# SPORTS CONDITIONING

## A comparison: moderate-intensity continuous activity and high-intensity intermittent activity

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Many individuals in the general population, including professionals in the fitness industry, still hear the message that exercise less than 20 minutes in duration is not beneficial for either the cardiovascular system or weight loss. Furthermore, where the goal is weight loss, that exercise should be low intensity. This thinking likely derived from the influence of the running craze of the early seventies and corresponding growth of sports science research. During such time, the term “aerobic” became synonymous with health and fitness and, although used much less, the term “anaerobic” was misunderstood and virtually given a “warning” label (in other words, avoid this type of activity). In a similar fashion, sports and activities were simplistically labeled aerobic or anaerobic, which is a clear misunderstanding of human metabolic pathways. The question is: has this thought process influenced the competitive world of sports?

During the time these misunderstandings developed, and with scant research from the new world of sport science, the sporting world continued to train utilizing the methods that developed over years of experience based largely on trial and error. Before training became popular, athletes developed most of their fitness by simply playing their sport. Consequently, many field and court sport athletes, as well as many other athletes, developed their fitness, almost exclusively, by engaging in high-intensity intermittent bursts of activity (i.e., activities inherent to their sport). Despite that, many field and court sport athletes today spend a significant amount of time utilizing low- to moderate-intensity continuous exercise in order to “build an aerobic base.” Given such disconnect, it is important for athletes and coaches to accurately understand the physiology of their sport so that they may make educated decisions in regard to their training.

An examination of the conditioning programs of high schools, colleges, and professional field and court sports teams reveals a wide variety of training philosophies, all of which include a significant amount of high-intensity intermittent training. The area wherein programs often differ is in the quantity of low- to moderate-intensity continuous “aerobic” training. Therefore, this article seeks to highlight recent research, which may influence athletes and coaches to examine the scientific literature and historical influence of early sport science and then question if the training methods they employ are optimal for the sport at hand. It is important to note, however, that this article is in no way intended to question the endurance training that is required by endurance athletes; rather, to present research causing endurance athletes to consider adding more high-intensity intermittent training to their programs without a concern for diminishing their endurance capacity. It is also accepted that low- to moderate-intensity activities are useful in recovering from high-intensity exercise and is a necessity in some sports that require repetitive practice to acquire skill. However, while the contention is not that low- to moderate-intensity continuous exercise can improve cardiovascular conditioning and weight loss, the need for significant quantities of this type of training for the field and court sport athlete is indeed challenged.

Early sports science laboratories demonstrated a common pattern. Central to these rooms was typically a treadmill, stationary bike, some Douglas bags (an out-dated tool for collecting expired ventilation), and instruments for analyzing the expired ventilation. What is important to realize is that, relative to today’s standards, the instrumentation available in earlier days was extremely limited in its ability to measure expired gases. Basically, the instrumentation could only determine oxygen consumption over extended periods. Consequently, research in the exercise sciences was biased toward examining steady-state continuous activity. The early recommendations of the American College of Sports Medicine[1] (ACSM) still ring loud, “Use rhythmical activities such as running, biking, or swimming, 3 to 5 times per week, for 20-60 minutes, at an intensity of 60-90% of one’s heart rate reserve.” Considering nearly all sport science research is conducted at academic institutions, it is noteworthy that the majority of the subjects in the early studies were students, whose classes tended to be 3-5 times per week for 30-60 minutes. Coincidence or bias? The recommended intensity was then likely a result of being slave to the dictated duration, the majority of humans will simply exercise at an intensity of 60-90% of the heart rate reserve given a duration of 20-60 minutes. The point is not that the research influencing the early recommendations of the ACSM was inaccurate, but that it unwittingly created a thought process within the fitness industry and sports world that non-continuous high-intensity activity was ineffective in promoting cardiovascular fitness. Further, there developed two commonly held misconceptions. First, metabolic pathways providing energy for muscular contraction respond in a definitive sequential manner. Second, the aerobic system responds slowly to the demands of exercise and, therefore, has little influence over intense exercise of short duration.

