a critical mistake. It is irrelevant whether fat is metabolized during exercise or after exercise. However, it should be noted that high intensity exercise involves the risk of injury and requires highly motivated participants. Therefore, careful pre exercise screening needs to be conducted by trained personnel. Designing a program with low to moderate continuous exercise may be necessary for unmotivated individuals or for rehabilitation purposes.

This study emphasizes the short term effect of exercise on EPOC because of the limitations of study design such as time restriction of subjects and regulation of food consumption after exercise. The purpose of this study is not to define the mechanisms of EPOC. However, if different intensities of exercise cause additional energy expenditure during recovery, exercise professionals should be aware of this.

## Chapter 3

### METHODS

This chapter will discuss subject recruitment, instrumentation, study design, and procedure.

#### Subject Recruitment

The target population was apparently healthy males and females who are classified in low risk of Initial ACSM Risk Stratification, and also between the ages of 18 and 30. Subjects were selected from Marshall University students or faculty who exercise regularly (2-3/week), and have been exercising for 5 months. Subjects who met these criteria were qualified to participate in further investigations. Anthropometric measurements were conducted at Marshall University Exercise Physiology Laboratory. Subjects were eliminated from this study if their BMI was more than 30 or they are not classified in low risk from the screening sheet based on ACSM guidelines. Investigators explained the benefits and risks of participating in this study, and time restrictions after subjects filled out the screening sheet (Appendix B). The informed consent form was given to interested subjects who met all criteria (Appendix C).

#### Instrumentation

The instruments used in this study were a metabolic cart (Vmax 29, SensorMedics, USA), a polar heart rate monitor (Polar Vantage XL, polar CIC INC), a treadmill (ST 65, Med Track), a bike ergometer (Ergomedic 828E, Monark) and scales. The metabolic cart was used to measure RMR and O<sub>2</sub> uptake after each exercise bout. A treadmill and bike were also used during VO<sub>2</sub> max test, and 3 exercise sessions. Anthropometric measurements, height and weight, were determined by scales. In addition, body fat

percentage was calculated by the Siri formula after the sum of 3-site skinfold including chest, abdominal and thigh for men, including triceps, suprailiac, and thigh for women was found (Pollock, Schmidt & Jackson, 1980).

### Study Design

This study employed a within-subjects repeated measurement design. Subjects participated in 3 different exercise sessions on separate days after the exercise intensity of each exercise session was found. Three exercise sessions were conducted at the same time of the day, but not on consecutive days. Subjects showed up at the Marshall University Exercise Physiology Laboratory in overnight fasting state (9 to 12 hours). Subjects were also asked to minimize physical activity to get the laboratory, in order to minimize possible effects on EPOC. Every exercise session was monitored by exercise professionals. Before the  $VO_2$  max test, and before every exercise session, subjects were prohibited from strenuous exercise within 24h. Subjects were required to measure their body weight before the every exercise sessions because fluctuation of body weight directly affects their RMR. A full convened review request was sent to the Institutional Review Board (IRB) at Marshall University along with study protocol, IRB abstract, screening sheet, informed consent, and flyer. The study was approved by the IRB as IRB study number 5009.

### Procedure

#### *VO<sub>2</sub> Max Test*

In the first part of this study, the subjects' substrate utilization was determined by R-value while walking on treadmill. The exercise protocol started with a base line which was established while the subjects sat on a bench. After normal values were obtained,

subjects started the warm up by walking at a comfortable speed. The test began with increasing the grade of treadmill each minute until subjects could not continue to exercise. During the test, subjects breathed through a rubber mouthpiece attached to a valve; therefore all communication was made by gesture (as previously explained to the subjects). Exhaled gas was measured by an O<sub>2</sub> and CO<sub>2</sub> analyzer in breath by breath method. Before the every test, the calibration of flow volume sensor was conducted by a 3.0 liter cylinder as well as an O<sub>2</sub> and CO<sub>2</sub> gas analyzer by known concentration of O<sub>2</sub> and CO<sub>2</sub>. The exercise intensity corresponding to R-value 0.9, 1.0 and 1.1 was recorded as the target exercise intensity of each exercise session. Three exercise sessions, all performed in same durations but at different intensities, were randomly scheduled at same time of the day, but not consecutive days.

#### *Aerobic Protocol*

In the aerobic protocol, subjects were lying in supine position while resting energy expenditure is measure by canopy dilution method. RMR was measured at least 20 minutes after normal resting value had been obtained. The average O<sub>2</sub> uptake of these 20 minutes was used as baseline data for determining EPOC. Subjects did a warm up for 5 minutes before starting the exercise. Exercise intensity was set at the R-value 0.9 that was continued for 20 minutes. During the exercise, HR was monitored to achieve the target exercise intensity previously found each minute. After the 20 minutes of exercise, subjects were asked to rest in supine position for 1 hour.

#### *Interval Protocol*

Subjects performed the interval protocol with exactly the same procedure of baseline and warm-up. During this exercise phase, subjects did exercise at intensity

corresponding to R-value 1.0 for 3 minutes with one minute active recovery pedaling bike. During the active recovery, the work rate was set at the same work rate of walking on the treadmill at 3.0 mph. This work rate was calculated with modified metabolic equations using the ACSM metabolic equation developed by Swain and Leutholtz (1997).

$$\text{Walking: } \text{VO}_2 = 3.5 + 2.68 * (\text{Speed in mph}) + 0.48 * (\text{Speed in mph}) * (\% \text{ of Grade})$$

$$\text{Leg Ergometry: } \text{VO}_2 = 3.5 + 2 * (\text{Work in kgm/min}) / (\text{Body weight in kg})$$

Therefore, the ratio of work and rest was 3:1; there were 5 cycles of this interval exercise which takes 20 minutes. Again, HR was monitored each minute. Subjects were also asked to rest in supine position for 1 hour after the exercise.

