Relationship of VO2 Peak, Body Fat Percentage, and Power Output Measured During Repeated Bouts of a Wingate Protocol

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ABSTRACT

Int J Exerc Sci 1(2) : 79-90, 2008. The principle of specificity would indicate that being aerobically trained would not necessarily enhance performance in events relying principally on oxygen-independent metabolic pathways (i.e. "anaerobic" exercise). Body fatness may be associated with aerobic and anaerobic performance. VO2 Peak was determined with a graded cycle ergometry and, in a separate session 4 consecutive Wingate power tests (3 min recovery) in 31 males. Pearson correlations were calculated for VO2 Peak and Body Fat Percentage with Peak Power, Mean Power, Minimum Power, Fatigue Index, Peak Heart Rate, and Recovery Heart Rate. No significant correlations were found for VO2 Peak or Body Fat Percentage with Peak Power on any bout ($p>0.05$). Significant correlations were found for VO2 Peak and Body Fat Percentage with Mean Power, Minimum Power, and Fatigue Index. Significant correlations were found for VO2 Peak with delta values of power performance and heart rates (peak and 3 min recovery). Results indicate that VO2 Peak is associated with repeated anaerobic performance, possibly due to greater capacity to recover between bouts. Body Fat Percentage was correlated with measures of power performance (strongest relationships existing in the earlier bouts), but is not strongly correlated with either the heart rate response to power performance or the change in performance over successive bouts.

KEY WORDS: Anaerobic power, power ergometry, exercise recovery

INTRODUCTION

The principle of specificity indicates that aerobic training would not necessarily enhance performance in events relying principally on oxygen-independent metabolic pathways (i.e. "anaerobic" exercise). Duffield et al. (8) estimated the aerobic energy system contribution during a 400-meter sprint event (~60 seconds) to be approximately 41% in males. Beneke et al. (4) evaluated the aerobic contribution to the Wingate anaerobic test and estimated the fraction to be approximately 18.6%, indicating that there is a significant contribution of the oxidative systems even in an event of this short duration (30 sec). Evidence has been provided by Granier et al. (14) that the aerobic energy contribution to the Wingate test varies depending upon the training state of the athlete (sprint vs middle-distance runners). In addition, it has been shown that aerobic metabolism can provide a significant part (~49%) of the energy utilized on a second bout of cycle ergometer sprint exercise (6). Characteristic of repeated bouts is that recovery from exercise is mediated aerobically (21). Therefore, even if aerobic fitness does not significantly enhance a single bout of exercise dominated by anaerobic energy production, it is plausible that greater oxidative capacity would benefit sprint performance if there were repeated bouts because of the aerobic nature of recovery.

Furthermore, the relative ATP contribution from oxygen-dependent metabolic pathways increases progressively in sequential high-intensity work bouts (2, 12, 26). This further emphasizes the potential importance of aerobic fitness in repeated sprint work.

In addition, elevated H+ concentration due to a high rate of lactate formation contributes to fatigue in various ways (10, 11, 25, 30, 31): reduced force per cross-bridge, inhibited sarcoplasmic reticulum Ca++ release, and reduced force generation at a given Ca++ concentration. Because lactate contributes to fatigue in skeletal muscle independent of associated reductions in pH (16), the ability to accelerate removal of lactate should augment performance in successive bouts of anaerobic exercise (1, 21). Lactate removal is an oxygen-dependent process and it is plausible that endurance-trained individuals may have a greater ability to remove lactate following intense exercise (3). Therefore, lactic acid clearance is another possible means by which oxidative capability may enhance performance of repeated sprint bouts. In other words, if a more "aerobically" trained individual is capable of faster lactate clearance, that person may perform better on subsequent bouts than a person that is less "aerobically" fit.

There are several anthropometric measures associated with athletic performance. Body fatness is often utilized as an indicator of disease risk (7), but also tends to be associated with both aerobic (32, 33) and anaerobic performance (34). Therefore, body fatness may be associated with both aerobic capacity and repeated anaerobic performance.

The Wingate protocol has been the subject of many studies, as well as a widely accepted paradigm for the study of variables related to anaerobic performance (13, 17-20, 22, 24, 27-28). Related to the current investigation, Riechman et al. (29) found that peak power on a modified 30-second Wingate test accounted for 75.7% of the variation in 2000-meter rowing performance, while 12.1% of the variance was accounted for by maximal oxygen uptake. In addition, Hoffman, et al. (15), studying basketball players, found little relationship between aerobic capacity (treadmill VO2 max) and recovery from high intensity exercise (Wingate power test and a line drill). However, only one Wingate bout was performed. In addition, Bentley and McNaughton (5) provided evidence that stage length during incremental (aerobic) cycling tests influences peak power and therefore its relationship to VO2 Peak. However, the protocol in that study was not a Wingate power test. The relationship of anaerobic power and oxygen consumption has been studied, but to the knowledge of the authors, there has been no direct investigation into the relationship of VO2 Peak and repeated performance of 30-second Wingate bouts.