Now that the techniques for analyzing human metabolism are more sophisticated, more research is being published about high-intensity intermittent training. A thorough review of the scientific literature reveals a significant body of research that contradicts the understanding of many athletes, coaches, and fitness professionals. Although it is possible to write an entire book on high-intensity intermittent exercise, this article endeavors to highlight only the most important elements of recent research.

Research has shown that subjects participating in games activities have gained similar improvements in cardiovascular fitness as subjects participating in traditional cardiovascular training[2]. With this is mind, one may argue that traditional cardiovascular training for field and court sport athletes is worthwhile since good cardiovascular conditioning in these sports is important. However, it has also been established that high-intensity intermittent training and moderate-intensity continuous training, despite having similar cardiovascular training effects, have different training effects on anaerobic capacity[3]. This study examined the effect of six weeks of moderate-intensity endurance training (70% $VO_2$ max, 60 minutes per day, 5 days per week) compared to six weeks of high-intensity intermittent training (170% $VO_2$ max, 7-8 sets of 20 seconds with 10 seconds recovery between bouts). Both training methods significantly increased the maximal oxygen uptake. However, while the endurance training had no impact on the anaerobic capacity, the high-intensity intermittent training increased the anaerobic capacity by 28%. It was, therefore, concluded that the high-intensity intermittent training imposed intensive stimuli on both energy systems. This finding has obvious implications to the field and court sport athlete, whom require both a high anaerobic capacity and the endurance to reproduce multiple repetitions of high energy output. This study was supported by a subsequent study that examined the anaerobic capacity of untrained, endurance-trained, and sprint-trained young men[4]. It was exhibited that there was no difference in anaerobic capacity between the untrained and endurance-trained subjects, whereas the anaerobic capacity of the sprinters was 30% greater. Both of these studies support the notion that significant endurance training can diminish an athlete's anaerobic capacity, while high-intensity intermittent training can simultaneously increase an athlete's anaerobic capacity and improve the athlete's endurance capacity.

Numerous studies examining the kinetics of oxygen uptake during short-term intense exercise reveal that the contribution of oxidative metabolism is early and significant. As early as the late 1980s, studies demonstrated oxidative contributions as high as 40% in intense exercise lasting 30 seconds and 50% lasting 1 minute[5,6]. More recent studies show an even greater contribution of oxidative pathways during high-intensity exercise. It has been demonstrated that a 3-fold increase in muscle oxygen uptake can take place within only 6 seconds of intense activity[7] (peaking at 50 seconds) and that oxidative pathways can contribute as much as 40% within 15 seconds of short-term exhaustive running[8] (peaking as soon as 25 seconds at 79% of $VO_2$ max). Further, using college sprinters, a comparison of the 30- second Wingate anaerobic power test, and a graded $VO_2$ max cycle ergometer test, showed a significant difference in muscle deoxygenation[9]. Using Near Infra-red Spectroscopy, the oxygen concentration of the vastus lateralis muscle was monitored at maximum intensity. The findings were as follows. First, during the Wingate test, deoxygenation reached 80% of the established maximum value; whereas in the $VO_2$ max test, deoxygenation reached only 36%. Second, and quite significantly, there was no delay in onset of deoxygenation in the Wingate test, while deoxygenation did not occur under low intensity work in the $VO_2$ max test.

Studies utilizing muscle biopsy samples also lend considerable support to the contribution of oxidative metabolism during high-intensity exercise. Completion of ten 6-second maximal sprints with 30 seconds of recovery between sprints, demonstrated that, while the mean power output of the tenth sprint was reduced to only 73% of the first, no change in muscle lactate concentration was observed indicating a significant contribution from aerobic metabolism[10]. Participation in lower body strength training has resulted in significant and equal increases in the cross-sectional area of both type I and type II fiber types[11], furthermore both fiber types contributed significantly to the anaerobic energy release at powers up to almost 200% $VO_2$ max[12]. Further, examination of mitochondrial function under exhaustive high-intensity intermittent exercise has shown that mitochondrial oxidative potential is maintained or even improved[13].