#### *High Intensity Interval Protocol*

In this protocol, subjects performed a high intensity interval protocol that duplicates the same procedure of baseline, warm-up and exercise in previous interval protocol. In the high intensity interval protocol, the work rate is set at R-value 1.1 with active recovery. The work rate of the active recovery was determined in the same procedure as well. The ratio of work and rest was 1:1 which continued for 20 minutes. Again, subjects were required to rest for 1 hour.

#### *EPOC calculation*

EPOC was calculated by subtracting baseline  $\text{VO}_2$  from gross  $\text{VO}_2$  each minute. This net  $\text{VO}_2$  was added up for one hour except when subjects'  $\text{VO}_2$  went back to less than the baseline value more than 10 consecutive minutes within one hour.

## Statistical Analysis

Collected data were entered in the Statistical Package for Social Science (SPSS). In this study, the independent variable is “intensity of exercise” and “type of exercise”, while the dependent variable is “EPOC”. ANOVA with repeated measures were used to analyze the differences of EPOC among the different exercises. When significant differences were obtained, pairwise comparisons were performed to identify the means difference. The rejected level of the ANOVA with repeated measurements was set at  $\alpha = 0.05$ .

## Chapter 4

### RESULTS

This chapter will present the data obtained from the study. The study planned to examine 4 males and 4 females, however, the final sample size (n = 8) was male (n = 5), and female (n =3). Although several female subjects were interested in participating in the study, they could not participate in this study due to the age limitations and class schedule issues. Therefore, one additional male subject was recruited to compensate for this problem.

#### Physical Characteristics of the Subjects

Physical characteristics are shown in Table 1. The mean of the total subjects' age was 23.5 ( $\pm$  1.2) years within the range of 19 to 25. The mean body weight, height and body fat percentage ( $\pm$  SD) were 71.2 ( $\pm$  14.88) kg, 171.1 ( $\pm$  5.38) cm, and 18.3( $\pm$  6.13) % respectively. All subjects are physically active, but not participating in regular athletic training or exercise programs prescribed by professionals.

Table 1

*Physical Characteristics of Subjects.*

Variable	N	Mean	SD
Age(yr)	8	23.5	1.2
Height(cm)	8	171.1	5.4
Weight(kg)	8	71.2	14.9
Fat (%)	8	18.3	6.1
VO <sub>2</sub> max (ml•kg <sup>-1</sup> •min <sup>-1</sup> )	8	40.6	6.3

### Baseline Data

The mean values of data obtained from resting state and exercise are presented in Table 2. The mean body weight ( $\pm$  SEM) for the subjects did not change over the three exercise sessions;  $70.9 \pm 14.8$ ,  $70.8 \pm 14.9$ , and  $70.9 \pm 14.9$  kg, respectively. The baseline  $\text{VO}_2$  values ( $\pm$  SEM) for each exercise session were  $0.207 (\pm 0.015)$ ,  $0.201 (\pm 0.015)$  and  $0.204 (\pm 0.014) \text{ l}\cdot\text{min}^{-1}$ , respectively. These values were not statistically different at  $p < 0.05$  level, therefore, further analysis was conducted. EPOC was calculated by subtracting baseline  $\text{VO}_2$  from recovery oxygen uptake in each exercise session.

### Exercise Data

The mean exercise intensities ( $\pm$  SEM) corresponding to R value 0.9, 1.0, and 1.1 were  $50.7 \pm (2.8519)$ ,  $70.1 \pm (2.3631)$  and  $93.7 \pm (0.9726)$  % respectively (Table 2). These values were calculated by R value corresponding to  $\text{VO}_2$  divided by  $\text{VO}_2$  max, and then multiplied by 100. Exercise duration was held 20 minutes, and intensity of exercise was adjusted by previously found HR with specific substrate utilization. The mean HR ( $\pm$  SEM) of each exercise were  $134.9 \pm 3.9$ ,  $149.9 \pm 5.3$ , and  $162.3 \pm 4.3$   $\text{beat}\cdot\text{min}^{-1}$  for aerobic exercise, IE, and HIIE, respectively. After the pairwise comparisons, significant differences ( $p < 0.05$ ) were found in each value. Therefore, we assume the difference in EPOC was caused by the difference in exercise intensity.

Table 2.

*Mean Values ( $\pm$  SEM) Obtained During Resting State and Exercises.*

Variable	Aerobic	IE	HIIE
Body weight (kg)	70.9 $\pm$ 5.25	70.8 $\pm$ 5.26	70.9 $\pm$ 5.26
Baseline VO <sub>2</sub> (l•min <sup>-1</sup> )	0.207 $\pm$ 0.0155	0.201 $\pm$ 0.0149	0.204 $\pm$ 0.0139
Exercise intensity (%)	50.7 $\pm$ 2.85*	70.1 $\pm$ 2.36**	93.7 $\pm$ .97***
HR(beat•min <sup>-1</sup> )	134.9 $\pm$ 3.866*	149.9 $\pm$ 5.259**	162.3 $\pm$ 4.271***

