

2014

Alterations and Specifications of Excess Post-Exercise Oxygen Consumption: A Review

Genevieve Kocoloski

Anne R. Crecelius

University of Dayton, acrecelius1@udayton.edu

Follow this and additional works at: http://ecommons.udayton.edu/hss_fac_pub



Part of the [Kinesiotherapy Commons](#), and the [Sports Sciences Commons](#)

eCommons Citation

Kocoloski, Genevieve and Crecelius, Anne R., "Alterations and Specifications of Excess Post-Exercise Oxygen Consumption: A Review" (2014). *Health and Sport Science Faculty Publications*. Paper 57.

http://ecommons.udayton.edu/hss_fac_pub/57

This Article is brought to you for free and open access by the Department of Health and Sport Science at eCommons. It has been accepted for inclusion in Health and Sport Science Faculty Publications by an authorized administrator of eCommons. For more information, please contact frice1@udayton.edu, mschlangen1@udayton.edu.

Alterations and Specifications of Excess Post-Exercise Oxygen Consumption: A Review

Genevieve Kocoloski^{1,2,3} and Anne R Crecelius¹

University of Dayton, 300 College Park, Dayton, OH, 45469

1. Department of Health and Sport Science
2. Berry Summer Thesis Institute
3. University Honors Program

Thesis Mentor: Anne R Crecelius, Ph.D.
Department of Health and Sport Science

Corresponding Author: Anne R Crecelius, Ph.D.
Email: acrecelius1@udayton.edu

Abstract

This review describes oxygen consumption, both in terms of a goal of weight management and aerobic training. It introduces excess post-exercise oxygen consumption (EPOC) and the benefits that can come from it. EPOC can aid in weight management as a means to continue to expend energy even after exercise has ceased. This review also discusses the many determinants of EPOC and analyzes the effects of various conditions on the elevated consumption. Such conditions include duration and intensity of exercise, training status, and supplementation. Later discussed are the possible underlying mechanisms and how they are responsible for EPOC. Although they have yet to be well-understood, these mechanisms provide insight into how EPOC is facilitated and why it occurs at all. More research is being conducted in attempts to better understand this concept and how EPOC can be advantageous to our human health.

Consistent aerobic exercise leads to many health benefits such as a positive impact on blood lipid levels and blood pressure, as well as increased energy expenditure for healthy weight management. Additionally, regular aerobic exercise can positively affect mental health, such as reducing depression and anxiety (Mersy, 1991). In general, aerobic exercise decreases the risk of the development of cardiovascular disorders, or disorders that affect the heart, blood vessels, or both. Given the significant burden of cardiovascular disease, aerobic exercise is a commonly prescribed lifestyle modification and is therefore important to fully understand.

Regular aerobic exercise causes the body to increase its oxygen consumption, otherwise known as its VO_2 . The consumption of oxygen fuels mitochondrial activity within muscle cells to produce ATP, the primary energy currency of all cells. During exercise, increased ATP is necessary in order to fuel contraction-relaxation cycles of muscles that together allow for body movement. With prolonged activity, the substrate for oxidative metabolism can come from stored energy sources, promoting weight loss. Ultimately it is the mass balance of energy intake versus energy expenditure that determines whether one will gain, lose, or maintain weight.

Exercise and Energy Expenditure

OVERVIEW

Given the hemodynamic and metabolic effects that aerobic exercise has on the body, there are two main reasons people engage in aerobic exercise. Some are simply looking to regulate or lose weight (Casazza, 2013), and aim to increase total energy expenditure in order to achieve net negative energy balance. However, others are high-performance athletes who depend on their ability to efficiently consume oxygen in order to perform the most work possible, or maintain high intensity levels. The ability to consume a lesser amount of oxygen to perform the same activities is advantageous for these athletes.

OXYGEN CONSUMPTION

The most efficient and most lucrative energy-producing pathway is aerobic metabolism, which uses oxygen to produce ATP. Aerobic exercise has been shown to improve cardiovascular structure and function while increasing energy expenditure and psychological benefit (Fletcher, 1996). When aerobic exercise is performed, VO_2 increases to provide the substrate for energy production. VO_{2max} , the maximal VO_2 achieved during progressive-intensity exercise, is a measure of aerobic capacity. A variety of factors can impact VO_{2max} including age (Fleg, 1988), sex (Hutchinson, 1991), training status (Sedlock, 1994; Caputo, 2004), environmental condition (Sawka, 1985) and supplementation (Brilla, 1990). Further, often times, submaximal exercise is quantified relative to one's VO_{2max} . Thus, in many ways, exercise and oxygen consumption are intimately related.

