## **Hormonal Weight Loss:**

Is there such a thing as the "Metabolic Effect?" Jade Teta ND, CSCS and Keoni Teta ND, LAc, CSCS

It is time to bring the science of weight loss out of the dark ages and apply a new understanding of exercise's impact on hormones and metabolism. The environment a person chooses dramatically affects the processing and use of energy he or she consumes. Intelligent exercise releases hormones in the body, and these chemical messengers translate movement into metabolic action. Hormonal signals are powerful determinants of which fuel our metabolic engine will use: sugar versus fat. Therefore, hormones manage much more than just caloric input and output. There is an optimal state of hormonal balance that enhances utilization of the body's fat stores; we call this the metabolic effect. The intelligent manipulation of lifestyle choices like exercise is the chief means of accessing this highly beneficial state of function.

To begin this discussion, let's take a look at how a strictly caloric model of metabolism holds up in examples of real people. It is useful to use athletes in this example since they are widely regarded as extremely functional and metabolically efficient. Among track athletes, both elite marathoners and sprinters are extremely lean. Any average person can quickly distinguish the difference between these two groups of athletes. One is muscular and lean while the other is more gaunt and wiry. Of these athletes, sprinters have less body fat and higher amounts of muscle mass, yet they burn far fewer calories when training for and engaging in their sport <sup>1-2</sup>. Sprinters engage in very short bursts of all out effort lasting seconds while marathoners run for hours and consume large amounts of caloric energy. If the calorie model is the final word on fat loss, why is there a discrepancy? Shouldn't marathoners be the leaner of the athletes?

To understand this glaring contradiction, the discussion must move to hormones and fuel metabolism. Hormones as described here simply refer to all signaling molecules in the body. In the case of weight loss, these chemical messengers are the ultimate predictors of the degree and type of energy used. The body is like an engine that can choose between two fuels. Fat is analogous to diesel fuel; it will get you far, but it wont provide much performance. Sugar is like high-octane and delivers exceptional performance but horrible mileage. Hormonal messengers determine which fuel dominates. In reality, the body burns both fuels all the time, but lifestyle choices elicit hormones that determine the amount of each fuel burned. The body becomes efficient at burning what you feed it and it preferentially replenishes used energy by refilling its tanks with the alternative fuel. In other words, eat sugar at a meal and you will burn sugar after the meal, but burn sugar during exercise and you will burn fat after. With this understanding, eating and exercise programs can be designed to release the optimal hormonal situation for accelerated fat loss we call the metabolic effect.

## **EPOC:** The metabolic effect of exercise

Exercise that modulates hormonal effects will burn more calories during activity and provide greater caloric benefit after exercise <sup>25</sup>. This increased energy use after an intelligent workout is referred to as *excess post-exercise oxygen consumption*, or EPOC. This is a measure of how much oxygen the body consumes in the hours and days after a workout. An example of EPOC in the acute sense is climbing a steep flight of stairs. While walking up the stairs breathing is labored, but respiration becomes most difficult after reaching the top. The body does this to recover the "debt" of oxygen created during activity. The EPOC created by climbing a flight of steps is an example of the much larger metabolic effect created from intelligent movement. Intelligent exercise, and creates sustained fat-burning after. The amount of oxygen consumed is directly correlated to how much energy is burned, but the hormonal situation determines whether that energy is mostly fat or sugar.

Most people wrongly assume that low intensity exercise burns more fat than higher intensity exercise. This is true only from a relative perspective. Relatively speaking, the lower the exercise intensity, the higher proportion of fat you burn compared to sugar. However, exercise of higher intensity and beyond the aerobic training zone burns more absolute energy and fat. Suppose two people go out an exercise for thirty minutes. Person A does aerobic exercise at an intensity of 60% max heart rate, while person B does interval training by exercising at an intensity of 60% max heart rate and then frequently (every few minutes) spikes the intensity above 85% for a short period and then returns to the lower intensity. Let's say Person A burned 200 calories total, 60% of which was fat and 40% of which was sugar. Therefore, Person A burned 120 total units of fat and 80 units of sugar. Person B, who exercised at a higher intensity with intervals, burned 50% fat and 50% sugar, but burned 300 calories total. This means Person B burned 150 units of fat and 150 units of sugar. We can see by this example, that Person B burns more energy (300 calories) and more total fat (150 units compared to 120 units) than person A despite a lower percentage of total energy coming from fat. This shows higher intensity exercise far exceeds its low intensity counterpart during exercise in addition to hormonal and EPOC benefits that last long after.