Note. IE = Interval exercise; HIIE = High intensity interval exercise  
 \* Significant different ( $P < 0.05$ ) from IT and HIIE (2-tailed)  
 \*\*Significant different ( $P < 0.05$ ) from Aerobic and HIIE (2-tailed)  
 \*\*\*Significant different ( $P < 0.05$ ) from Aerobic and IT (2-tailed)

#### Magnitude of EPOC

Total one hour EPOC is represented in Figure 1. There was a significant difference ( $p < 0.05$ ) among the exercise sessions. In order, the mean values ( $\pm$  SEM) for EPOC of aerobic exercise, IE and HIIE were 2.106 ( $\pm$  0.219), 2.846 ( $\pm$  0.309) and 4.969 ( $\pm$  0.522) l•hour<sup>-1</sup>. Mean EPOC of HIIE was more than twice as much as EPOC of aerobic exercise. Table 3 presents the result of the statistical analysis of variance,  $F(2, 14) = 23.91$ , which was higher than 3.74 at  $p < 0.05$  level indicating a significant difference with observed power = 1.00 and  $\omega^2 = 0.73$ . After this was found, pairwise comparisons were conducted to assess which means differ from each other. There was no significant difference ( $p < 0.05$ ) between mean EPOC of aerobic exercise and IE. However, a significant difference ( $p < 0.05$ ) was found in mean EPOC of HIIE when it was compared with both aerobic exercise and IE (Table 4). During the course of the hour VO<sub>2</sub> changes

were plotted for each exercise (Figure 2). Rapid decline was observed first ten minutes, however,  $VO_2$  from HIIE remained elevated more than both aerobic and IE  $VO_2$ .

Table 3.

*Tests of Within Subjects Effects*

Source	Sum of squares	<i>df</i>	Mean square	<i>F</i>	<i>p</i>
EPOC	35.341	2	17.670	23.914	0.000*
Error(EPOC)	10.345	14	0.739		
Totals	45.686	16			

Note. \* significant different  $p < 0.05$   
 $\omega^2 = 0.73$   
observed power = 1.00

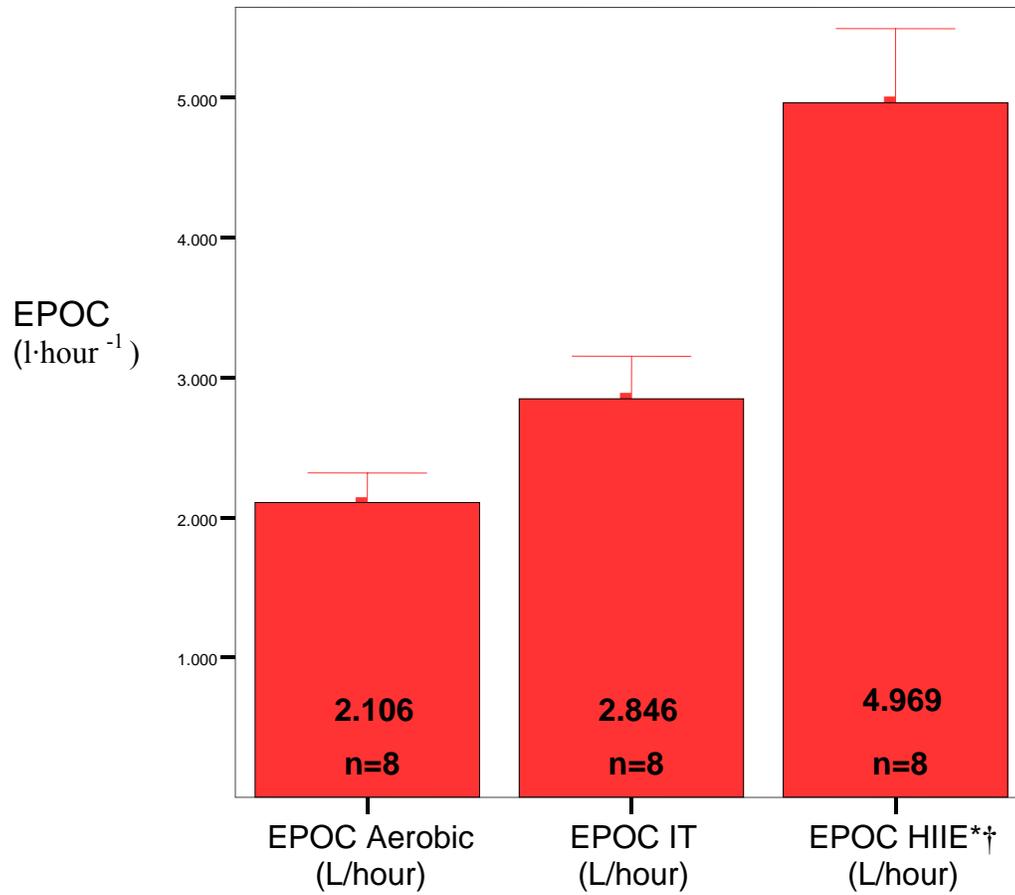
Table 4.

