Fatmax: A New Concept to Optimize Fat Oxidation During Exercise?

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It has been recognized that facilitation of fat metabolism is important for both performance and the health-related aspects of exercise. Although several studies have described the relationship between exercise intensity and fat oxidation, few studies have studied this relationship over a wide range of intensities. In absolute terms, carbohydrate oxidation will increase proportionally with exercise intensity, whereas the rate of fat oxidation will initially increase but will decrease again at high exercise intensities. Here we have defined the exercise intensity at which maximal fat oxidation is observed as *Fatmax*. This exercise intensity may have importance for weight loss programs, health-related exercise programs, and endurance training. Future research should focus on developing an exercise test with which *Fatmax* can be accurately determined. Further research should investigate the efficacy of training at *Fatmax* in a variety of conditions.

Key Words: exercise intensity, fuel oxidation, fat metabolism

Key Points:

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Introduction

Much of the emphasis of exercise programs for obese patients, and endurance training for athletes, is on cardiovascular and respiratory benefits, and increasing the oxidative capacity of skeletal muscle (3, 11). Fat metabolism, however, is an area that has received less attention, even though its importance has been recognized for both performance and the health-related aspects of exercise (14-17, 25).

Endurance performance is largely determined by the ability of the athlete to mobilize and oxidize fat to spare reserves of carbohydrates, and this is most likely susceptible to specific training (13). In weight control programs, exercise is used alone or in conjunction with a dietary restriction. When exercise is the only intervention, the main goal is to reduce body fat (9, 22, 30). When combined with a diet program, it is mainly used to counteract the decrease in fat oxidation often

seen after weight loss (2, 7, 23). In a variety of clinical conditions, exercise may be beneficial through changes in fat metabolism. For example, an increased capacity to oxidize fat as a fuel in diabetic patients will make them less dependent on glucose as a source of energy (29). Although it is generally agreed that exercise programs will increase the capacity to oxidize fat, there is controversy as to what exercise intensity or training program should be employed in order to achieve the aims of all three groups, and guidelines are generally inconsistent.

The purpose of this short review is not to describe new factual information but to provide new ideas, introduce a new concept, and expand the boundaries of our thinking about exercise training in relation to fat metabolism.

Fat Oxidation During Exercise

Effect of Exercise Intensity on Fat Oxidation

Already in 1939, Christensen and Hansen (4) observed that changes in the exercise intensity induced changes in substrate utilization. With increasing exercise intensity, the relative contribution of carbohydrate as a fuel will increase; concomitantly, the relative contribution of fat oxidation decreases. However, in absolute terms, carbohydrate oxidation will increase proportionally with the exercise intensity, whereas the rate of fat oxidation will initially increase but will decrease again at high exercise intensities (21). At intensities where the rate of glycolysis has increased considerably (at exercise intensities above lactate threshold), relatively less fatty acids will be used as a fuel, and although the energy expenditure is increasing, absolute rates of fat oxidation will actually decrease. In Figure 1, fat oxidation is depicted versus the exercise intensity expressed as VO₂. The data are based on the theoretical calculation based on stoichiometric equations (8), where the respiratory exchange ratio (RER) increases linearly from 0.86 (an average resting RER) to 1.00 as VO₂ increases linearly from 1.2 to 4.0 L/min. The exercise intensity at which fat oxidation is optimal will thus be somewhere between the low and the high intensity exercise. Here, we would like to introduce the term Fatmax to describe the exercise intensity at which fat oxidation is maximal. To our knowledge, at present, no studies have attempted to identify this exercise intensity systematically and accurately.

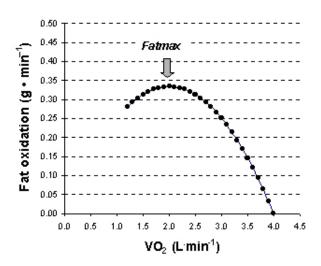


Figure 1 — Theoretical curve indicating fat oxidation as a function of exercise intensity (VO₂). The exercise intensity at which the highest rate of fat oxidation is observed is referred to as *Fatmax*.