It was hypothesized that there is some positive effect of possessing higher maximal oxygen consumption during repeated Wingate cycling even though it is considered an "anaerobic" test. Because the potential benefit of enhanced oxidative capacity may only be realized when acute recovery is an issue, the current study employed several anaerobic bouts separated by 3 min recovery. In addition to VO2, anthropometric qualities of the performer may be associated with the observed repeated Wingate performance. Therefore, the purpose of this study was to investigate the relationship of VO2 peak and body fat percentage with performance of four successive 30-sec bouts of intense anaerobic exercise.

METHOD

Subjects

Thirty-one college-aged males volunteered to participate in the study. Only male subjects were recruited in order to insure homogeneity of the population due to expected large differences between male and female VO2 Peak, body fatness, and power output capabilities. Although the choice of exclusively using males limits applicability to other populations, quality of the results is enhanced. Subjects were recruited via word-of-mouth. Each subject signed an informed consent and completed a health history questionnaire prior to the study, with only apparently healthy individuals being permitted to participate. The study was approved for use of human subjects by the local Institutional Review Board.

Methodology

Subjects participated in two laboratory sessions (one session involved descriptive data collection and a graded cycle ergometer test to determine VO2 Peak and one session involved 4 Wingate trials). Height was measured using a standard stadiometer, followed by mass to the nearest 0.11 kg on a standard balance scale (Detecto-Medic, Detecto Scales Inc., Brooklyn, NY). Body fat percentage was measured with skinfold calipers (Lafayette Instrument Company, Lafayette, IN) using the three-site (chest, abdomen, thigh) skinfold method (35).

In order to determine VO2 peak, subjects were fitted with an appropriately sized air-cushioned facemask and asked to pedal at 60 rpms on a cycle ergometer (Monark Ergomedic 824E, Sweden) while metabolic data were collected using a Vacu·med Vista mini cpx system interfaced with Turbofit software (Vacu·med, Ventura, CA, USA). Heart rate was monitored using a Polar heart rate monitor transmitter (Stamford, CT, USA) positioned at the level of the sternum. Subjects performed a 2 min warm up at 0 watts. At the end of 2 min resistance was increased 50 watts every 2 min until volitional exhaustion.

For Wingate trials subjects completed a warm-up consisting of 4 min of cycling at 50 watts (50 rpms, 1.0 kp) on a Monark 824 cycle ergometer designed for immediate-load resistance and equipped with toe clips to prevent foot slippage. The resistance for the Wingate testing was determined by computer software (SMI Power 5.2, Sports

Medicine Industries), using 7.5% of body mass. Even though 7.5% of body mass may be too low for optimization of power in some adults (18), it was used for this study due to the intense physical challenge of repeated Wingate bouts. The subjects began pedaling as fast as possible with no resistance. When maximum rev·min^{-1} were reached, the weight basket was dropped. Subjects were verbally encouraged to provide maximal effort throughout each 30-second test. The first Wingate trial (W1) was followed by three identical Wingate tests (W2 ,W3, W4), as described above, with three minutes recovery between each trial.

During the Wingate bouts, data for Peak Power, Mean Power, Minimum Power, and Fatigue Index were collected at 1-second intervals via an optical sensor (OptoSensor, Sports Medicine Industries) interfaced with computer software (SMI Power 5.2, Sports Medicine Industries). The highest heart rate attained during or immediately after the conclusion of each individual Wingate bout was recorded as Peak Heart Rate. Recovery Heart Rate was recorded at the end of each 3 min recovery period. The recovery period consisted of passive seated rest on the cycle ergometer, as evidence has been provided that passive (vs active) recovery restores perfromance capability more effectively in situations of repeated bouts with short recovery intervals (9).

Table 1. Correlations of Body Fat Percentage and VO_2 Peak with power performance indicators.