*Mean Difference Values for Pairwise Comparisons*

	Aerobic	IE	HIIE
Aerobic	0.00000	0.74000	2.86312*
IE		0.00000	2.12313*
HIIE			0.00000

Note. \* significant different  $p < 0.05$

Figure 1. Mean EPOC ( $\pm$  SEM)



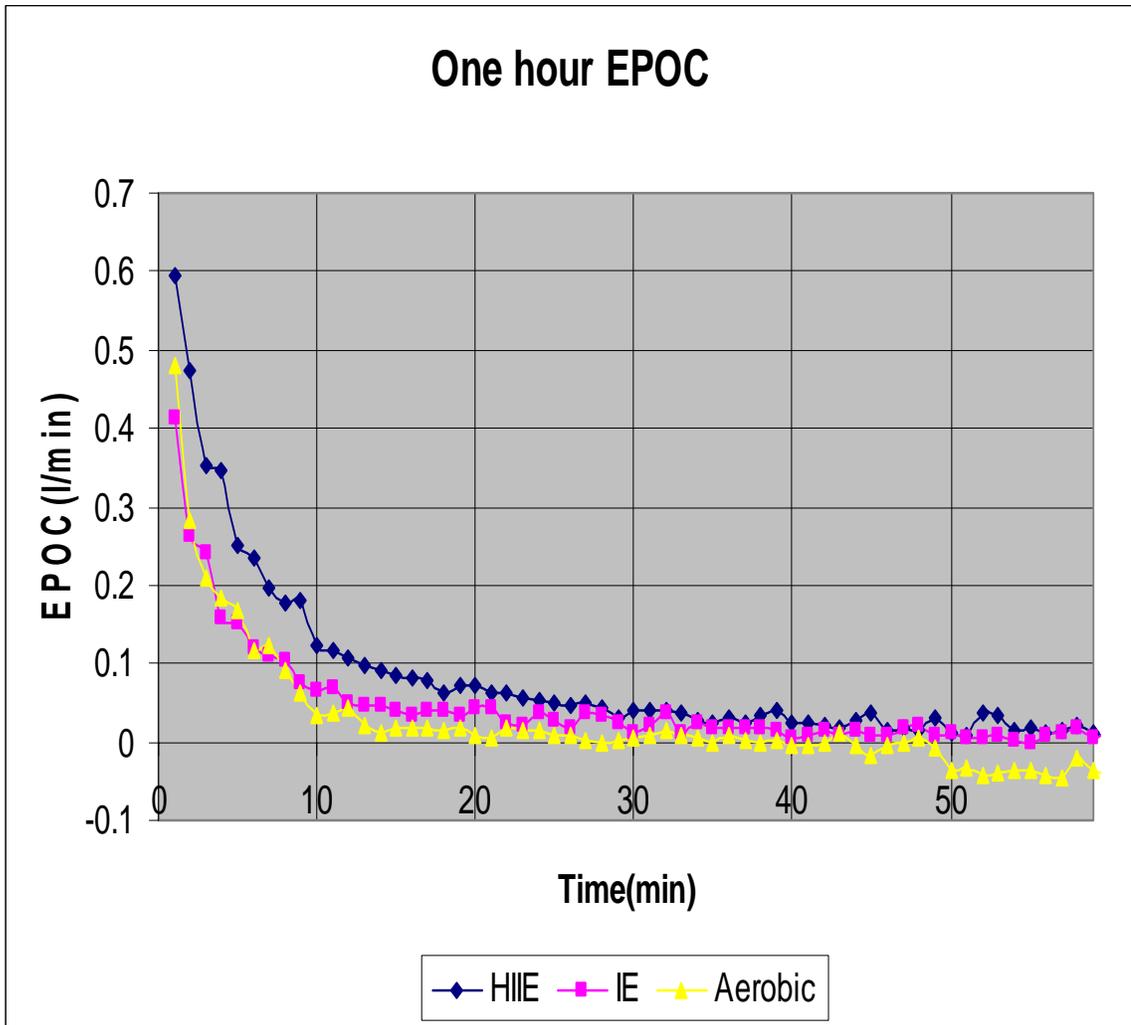
Error Bars show Mean  $\pm$  1.0 SEM

Bars show Means

\*significant difference from aerobic

†significant difference from IT

Figure 2. Mean postexercise oxygen consumption over one hour.



## Chapter 5

### SUMMARY AND CONCLUSION

This chapter discusses the results of this study and its practical applications. The purpose of this study was to determine the effect of EPOC on different intensities of exercise.

#### Summary

The primary purpose of this study was to determine if a selection of exercise intensities would beneficially affect post energy metabolism as measured by one hour of EPOC. The results of this study revealed that mean one hour EPOC ( $\pm$  SEM) were 2.076 ( $\pm$  0.223), 2.847 ( $\pm$  0.309) and 4.959 ( $\pm$  0.522), respectively that indicated HIIE produced the highest EPOC in apparently healthy individuals who regularly participate in exercise.

Statistical analysis indicated that there was a significant difference ( $p < 0.05$ ) with effect size = 0.72. In addition, it revealed high observed power = 1.00 due to repeated measurements. According to Cohen (1988), effect size = 0.2, 0.5, 0.8 represented small, moderate and large differences. Generally, the power of a study should be at least 0.8 or 80 % (Greenfield, Kuhn, & Wojtys, 1997). Therefore, this study revealed a significant difference with moderate effect size and an acceptable power value.

One limitation of this study was the small sample size recruited from one local area. Also, both genders were not equally represented. As far as we know, female gender specific studies show similar results, however, a percentage of difference between males and females may exist. Another limitation to be considered is homogeneity of pre exercise conditions. Børsheim and Bahr (2003) stated that body weight, food intake, and exercise should be controlled. It also suggested that subjects should sleep overnight in the

laboratory and that menstrual cycles may need to be controlled for female subjects, however due to limitations of laboratory capacity and budget constraints, some items were difficult to control. In summary, the null hypothesis that there will be no difference in EPOC among 3 different exercise sessions needs to be rejected.

