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A Comparison of Blood Lactate Level and Heart Rate Following a Peak Anaerobic Power Test in Different Exercise Loads

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ABSTRACT

The purpose of the present research was to compare blood lactate level and heart rate following three loads of the Wingate test for anaerobic power. The sample of the research consisted of 11 physical education students with the experience of participating in national and international long-distance track events who voluntarily participated in the study (23.00 ± 1.78 years old; 174.36 ± 6.24 cm height; 66.58 ± 5.76 kg weight). The participants underwent three loads of the Wingate test. The exercise loads were performed with 2 days interval. Before, during, and after each test the blood lactate level and heart rate of the participants were measured. In addition, anaerobic power, anaerobic capacity, and fatigue index of the participants were calculated after each test. The data was analyzed in SPSS 16. The mean and standard deviation of all the variables were calculated. One-way ANOVA was applied to examine significant differences between the variables and Fisher's Least Significant Difference (LSD) test was applied to find the reason for possible differences. A significant difference was observed in cardiac output of the participants at different loads of the Wingate test. However, there was no significant difference in blood lactate acid at different exercise protocols. Moreover, no significant difference was observed in anaerobic power, anaerobic capacity, and fatigue index at different exercise loads. The maximum anaerobic power and capacity was observed in age group 2 (85 kg) (anaerobic power: 79 ± 99.84 W, -9.57 ± 0.88 W/Kg; anaerobic capacity: 512.76 ± 69. 05 W, 7.67 ± 0.61 W/Kg). In addition, it was revealed that heart rate increases with exercise load in the Wingate test.

Keywords: Wingate test, blood lactate, heart rate, anaerobic power, anaerobic capacity

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INTRODUCTON

Anaerobic power and anaerobic capacity are essential elements in many sports. Anaerobic performance is one of the main characteristics of brief and explosive exercise in different sports. Anaerobic power is the ability to generate energy through the phosphagen system in brief and intensive exercises and anaerobic capacity is the ability to combine the energies from glycolysis and the phosphagen system [1, 2, 3]. There are many tests for measuring anaerobic performance, but the Wingate test is the most widely used and the most efficient of these tests [4]. This test involves 30 seconds of quick pedaling on a bicycle ergometer. The amount of workload in the Wingate test is 0.075 kgf per kg bodyweight of the subject. This value has been determined based on experiments on a group of inexperienced youths [4, 5]. In many other studies, different workloads have been employed with different results [6, 7, 8].

High intensity exercise can be performed continuously only for a short period of time and energy demand fluctuate from a high to low level between the work and rest periods. Work of high intensity that it can be performed continuously only for a short period of time is accompanied by a high rate of glycogen depletion, lactate accumulation and a greater contribution of carbohydrate to oxidative metabolism.

The lactate formed in fast twitch muscle fibers can diffuse out of the muscle and enter the blood or it can shuttle directly to adjacent slow twitch muscle fibers where the lactate can be consumed as a fuel. Blood lactate levels can be used to guide training intensity because effective training occurs when an individual trains at an exercise intensity that corresponds to the lactate threshold i.e. the exercise intensity at which lactate begins to build up in the blood [9]. Heart rate recovery (HRR) is the rate at which the heart rate (HR) decreases (i.e., the time taken for HR to recover) following a moderate to heavy exercise in response to a combination of parasympathetic activation and sympathetic withdrawal [10, 11, 12, 13]. HRR is known to change in response to acute and seasonal changes in training load [14, 15, 16]. However, there are no clear data indicating whether HRR is a sensitive measure of autonomic control and, in particular, whether HRR can be used as an index representing the body's capacity to respond to training. Aerobic fitness is another variable that could influence HRR response. The recovery period after exercise is an important performance factor in the repetitive exercises [17].

Short and Sedlock showed that throughout the recovery period the group of trained athletes with superior aerobic capacity had a consistently lower HR as compared to untrained subjects, but there is still no clear explanation of this phenomenon. HRR after maximal exercise has been neither studied in elite athletes characterized by superior aerobic fitness nor compared it with athletes characterized by lower aerobic capacity [18]. The evaluation of the post-exercise HRR can be quantified by different methods, including the absolute difference between the final HR at exercise completion and HRR recorded following 60 s of recovery (HRR60s), first 30 s of HRR via semi-logarithmic regression analysis (T30), or the time constant of the HR decay obtained by fitting the post-exercise HRR by a first-order exponential decay curve [19, 20, 21].