		Body Fat Percentage		**VO_2 Peak**	
	Mean + SD	11.7 $\pm$ 6.1		46.6 $\pm$ 9.3	
		r	**p**	**r**	**p**
Bout 1					
Peak Watts/kg	10.9 + 2.2	-0.242	0.198	-0.200	0.282
Mean Watts/kg	7.6 $\pm$ 1.5	-0.540*	0.002	0.104	0.578
Min Watts/kg	5.3 + 1.3	-0.610*	0.000	0.254	0.168
Fatigue Index	50.0 $\pm$ 11.4	0.481*	0.007	-0.430*	0.016
Bout 2					
Peak Watts/kg	10.4 $\pm$ 2.1	-0.311	0.100	-0.028	0.884
Mean Watts/kg	6.4 $\pm$ 1.4	-0.657*	0.000	0.483*	0.007
Min Watts/kg	4.1 $\pm$ 1.4	-0.590*	0.001	0.603*	0.000
Fatigue Index	59.4 $\pm$ 12.1	0.422*	0.022	-0.675*	0.000
Bout 3					
Peak Watts/kg	9.4 $\pm$ 2.2	-0.216	0.278	0.016	0.934
Mean Watts/kg	5.5 + 1.4	-0.543*	0.003	0.527*	0.004
Min Watts/kg	3.5 $\pm$ 1.3	-0.517*	0.007	0.595*	0.001
Fatigue Index	61.0 + 13.9	0.404*	0.041	-0.489*	0.010
Bout 4					
Peak Watts/kg	8.5 $\pm$ 2.4	-0.296	0.143	0.185	0.366
Mean Watts/kg	5.0 $\pm$ 1.5	-0.485*	0.012	0.573*	0.002
Min Watts/kg	3.3 $\pm$ 1.3	-0.354	0.076	0.611*	0.001
Fatigue Index	60.3 $\pm$ 13.0	0.153	0.455	-0.525*	0.006

*Significant Pearson Product Moment correlation ($p \leq 0.05$).

Statistical Analysis

Data were analyzed using SPSS 10.0. Pearson Product Moment correlations were calculated for VO2 Peak, Body Fat Percentage, Heart rate, Peak Power, Mean Power, Minimum Power, and Fatigue index ((Peak Power-Minimum Power)/Peak Power*100) for each bout. In some instances, fatigue index provides limited insight because it is only indicative of a single trial. In other words, there may be an advantage in each individual trial, but not necessarily an advantage in fatigue index in successive bouts. Therefore, delta values for peak, mean, and minimum power [(Power 1 – Power 2)/Power 1 * 100; (Power 1 – Power 3)/Power 1 * 100; and (Power 1 – Power 4)/Power 1 * 100] were analyzed to compare power variables in each bout to those in the first bout. This procedure permits repeated trials to be compared using the initial trial performance as a criterion standard (22, 23). Results were considered significant at $p \leq 0.05$.

RESULTS

The subjects were found to have an average age of 22.7±2.9 years and average VO2 of 46.6±9.3 ml/kg/min (range = 40.5). The subjects had a mean height of 177.9±7.3 cm, with an average mass of 80.4±16.0 kg and a mean body fat percent of 11.7±6.1 (range = 21.4).

Table 1 displays correlations of VO2 Peak and Body Fat Percentage with power performance indicators including peak watts/kg, mean watts/kg, minimum watts/kg and fatigue index. Representative data are displayed graphically in Figures 1 through 4. A significant correlation was found between VO2 Peak and Body Fat Percentage (r = -0.534; p = 0.002). No significant correlations were found for VO2 Peak or Body Fat Percentage with Peak Power on any Wingate bout (p>0.05).

Table 2 displays correlations of VO2 Peak and Body Fat Percentage with delta values of power performance indicators. No significant correlations were found for Body Fat Percentage with delta values of power performance indicators on any Wingate bout (p>.005).

Table 2. Correlations of Body Fat Percentage and VO_2 Peak with power performance indicator delta values.

	Body Fat Percentage		VO_2 Peak	
	r	p	r	p
Peak Power				
Delta 1vs2	0.124	0.522	-0.285	0.127
Delta1vs3	-0.231	0.247	-0.150	0.447
Delta 1vs4	0.213	0.308	-0.414*	0.036
Mean Power				
Delta 1vs2	0.132	0.494	-0.547*	0.002
Delta1vs3	-0.142	0.481	-0.370	0.053
Delta 1vs4	0.172	0.410	-0.453*	0.020
Minimum Power				
Delta 1vs2	0.124	0.522	-0.456*	0.011
Delta1vs3	0.127	0.537	-0.391*	0.044
Delta 1vs4	0.062	0.762	-0.370	0.058

*Significant Pearson Product Moment correlation ($p \leq 0.05$).

Table 3 displays correlations of VO2 Peak and Body Fat Percentage with Peak and Recovery Heart rates for all four bouts. Representative data is displayed graphically in Figure 5. Significant correlations were found for VO2 Peak and Recovery Heart Rate (p<0.05). No significant correlations were found for Body Fat Percentage and Heart Rate on any Wingate bout (p>0.05).

