LEADING ARTICLE

Sports Med 2001; 31 (1): 1-11
0112-1642/01/0001-0001/$22.00/0

The Relationship Between Aerobic Fitness and Recovery from High Intensity Intermittent Exercise

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Abstract

A strong relationship between aerobic fitness and the aerobic response to repeated bouts of high intensity exercise has been established, suggesting that aerobic fitness is important in determining the magnitude of the oxidative response. The elevation of exercise oxygen consumption ($\dot{V}O_2$) is at least partially responsible for the larger fast component of excess post-exercise oxygen consumption (EPOC) seen in endurance-trained athletes following intense intermittent exercise.

Replenishment of phosphocreatine (PCr) has been linked to both fast EPOC and power recovery in repeated efforts. Although ^{31}P magnetic resonance spectroscopy studies appear to support a relationship between endurance training and PCr recovery following both submaximal work and repeated bouts of moderate intensity exercise, PCr resynthesis following single bouts of high intensity effort does not always correlate well with maximal oxygen consumption ($\dot{V}O_{2max}$). It appears that intense exercise involving larger muscle mass displays a stronger relationship between $\dot{V}O_{2max}$ and PCr resynthesis than does intense exercise utilising small muscle mass.

A strong relationship between power recovery and endurance fitness, as measured by the percentage $\dot{V}O_{2max}$ corresponding to a blood lactate concentration of 4 mmol/L, has been demonstrated. The results from most studies examining power recovery and $\dot{V}O_{2max}$ seem to suggest that endurance training and/or a higher $\dot{V}O_{2max}$ results in superior power recovery across repeated bouts of high intensity intermittent exercise.

Some studies have supported an association between aerobic fitness and lactate removal following high intensity exercise, whereas others have failed to confirm an association. Unfortunately, all studies have relied on measurements of blood lactate to reflect muscle lactate clearance, and different mathematical methods have been used for assessing blood lactate clearance, which may compromise conclusions on lactate removal.

In summary, the literature suggests that aerobic fitness enhances recovery from high intensity intermittent exercise through increased aerobic response, improved lactate removal and enhanced PCr regeneration.

The ability to recover quickly is critical if subsequent bouts of all-out activity are required, as in many team sports. It has been suggested that adaptations associated with endurance training should enhance recovery from high intensity intermittent exercise,[1,2] and since the theoretical basis is so compelling, coaches and athletes alike invest a great deal of time into training and maintaining aerobic fitness. As will be seen, the literature supports aerobic fitness as a means of improving recovery from high intensity intermittent exercise.

1. Exercise Recovery

The return of the muscle to its pre-exercise state following exercise is a process known as recovery. The recovery process is biphasic, with an initial rapid phase of recovery lasting 10 seconds to a few minutes followed by a slower second recovery phase lasting anywhere from a few minutes to a number of hours.[3] During recovery, oxygen consumption ($\dot{V}O_2$) is elevated to help restore metabolic processes to pre-exercise conditions. The post-exercise $\dot{V}O_2$ beyond that required at rest has been termed excess post-exercise oxygen consumption (EPOC).[3]

The fast phase of recovery is marked by rapidly declining $\dot{V}O_2$ and heart rate. It is during this period that tissue stores of oxygen are quickly replenished,[3] and most of the ATP and phosphocreatine (PCr) depleted in the muscle are restored, with 70% of the phosphagens restored within 30 seconds and 100% restored within 3 to 5 minutes.[4] Once depleted, PCr is not restored until exercise concludes.[5] Additionally, no replenishment of PCr occurs when the circulation is occluded,[6,7] suggesting that oxygen is required for the process.

The increased metabolism marking the slow recovery period has been associated with the removal of lactate and H^+,[3,8] elevated body temperature,[9] the cost of increased respiratory and cardiac functions,[3] the effect of catecholamines[10] and the cost of glycogen resynthesis.[3] Recovery is not complete until metabolism has returned to pre-exercise levels.

2. Recovery from High Intensity Intermittent Exercise

The ability to perform maximally on repeated exercise bouts is influenced by the nature of both exercise and recovery periods. Generally, the more that exercise disrupts homeostasis, the greater the effect on recovery metabolism.[11] The more complete these restorative processes, the greater the ability to generate force or maintain power on subsequent work intervals.

Although a single bout of high intensity exercise lasting a few seconds results in decreased ATP/PCr stores, if the bout exceeds more than a few seconds anaerobic glycolysis will also be required to provide energy.[12] The metabolic consequence of increased anaerobic glycolysis is an increase in H^+ concentration and depressed pH, which may adversely affect performance by disrupting contractile processes.[8] Following exercise, complete phosphagen recovery may require 3 to 5 minutes,[4] but complete restoration of pH and lactate to pre-exercise levels may take an hour or more.[13] In repeated exercise bouts, if the subsequent recovery interval is less than a few minutes long, as in many team sports,[14,15] the ATP/PCr stores may be only partially restored before the onset of subsequent exercise demands, resulting in compromised performance on successive bouts. Moreover, as ATP/PCr stores are progressively depleted with subsequent high intensity work bouts,[12,16] there will be increased reliance on anaerobic glycolysis.[17] Following exercise that results in both the depletion of ATP/PCr stores and increased lactate and H^+ accumulation, it will require longer to return to the pre-exercise state. Once H^+ has accumulated, existing transport and metabolic pathways are less efficient, slowing the rate of recovery from exercise.