### Discussion

Both exercises are at or below the ventilatory threshold which indirectly may reflect the onset blood lactate accumulation, inducing significantly lower EPOC. The elevation of one hour EPOC may be explained by oxidation of blood lactate from the exercise even though we did not directly measure the blood lactate level. This has been known as one of the factors that induce elevated recovery oxygen uptake (Børsheim & Bahr, 2003; Gaesser & Brooks, 1984).

Previous research examining the effect of EPOC on duration and intensity of exercise has shown a direct correlation between exercise duration, intensity and EPOC (Bahr, & Sejersted, 1991; Bahr et al, 1987; Quinn et al, 1993; Sedlock et al, 1989; Hagberg et al, 1980; Maehlum et al, 1986; Chad & Wenger, 1988; Laforgia, Withers, Shipp & Gore, 1997). However, the duration and magnitude of EPOC is not clearly understood. As mentioned in the previous chapter, the comparison of these results is very difficult because of methodological differences. Studies confirming one hour EPOC or less than one hour EPOC are presented in Table 5 (modified from Børsheim & Bahr, 2003, Table I ). The range of EPOC is 1.4 liters to 6.9 liters from exercise intensity of 25% to 80 %. Therefore, this study agreed fairly well with the other studies. Laforgia et al, (1997) reported EPOC of 6.9 liters for one hour, however, they used highly trained men who exercised at 70% of  $VO_2$ max for 30 minutes. Both exercise intensity and

duration are higher than this study; in addition, subjects were highly trained athletes who can accomplish a higher work. Burleson, O'Bryant, Stone, Collins and Triplett-McBride (1998) reported 3.4 liters of EPOC from exercise at 45% of  $VO_2$  for 27 minutes. Our study showed a slightly lower value from aerobic exercise which was 50% of  $VO_2$  max for 20 minutes. Again many things can influence EPOC; especially since the mean body weight of the subjects in the study (Burleson et al., 1998) was approximately 10 kg heavier than our subjects.

The finding of this study is that manipulating exercise intensity above ventilatory threshold has a significant effect on EPOC which lasts more than one hour. The results of this study have a precise practical implication related to exercise prescription for an individual who is concerned with weight control. In spite of the scientific evidence, few implementations have been utilized in the practical field. However, high intensity exercise possesses a higher risk of injury, and thus may not be appropriate in a clinical setting. In addition, obese people who are not used to exercise may need to begin with low intensity exercise that gradually progresses in intensity, duration and frequency of exercise.

#### Implication

Practically speaking, approximately extra 25 kcal is used after the HIIE while 10 kcal is used after the aerobic exercise. If the HIIE protocol was used for a weight reduction program instead of aerobic exercise, it would induce a 2.5 times greater energy expenditure than aerobic exercise. Considering only postexercise energy expenditure, by the time an aerobic exercise participant achieves a 1 pound loss, the HIIE participant will have lost 2.5 pounds.

Exercise prescriptions should undergo a reevaluation. Typical fat burning programs have only a small effect on weight control when observed from a holistic perspective. It may be that emphasis should not be placed on the exercise itself, but on post exercise energy expenditure.

### Conclusion

In summary, the results of this study suggest that exercise intensity exponentially affects EPOC. Therefore, it may be appropriate to prescribe high intensity exercise instead of aerobic exercise to apparently healthy individuals who are concerned with their body weight. Although it is difficult to quantify the exact amount of calories from EPOC due to inter individual variability, it does appear to provide additional energy expenditure after the exercise. This benefit should be considered not only when focusing on energy expenditure and substrate utilization during exercise because weight fluctuation is based on the law of thermodynamics.

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## APPENDICES

## Appendix A

Studies on the Effect of Aerobic Exercise on Postexercise  $\text{VO}_2$  ( $\leq 1$  hour)

Studies on the effect of aerobic exercise on post-exercise VO<sub>2</sub> (≤ one hour)

Study	Year	Study Participant	Exercise mode, duration and intensity	EPOC
Pacy et al.	1985	2F	Cycling: 35-55% for 20 min X 4	< 2.2L, < 60 min
		2M		
Freedman-Akabas et al.	1985	13F(UT + T)	TM: Anaerobic threshold, 20 min	< 4.2 L, <40 min
		10M(UT + T)		
Elliot et al.	1988	3F	Cycling: 80%, 10 min	~ 2.37 L(11.4 kcal), 30 min
		3M	Cycling: 80%, 30 min	
Kaminisky et al.	1990	6F	TM: 70%, 50 min	1.4L, <30min
			TM: 70%, 2 X 25 min	3.1L, <30min
Gore & Withers	1990	9M	TM: 30%, 20, 50, 80min	1.0, 1.4,1.0L <1h
			TM: 50%, 20, 50min	3.1,5.2L <1h
Berg	1991	5F,5M	TM:40% Male	~1.4L <1h
			TM:40% Female	~2.2L <1h