The idea of hormonal influences on calorie burning is a novel concept to some, and is far more complex than simple one-dimensional models of hormonal metabolism. For example, we know that exercise of sufficient intensity elevates stress hormones like adrenaline, nor-adrenaline, and cortisol. As an innate physiological response to stress, these hormones are generated during a "fight or flight" response. Together they ensure the switch to high-octane sugar usage which historically supplied the energy to fight for our lives or run like hell. As we run faster and harder the body's supply of oxygen drops off. Since sugar is a fuel that can be burned in the absence of oxygen, highly intense activity depletes sugar stores. This increase in anaerobic metabolism generates lactic acid which is far more than a waste product, but also a buffering aid and likely signaling molecule <sup>26-28</sup>. As lactic acid builds up to extreme levels, it is correlated with powerful metabolic stimulants like testosterone and human growth hormone <sup>22-24</sup>. The total

hormonal environment created acts synergistically to produce a leaner and more functional physiology.

The effect of these hormonal messengers persists after activity, and that coupled with empty energy reserves delivers signals that rebuild, regenerate, and recycle energy. Since sugar stores are depleted during intense exercise, fat is used after to repair the body and regenerate sugar reserves. In this way, the body becomes a fat burning machine through the hormonal metabolic effect and the ensuing EPOC. This finely orchestrated hormonal response creates the perfect scenario for fat burning and muscle building and ensures survival by generating a leaner, faster, and stronger body. It is useful to point out that humans in natural conditions did low intensity activity all day everyday. However, the last activity one should choose when confronted with stress and high blood sugar is low intensity exercise. This runs counter to inherited physiology and biochemical understanding. Our genes and metabolic processes are still tuned to a fight or flight reality. Intelligent hormonal exercise works along with this ancient machinery.

Interestingly, the scenario above describes the type of exercise sprinters use in their training. It is important to point out the rise in cortisol many people fear is only a problem when it is unopposed by growth hormone and testosterone<sup>3-6</sup>. Hormones do not work in isolation, and like people they will behave differently depending on the social environment they find themselves in. When cortisol is "socializing" with testosterone and growth hormone, its muscle breakdown is blocked, fat storing at the belly is reversed, and the three synergistically enhance fat burning <sup>3-6</sup>. Attempting to blunt the cortisol response to high intensity exercise is counterproductive for fat burning and not necessary in the context of growth hormones <sup>7-10</sup>. Long duration and lower intensity cardiovascular exercise is more the problem because it causes cortisol to rise unopposed by the growth promoting hormones. This may explain why standard aerobic prescriptions are not as effective for optimal body composition and why marathon runners exhibit frail bodies devoid of muscle <sup>14-17, 20</sup>. Duration of exercise and not the intensity is the most salient issue in regards to cortisol <sup>21</sup>.

## **Intelligent Exercise:**

The description above dictates that intelligent exercise must be intense enough to elicit the hormonal metabolic effect described. There are many tools and techniques to generate this effect with exercise, but none of them include long duration or "aerobic zone" training. This new technology and understanding dictates that the real fat burning zone exists at higher intensities. Breaching 85% to 90% of maximum heart rate ensures adequate intensity and can easily be managed with short duration interval training. This level of exertion correlates well with the ability to speak during exercise <sup>32</sup>. In addition, a weight training program that uses full body movements, short rest periods, and forces both mechanical and metabolic muscle failure will cause a ripple effect lasting long after exercise has ended <sup>18-19</sup>.

So how long does this metabolic effect last? When the tools and techniques described are used appropriately the magnitude and duration of EPOC is substantial. Two resistance

training studies that combined many of the elements described above showed a sixteen hour elevation for women and a forty-eight hour elevation for men <sup>18-19</sup>. Studies on interval training show similar effects <sup>11-13</sup>. This is admittedly hard to swallow when one considers exercisers spend countless hours doing aerobic workouts which are largely ineffective for weight loss <sup>14-17</sup>.