Mechanisms Behind Fatmax

Most likely, *Fatmax* will be just below the intensity where the rate of glycolysis commences to increase markedly. Recently, we and others have shown that increasing the rate of glycolysis by feeding subjects high carbohydrate pre-exercise feedings (6) or by increasing the exercise intensity from 40 to 70% VO_{2max} (24) decreases the oxidation of long chain fatty acids but not of medium chain fatty acids. This suggested that increasing rates of glycolysis inhibit fat oxidation by inhibiting the CPT-1 and carnitine-mediated transport of fatty acids into mitochondria. In addition, several studies reported a decreased rate of appearance of fatty acids into the circulation during intense exercise, suggesting that the mobilization of fatty acids is decreased. This could be linked to reduced rates of lipolysis or reduced blood flow to the adipose tissue (for review, see 14–17). Because fat oxidation is directly dependent on the rate of glycolysis, *Fatmax* might coincide with the onset of blood lactate accumulation. This hypothesis has not been tested.

Potential Applications of Fatmax

Exercise training programs at an intensity that elicits maximal rates of fat oxidation (i.e., *Fatmax*) may be useful in a variety of conditions and for a variety of populations. For example, it could have implications for exercise programs to treat and prevent cardiovascular diseases, obesity, and non-insulin-dependent Diabetes Mellitus (NIDDM). It could also be used in general weight reduction programs and for athletes as a means to increase one's capacity to oxidize fat.

Training Programs for Athletes. One of the main adaptations to endurance training is a shift from carbohydrate towards fat metabolism (12, 13, 28). The ability to oxidize fat as a fuel is also highly correlated with exercise performance (12, 13). Training of endurance athletes therefore often includes training sessions that specifically aim to improve fat metabolism. Training at an exercise intensity at which metabolic pathways involved in fat metabolism are maximally activated could, at least theoretically, result in optimal adaptations in these pathways. This however has not yet been investigated.

Exercise Programs to Lose Weight or Body Fat. A recent meta-analytical review reported that a decrease in bodyweight of 0.2 kg/week can be achieved by following an exercise program (19). The studies showed great variety in the exercise prescription for the obese population. The same review also suggested that exercise is critical in weight maintenance. A smaller percentage of the weight loss induced by a restricted diet will be regained when an exercise program is followed during and after the diet period. However, the best exercise type, intensity, and duration are still unclear. The latest recommendation of an expert panel from the Centers for Disease Control and the American College of Sports Medicine consist of 30 min or more of moderate-intensity physical activity on most days of the week (1). The objective of this recommendation is to encourage more participation in physical activity. For obese individuals, the frequency, intensity, and duration of exercise are more focussed on increasing energy expenditure than on increasing fat oxidation. Finding the optimal intensity for fat oxidation might increase weight loss and support weight maintenance.

Exercise Programs to Treat or Prevent Welfare Diseases. There is considerable interest in finding treatments and preventive measures to avoid overweight, obesity, and associated diseases like NIDDM, arteriosclerosis, and hypertension (26). Exercise has been shown to significantly decrease the risk of developing such diseases (18, 27). However, there is controversy as to what

type of exercise and what exercise intensity would be most effective in reducing this risk. Since many of these disease states are linked to high levels of circulating triglycerides and disturbance in fat metabolism (5, 10, 20, 29), it is tempting to think that exercise intensities at which a maximal amount of fat is oxidized (*Fatmax*) would be a preferred intensity. Therefore, determination of *Fatmax* could also be a useful tool to design exercise programs in order to obtain optimal health benefits.

Conclusions and Directions for Future Research

The concept of *Fatmax* seems promising and may be an efficient exercise intensity for weight loss programs, health-related exercise programs, and endurance training. Future research should focus on developing an exercise test with which *Fatmax* can be accurately determined, and such a test needs to be validated and tested for reliability. Further research should investigate the efficacy of training at *Fatmax* under a variety of conditions, and methods should be developed to monitor exercise intensity during training.

References

1. ACSM, CDC. 1995. Physical activity and public health—a recommendation from the centers for disease control and prevention and the american college of sports medicine. JAMA 273:402-7.

2. Astrup A. 1993. Dietary composition, substrate balances and body fat in subjects with a predisposition to obesity. Int J Obes 17(suppl. 3):S32-S36.

3. Blair SN, Kampert JB, Kohl HW, Barlow CE, Macera CA, Paffenbarger RS, Jr., Gibbons LW. 1996. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA 276:205-10.

4. Christensen E, Hansen O. 1939. Arbeitsfahigkeit und ernahrung. Scand Arch Physiol 81:160-71.

5. Coppack SW, Jensen MD, Miles JM. 1994. In vivo regulation of lipolysis in humans. J Lipid Res 35:177-93.

6. Coyle EF, Jeukendrup AE, Wagenmakers AJM, Saris WHM. 1997. Fatty acid oxidation is directly regulated by carbohydrate metabolism during exercise. Am J Physiol 273:E268-E275.