Excess Post-Exercise Oxygen Consumption

OVERVIEW

When exercise occurs, the demand for oxygen immediately rises, while the increase in oxygen consumption is somewhat delayed. Thus, an oxygen deficit is created in which the body's amount of oxygen consumption does not match the demand driving ATP production. Eventually, steady-state conditions are reached whereby the supply and demand is well-matched. Upon cessation of exercise, the demand for oxygen immediately declines. However, VO_2 remains elevated even after the exercise is over. It is thought that this excess post-exercise oxygen consumption (EPOC) occurs in order to make up for the inadequate oxygen consumption at the beginning of exercise. EPOC can be especially useful in weight control (Baum, 2008), but the specific mechanisms that facilitate prolonged EPOC remain unknown (Børsheim, 2003).

THE EFFECT OF DURATION AND INTENSITY

Both the duration and intensity of a given exercise bout, and the steady-state VO_2 levels reached can impact EPOC. Intensity of exercise has been shown to affect both duration and magnitude of EPOC, whereas exercise duration only affects duration of EPOC (Sedlock, 1989). Average EPOC after high-intensity exercise is higher than at low-intensity, and at the end

of a 3-hour period, EPOC remained elevated after high-intensity but not low-intensity exercise (Phelain, 1997).

Additionally, multiple, shorter bouts of exercise produce a greater EPOC response than one longer, continuous bout. Subjects cycling at 70% VO_{2max} twice for 15 minutes produce a greater magnitude of EPOC after the two bouts than those cycling once for 30 minutes, as seen in Figure 1 (Almuzaini, 1998). Therefore, repeated shorter bouts of exercise are more beneficial to those trying to lose or regulate weight than a longer, continuous bout.

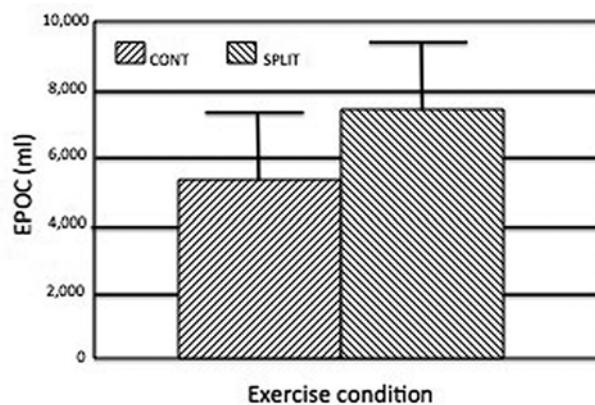


Figure 1. The differences in magnitude of EPOC between split and continuous exercise bouts. (Almuzaini, K.S., Potteiger, J.A., & Green, S.B. (1998). Effects of split exercise sessions on excess postexercise oxygen consumption and resting metabolic rate. *Canadian Journal of Applied Physiology*, 23(5), 433-443. © Canadian Science Publishing or its licensors.)

THE EFFECT OF TRAINING STATUS

It is difficult to examine the effect of training on EPOC response due to the differences in relative and absolute workloads between trained and untrained individuals. When the two groups work at the same relative workload, the absolute workloads differ and vice versa. When trained and untrained males exercised at the same relative workload of 50% VO_{2max} until the same energy expenditure was reached, no difference in the magnitude or duration of the resultant EPOC was observed (Sedlock, 1994). The difficulty of determining direct impacts of various conditions (training, environmental, supplement, etc.) on EPOC is that many of these conditions impact the intensity (relative to VO_{2max}) of the exercise bouts themselves. Thus, dissociating the direct impacts on EPOC versus the impact of the initial exercise but has proved difficult.

MECHANISTIC PROPERTIES OF EPOC

Rapid EPOC observed soon after the end of exercise can be attributed to a number of underlying mechanisms in the body. During exercise, oxygen deficits are created due to the widespread demand of this substrate. The oxygen consumed post-exercise is used to metabolize lactate to produce energy in the mitochondria and to replenish the depleted oxygen levels in skeletal muscle and blood (Gaesser, 1984).