Aerobic interval training is an important training tool for developing endurance performance because athletes can perform more high intensity work, i.e. a greater training stimuli, than what can be tolerated with continuous, constant-intensity exercise. Aerobic interval training consists of five variables: work interval intensity and duration, recovery interval intensity and duration, and total work duration [22]. Whilst other variables during interval training session are easily construed, determining the recovery intensity and duration is much more difficult in practice [23].

The practice is to delay performing the next interval run until the athlete's HR during the recovery period in-between bouts has lowered to between 100 and 140 beats per minute (beats min–1) (Peak Running Performance Research Fox 1979), [24]. Proponents of this practice deemed that lowering the athlete's HR to the aforementioned range would indicate that the body has reached an optimal state of readiness for the next work bout. However, to our knowledge, there is no research evidence to support this widely used practice.

A major presumption of using HRrec as a recovery tool is that an aerobically fitter individual would have a faster HRrec [25]. Indeed, several previous studies have shown a relationship between aerobic fitness and HRrec. For example, (Sugawara et al., 2001) found that HRrec from submaximal exercise was moderately correlated with V·O2max in college-aged students ($r=-0.75$ and 0.58 respectively, $p<0.05$). Similarly, HRrec post submaximal exercise was also found to be significantly faster in trained compared to sedentary individuals (Du et al. 2005). Importantly, not only does HRrec appear to be sensitive in cross-sectional studies, but HRrec has also been found to be accelerated after short-term or long-term endurance training and too subsequently decline in parallel with detraining collectively, these findings suggest that aerobic fitness is influential in the dynamics of HRrec [26, 27].

The Wingate test and lactic acid (LA) concentration measurement after maximal effort are frequently used for anaerobic capacity evaluation. The anaerobic capacity determinants in the Wingate test are maximal power, mean power and fatigue index. Some discordant data considering changes of power produced in the Wingate test consisting of short but maximal intensity efforts on the cycloergometer are presented in the references. Allemeier et al., (1994); Rodas et al., (2000), reported that sprint training on the cycloergometer did not cause changes of power measured in the Wingate test [28, 29]. In the work of Parry et al., (2000), no post-training changes of the peak power or average power measured in the Wingate test were observed in a group of subjects practicing every day for 2 weeks. These values increased after 30 s activity by 20 and 14% respectively in the second group, which had a 2-day break between training sessions [30].

Recovery encompasses active process of re-establishing physiological and psychological resources and states that allow the individual to use these resources again [31]. A return to pre exercise levels of blood lactate usually occurs within an hour and light activity during the post-exercise period has been shown to accelerate this clearance [32].

The recovery strategies are designed to maintain a high rate of blood flow to the working muscles, to expedite lactic acid translocation from the muscle cell to the blood, to accelerate the resynthesis of high energy phosphate, and to replenish oxygen in the blood, bodily fluids and muscle myoglobin. The recovery should be sufficiently long enough to allow the next repetition to be at the same or above the level as the previous effort, but a longer recovery should

be avoided if optimum training benefit is to result.As a rule of thumb, the heart rate should drop to approximately 120-bpm near to the end of the recovery interval [33].

Some significant or insignificant post-training changes of maximal power were noted depending on the exercises performed. Nonetheless, in the works of Linossier et al. and Stathis et al. brief efforts with high intensity performed on the cycloergometer elicited the maximal power and increase of performed work.

It is believed that the post-exercise concentration of lactic acid (LA) is an indicator of adaptation to very intense training [34, 35].

According to Lutosławska et al. the relation between lactic acid and anaerobic work may change contingently upon applied training; however, there are only a few works relating to this subject in the literature [36]. Significant correlations of lactic acid concentration and anaerobic test results were reported in the papers of, Granier et al., (1995); Lutosławska et al., (1998); Gratas-Delamarche et al., (1994), found a significant correlation between the LA concentration and work performed in the Wingate test in female sprinters but did not observe such a correlation in male athletes [36, 37, 38]. No relation between LA concentration and power in 10 and 30 s test conducted on men and women was observed by Jacobs et al. [39].

The ability to increase cardiac output by augmenting stroke volume early in exercise may be training-specific because researchers have not been able to observe this phenomenon in untrained subjects [40].

The anaerobic threshold has been studied thoroughly during the past decades, and it has been described using a variety of definitions. Since the introduction of the term anaerobic threshold by Wasserman et al. (1978), AT has been associated with an increased blood lactate concentration, increased ventilation, increased CO2 excretion and a decreased bicarbonate concentration. Researchers have suggested that these increases reflect a shift from aerobic to anaerobic metabolism [41].