Another misconception about exercise and metabolism concerns substrate utilization. Because it has been shown that low-intensity exercise uses a higher percentage of lipid oxidation than high-intensity exercise, it is commonly accepted that low-intensity continuous activity is the most effective form of exercise to "burn" fat. This logic does not, however, account for high-intensity exercise eliciting a higher energy expenditure than low-intensity exercise, during both exercise and over a 24-hour period. This finding is true even when work-output is equalized[14]. It has been demonstrated that low-intensity, long-duration exercise results in a greater total fat oxidation than moderate-intensity exercise of similar caloric expenditure[15]. However, when endurance training is compared to high-intensity intermittent training, the findings differ. The effect of a 20-week endurance-training program (mean estimated energy cost - 120.4 MJ) upon body fatness and muscle metabolism was compared to a 15-week high-intensity intermittent-training program (mean estimated energy cost - 57.9 MJ)[16]. Despite the lower energy cost of the high-intensity program, it induced a more pronounced reduction in subcutaneous fat compared with the endurance program. When corrected for the energy cost of training, the reduction induced by the high-intensity program was nine-fold greater than the endurance program. Muscle biopsies taken before and after the training programs revealed that the high-intensity intermittent-training program increased the enzyme activity of a betaoxidation (fat metabolism) marker. It was concluded that for a given level of energy expenditure, vigorous exercise favors negative energy and lipid balance to a greater extent than exercise of low to moderate intensity. Further, metabolic adaptations in skeletal muscle in response to high-intensity intermittent-training appeared to favor lipid oxidation. These findings have been validated with subsequent studies[17,18]. It has also been found that only during high intensity exercise is triglyceride within the muscle hydrolyzed to release fatty acids for subsequent direct oxidation[19,20].

When examining the effect of training intensities upon energy expenditure, the effect the type of training has upon the post-exercise metabolism is often overlooked. It is well documented that high-intensity exercise, either intermittent or continuous, increases recovery oxygen consumption more than prolonged low-intensity exercise[21,22,23,24,25,26]. In addition, for a single bout of maximal exercise, it has been shown that sixty seconds is optimal to maximize the excess post-exercise oxygen consumption (EPOC)[27]. Compared to one continuous bout of exercise, the magnitude of EPOC is significantly elevated by splitting the equivalent exercise into two sessions[28], supporting the benefits of interval training when the aim is to increase overall energy expenditure. It has also been demonstrated that following high-intensity exercise, but not low-intensity exercise, oxygen consumption remains elevated above resting levels at 3 hours post-exercise; further, at this 3-hour time point, the rate of fat oxidation was higher after high-intensity exercise as compared to low-intensity exercise[29].

Based on these findings, it perhaps should not have been surprising that prescribing exercise in several short bouts versus one long-bout per day produced similar changes in cardiorespiratory fitness and had a trend towards greater weight loss[30]. This study did appear to have somewhat of an effect upon the thinking towards continuous exercise by altering the exercise recommendations of the fitness industry. In fact, even prior to this study, a group of experts was brought together in 1995 by the Centers for Disease Control and Prevention (CDC) and the ACSM to review the pertinent scientific research and to develop a clear, concise "public health message" regarding physical activity[31]. The panel concluded that every US adult should accumulate 30 minutes or more of moderate-intensity physical activity on most, preferably all, days of the week. The acknowledgment that the activity did not need to be continuous was a major shift from the initial recommendations of the ACSM. It was even stated, "accumulation of physical activity in intermittent, short bouts is considered an appropriate approach to achieving the activity goal". This concept was validated in another study that demonstrated that three 1-minute bouts of maximal intensity exercise, separated by 1-hour recoveries, constituted 74% of the oxygen uptake of 20 minutes of low- to moderate-intensity exercise[32]. The idea of extending recoveries to allow for maximal performance in subsequent bouts of intermittent training may be useful for training anaerobic capacity. It has been shown that following 30 seconds of maximal isokinetic cycling, 4 minutes of recovery is sufficient to almost completely restore AdenosineTriphosphate (ATP) and mixed-muscle phosphocreatine (PCr) in type I muscle fibers, but not type II muscle fibers[33]. Furthermore, the restoration of ATP and PCr correlated positively with total work production of a subsequent 30-second bout. Consequently, the inclusion of training sessions in ones overall program where recoveries are extended to allow for total restoration of type II muscle fiber ATP and PCr content may improve muscle training for high-intensity performance.