Continued

<b>Study</b>	<b>Year</b>	<b>Study Participant</b>	<b>Exercise mode, duration and intensity</b>	<b>EPOC</b>
Brochman et al.	1993	5F	TM: 25%, 2h	12%, 40min
			TM: 81%, 10min	23%, >1h
			TM: 89%, 7 X 2min with 2 min break	44%. >1h
Kaminsky & Whaley	1993	10F(5 obese, 5 lean )	TM: Alternating 3 min bout at 30% and	
			90%, 36 min	~3.6L(17.4 kcal), 37.5 min
			60%, 36 min	~1.9L(9.0 kcal), 16.5 min
Frey et al.	1993	13F (7UT +6 T)	60%, 36min	
			Cycling:65% 45 min(until 300kcal)	UT: 4.0 L, >1h T: 4.7L, 50min
			Cycling:80% 24 min(until 300kcal)	UT: 5.9L, >1h T: 5.6L, 40min
Sedlock	1994	10M(5UT + 5T)	Cycling 50% ~300kcal	UT: ~2.5L(12.2kcal), 20.4 min
				T:~2.5L(12.2kcal), 16,6

Continued

<b>Study</b>	<b>Year</b>	<b>Study Participant</b>	<b>Exercise mode, duration and intensity</b>	<b>EPOC</b>
Dawson et al.	1996	8F	Cycling 67%, 34 min	3.6L, 13.9 min
			Cycling 55%, 41 min	2.6L, 14min
			Cycling 45%, 49 min	2.4L, 13.2min
Trost et al.	1997	5M	Cycling: 65%, 60min	1h EPOC 5.5L, >1h
Laforgia et al	1997	8M(T)	TM 70%, 30min	6.9L, 1h

Note: EPOC = Excess post exercise oxygen consumption; M = Male; F = Female; UT = Untrained; T = Trained; TM = Treadmill;

Appendix B  
Screening Sheet

## Screening sheet

All information given is personal and confidential. The information will give us to better understanding of your health and fitness level.

**Name** \_\_\_\_\_ **Age** \_\_\_\_\_

**Current address** \_\_\_\_\_

**Phone (home, business)** \_\_\_\_\_

**E-mail address** \_\_\_\_\_

**Emergency contact information (name, address, phone)** \_\_\_\_\_

1. Do you have chest pain or discomfort in the chest, neck, jaw and arms during exercise?  
YES                      NO
2. Do you have shortness of breath at rest or with mild exertion?  
YES                      NO
3. Have you ever fainted or experienced dizziness?  
YES                      NO
4. Do you have trouble breathing when you lie down or sleep?  
YES                      NO
5. Do you have swollen ankles?  
YES                      NO
6. Do you have a fast heart beat?  
YES                      NO
7. Do you experience intermittent cramps in your legs while walking?  
YES                      NO
8. Do you have a known heart murmur?  
YES                      NO
9. Do you feel unusual fatigue or shortness of breath with usual activities?  
YES                      NO

**Family History**

Has your father had myocardial infarction, coronary revascularization or sudden death before age 55 or your mother before age 65?

YES                      NO

**Cigarette smoking**

Do you smoke cigarettes or did you quit in the previous 6 months?

YES                      NO

**Hypertension**

Has your doctor ever told you that you have high blood pressure?

YES                      NO

**Hypercholesterolemia**

Has your doctor ever told you that you have abnormal cholesterol levels?

YES                      NO

**Impaired fasting glucose**

Has your doctor ever told that you have high blood glucose (sugar)?

YES                      NO

**Obesity**

Is your BMI more than 30 or your waist circumference more than 100cm?

YES                      NO

**Sedentary lifestyle**

Do you participate in an exercise program or recreational activity regularly?

YES                      NO

Do you have any other reasons that limit your exercise program?  
(Orthopedic problems, metabolic problems)? If so, please list them below:

---

---

Is there any chance that you are pregnant?

Yes            NO

Do you currently exercise?

Yes            NO

If yes, how often do you exercise and how long have you been exercising?

---

---

---

What type of exercise do you do? (e.g. running, bike, rowing, free weights, machine etc...)

---

Do you take any medication or supplements?

Yes            NO

If yes, please list the names of drugs, purpose of drugs, and prescribed or OTC?

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I have answered the above questions honestly, and to the best of my knowledge.

Name \_\_\_\_\_

Signature \_\_\_\_\_

Date \_\_\_\_\_

Appendix C  
Informed Consent

## **Excess Postoxygen Consumption and Interval Training**

**Jeff, Chandler, Ed. D:** Principal Investigator

**Eric, Arnold, ABD:** Co-Investigator

**Shinichi Asano:** Co-investigator

You are invited to be in a research study. Research studies are designed to gain scientific knowledge that may help other people in the future. You may or may not receive any benefit from being part of the study. There may also be risks associated with being part of research studies. Your participation is voluntary. Please take your time to make your decision, and ask your research doctor or research staff to explain any words or information that you do not understand.

### 1. Purpose and explanation of study

You have been asked to participate in a study generated by the investigator for graduate research. This study is designed to compare the effects of different types of exercise on you, and also to find out which one is the most appropriate. You will perform an exercise test and 3 exercise sessions on a treadmill. The test starts at a low intensity and gradually increases exercise intensity. In the 3 exercise sessions, you will perform aerobic, interval, and high intensity interval exercise for 20 minutes on separate days. For both the test and the 3 exercise sessions, you will be connected to a metabolic cart that measures oxygen consumption. This will give you an accurate maximal oxygen consumption ( $VO_2$  max: maximum exercise capacity) measurement, and specific energy expenditure corresponding to exercise intensity. We may stop the test and 3 exercise sessions at any time because of abnormal vital signs such as heart rate (HR), or blood pressure (BP). You may also stop at any time due to any symptoms or discomfort. The exercise test and 3 exercise sessions will be conducted at the same time of the day, but not on consecutive days. The schedule of testing and exercise sessions will be discussed with the investigators; the study will be completed in no more than 4 weeks

### 2. Attendant Risk and Discomfort

Since the test requires maximal effort, physical discomfort may occur such as high BP, fainting, and irregular or fast heart rhythms. In rare cases, there may be a risk of heart attack, stroke or death. Emergency equipment and trained personnel are available to deal with medical emergencies. In the unlikely event of an emergency while participating in this study, emergency help will be available at the subject's expense or the subject's insurance carrier. No compensation, financial or otherwise, will be provided by the investigators or Marshall University.