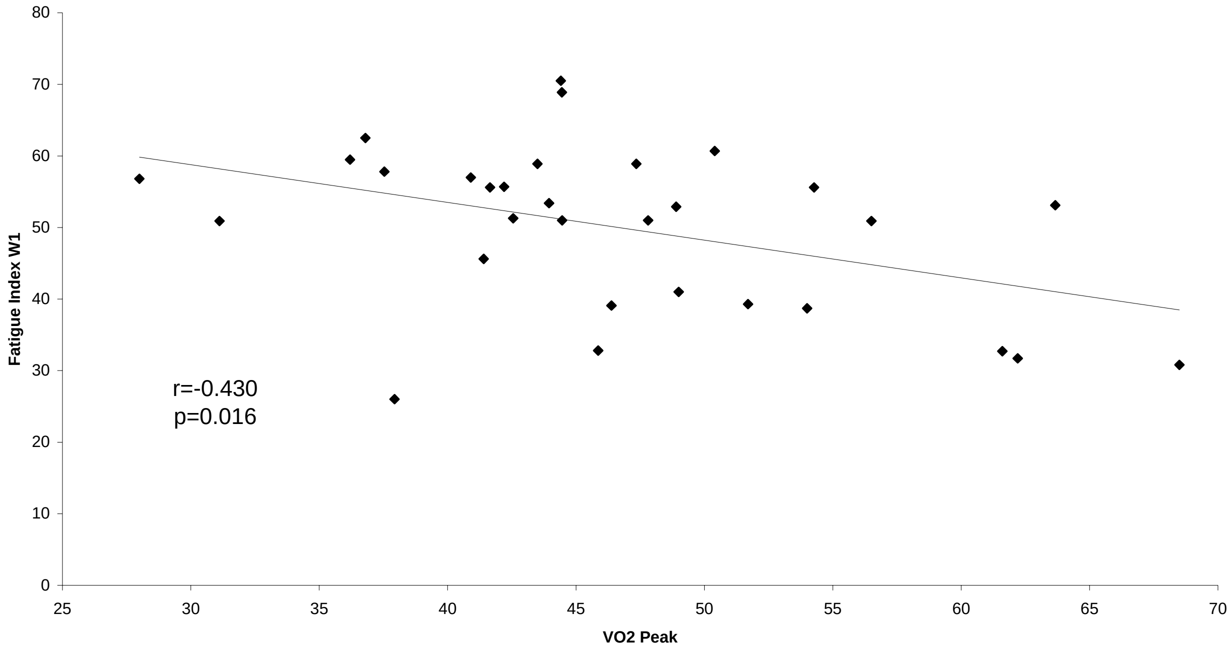

Figure 1. Correlation of VO_2 Peak and Fatigue Index on Wingate Bout 1.

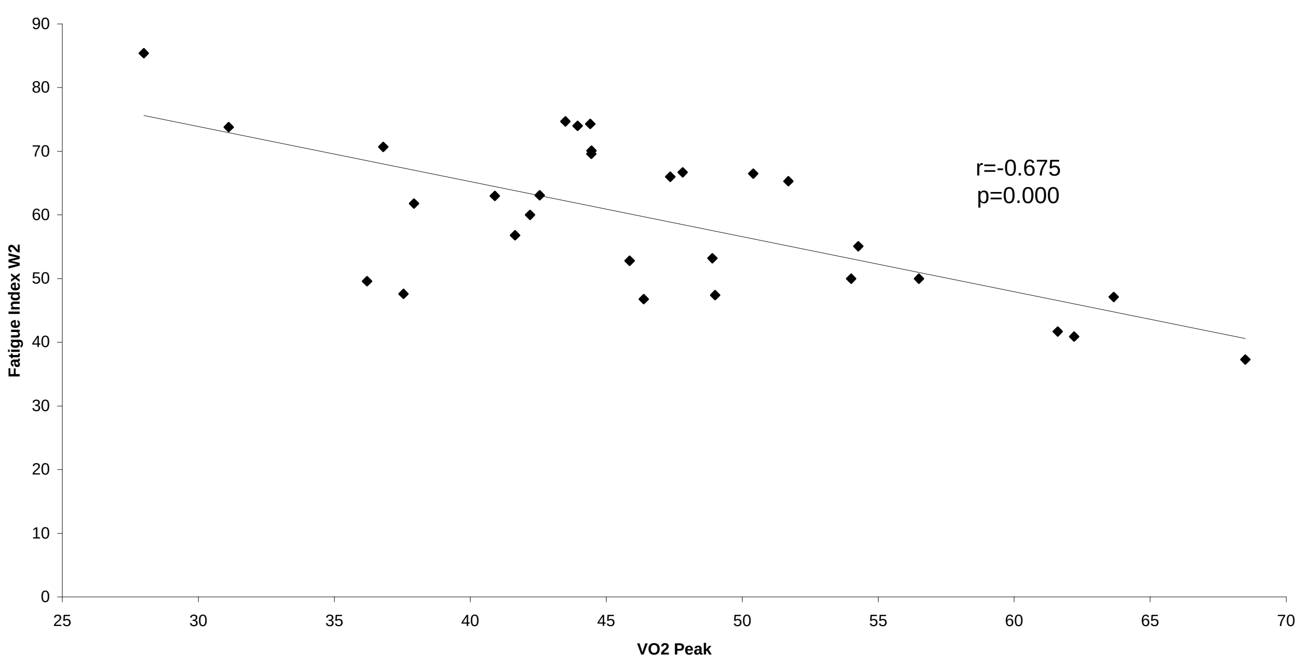

Figure 2. Correlation of VO_2 Peak and Fatigue Index on Wingate Bout 2.