Of paramount importance to nearly all athletes, but particularly to athletes whose sports require a high anaerobic capacity, is the ability to tolerate lactate. Thus, the effect that training methods have on the ability to tolerate lactate has important implications to both athlete and coach. Regulation of skeletal muscle internal pH (pHi) depends on continuous activity of membrane transport systems that mediate an outflux of hydrogen ions (H+, (or bicarbonate influx)), whereby the acid load is counterbalanced. The dominant acid extruding system associated with intense exercise is the lactate/H+ transporter which has been shown to be upregulated with training[34]. The oxidative fibers of skeletal muscle use lactic acid as a respiratory fuel. It has now been shown that skeletal muscle contains proton-linked monocarboxylate transporters (MCTs) that transport lactic acid across the muscle fiber plasma membrane. It has further been established that two isoforms exist in skeletal muscle, MCT1 and MCT4, and that the distribution of these isoforms is fiber dependent. MCT1 is primarily found in type I oxidative fibers, whereas MCT4 is primarily found in type II glycolytic fibers[35]. Studies are now emerging on the effect of training intensity upon these transporters. Three weeks of moderate-intensity training did not increase MCT1 or skeletal muscle lactate uptake, whereas 3 weeks of high-intensity training did increase both MCT1 and lactate uptake[36]. Further research supports this finding as well as demonstrating that intense exercise increases MCT4 as well as MCT1[37]. As a general rule of human physiology, we adapt to stress. Accordingly, it makes sense that, if athletes need to develop lactate tolerance, they should produce high amounts of lactate in their training. The current research supports this notion.

To this point, comparisons of high-intensity training and low- to moderate-intensity training and the effect these different intensities have on parameters of obvious concern to field and court sport athletes and coaches has been addressed. It is worth, however, highlighting a few broader areas of interest that may not usually be discussed. It has been shown that increases in high-density lipoprotein cholesterol levels (the “good cholesterol”) have occurred as a result of intermittent exercise, but not as a result of continuous exercise[38]. Beta-endorphin levels, which are associated with positive changes in mood state, have been shown to increase following incremental graded and short term anaerobic exercise, the extent correlating with the lactate concentration. However, in endurance exercise performed at a steady-state between lactate production and elimination, blood beta-endorphin levels do not increase until exercise duration exceeds approximately 1 hour – with the increase being exponential thereafter[39]. Plasma glutamine, an essential amino acid for the normal functioning of the immune system, was decreased in over-trained athletes and after prolonged exercise (intermittent and continuous) but increased after short-term, high intensity exercise[40,41]. The total antioxidant capacity of marathon runners cannot prevent exercise-induced lipid peroxidation following a half-marathon run, and at rest have demonstrated significantly elevated levels of conjugated dienes (an index of lipid peroxidation) as compared to sprint-trained athletes and controls[42,43]. These latter findings all lend support to some benefits of high-intensity exercise that might not normally be considered.

In conclusion, the research is extensive in its support for the notion that high-intensity intermittent training should be the predominant method employed by the field and court sport athlete. It has been established that this type of exercise can have an equal or even greater training effect on the cardiovascular system than continuous endurance training, while also increasing the anaerobic capacity. This form of training also produces a more favorable body composition, and better improves the ability of the athlete to tolerate lactate. Research also supports the fact that there are a number of additional benefits resulting from participation in high-intensity training that are not evident with long continuous exercise. Standing back from the research, a coach might simply question from which track and field event an athlete would be selected, that would be best suited to participate in a field sport. The answer should provide clues as to the type of training the games player should use.

1. The recommended quality and quantity of exercise for developing and maintaining fitness in healthy adults. Med. Sci. Sports. 1978: 10(3): vii.

2. Hannon JC, Pellett TL. Comparison of heart-rate intensity and duration between sport games and traditional cardiovascular activities. Percept. Mot. Skills. 1998; 87(3 Pt2):1453-4.

3. Tabata I, Nishimura K, Kouzaki M, Hirai Y, Ogita F, Miyachi M, Yamamoto K. Effects of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and VO2max. Med. Sci. Sports Exerc. 1996; 28(10):1327-30.