Initial \_\_\_\_\_

### 3. Responsibility of Participant

The responsibility of the participant is to fully disclose your medical history and physical symptoms during the test and exercise sessions.

### 4. Expected Benefits

Information from the test will show your fitness level and the most accurate exercise intensity for you. Information from the exercise sessions will give you a precise energy expenditure that you may use in your daily exercise program. Also, the results of this study will be a future reference for your health status; however, this study is not a diagnostic procedure such as stress test.

### 5. Number of subjects in this study

A total of 10 subjects are the most that would be able to enter the study

### 6. Cost/compensation

There are no costs to your for taking part in this study. You will receive no payment or other compensation for taking part in this study.

### 7. Use of Medical Records.

Obtained information will be treated as confidential and will not be released to anyone without your permission. It will be used for statistical analysis as privileged data only. However, we can not guarantee absolute confidentiality. Certain people other than your researchers may also need to see your study record. These include certain university and government agencies that need to know more about the study. For example, individuals who provide oversight on this study such as the Marshall University Institutional Review Board and the Office of Research Integrity may need to look at your records. This is done to make sure that we are doing the study in the right way, and also we are protecting your rights and your safety.

Initial\_\_\_\_\_

8. Inquiries.

You may ask any question about this study to the investigator, Shinichi Asano, or his advisor Jeff Chandler, EdD., of the Department of Exercise Science Sports and Recreation, Marshall University, and Eric Arnold, ABD, the director of the Exercise Physiology Laboratory, Marshall University. **The Office of Research Integrity at Marshall University or IRB#1 Chairman Dr. Henry Driscoll can provide you with general information about the rights of human subjects in research.**

**Phone: (304)696-7320.**

Contact Information:

**Jeff Chandler, EdD. (304)696-2924**

**Eric Arnold, ABD, (304)696-2925**

**Shinichi Asano, (304)696-3672**

9. Freedom of Consent

You may refuse to participate in this study. If you refuse or quit during the study at any time, you will not be penalized or lose the benefits you already have.

Your participation in this study is voluntary. You may refuse to participate in this study without penalty. If you decide to participate, you may withdraw from the study at anytime without penalty and without loss of benefits to which you are otherwise entitled. The information in this study records will be kept confidential.

I have read the information about this study, and I understand the test procedure. I consent to participate in this study.

Name \_\_\_\_\_  
Signature \_\_\_\_\_  
Date \_\_\_\_\_

Witness \_\_\_\_\_  
Signature \_\_\_\_\_  
Date \_\_\_\_\_

Person Obtaining Consent  
Signature \_\_\_\_\_  
Date \_\_\_\_\_

Initial \_\_\_\_\_

## Appendix D

### Graded Exercise Test and EPOC Measurement



# Baseline Data

Name \_\_\_\_\_

Age \_\_\_\_\_

Gender \_\_\_\_\_

Date \_\_\_\_\_

Protocol	Aerobic	IT	HIIT
Date			
BW			

Time	VO2	RER		VO2	RER		VO2	RER
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								
11								
12								
13								
14								
15								
16								
17								
18								
19								
20								
21								
22								
23								
24								
25								
Average								

**Comment** \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

<b>Fasting?</b>	Yes	No
<b>Sleep during test?</b>	Yes	No
<b>Bed Rest?</b>	>30	<30

# Exercise Date

Name \_\_\_\_\_

Age \_\_\_\_\_

Gender \_\_\_\_\_

Date \_\_\_\_\_

Protocol    Aerobic                      IT                      HIIT

Date                      \_\_\_\_\_                      \_\_\_\_\_                      \_\_\_\_\_

TRH                      \_\_\_\_\_                      \_\_\_\_\_                      \_\_\_\_\_

Time	HR			HR			HR	
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								
11								
12								
13								
14								
15								
16								
17								
18								
19								
20								

Average

**Comment** \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

# EPOC

Name \_\_\_\_\_

Protocol **Aerobic**

Date \_\_\_\_\_

Total EPOC \_\_\_\_\_

Average R \_\_\_\_\_

L/min					L/min				
Time	VO2	BL	EPOC	R	Time	VO2	BL	EPOC	R
1					31				
2					32				
3					33				
4					34				
5					35				
6					36				
7					37				
8					38				
9					39				
10					40				
11					41				
12					42				
13					43				
14					44				
15					45				
16					46				
17					47				
18					48				
19					49				
20					50				
21					51				
22					52				
23					53				
24					54				
25					55				
26					56				
27					57				
28					58				
29					59				
30					60				

# EPOC

Name \_\_\_\_\_

Protocol Interval exercise

Date \_\_\_\_\_

Total EPOC \_\_\_\_\_

Average R \_\_\_\_\_

L/min					L/min				
Time	VO2	BL	EPOC	R	Time	VO2	BL	EPOC	R
1					31				
2					32				
3					33				
4					34				
5					35				
6					36				
7					37				
8					38				
9					39				
10					40				
11					41				
12					42				
13					43				
14					44				
15					45				
16					46				
17					47				
18					48				
19					49				
20					50				
21					51				
22					52				
23					53				
24					54				
25					55				
26					56				
27					57				
28					58				
29					59				
30					60				