During exercise, the amount of ATP and creatine phosphate in the blood deplete significantly, but they are replenished in the recovery period when VO_2 is elevated. Also during recovery, the 70% of lactate accumulated in the muscles is removed and released into the blood, further producing energy (Bangsbo, 1990).

Prolonged EPOC is not yet well-understood, but the elevated levels of ventilation and circulation, as well as an increased heart rate, can contribute (Bahr, 1992). The oxygen consumed post-exercise is important in order to return the body to its resting state. A possible mechanism for prolonged EPOC is β -adrenergic stimulation (Børsheim, 1998). Catecholamines such as epinephrine and norepinephrine are elevated in the bloodstream during exercise. Energy expenditure is stimulated by catecholamines, through these β -adrenoreceptors. It takes time for these catecholamine levels to return to baseline following the end of exercise, causing a prolonged, elevated energy expenditure post-exercise.

Additionally, during this state of EPOC, reduced blood pressure levels are seen following the

cessation of exercise. It is unlikely that these reduced levels are seen because of an increased production of NO (Halliwill, 2000). However, like many mechanisms of EPOC, it has yet to be determined what causes these reduced blood pressures.

ALTERING EPOC

EPOC can be affected through many factors such as intensity and duration of exercise, as discussed above. Additionally, given that cardiac output is a primary determinant of VO_2 (along with the arterial-venous oxygen content difference), factors that impact cardiac output may lead to alterations in VO_2 if not reflexively compensated for. At the level of the muscle, any substance capable of modulating mitochondrial respiration will impact VO_2 as well. One such substance that is capable of both hemodynamic and metabolic effects is the gaseous signaling molecule nitric oxide. Nitric oxide can be synthesized within the body via nitric oxide synthase during the conversion of L-arginine to L-citrulline. Additionally, the reduction of nitrate to nitrite to nitric oxide can also increase NO bioavailability.

Nitrates in the Body

NITRATES PATHWAY TO NITRIC OXIDE

The two main sources of nitrate in the body are dietary intake and the oxygen-dependent L-arginine-NO synthase pathway. We can either consume nitrates in the foods we eat or our bodies can convert L-arginine to nitric oxide. As seen in *Figure 2*, when inorganic nitrates (NO_3^-)

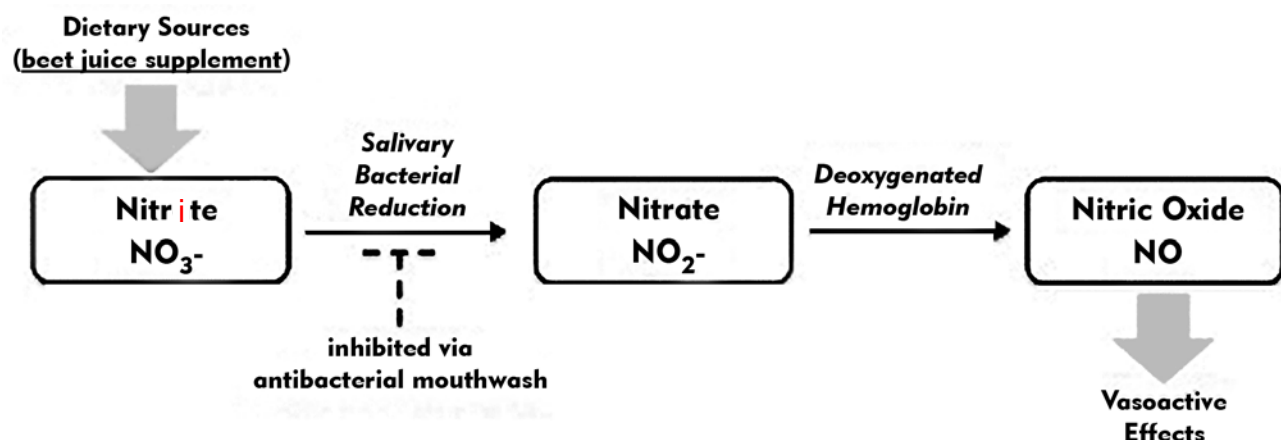


Figure 2. The pathway showing the reduction of ingested dietary nitrates to the gaseous nitric oxide. Figure courtesy of Genevieve Kocoloski, 2014.

are consumed, salivary bacteria convert them to nitrite (NO_2^-). Deoxygenated hemoglobin further convert the nitrites to nitric oxide (NO). If at any point this pathway is interrupted, the effects of ingesting nitrates will not be seen.