Anaerobic threshold is commonly defined as the highest metabolic rate at which blood lactate concentration is maintained at a steady state during prolonged exercise. This definition developed by Wasserman et al. (1978) is based on the assumption that exercise above a specific work rate stimulates recruitment of anaerobic, lactic acid producing energy metabolism; therefore, AT has been accepted as a tool for predicting endurance performance and for the development of training strategies for athletes [41].

When an individual exercises with progressive intensity, that individual experiences marked increases in blood lactate concentration (Green et al., 1983; Stegman et al., 1980) and ventilatory measures (Caiozzo et al., 1982; Wasserman et al., 1967). Using blood lactate concentration and ventilatory threshold, researchers can examine the various parameters within the blood concentration and respiration in order to determine the critical exercise intensity at which an individual can exercise maximally for extended periods of time [42, 43, 44, 45].

MATERIALS AND METHODS

Population and sample

The purpose of this research was to compare the lactic acid level and cardiac output of endurance runners in three exercise protocols using the Wingate test for Anaerobic Power. The sample of the research consisted of 11 runners who studied at the Faulty of Physical Education and Sport Science of Firat University. The subjects had the experience of participating in national and international tournaments and they all volunteered to take part in the study. The subjects were informed about the procedure and possible risks of the research and completed the consent from.

Procedure

The subjects underwent three exercise loads in Wingate test for anaerobic power. The exercise load was 75 g, 85 g, and 95 g for each kilograms of body weight. Each load was administered at a certain hour with two-day intervals. The subjects were instructed to have passive rest with proper nutrition from 3-4 hours before the test and to abstain from alcohol and cigarettes. Measuring heart rate and blood pressure and blood sampling were done before warm-up, 5 minutes after passive rest, at the end of the test, and 5 minutes after the test.

Measurements and tests

The height of the subjects was measured in the standing position by a stadiometer with a measurement error of ±11 mm. The weight of the subjects was measured by Tanita Digital Scale with a measurement error of 0.5 kg. Body fat and BMI were measured using Tanita Body Composition Analysis. The heart rate of the subjects was measured

using a digital heart monitor (Polar S720i Heart Rate Monitor, Finland). Systolic and diastolic blood pressure was measured by Omron M2 Compact Blood Pressure Monitor (HEM-7102-E). Blood lactate level was measured through finger-stick (capillary) blood sampling. The samples were immediately analyzed by Nova Biomedical Blood Analyzer [9].

Measuring anaerobic capacity and power

Anaerobic capacity and power were measured by an ergometer (Monark 894 E Peak Bike) according to Wingate test protocol. Wingate test involves 40 seconds of pedaling on a bicycle ergometer with maximum speed against an external force. After explaining the test procedure to the subjects, they performed a warm-up for 4-5 minutes that included 2 or 3 pedaling cycles with 60-70 percent intensity and with 60-70 cycles per minute. A 3-5 minute rest followed the warm-up. The test started after the required load for each participant was determined and transferred from the computer to the bicycle ergometer. The participants were asked to pedal as fast as they can in the shortest time possible without any load. When pedaling reached the maximum speed possible, the exercise intensity was manually decreased and the test began again. The participants continued to cycle maximally for 30 seconds against an external load. During the test, the power parameters were given to the computer by RS32 application. All the measurements were done by the computer. Some of the measures obtained from this test were mean and peak power (expressed in Watts), relative peak power (determined by dividing peak power by body mass, expressed as W/kg), mean peak power, minimum peak power, and a fatigue index determined from the decline in power [B, C].

Fatigue Index = [(Peak Power Output - Min Power Output) / Peak Power Output] x 100

Statistical analysis

The arithmetic mean and standard deviation of all the variables were calculated. One-way analysis of variance was used to examine any significant differences between the data and Fisher's Least Significant Difference (LSD) test was applied to find the differences between various exercise intensities. All the operations were done in SPSS 16.