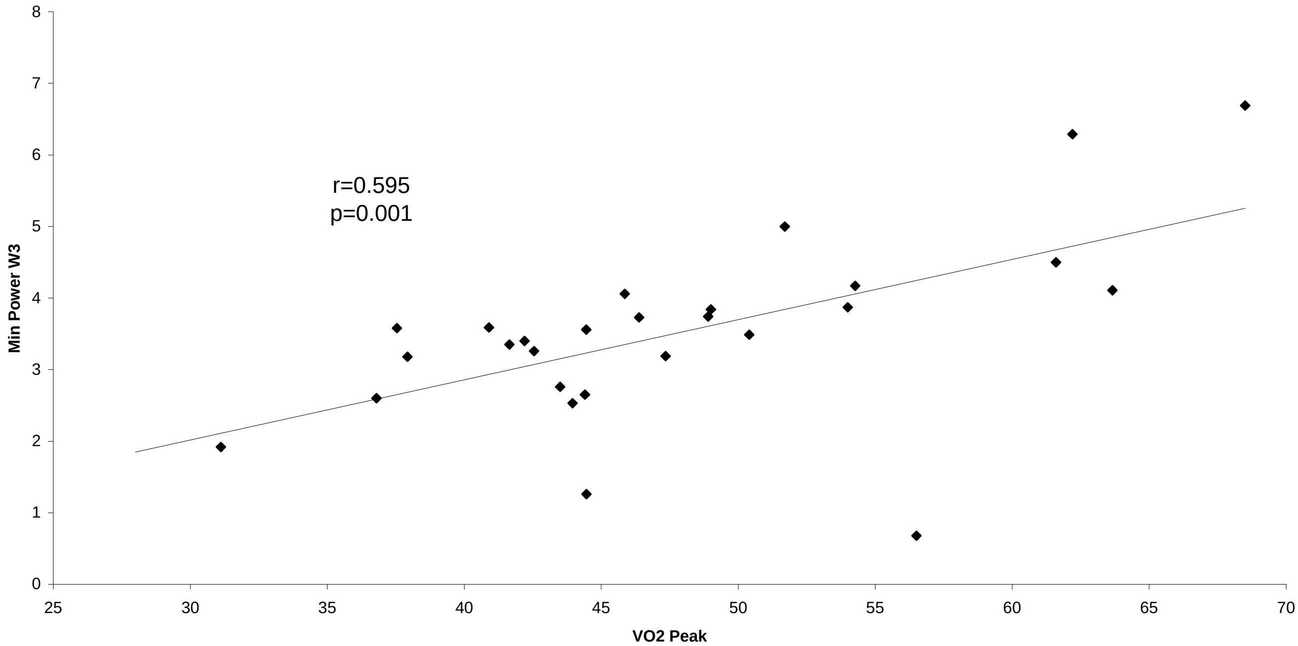

Figure 3. Correlation of VO_2 Peak and Minimum Power on Wingate Bout 3.