7. Flatt JP. 1995. Use and storage of carbohydrate and fat. Am J Clin Nutr 61:952S-959S.

8. Frayn KN. 1983. Calculations of substrate oxidation rates in vivo from gaseous exchange. J Appl Physiol 55:628-34.

9. Garrow JS, Summerbell CD. 1994. Meta-analysis on the effect of exercise on the composition of weight loss. Int J Obes 18:516-17.

10. Groop LC, Bonadonna RC, Simonson DC, Petrides AS, Shank M, DeFronzo RA. 1992. Effect of insulin on oxidative and nonoxidative pathways of free fatty acid metabolism in human obesity. Am J Physiol 263:E79-E84.

11. Henriksson J. 1977. Training induced adaptation of skeletal muscle and metabolism during submaximal exercise. J Physiol (Lond) 270:661-75.

12. Holloszy JO, Coyle EF. 1984. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol 56:831-38.

13. Jansson E, Kaijser L. 1987. Substrate utilization and enzymes in skeletal muscle of extremely endurance-trained men. J Appl Physiol 62:999-1005.

14. Jeukendrup AE. 1999. Dietary fat and physical performance. Curr Opin Clin Nutr 2:521-26.

15. Jeukendrup AE, Saris WHM, Wagenmakers AJM. 1998. Fat metabolism during exercise: a review. Part I: Fat mobilization and muscle metabolism. Int J Sports Med 19:231-44.

16. Jeukendrup AE, Saris WHM, Wagenmakers AJM. 1998. Fat metabolism during exercise: a review. Part II: regulation of metabolism and the effects of training. Int J Sports Med 19:293-302.

17. Jeukendrup AE, Saris WHM, Wagenmakers AJM. 1998. Fat metabolism during exercise: a review. Part III: effects of nutritional interventions. Int J Sports Med 19:371-79.

18. Krotkiewski M, Mandroukas K, Sjostrom L, Sullivan L, Welterqvist H, Bjorntorp P. 1994. Effects of long-term physical training on body fat, metabolism, and blood pressure in obesity. Metabolism 28:650-58.

19. Miller WC, Koceja DM, Hamilton EJ. 1997. A meta-analytical of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. Int J Obes 21:941-47.

20. Nestel PJ, Ishikawa T, Goldrick RB. 1978. Diminished plasma free fatty acid clearance in obese subjects. Metabolism 27:589-97.

21. Romijn J, Coyle E, Sidossis L, Gastadeldelli A, Horowitz J, Endert E, Wolfe R. 1993. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. Am J Physiol 265:E380-E391.

22. Saris WHM. 1993. The role of exercise in the dietary treatment of obesity. Int J Obes 17:(suppl. 1):S17-S21.

23. Schutz Y, Tremblay A, Weinsier RL, Nelson KM. 1992. Role of fat oxidation in the long-term stabilization of body weight in obese women. Am J Clin Nutr 55:670-74.

24. Sidossis L, Gastaldelli A, Klein S, Wolfe RR. 1997. Regulation of plasma fatty acid oxidation during low- and high-intensity exercise. Am J Physiol 272:E1065-E1070.

25. Tappy L, Felber JP, Jéquier E. 1991. Energy and substrate metabolism in obesity and postobese state. Diabetes Care 14:1180-88.

26. Thompson DL, Townsend KM, Boughey R, Patterson K, Basset DR. 1998. Substrate use during and following moderate- and low-intensity exercise: implications for weight control. Eur J Appl Physiol 78:43-49.

27. Tremblay A, Despres JP, Pouliot MC. 1991. Normalization of the metabolic profile in obese women by exercise and a low-fat diet. Med Sci Sports Exerc 23:1326-31.

28. Turcotte LP, Richter EA, Kiens B. 1992. Increased plasma FFA uptake and oxidation during prolonged exercise in trained vs. untrained humans. Am J Physiol 262:E791-E799.

29. Walberg-Henriksson H, Rincon J, Zierath JR. 1998. Exercise in the management of non-insulin-dependent diabetes mellitus. Sports Med 25:25-35.

30. Zachweija JJ. 1996. Exercise as treatment for obesity. Endocrinol Metab Clin North Am 25:965-88.

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