RESULTS

Table 1: The characteristics of the subjects (N = 11)

Variable (N:11)	Mean ± Sd
Age(year)	23.00±1.78
Sport Age (year)	8.45±1.96
Height (cm)	174.36±6.24
Weight (kg)	66.58±5.76
Fat %	6.87±2.42
BMI	21.92±1.99

Table 2: Variables obtained from different exercise loads in Wingate test

Variable (n:11)	Load-1 (%75)	Load-2 (%85)	Load-3 (%95)	F	P
Peak power (w)	610.56±114.93	640.79 ±99.84	619.61 ±87.17	0.258	0.774
Peak power (w/kg)	9.20 ±1.24	9.57 ±0.88	9.48 ±0.85	0.411	0.666
Average Power (w)	474.23 ±71.19	512.76 ±69.05	495.06 ±55.21	0.952	0.397
Average Power (w/kg)	7.15 ±0.67	7.67 ±0.61	7.56 ±0.54	2.201	0.128
Min.Power (w)	321.95 ±93.52	339.71 ±57.82	300.77 ±75.93	0.702	0.503
Min.Power (w/kg)	4.78 ±1.24	5.09 ±0.72	4.64 ±1.23	0.491	0.617
Fatique Index	47.21 ±9.60	46.62 ±7.42	50.75 ±13.26	0.509	0.606

P>0.05

There aren't any significant differences between peak power, average power and minimal power in load-1T load -2 and load -3 conditions.

There is significant difference in finish heart rate in there conditions of loads1, 2 and 3. but this differences weren't significant in an other variables (including lactate T systolic blood pressure and diastolic blood pressure) in there periods of rest , finish and recovery.

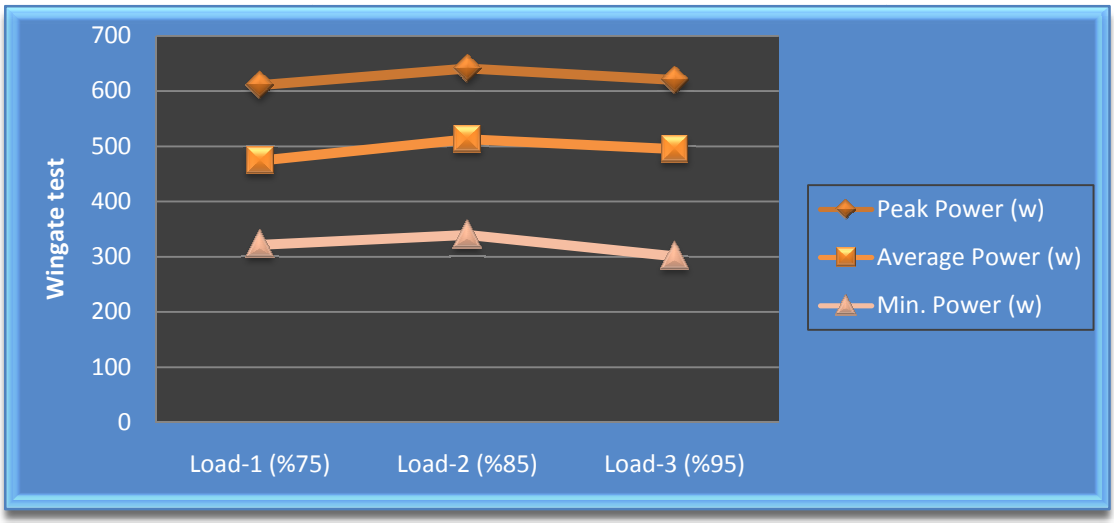

Figure 1: Anaerobic power variables obtained from different exercise loads in Wingate test

Table 3: Hear rate, blood lactate level, and systolic and diastolic blood pressure measured before, during, and after Wingate test with different loads

Variable (n:11)		Load-1 (%75)	Load-2 (%85)	Load-3 (%95)	F	P	LSD
Heart Rate	**Rest**	56.72±6.10	58.27±6.82	62.45±8.18	0.716	0.497	**L1 - L3***
	Finish	164.90±7.07	170.36±6.60	172.54±7.62	3.363	0.048*	
	Recovery	93.90 ±16.66	96.81 ±16.04	90.90 ±17.96	0.336	0.717	
Lactate Mmo5l	**Rest**	1.72±0.28	1.47±0,27	1.60±0.27	2.275	0.120	
	Finish	14.06±3.08	13.44±3.55	13.00±2.35	0.340	0.714	
	Recovery	13.56 ±2.82	13.81 ±2.11	13.09 ±2.30	0.253	0.778	
Systolic Blood Pressure (mmHg)	**Rest**	127.90±13.71	128.90±11.33	126.00±6.48	0.201	0.819	
	Finish	145.72±8.36	152.54±20.18	145.18±12.04	0.893	0.420	
	Recovery	128.18±21.04	135.45±15.17	130.27±13.89	0.534	0.592	
Diastolic Blood Pressure (mmHg)	**Rest**	67.18±12.01	62.63±12.27	69.27±8.92	1.014	0.375	
	Finish	69.27±6.87	71.81±10.04	68.09±9.06	0.469	0.630	
	Recovery	63.18±9.65	67.27±11.68	64.63±8.22	0.477	0.625	