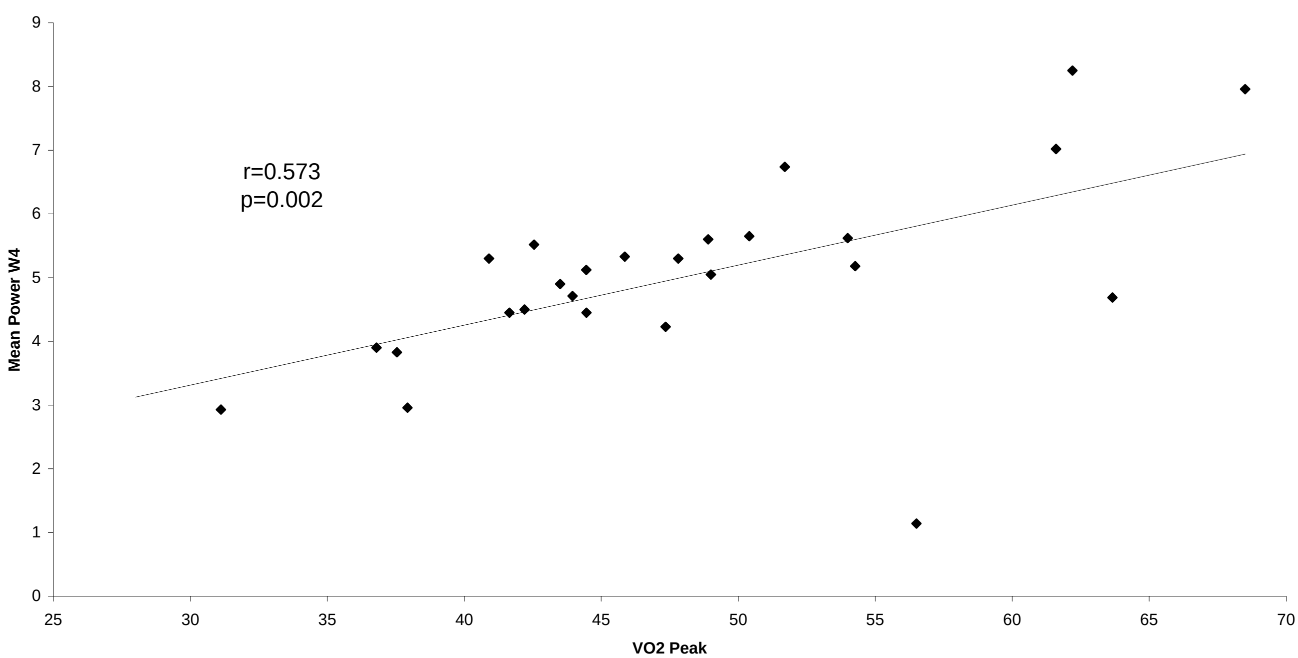

Figure 4. Correlation of VO_2 Peak and Mean Power on Wingate Bout 4.

Table 3. Correlations of Body Fat Percentage and VO_2 Peak with peak and recovery heart rates.

	Body Fat Percentage		VO_2 Peak	
	r	p	r	p
Bout 1				
Peak HR	0.208	0.271	-0.596*	0.000
Recovery HR	0.233	0.215	-0.501*	0.004
Bout 2				
Peak HR	0.052	0.785	-0.421*	0.021
Recovery HR	0.180	0.360	-0.383*	0.040
Bout 3				
Peak HR	0.260	0.190	-0.521*	0.004
Recovery HR	0.199	0.340	-0.567*	0.003
Bout 4				
Peak HR	0.199	0.329	-0.431*	0.025
Recovery HR	0.211	0.300	-0.502*	0.008

*Significant Pearson Product Moment correlation ($p \leq 0.05$).

DISCUSSION

The purpose of this study was to investigate the relationship of VO2 Peak and body fatness with performance on successive 30sec bouts of intense anaerobic exercise. The results indicate that both VO2 Peak and body fatness are significantly correlated with anaerobic performance, but remain independent correlates of various aspects of repeated Wingate performance. However, the strongest relationships of VO2 Peak and body fatness with measures of power performance occur at different points and are associated with different physiological indicators.

As consistent with previous studies, body fat percentage was significantly negatively correlated with VO2 Peak (r=-0.534; p=0.002). An individual with a greater aerobic capacity most likely engages in enough physical activity to alter their body composition in a favorable manner. Engaging in exercise of a more anaerobic nature probably induces positive adaptations in a person's body composition. Therefore, it may also be expected that body fat percentage would be correlated significantly with power performance variables in general (simply due to greater physical fitness). However, the less obvious aspect of the findings is that the correlations do not necessarily hold true for Peak Power, power performance delta values (Table 2), or heart rate response to repeated bouts (Table 3).

Body fat percentage and Peak Power output per kilogram were not significantly correlated on any bout. In addition, body fat percentage displayed no significant correlation with the change in power performance over successive bouts (Table 2). These are interesting in light of the finding that body fat percentage was significantly correlated with Mean Power, Minimum Power, and Fatigue Index in power on every bout. It is also interesting that the correlation of body fat percentage with power performance variables becomes weaker during successive bouts, even as VO2 Peak is becoming a stronger correlate with power performance. Pertaining to this notion it is interesting to observe the correlations (r values) and the significance of those correlations (p values) for VO2 Peak and body fatness from one bout to the next (Table 1). These data could indicate that possession of more relative lean muscle tissue is an advantage on any given bout of exercise, be it "aerobic" or "anaerobic". However, repeated bouts of exercise require a greater aerobic fitness component with each successive bout (6, 29). Therefore with each subsequent bout, the advantage to being "lean" in general is slowly overtaken in importance by the advantage of being lean due to a specific type of fitness (in this case, aerobic fitness).

Previous studies have concluded the aerobic metabolic pathways contribute an increasingly lager portion of ATP when high intensity bouts are repeated in succession (2, 12, 26). In this study, significant correlations were found for VO2 Peak and measures of anaerobic performance on successive bouts (Table 1), performance delta values (Table 2), and heart rate response (Table 3). These data are consistent with the previous finding that aerobic mechanisms contribute significant amounts of energy after the first bout (6); and therefore an enhanced oxidative capacity would be an advantage during repeated sprint activities. In addition, if endurance trained individuals are capable of faster lactate clearance (3); there should be some advantage because of an attenuated decrement in force in successive bouts. Though lactate was not measured during this investigation, it is safe to speculate that blood lactate can reach extremely high levels during exercise of this nature. Research has indicated that elevated H+ concentration due to a high rate of lactate formation contributes to fatigue in various ways (10-11, 25, 30-31), all of which lead to impaired force production. Therefore, it stands to reason that greater lactate clearance capability (which can only occur via oxidative means) should enhance performance on repeated bouts of anaerobic exercise.