## Some Studies:

A 2001 study in the American College of Sports Medicine's flagship journal, Medicine and Science in Sports and Exercise illustrates the point nicely <sup>31</sup>. This study compared two groups of women. One group exercised using standard zone aerobic training while the other group used anaerobic interval exercise. The anaerobic interval group exercised for 2 minutes at a highly intense 97% max heart rate. They then rested by doing three minutes of low intensity activity. The first, more aerobic group performed moderately intense activity at close to 70% of max heart rate. The researchers made sure that each group burned exactly 300 calories. Despite exercising longer and burning the same amount of calories, the aerobic group lost less body fat at the end of the study compared to the interval group. In addition, fitness in the interval group was substantially greater than in the aerobic group. This study demonstrates the effect of EPOC and shows that something other than just calories is driving metabolism.

A similar study published in the same journal in 1996 showed that an anaerobic trained interval group burned significantly more fat than their aerobically trained counterparts <sup>30</sup>. Not only did the interval group burn more fat during exercise, but they exhibited increased fat burning effects that persisted for 24 hours after the exercise had stopped. These results clearly show that high intensity interval training burns more overall fat and calories during exercise, and demonstrate EPOC leads to a continued fat burn after exercise as well. Perhaps the most interesting thing about this study is that the interval group was able to accomplish all this with an exercise session that was a full 15 minutes shorter than the aerobic group. This shows that intelligent exercise moving away from the aerobic paradigm allows exercisers to have their cake and eat it too.

Perhaps the most telling study on the effects of high intensity exercise vs. aerobic training came in 1994 in the journal Metabolism <sup>29</sup>. This study tracked two groups of people undergoing different modes of exercise. Group 1 did zone aerobic training for a period of 20 weeks, while Group 2 did 15 weeks of a high intensity interval program. The researchers wanted to see how each program would affect body fatness and metabolism. The results showed that the aerobic group burned 48% more calories than the interval group (120.4 MJ vs 57.9MJ) over the course of the study. However, despite the huge caloric disadvantage, the interval group enjoyed a 9 fold greater loss in subcutaneous fat (fat under the skin). Most remarkably, resting levels of 3-hydroxyacyl coenzyme A dehydrogenase (HADH), an enzymatic marker of fat burning, were significantly elevated in the interval group trained 5 weeks less than the aerobic group, had shorter workouts, and yet far exceeded the aerobic group in fat burning at rest and during exercise. The

measurement of fat burning enzymes in this study shows for the first time that this new exercise technology can "teach" the body to be a more efficient fat burning machine.

The current exercise environment for weight loss is still rooted in the low intensity, single mode and calorie burning paradigms. This approach is successful for some, yet fails the vast majority. New models for exercise are needed to combat the growing epidemic of obesity and chronic disease. Short duration, high intensity exercise offers a clear departure from current weight loss models. Those that desire real transformations, and are frustrated by cook book exercise prescriptions, need new and improved approaches for overcoming obesity. Training for the metabolic effect offers healthcare providers, trainers, and gym managers alike new and effective exercise techniques to combat obesity and ensure weight loss.



References:

- 1) Spenst ET AL. (1993) Muscle Mass of Competitive Male Athletes. Journal of Sports Science.11(1):3-8.
- 2) Barnard ET AL. (1979) Physiological characteristics of sprint and endurance masters runners. Medicine and Science in Sports and Exercise.11(2):167-71.
- Ottosson ET. AL. (2000). Effect of Cortisol and Growth Hormone on Lipolysis in Human Adipose Tissue. J Clin Endocrinol Metab. 85(2):799-803.
- Crawford ET AL. (2003). Randomized Placebo-Controlled trial of androgen Effects in Muscle & Bone in Men Requiring Long-Term Glucocorticoid Treatment. J Clin Endocrinol Metab. 88(7):3167-3176.
- 5) Bjorntorp ET AL. (1997) Hormonal Control of Regional Fat Distribution. Hum Reprod. Suppl 1:21-25.
- 6) McCarty ET AL. (2001). Modulation of adipocyte lipoprotein lipase expression as a strategy for preventing or treating visceral adiposity. Med Hypotheses. 57(2):192-200.
- 7) Ottosson Et AL. (1995). Growth hormone inhibits lipoprotein lipase activity in human adipose tissue. Journal of Clinical Endocrinology and Metabolism, 80, 936-941
- 8) Samra Et AL. (1998). Effects of physiological hypercortisolemia on the regulation of lipolysis in subcutaneous adipose tissue. Journal of Clinical Endocrinology and Metabolism, 83, 626-631
- 9) Djurhuus ET AL. (2004). Additive effects of cortisol and growth hormone on regional and systemic lipolysis in humans. American Journal of Physiology, E286, 488-494
- 10) Djurhuus ET AL. (2002). Effects of cortisol on lipolysis and regional interstitial glycerol levels in humans. American Journal of Physiology, E283, 172-177
- 11) Kraemer ET AL. (1991). Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. International Journal of Sports Medicine, 12:228-235.
- 12) Osterberg ET AL. (2000). Effect of acute resistance exercise on postexercise oxygen consumption and resting metabolic rate in young women. International Journal of Sport Nutrition and Exercise Metabolism, 10:71-81.
- 13) King ET AL. (2001) A comparison of high intensity vs. low intensity exercise on body composition in overweight women. Medicine and Science in Sports and Exercise, 33:A2421.
- 14) Miller ET AL. (1997). A meta analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. International Journal of Obesity, 21:941-947.