**P<0. 05*

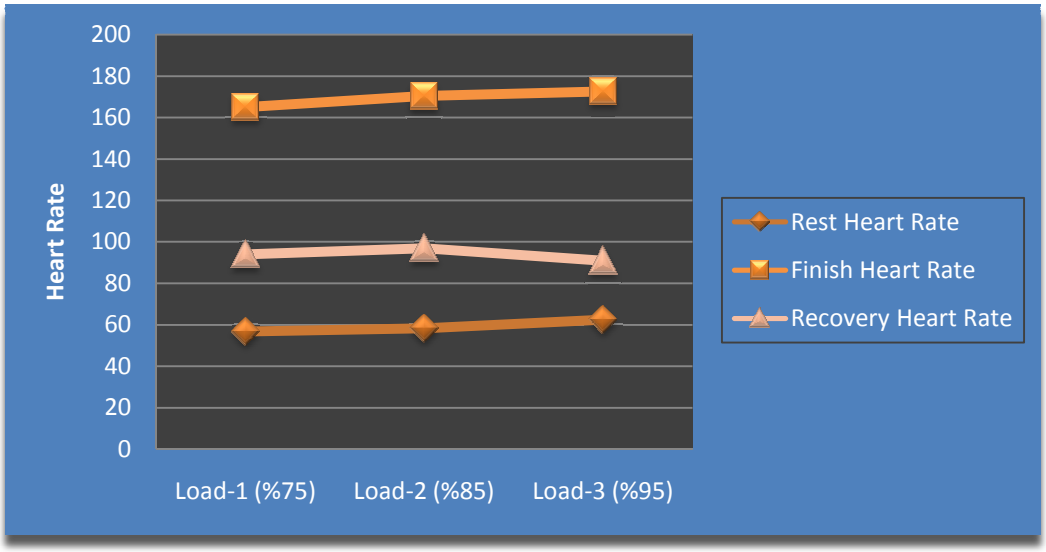

Figure 2: Heart rate measured before and after Wingate test and during recovery

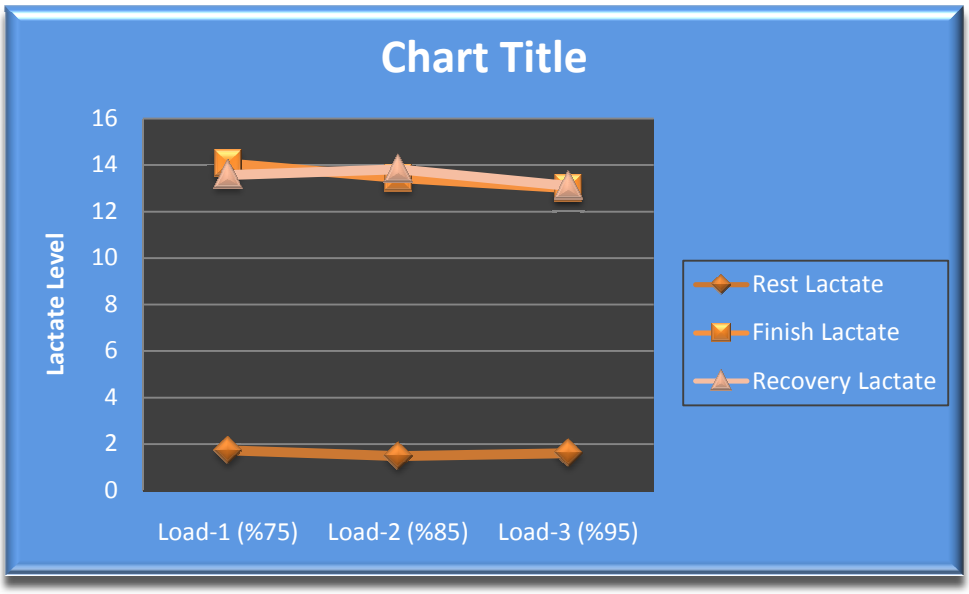

Figure 3: Lactate acid level obtained before and after Wingate test and during recovery