# EPOC

Name \_\_\_\_\_

Protocol **HIE**

Date \_\_\_\_\_

Total EPOC \_\_\_\_\_

Average R \_\_\_\_\_

L/min					L/min				
Time	VO2	BL	EPOC	R	Time	VO2	BL	EPOC	R
1					31				
2					32				
3					33				
4					34				
5					35				
6					36				
7					37				
8					38				
9					39				
10					40				
11					41				
12					42				
13					43				
14					44				
15					45				
16					46				
17					47				
18					48				
19					49				
20					50				
21					51				
22					52				
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24					54				
25					55				
26					56				
27					57				
28					58				
29					59				
30					60				

# Curriculum Vitae

**Shinichi Asano, CSCS**

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## Home:

1539 4<sup>th</sup> Avenue apartment 9  
Huntington, WV 25701  
Phone: 304-697-6836  
E-mail: [asano1@marshall.edu](mailto:asano1@marshall.edu)

## Education:

Master of Science, Exercise Science  
Emphasis in Exercise Physiology  
Marshall University, Huntington, West Virginia: 2005 May anticipated.  
Thesis: "Excess post exercise oxygen consumption and interval training"

Post Bachelor of Science, Physical Education  
Emphasis in Athletic Training  
Salem International University, Salem, West Virginia:

Bachelor of Arts, Intercultural Studies  
Teikyo University, Tokyo, Japan: 2000 March

## Work Experience:

Spring 2005-present Graduate Assistant  
Marshall University  
Exercise Physiology/Bioenergetics Laboratory

As a graduate assistant and laboratory coordinator, duties include coordinating Exercise Physiology/Bioenergetics Laboratory, implementing advanced exercise tests (Blood lactate analysis, resting/exercise 12 lead EKG, VO<sub>2</sub> max test, Resting metabolic rate, Hydrostatic weighing, Wingate, Pulmonary function test), assisting lab graduate/undergraduate class, teaching graduate assistants/undergraduate volunteers basic fitness tests, managing data, and assisting with coverage of various laboratory events as needed.

Fall 2004: Graduate Assistant  
Marshall University  
Exercise Physiology

As a graduate assistant, duties included assisting the laboratory assistant director, implementing basic exercise tests (cardiovascular, muscular strength, and body composition assessment/counseling), providing laboratory training sessions to graduate and undergraduate students, and managing data.

Fall, 2004: Graduate Assistant  
Marshall University  
Strength and Conditioning

As a graduate assistant, duties included assisting strength and conditioning coaches, teaching Olympic weight lifting, developing and implementing pre/in season strength and conditioning programs, and assisting with coverage of various weight room events as needed.  
Primarily responsible for women's tennis, golf, diving, and cross country teams.

Spring, 2004: Graduate Assistant  
Marshall University  
Diabetes Exercise and Cardiac Rehabilitation Center

As a graduate assistant, duties included monitoring blood pressure and heart rate for phase III cardiovascular patients at rest and during rehabilitation exercises, monitoring blood glucose for diabetic patients at rest and during rehabilitation exercises, teaching exercise techniques, and data management.

Spring, 2003 – fall, 2003: Graduate Assistant  
Marshall University  
Fitness Center

As a graduate assistant, duties included maintaining the fitness center, and monitoring the exercise of student and faculty.

**Related Experience:**

Summer 2004: Football Summer Conditioning, Marshall University  
Summer 2004: Guest Speaker at Diabetes/Cardiac Exercise Center, Marshall University  
Spring 2004: HIT Center, Huntington, WV  
Summer 2002: Football Summer Conditioning, Fairmont State University, WV

**Research Experience:**

Mak, J. Y., & Kim, C. (in progress). *Differences among the selected socio-demographic variables to the transformational leadership style*. Assisted with data management

Asano, S. (2005) *Excess post exercise oxygen consumption and interval training*.

**Certifications/Memberships:**

Member of American College of Sports Medicine

Member of National Strength and Conditioning Association

Certified Strength and Conditioning Specialist, certification number 200322232

American Heart Association CPR/AED

American Red Cross: Community First AID and Safety, Adult CPR

**Awards:**

2005: Taylor Scholarship Award

1995-1999: Aichi Prefecture Boxing Champion: Bantam Weight

1995-1999: Tokai Regional Boxing Champion: Bantam Weight

1995: Japan Olympic Committee Junior Olympic Cup: 3<sup>rd</sup> place

1995: National High School Boxing Tournament: 3<sup>rd</sup> place

1995: 50<sup>th</sup> National Athletic Festival: Junior: 2<sup>nd</sup> place

1995: Japan Amateur Boxing Federation Junior Ranking: Bantam Weight 2<sup>nd</sup> place

1996: Member of National Team for Sydney Olympics 2000

1996: 51<sup>st</sup> National Athletic Festival: Senior: 5<sup>th</sup> place

1998: National All Japan Boxing Tournament: 3<sup>rd</sup> place

1998: Japan Amateur Boxing Federation Senior Ranking: Bantam Weight 3<sup>rd</sup> place

1999: National All Japan Boxing Tournament: 5<sup>th</sup> place

1999: Japan Amateur Boxing Federation: Senior Ranking: Feather Weight: 7<sup>th</sup> place