4. Medbo JI, Burgers S. Effect of training on the anaerobic capacity. Med. Sci. Sports Exerc. 1990; 22(4):501-7.

5. Medbo JI, Tabata I. Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. Appl. Physiol. 1989; 67(5):1881-6.

6. Serresse O, Lortie G, Bouchard C, Boulay MR. Estimation of the contribution of the various energy systems during maximal work of short duration. Int. J. Sports Med. 1988; 9(6):456-60.

7. Bangsbo J, Krustrup P, Gonzalez-Alonso J, Boushel R, Saltin B. Muscle oxygen kinetics at onset of intense dynamic exercise in humans. Am. J. Physiol. Regul. Integr. Comp. Physiol. 2000; 279(3):R899-906.

8. Nummela A, Rusko H. Time course of anaerobic and aerobic energy expenditure during short-term exhaustive running in athletes. Int. J. Sports Med. 1995;16(8):522-7.

9. Nioka S, Moser D, Lech G, Evengelisti M, Verde T, Chance B, Kuno S. Muscle deoxygenation in aerobic and anaerobic exercise. Adv. Exp. Med. Biol. 1998; 454:63-70.

10. Gaitanos GC, Williams C, Boobis LH, Brooks S.J. Human muscle metabolism during intermittent maximal exercise. Appl. Physiol. 1993; 75(2):712-9.

11. Chilibeck PD, Syrotuik DG, Bell GJ. The effect of strength training on estimates of mitochondrial density and distribution throughout muscle fibres. Eur. J. Appl. Physiol. Occup. Physiol. 1999; 80(6):604-9.

12. Vollestad NK, Tabata I, Medbo JI. Glycogen breakdown in different human muscle fibre types during exhaustive exercise of short duration. Acta. Physiol. Scand. 1992;144(2):135-41.

13. Tonkonogi M, Walsh B, Tiivel T, Saks V, Sahlin K. Mitochondrial function in human skeletal muscle is not impaired by high intensity exercise. Pflugers Arch. 1999; 437(4):562-8.

14. Treuth MS, Hunter GR, Williams M. Effects of exercise intensity on 24-h energy expenditure and substrate oxidation. Med. Sci. Sports Exerc. 1996; 28(9):1138-43.

15. Thompson DL, Townsend KM, Boughey R, Patterson K, Bassett DR Jr. Substrate use during and following moderate- and low-intensity exercise: implications for weight control. Eur. J. Appl. Physiol. Occup. Physiol. 1998; 78(1):43-9.

16. Tremblay A, Simoneau JA, Bouchard C. Impact of exercise intensity on body fatness and skeletal muscle metabolism. Metabolism. 1994; 43(7):814-8.

17. Chilibeck PD, Bell GJ, Farrar RP, Martin TP. Higher mitochondrial fatty acid oxidation following intermittent versus continuous endurance exercise training. Can. J. Physiol. Pharmacol. 1998; 76(9):891-4.

18. Yoshioka M, Doucet E, St-Pierre S, Almeras N, Richard D, Labrie A, Despres JP, Bouchard C, Tremblay A. Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness. Int. J. Obes. Relat. Metab. Disord. 2001; 25(3):332-9.

19. Romijn JA, Coyle EF, Sidossis LS, Gastaldelli A, Horowitz JF, Endert E, Wolfe RR. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. Am. J. Physiol. 1993; 265(3 Pt 1): E380-E391.

20. Wolfe RR. Fat metabolism in exercise. Adv. Exp. Med. Biol. 1998; 441:147-156.

21. Brockman, L. et al. Oxygen uptake during recovery from intense intermittent running and prolonged walking. J. Sports Med. Phys. Fitness. 1993; 33(4): 330-336.

22. Bahr R et al. Effect of supramaximal exercise on excess postexercise $O_2$ consumption. Med. Sci. Sports Exerc. 1992; 24(1): 66-71.

23. Bahr R et al. Effect of intensity of exercise on excess postexercise $O_2$ consumption. Metabolism. 1991; 40(8): 836-841.

24. Broeder CE et al. The metabolic consequences of low and moderate intensity exercise with or without feeding in lean and borderline obese males. Int. J. Obesity. 1991; 15: 95-104.