As was expected, higher VO2 Peak was not significantly correlated with higher Peak Power output. In line with the principle of specificity, adaptations gained as a result of aerobic exercise (e.g. changes in muscle fiber characteristics) are not necessarily positive adaptations in terms of maximal power production. This finding also helps to explain the lack of correlation between body fat percentage and Peak Power. The subjects recruited for this study were not necessarily "aerobically" or "anaerobically" trained. Therefore, a subject in our study did not necessarily possess lower body fatness due to anaerobic training. It follows that there is no reason to expect higher Peak Power production (or greater performance from bout to bout) simply because the subject has lower body fatness. However, it is a safe assumption that those subjects in our sample with higher VO2 Peaks (e.g. in the 50-60 ml/kg/min range) probably were engaging regularly in aerobic exercise. Therefore, subjects on the higher end (aerobically) likely have the adaptations that would be advantageous in terms of performing repetitive bouts. Significant correlations of VO2 Peak with both Fatigue Index and Minimum Power provide evidence of this assumption. We would not expect a high VO2 Peak to translate into high Peak Power output, but having a greater Minimum Power and Lower Fatigue Index is indicative of a person capable of maintaining a given power output over the course of the bout (or several bouts).

The above data also lend strength to the finding that body fat percentage was not significantly correlated with heart rate response, while VO2 Peak was significantly correlated with heart rate on every bout (Table 3). In other words, the higher the VO2 Peak the more likely that a subject engages in regular aerobic exercise and therefore possesses cardiovascular and body composition adaptations to that exercise. These adaptations then transfer into greater performance during repeated bouts of high-intensity exercise, with aerobic adaptations being more important with each successive bout. However, if a subject possesses lower body fatness it does not necessarily mean that the subject is a regular "aerobic" exerciser. Possibly explaining the reason for correlations for body fatness and power performance variables tend to be highest in the earlier bouts.

In summary, results indicate that VO2 Peak is associated with anaerobic performance (with the strongest correlations occurring in later bouts), possibly due to greater capacity to recover between high intensity bouts and an increasing reliance on aerobic metabolism with each repeated bout. Body fat percentage is also correlated with power performance, with the strongest correlations existing in the earlier bouts. Body fat percentage is not strongly correlated with either the heart rate response to power performance or the change in power performance across successive bouts. Future studies should incorporate lactate and simultaneous metabolic analysis during the Wingate bouts and recovery periods. In addition, it would useful to repeat the current study with the use of subjects that are known to be "aerobically trained" and "anaerobically trained".

REFERENCES

1. Ahmaidi S, Granier P, Taoutaou Z, Mercier J, Dubouchaud H, Prefaut C. Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. Med Sci Sports Exerc 28: 450-456, 1996.

2. Bangsbo J, Krustrup P, Gonzalez-Alonso J, Saltin B. ATP production and efficiency of human skeletal muscle during intense exercise: effect of previous exercise. Am J Physiol Endocrinol Metab 280(6): E956-E964, 2001.

3. Bassett DR, Merrill PW, Nagle F., Agre JC, Sampedro R. Rate of decline in blood lactate after cycling exercise in endurance-trained and untrained subjects. J Appl Physol 70: 1816-1820, 1991.

4. Beneke, R., C. Pollmann, I. Bleif, R.M. Leithäuser, and M. Hütler. How anaerobic is the Wingate Anaerobic Test for humans? Eur J Appl Physiol 87(4-5): 388-92, 2002.

5. Bentley DJ, McNaughton LR. Comparison of W (peak), VO2 (peak) and the ventilation threshold from two different incremental exercise tests: relationship to endurance performance. J Sci Med Sport 6(4): 422-435, 2003.

6. Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. J Appl Physiol 80(3): 876-84, 1996.

7. Christou DD, Gentile CL, DeSouza CA, Seals DR, Gates PE. Fatness is a better predictor of cardiovascular disease risk factor profile than aerobic fitness in healthy men. Circulation 111(15): 1904-14, 2005.

8. Duffield R, Dawson B, Goodman C. Energy system contribution to 400-metre and 800-metre track running. J Sports Sci 23(3): 299-307, 2005.

9. Dupont, G., W. Moalla, R. Matran, and S. Berthoin. Effect of short recovery intensities on the performance during two Wingate tests. Med Sci Sports Exerc 39(7): 1170-6, 2007.

10. Fitts RH. Cellular mechanisms of muscle fatigue. Physiol Reviews 74: 49-94, 1994.

11. Fuchs F, Reddy V, Briggs FN. The interactions of cations with the calcium-binding site of troponin. Biochem Biophys Acta 221: 407-409, 1970.

12. Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent

maximal exercise. J Appl Physiol 75(2): 712-719, 1993.

13. Gordon SE, Kraemer WJ, Vos NH, Lynch JM, Knuttgen HG. Effect of acid-base balance on the growth hormone response to acute high-intensity cycle exercise. J Appl Physiol 76(2): 821-829, 1994.

14. Granier, P., B. Mercier, J. Mercier, F. Anselme, and C. Préfaut. Aerobic and anaerobic contribution to Wingate test performance in sprint and middle-distance runners. Eur J Appl Physiol Occup Physiol 70(1): 58-65, 1995.

15. Hoffman, J.R., S. Epstein, M. Einbinder, and Y. Weinstein. The influence of aerobic capacity on anaerobic performance and recovery indices in basketball players. J Strength Cond Res 13(4): 407-411, 1999.

16. Hogan MC, Gladden LB, Kurdak SS, Poole DC. Increased [lactate] in working dog muscle reduces tension development independent of pH. Med Sci Sports Exerc 27: 371-377 1995.

17. Inbar O, Bar-Or O. Anaerobic characteristics in male children and adolescents. Med Sci Sports Exerc 18(3): 264-269, 1986.

18. Inbar O, Bar-Or O, Skinner JS. The Wingate Anaerobic Test. Human Kinetics, Champaign, IL, 1996.

19. Kraemer WJ, Harman FS, Vos NH, Gordon SE, Nindl BC, Marx JO, Gomez AL, Volek JS, Ratamess NA, Mazzetti SA, Bush JA, Dohi K, Newton RU, Hakkinen K. Effects of exercise and alkalosis on serum insulin-like growth factor I and IGF-binding protein-3. Can J Appl Physiol 25(2): 127-138, 2000.

20. Marx JO, Gordon SE, Vos NH, Nindl BC, Gomez AL, Volek JS, Pedro J, Ratamess N, Newton RU, French DN, Rubin MR, Hakkinen K, Kraemer WJ. Effect of alkalosis on plasma epinephrine responses to high intensity cycle exercise in humans. Eur J Appl Physiol 87(1): 72-77, 2002.

21. McArdle WD, Katch FI, Katch, VL. Exercise Physiology: Energy, Nutrition and Human Performance, 5th Edition. Williams and Wilkins, Baltimore, MD, 2001.

22. McLester JR, Green JM, Chouinard JL. Effects of standing vs. seated posture on repeated Wingate performance. J Strength Cond Res18(4): 816-820, 2004.

23. Michael T. Percent fatigue for repeated wingate tests. Med Sci Sports Exerc 32(5): S310, 2000.

24. Patton JF, Duggan A. An evaluation of tests of anaerobic power. Aviat Space Environ Med 58(3): 237-242, 1987.

25. Powers SK, Howley ET. Exercise physiology: Theory and Applicationto Fitness and Performance 5th Edition, McGraw-Hill, New York, NY, 2004.

26. Putman CT, Matsos MP, Hultman E, Jones NL, Heigenhauser GJ. Pyruvate dehydrogenase activation in inactive muscle during and after maximal exercise in men. Am J Physiol 276: E483-488, 1999.

27. Reiser RF, Maines JM, Eisenmann JC, Wilkinson JG. Standing and seated Wingate protocols in human cycling. A comparison of standard parameters. Eur J Appl Physiol 88(1-2): 152-157, 2002.

28. Reiser RF, Peterson ML, Broker JP. Influence of hip orientation on Wingate power output and cycling technique. J Strength Cond Res 16(4): 556-560, 2002.

29. Riechman SE, Zoeller RF, Balasekaran G, Goss FL, Robertson RJ. Prediction of 2000 m indoor rowing performance using 30 s sprint and maximal oxygen uptake. J Sports Sci 20(9): 681-7, 2002.

30. Sahlin K. Metabolic factors in fatigue. Sports Med 13: 99-107, 1992.

31. Sale DG. Influence of exercise and training on motor unit activation. In Exerc Sports Sci Reviews, KB Pandolf (ed.), 15: 95-151, New York: Macmillan, 1987.

32. Slater GJ, Rice AJ, Mujika I, Hahn AG, Sharpe K, Jenkins DG. Physique traits of lightweight rowers and their relationship to competitive success. Br J Sports Med 39(10): 736-741, 2005.

33. Sleivert GG, Rowlands DS. Physical and physiological factors associated with success in the triathlon. Sports Med 22(1): 8-18, 1996.

34. Thorland WG, Johnson GO, Cisar CJ, Housh TJ, Tharp GD. Strength and anaerobic responses of elite young female sprint and distance runners. Med Sci Sports Exerc 19(1): 56-61, 1987.

35. Whaley MH (ed). American College of Sports Medicine Guidelines for Exercise Testing and Prescription (7th Edition). Lippincott Williams & Wilkins, Baltimore, MD, 2006.