NITRIC OXIDE'S ROLE IN THE BODY

Nitric oxide is the substance responsible for relaxation of blood vessels, which will in turn increase oxygen delivery to the rest of the body. In addition to its hemodynamic effects, it has also been shown to stimulate mitochondrial biogenesis (Clementi, 2005; Nisoli, 2004), or production of new mitochondria.

More recently discovered is NO's effect on oxygen consumption during exercise. A recent study has shown that the ingestion of nitrate supplements can both decrease VO_2 and improve athletic performance (Cermak, 2012). However, because the amount of nitrates necessary to see these effects translates to 200-300 grams of spinach or beet root, concentrated supplements are commonly used to provide a more practical means of ingesting the substance.

Nitrate Supplements

ORGANIC NITRATES

Many organic vegetables, such as beet root and leafy greens, are good sources of nitrates. However, in order to obtain significant increases in plasma nitrates from these organic foods, one would have to consume two to four times the recommended serving size (Cermak, 2012). Instead, nitrate supplements such as concentrated beet root juice are commonly used.

EFFECT OF DOSAGE ON RESULTS

Since nitrates have been shown to improve athletic performance with their effects peaking around 2.5 hours after ingestion, it would be ideal to consume the supplements relatively close to the start of an event. However, the consumption of the beet juice this close to competition may not be ideal for many athletes. This led to a study that determined whether the duration of dosage of the nitrate supplement played a role in the magnitude of its effects.

Chronic ingestion of beetroot juice has been shown to improve exercise efficiency and peak power during a maximal effort, incremental

exercise test. After 15 days of consuming beet root juice, $\text{VO}_{2\text{max}}$ significantly decreased while the peak power significantly increased (Jones, 2012). Significant effects were not observed 2.5 hours or 5 days after ingestion. This increase in exercise tolerance can be attributed to a constant source of nitrates available to the body.

USEFULNESS OF SUPPLEMENTATION

Nitrate supplementation has been shown to result in many different hemodynamic and metabolic effects. These effects include a decrease in blood pressure, increase in heart rate, and decrease in $\text{VO}_{2\text{max}}$ at a given workload. Because a decrease in VO_2 is coupled with a decrease in energy expenditure, supplementing would not be useful when trying to lose weight, but more beneficial to those trying to improve athletic performance. Nitrate supplementation can be beneficial to performance, but it is important to consider the goal of the exercise when deciding if it is an appropriate supplement.

Conclusions

As stated above, exercise can provide many health benefits for those seeking to manage their weight or improve their athletic performance. Due to these important benefits, research has been done to find ways in which oxygen consumption can be altered to positively impact health. EPOC, a measure of oxygen consumption post-exercise has been found to play a key role in weight management, and thus has received much attention. Various substances such as nitric oxide have been found to impact oxygen consumption both during and after exercise.

Nitric oxide, which can be obtained through organic nitrate sources, has been shown to decrease $\text{VO}_{2\text{max}}$ and also decrease blood pressure when ingested prior to exercise. It has also been shown to improve performance in a competitive atmosphere. Effects of nitrate supplementation are continuing to be investigated, but evidence so far indicates its usefulness in performance exercise.

Aerobic exercise can help decrease the risk of many health issues, including but not limited to cardiovascular disorders. It's important to be aware of the many factors that can impact aerobic fitness and how they play a role in human health. When considering possible methods

to impact VO_2 or fitness in general, it's important to consider all of the effects of the intervention to determine whether or not that route is appropriate in achieving the ultimate health goal.