DISCUSSION AND CONCLUSION

The purpose of this research was to compare the lactic acid level and cardiac output of endurance runners in three exercise protocols using the Wingate test for Anaerobic Power. The sample of the research consisted of 11 runners who studied at the Faulty of Physical Education. The subjects had the experience of participating in national and international tournaments and they all volunteered to take part in the study. The subjects were informed about the procedure and possible risks of the research and completed the consent from. The results this study show, (Peak power (w) p : 0.774 and Peak power (w/kg) p: 0.666) also (Average Power (w) p: 0.397 and Average Power (w/kg) p: 0,128) and results, (Min.Power (w) p: 0.503; Min.Power (w/kg), p: 0.617 and Fatique Index, p: 0.606). There aren't any significant differences between peak power, average power and minimal power in load-1T load -2 and load -3 conditions, which is consistent with the results Sands, W. A.etal., (2004); Souissi, Net al., (2004) Üçok, K., et al., (2005). And other researches, Allemeier et al., (1994); Rodas et al., (2000), reported that sprint training on the cycloergometer did not cause changes of power measured in the Wingate test. In the work of Parry et al., (2000), no post-training changes of the peak power or average power measured in the Wingate test were observed in a group of subjects practicing every day for 2 weeks. These values increased after 30 s activity by 20 and 14% respectively in the second group, which had a 2-day break between training sessions [30].

Also another theory among; Hear rate, blood lactate level, and systolic and diastolic blood pressure measured before, during, and after Wingate test with different loads,showed: There is significant difference in finish heart rate in there conditions of loads1, 2 and 3. But this differences weren't significant in an other variables (including lactate T systolic blood pressure and diastolic blood pressure) in there periods of rest , finish and recovery.

Other research indicates that researchers, The evaluation of the post-exercise HRR can be quantified by different methods, including the absolute difference between the final HR at exercise completion and HRR recorded following 60 s of recovery (HRR60s), first 30 s of HRR via semi-logarithmic regression analysis (T30), or the time constant of the HR decay obtained by fitting the post-exercise HRR by a first-order exponential decay curve (Buchheit et al., 2006; Imai et al., 1994; Perini et al.,)

And other researches, Lutosławska et al. the relation between lactic acid and anaerobic work may change contingently upon applied training; however, there are only a few works relating to this subject in the literature [36]. Significant correlations of lactic acid concentration and anaerobic test results were reported in the papers of, Granier et al., (1995); Lutosławska et al., (1998); Gratas-Delamarche et al., (1994), found a significant correlation between the LA concentration and work performed in the Wingate test in female sprinters but did not observe such a correlation in male athletes. No relation between LA concentration and power in 10 and 30 s test conducted on men and women was observed by Jacobs et al. [39].

During exercise, particularly short-term high intensity exercise, muscles produce lactate rapidly, whereas lactate clearance is slowed. Later during recovery from short- term exercise, there is net lactate uptake from the blood by resting muscles or other muscles that are doing mild to moderate exercise [46]. The ability to maintain a high power

output during high-intensity intermittent exercise was impaired when oxygen availability was reduced by acute hypoxia, which was associated with a higher accumulation of blood lactate [47]. Therefore, lactic acid removal after exercise had been considered critical for the resumption of exercise, especially during athletic competition involving repetitive high-intensity activities. Since it has been suggested that muscles engaged in heavy exercise will negatively affect the performance in other non-exercised muscles, the use of non-maximally exercised muscles during active recovery of low-intensity i.e. below the lactate threshold theoretically could be beneficial on the subsequent repeated performances of the maximally exercised muscle groups [48].

To our knowledge, this is the first study that has directly revealed the influence of aerobic capacity on the ultra short-term post-exercise HR in athletes. The results obtained in this study generally suggest that no compressions between HRR in athletes with similar training status.

The rapid recovery of HR following a moderate to- heavy exercise may be an important mechanism in preventing excessive cardiac work, which could also have important implications for athletic training. Several investigators have shown that the endurance trained athletes have faster HR responses after the cessation of exercise as compared with sedentary subjects [49]. Of importance here could be that the parasympathetic activation is considered to be the main mechanism underlying exponential cardiodeceleration following the exercise.

It does, however, remain possible that HRrec varies amongst athletes who participate in contrasting sports, for example, between predominantly endurance sports (e.g. triathlon, rowing) and speed/power-oriented sports or between athletes of contrastingly different training status, for example, professional (well trained) vs. recreational (moderately trained) soccer players (Edwards et al. 2003). These studies appear to suggest that beyond a certain threshold level of aerobic fitness, which is presently undetermined, the differences in HRrec could be observed. That no relationship was observed between HRrec and V·O2max in this study could be due solely to the fact that the sample in the current study was homogeneous [50].