25. Smith J et al. The effects of intensity of exercise on excess postexercise oxygen consumption and energy expenditure in moderately trained men and women. Eur. J. Appl. Physiol. 1993; 67(5):420-425.

26. Laforgia J. et al. Comparison of energy expenditure elevations after submaximal and supramaximal running. J. Appl. Physiol. 1997; 82(2):661-666.

27. Withers RT et al. Oxygen deficits incurred during 45, 60, 75 and 90-s maximal cycling on an air-braked ergometer. Eur. J. Appl. Physiol. 1993; 67(2): 185-91.

28. Almuzaini KS et al. Effects of split exercise sessions in excess postexercise oxygen consumption and resting metabolic rate. Can. J. Appl. Physiol. 1998; 23(5):433-443.

29. Phelain JF, Reinke E, Harris MA, Melby CL. Postexercise energy expenditure and substrate oxidation in young women resulting from exercise bouts of different intensity. J. Am. Coll. Nutr. 1997; 16(2):140-6.

30. Jakicic JM, Wing RR, Butler BA, Robertson RJ. Prescribing exercise in multiple short bouts versus one continuous bout: effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. Int. J. Obes. Relat. Metab. Disord. 1995; 19(12):893-901

31. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA. 1995 Feb 1;273(5):402-7.

32. Darby M, Smith MJ, Melby C[§], Gotshall R. Comparison of the effect of long-bout exercise with repeated short-bout exercise on oxygen consumption. 1998; Masters thesis: Departments of Exercise & Sport Science and Food Science and Human Nutrition[§]. Colorado State University, Fort Collins, CO.

33. Casey A, Constantin-Teodosiu D, Howell S, Hultman E, Greenhaff PL. Metabolic response of type I and II muscle fibers during repeated bouts of maximal exercise in humans. Am. J. Physiol. 1996; 271(1 Pt 1):E38-E43.

34. Juel C. Muscle pH regulation: role of training. Acta. Physiol. Scand. 1998; 162(3):359-366.

35. Juel C, Halestrap AP. Lactate transport in skeletal muscle - role and regulation of the monocarboxylate transporter. J. Physiol. 1999; 517(Pt 3):633-42.

36. Baker SK, McCullagh KJ, Bonen A. Training intensity-dependent and tissue -specific increases in lactate uptake and MCT-1 in heart and muscle. J. Appl. Physiol. 1998; 84(3):987-994.

37. Pilegaard H, Domino K, Noland T, Juel C, Hellsten Y, Halestrap AP, Bangsbo J. Effect of high-intensity exercise training on lactate/H+ transport capacity in human skeletal muscle. Am. J. Physiol. 1999; 276(2 Pt 1):E255-61.

38. Ebisu T. Splitting the distance of endurance running: on cardiovascular endurance and blood lipids. Jpn. J. Phys. Educ. 1985: 30: 37-43.

39. Schwarz L, Kindermann W. Changes in beta-endorphin levels in response to aerobic and anaerobic exercise. Sports Med. 1992; 13(1): 25-36.

40. Parry-Billings M, Budgett R, Koutedakis Y, Blomstrand E, Brooks S, Williams C, Calder PC, Pilling S, Baigrie R, Newsholme EA. Plasma amino acid concentrations in the overtraining syndrome: possible effects on the immune system. Med. Sci. Sports Exerc. 1992: 24(12): 1353-1358.

41. Walsh NP, Blannin AK, Clark AM, Cook L, Robson PJ, Gleeson M. The effects of high-intensity intermittent exercise on the plasma concentrations of glutamine and organic acids. Eur. J. Appl. Physiol. Occup. Physiol. 1998; 77(5):434-8.

42. Child RB, Wilkinson DM, Fallowfield JL, Donnelly AE. Elevated serum antioxidant capacity and plasma malondialdehyde concentration in response to a simulated half-marathon run. Med. Sci. Sports Exerc. 1998; 30(11):1603-1607.

43. Marzatico F, Pansarasa O, Bertorelli L, Somenzini L, Della Valle G. Blood free radical antioxidant enzymes and lipid peroxides following long-distance and lactacidemic performances in highly trained aerobic and sprint athletes. J. Sports Med. Phys. Fitness. 1997; 37(4):235-239.

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