References

- Almuzaini, K. S., Potteiger, J. A., & Green, S. B. (1998). Effects of split exercise sessions on excess postexercise oxygen consumption and resting metabolic rate. *Canadian Journal of Applied Physiology*, 23(5), 433-443.
- Bahr, R. (1992). Excess postexercise oxygen consumption: Magnitude, mechanisms and practical implications. *Acta Physiol. Scand.*, 144(Suppl. 605), 1-70.
- Bangsbo, J., Gollnick, P.D., Graham, T.E., *et al.* (1990). Anaerobic energy production and O_2 deficit-debt relationship during exhaustive exercise in humans. *J Physiol*, 422, 539-59.
- Baum, K., Schuster, S. (2008). Excess post-exercise energy expenditure: A significant contribution to weight loss? *Deutsche Zeitschrift Fur Sportmedizin*, 59(5), 110-114.
- Børsheim, E., Knardahl, S., Høstmark, A., & Bahr, R. (1998). Adrenergic control of post-exercise metabolism. *Acta Physiologica Scandinavica*, 162(3), 313-323.
- Børsheim, E., & Bahr, R. (2003). Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. *Sports Medicine*, 33(14), 1037-1060.
- Brilla, L. R., & Landerholm, T. E. (1990). Effect of fish oil supplementation and exercise on serum lipids and aerobic fitness. *The Journal of Sports Medicine and Physical Fitness*, 30(2), 173-180.
- Caputo, F., & Denadai, B. S. (2004). Effects of aerobic endurance training status and specificity on oxygen uptake kinetics during maximal exercise. *European Journal of Applied Physiology*, 93(1-2), 87-95.
- Casazza, K., Fontaine, K. R., Astrup, A., Birch, L. L., Brown, A. W., Brown, M. M. B., *et al.* (2013). Myths, presumptions, and facts about obesity. *N Engl J Med*, 368(5), 446-454.
- Cermak, N. M., Gibala, M. J., & van Loon, L. J. (2012). Nitrate supplementation's improvement of 10-km time-trial performance in trained cyclists. *International Journal of Sport Nutrition and Exercise Metabolism*, 22(1), 64.
- Clementi, E., & Nisoli, E. (2005). Nitric oxide and mitochondrial biogenesis: A key to long-term regulation of cellular metabolism. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 142(2), 102-110.
- Fleg, J. L., & Lakatta, E. G. (1988). Role of muscle loss in the age-associated reduction in VO_2 max. *Journal of Applied Physiology*, 65(3), 1147-1151.
- Fletcher, G. F., Balady, G., Blair, S. N., Blumenthal, J., Caspersen, C., Chaitman, B., *et al.* (1996). Statement on exercise: Benefits and recommendations for physical activity programs for all americans. A statement for health professionals by the committee on exercise and cardiac rehabilitation of the council on clinical cardiology, american heart association. *Circulation*, 94(4), 857-862.
- Gaesser, G. A., & Brooks, G. A. (1984). Metabolic bases of excess post-exercise oxygen consumption: A review. *Medicine and Science in Sports and Exercise*, 16(1), 29-43.
- Halliwill, J. R., Minson, C. T., & Joyner, M. J. (2000). Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. *Journal of Applied Physiology* (Bethesda, Md.: 2000), 89(5), 1830-1836.
- Hutchinson, P., Cureton, K., Outz, H., & Wilson, G. (1991). Relationship of cardiac size to maximal oxygen uptake and body size in men and women. *Int J Sports Med*, 369, 373.
- Jones, A. M., Bailey, S. J., & Vanhatalo, A. (2012). Dietary nitrate and O_2 consumption during exercise. *Medicine and Sport Science*, 59, 29-35.
- Mersy, D. J. (1991). Health benefits of aerobic exercise. *Postgraduate Medicine*, 90(1), 103-7, 110-2.
- Nisoli, E., Falcone, S., Tonello, C., Cozzi, V., Palomba, L., Fiorani, M., *et al.* (2004). Mitochondrial biogenesis by NO yields functionally active mitochondria in mammals. *Proceedings of the National Academy of Sciences of the United States of America*, 101(47), 16507-16512.
- Phelain, J.F., Reinke, E., Harris, M.A., *et al.* (1997). Postexercise energy expenditure and substrate oxidation in young women resulting from exercise bouts of different intensity. *J Am Coll Nutr*, 2, 140-6.
- Sawka, M. N., Young, A. J., Cadarette, B. S., Levine, L., & Pandolf, K. B. (1985). Influence of heat stress and acclimation on maximal aerobic power. *European Journal of Applied Physiology and Occupational Physiology*, 53(4), 294-298.
- Sedlock, D. A. (1994). Fitness level and postexercise energy expenditure. *The Journal of Sports Medicine and Physical Fitness*, 34(4), 336-342.
- Sedlock, D. A., Fissinger, J. A., & Melby, C. L. (1989). Effect of exercise intensity and duration on postexercise energy expenditure. *Medicine and Science in Sports and Exercise*, 21(6), 662-666.