Recovery depends on the magnitude of fatigue from the preceding exercise and, consequently, it might be argued that the HIGH V · O2max group performed relatively more work than the LOW V · O2max group since the former, on average, ran for much longer on the treadmill than the latter. This could have increased the ardiovascular demand and production of metabolic byproducts (e.g. heat, stress hormones) in the HIGH V · O2max group, which might have influenced HRrec (i.e. it remained elevated for longer post exercise) and compounded the relationship between V·O2max and HRrec.

For endurance sport athletes, during the intervening recovery periods of an aerobic interval training session, coaches have traditionally used the HRrec method of allowing HR to return to between 100 and 140 beats min–1 before commencing the subsequent intervals (Peak Running Performance Research, Snell 1990; Fox 1979). In the case of team sport athletes, it is common practice among coaches to group the aerobically fitter athletes together because it is perceived that these athletes possess the enhanced ability to recover faster after each interval run [51].

REFERENCES

[1] A. Robert, J. Steven. Exercise Physiology., **2003,** S.123, New York.
[2] A. Özkan, Y. Köklü, G.Ersöz. Wingate anaerobik güç testi; Uluslararası İnsan Bilimleri Dergisi.Cilt:7 Sayı:1, **2010.**
[3] R.F. Reiser, J. M. Maines, J.C. Eisenman, J.G. Wilkinson. European Journal of Applied Physiology., **2002,** 88, 152-157.
[4] O. Inbar, O. Bar-Or, JS. Skinner. The Wingate Anaerobic Test; Champaign, IL: Human Kinetics Books; **1996.**
[5] O. Bar-Or. Sports Medicine., **1987,** 4, 381-394.
[6] W.A. Sands, J.R. McNeal, M.T. Ochi, M.J. Urbanek, M. Jemni, M.H. Stone. Journal of Strength and Conditioning Research; **2004,** 18: 810-815.
[7] N. Souissi, A.Gauthier, B. Sesboüé, J. Larue, D. Davenne. International Journal of Sports Medicine; **2004,** 25:14-19.
[8] K. Üçok, H. Gökbel, N. Okudan. European Journal General Medicine., **2005,** 2(1), 10-13.
[9] S. Kulandaivelan, S.K. Verma, S. Mukhopadhyay, N. Vignesh. J of Exe Sci and Physiotherapy., **2009,** Vol. 5, No. 1: 30-33.
[10] J. Borresen, M.I. Lambert. Sports Med., **2008,** 38: 633-646.
[11] P.J. Kannankeril, J.J. Goldberger. Am. J.Physiol. Heart Circ. Physiol., **2002,** 282: H2091-H2098.
[12] P.J. Kannankeril, F.K. Le, A.H. Kadish, J.J. Goldberger. J. Investig., **2004,** Med. 52: 394-401.
[13] G.L. Pierpont, D.R. Stolpman, C.C. Gornick, .J. Auton. Nerv., **2000,** Syst. 80: 169-174.
[14] J. Borresen, M.I. Lambert. Eur. J. Appl. Physiol., **2007,** 101: 503-511.