 Sjodin ET AL. (1996). The influence of physical activity on BMR. Medicine and Science in Sports and Exercise, 28:85-91.

Xiser

- 16) Kraemer ET AL. (1999). Influence of exercise training on physiological and performance changes with weight loss in men. Medicine and Science in Sports and Exercise, 31:1320-1329.
- 17) Wilmore ET AL. (1999). Alterations in body weight and composition consequent to 20 wk of endurance training: the HERITAGE Family Study. American Journal of Clinical Nutrition, 70:346-352.
- Osterberg ET AL. (2000) Effect of acute resistance exercise on postexercise oxygen consumption and resting metabolic rate in young women. International Journal of Sport Nutrition and Exercise Metabolism.10(1):71-81.
- Schuenke ET AL. (2002) Effect of an acute period of resistance exercise on excess post-exercise oxygen consumption: Implicationsfor body mass management European Journal of Applied Physiology. 86:411-417.
- 20) Kraemer ET AL. (1997). Physiological adaptations to a weight-loss dietary regimen and exercise programs in women. Journal of Applied Physiology, 83:270-279.
- Jacks ET AL. (2002) Effect of exercise at three exercise intensities on salivary cortisol. Journal of Strength and Conditioning Research. 16:286-289.
- 22) Turner ET AL. (1995). Effect of graded epinephrine infusion on blood lactate response to exercise. J Appl Physiol,79(4):1206-11.
- 23) Takahashi ET AL.(1995). Relationship among blood lactate and plasma catecholamine levels during exercise in acute hypoxia. Applied Human Sci,14(1):49-53.
- 24) Kaiser ET AL. (1983). Effects of acute beta-adrenergic blockade on blood and muscle lactate concentration during submaximal exercise. International Journal Sports Med, 4(4):275-7.
- Bell ET AL. (2000). Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. European Journal of Applied Physiology, 81:418–427.
- Gladden (2004). Lactate Metabolism: A new paradigm for the third millennium. Journal of Physiology. 558(1):5-30.
- 27) Chawalbinska-Moneta ET AL (1996). Threshold increases in plasma growth hormone in relation to plasma catecholamine and blood lactate concentrations during progressive exercise in endurance-trained athletes. European Journal of Applied Physiology. 73(1-2):117-120
- Godfrey ET AL (2003). The exercise-induced growth hormone response in athletes. Sports Medicine. 33(8):599-613
- Tremblay ET AL. (1994). Impact of exercise intensity on body fatness and skeletal muscle metabolism. Metabolism. 43:814-818
- 30) Treuth ET AL. (1996). Effects of exercise intensity on 24-h energy expenditure and substrate oxidation. Medicine and Science in Sports and Exercise, 28, 1138-1143
- 31) King ET AL. (2001). A comparison of high intensity vs. low intensity exercise on body composition in overweight women. Medicine and Science in Sports and Exercise, 33, A2421
- 32) Meckel ET AL. (2002). The effects of speech production on physiological responses during submaximal exercise. Medicine and Science in Sports and Exercise. 34(8):1337-1343.

WWW.MYXISER.COM

The Worlds Best HIIT Mini Stepper Xiser