[15] R. Perini, A. Tironi, M. Cautero, A. Di Nino, E. Tam, C. Capelli. Eur. J. Appl. Physiol., **2006,** 97: 395-403.
[16] K.Yamamoto, M. Miyachi, T. Saitoh, A. Yoshioka, S. Onodera. Med. Sci. Sports Exerc., **2001,** 33: 1496-1502.
[17] A. Taheri, A. Habibi, M. Ghanbarzadeh, P. Ramezani. International journal of sport studies**., 2012,** Vol., 2 (4), 180-185.
[18] K.R. Short, D.A. Sedlock. J. Appl. Physiol., **1997,** 83: 153-159.
[19] M. Buchheit, C. Gindre. Am. J. Physiol. Heart Circ. Physiol., **2006,** 291: H451-H458,.
[20] K. Imai, H. Sato, M. Hori, H. Kusuoka, H. Ozaki, H.Yokoyama, H. Takeda, M. Inoue, T. Kamada. J. Am. Coll. Cardiol., **1994,** 24: 1529-1535.
[21] R.Perini, C. Orizio, A. Comande, M. Castellano, M. Beschi, A. Veiscteinas. Eur. J. Appl.Physiol. Occup, Physiol., **1989.** 58: 879-883.
[22] S. Seiler, KJ. Hetlelid. Med Sci Sports Exerc., **2005**, 37:1601-7.
[23] T. Karu, A. Nurmekivi, E. Lemberg, E. Pihl, V. Jürimäe. Scand J Med Sci Sports., **2000,** 10:33-6.
[24] E.L. Fox. Interval training. Bull Hosp Joint Disp., **1979,** 40:64–71.
[25] R.P. Lamberts, KA.PM. Lemmink, J.J. Durandt, M.I. Lambert. J Strength Cond Res., **2004**, 18:641–5.
[26] N. Du, S. Bai, K. Oguri, Y. Kato, I. Matsumoto, H. Kawase, T. Matsuoka. J Sports Sci Med., **2005,** 4:9–17.
[27] J. Sugawara, H. Murakami, S. Maeda, S. Kuno, M. Matsuda. Eur J Appl Physiol., **2001,** 85:259–63.
[28] C.A. Allemeier, A.C. Fry, P. Johnson, R.S. Hikida, F.C. Hagerman, R.S. Staron. J. Appl. Physiol., **1994,** 77:2385-2390.
[29] J. Parra, J.A. Cadefau, G. Rodas, N. Amigó, R. Cussó. Acta Physiol. Scand., **2000,** 169:157- 165.
[30] G. Rodas, J.L. Ventura, J.A. Cadefau, R. Cussó, J. Parra. Eur. J. Appl. Physiol., **2000,** 82:480- 486.
[31] M. Kellmann, K.W. Kallus. Kluwer Academic/Plenum Publishers., **1999,** pp:101-117.
[32] P.D. Gollnick, W.M. Bayly, D.R. Hodgson. Med Sci Sports Exerc., **1986,** J;18(3):334-340.
[33] S.K. Powers, E.T. Howley. 2nd ed. Brown, Benchmark Publishers; Madison, Wis., **1994,** pp.171-258.
[34] M.T. Linossier, C. Denis, D. Dormois, A. Geyssant, J.R. Lacour. Eur. J. Appl. Physiol., **1993,** 67:408-414.
[35] C.G.A. Stathis, M.A. Febraio, M.F. Carey, R.J. Snow. J. Appl. Physiol., **1994,** 76:1802-1809.
[36] G. Lutosławska, E. Hübner-Woźniak, D. Sitkowski, L. Borkowski. Biol Sport., **1998,** 15:67-74.
[37] P. Granier, B. Mercier, J. Mercier, F. Anselme, C. Préfaut. Eur. J. Appl. Physiol., **1995,** 70:58-65.
[37] A. Gratas-Delamarche, R. Le Cam, P. Delamarche, M. Monnier, H. Koubi. Eur. J. Appl. Physiol., **1994,** 68:362-366.
[39] I. Jacobs, P.A. Tesch, O. Bar-Or, J. Karlsson, R. Dotan. J. Appl. Physiol., **1983,** 55:365-367.
[40] J.Schairer, D. Briggs, T. Kono. Cardiology., **1991,** 79, 284-289.
[41] K. Wasserman. New England Journal of Medicine., **1978,** 298, 780-785.
[42] H. Green, R. Hughson, G.Orr, D. Ranney. Journal of Applied Physiology, **1983,** 54,1032-1038.
[43] H. Stegmann, W. Kinderman, A. Schrakel. International Journal of Sports Medicine., **1980,** 2, 160-165.
[44] V. Caiozzo, J. Davis, J. Ellis, J. Azus, R. Vandagriff, C. Prietto, W. McMaster. Journal of Applied Physiology., **1982,** 53, 1184-1189.
[45] K.Wasserman, A.Van Kessel, G. Burton. Journal of Applied Physiology., **1967,** 22, 71-85.
[46] LB. Gladden, Med Sci Sport Exerc., **2000,** 32(4):764-771.
[47] P.D. Balosm, J.Y. Seger, B. Sjodin, B. Int J Sports Med., **1992,** 13(7):528-533.
[48] J. Karlsson, F. Bonde-Petersen, J. Henriksson, HG. Knuttgen. J Appl Physiol., **1975,** 38(5):763-767.
[49] K.C. Darr, D.R. Bassett, B.J. Morgan, D.P. Thomas. Am. J. Physiol., **1988,** 254: H340-H343.
[50] A.M. Edwards, A.M. MacFadyen, N. Clark. J Sports Med Phys Fitness., **2003,** 43:14–20.
[51] P. Snell. Middle distance running. Physiology of Sports. New York: E & FN Spon., **1990,** p 112.