The length of the recovery interval between repeats of high intensity bouts of exercise will also affect recovery. Wooton and Williams[17] found that, although power output decreased over repeated 6-second all-out sprints with either 30 or 60 seconds of recovery between sprints, power output declined less when 60 seconds of recovery was allowed. A longer recovery interval ensures more complete

recovery; however, in sports requiring intermittent bursts of all-out effort the recovery periods may last only a few seconds, so performance on subsequent bouts may suffer. During brief intervals of recovery, at least some of the ATP, PCr and oxymyoglobin is restored. While restoration of the oxygen-myoglobin stores can take 10 to 80 seconds[18] complete phosphagen recovery may require 3 to 5[4] or even 8[6] minutes. The rate of post-exercise PCr resynthesis appears to be controlled by the rate of oxidative metabolism within the muscle,[19] and in the absence of circulation, little PCr is regenerated.[7,20]

The ability to recover from exercise resulting in lactate production depends on the capacity to tolerate, buffer and/or rapidly remove H^+ from working muscle.[21] Important buffers within muscle include PCr, inorganic phosphate, protein-bound histidine residues and carnosine.[22] Once in the blood, lactic acid is effectively buffered by sodium bicarbonate. Approximately 65% of the lactate is converted to pyruvate by lactate dehydrogenase (LDH), then undergoes aerobic degradation via the Krebs cycle and electron transport system, with the remaining 35% converted to glucose and/or glycogen, secreted in urine and sweat or converted to protein.[22] Most of lactate oxidation occurs in skeletal muscle, particularly the slow-twitch fibres.[23] The restoration of muscle pH is critical for optimal force production on subsequent exercise, since the rate of PCr resynthesis is influenced by the metabolic environment of muscle, especially the concentration of H^+[24] but also the ATP concentration and the rate of oxidative phosphorylation within working muscle.[25,26]

3. Possible Role of Aerobic Fitness in Enhancing Recovery

The most widely accepted measure of aerobic fitness, $\dot{V}O_{2max}$, represents the maximum rate at which aerobic metabolism can supply energy.[1] Another widely used index of aerobic fitness, aerobic capacity, identifies by blood lactate or ventilatory parameters the maximal steady-state exercise speed or workload that can be sustained for extended periods of time. Increases in $\dot{V}O_{2max}$ and aerobic capacity result from endurance training.[27] Aerobic capacity measures have proven useful in predicting success in distance running events,[28,29] with some scientists[30] regarding it as superior to $\dot{V}O_{2max}$ as a measure of endurance fitness.

Thoden[1] proposes that aerobic training may enhance the ability of the muscle to recover following anaerobic exercise, suggesting that an athlete with higher aerobic fitness will tax nonoxidative sources less and thereby recover at a more rapid rate from exercise. Theoretically, an increase in aerobic fitness could enhance recovery from anaerobic performance both by supplementing anaerobic energy during the exercise and by providing aerobically derived energy at a faster rate during the recovery period. Additionally, any improvements that aid in transport to or from the muscle, such as increased blood flow, could enhance the removal of lactate, H^+ and heat.

Individuals with high maximal aerobic power exhibit increased concentrations of aerobic enzymes, increased mitochondrial number, size and surface area[31] and increased myoglobin,[32] all contributing to improved oxygen extraction by muscle. Aerobic training also results in increased muscle blood flow, which is accomplished through elevated cardiac output,[33] increased capillarisation of muscle tissue[32,34] and an improved ability to vasodilate.[35] Oxygen delivery in the endurance-trained athlete is further improved by increases in blood volume and total haemoglobin volume.[36] Together, these enhancements result in an increased rate of $\dot{V}O_2$ during high intensity exercise[27] and decreased time to reach peak $\dot{V}O_2$ during exercise,[37] which may result in less lactic acid accumulation.[38] In conjunction with enhanced ATP/PCr stores[39] and elevated myokinase and creatine kinase concentrations[40] seen in trained athletes, these adaptations should result in an ability to supply more energy through the phosphagen and aerobic systems, thus decreasing the reliance on anaerobic glycolysis and thereby stemming the rise in H^+ during high intensity intermittent work. Endurance training results in lower blood and muscle lactate levels for the same absolute submaximal workload[41] because of decreased production of lactate as a result of in-

creased reliance on other energy systems[31] and/or increased lactate clearance.[42] With reduced anaerobic glycolysis during exercise, less energy is required during the recovery period to rid the muscle of H^+ and lactate, potentially hastening the recovery process.

Lactate removal from muscle is enhanced by increased buffering capacity and increased blood flow. Increased capillary density, as seen in endurance-trained individuals, provides a decreased diffusion distance between capillaries and muscle fibres, enhancing movement of oxygen and nutrients to, and the removal of H^+ and lactate from, the muscle.[31] Enhanced oxygen delivery to muscles post-exercise potentially accelerates the rate of PCr resynthesis, an oxygen-dependent process.[6,20] Tesch and Wright[26] found a significant correlation between capillary density and blood lactate concentration, suggesting an improved efflux of lactate as a result of increased capillary density.

Other training effects seen in aerobically trained individuals that may hasten recovery are improved temperature regulation during and after exercise,[43] better mobilisation and utilisation of fuel substrates[44] and increased hypertrophy of and selective recruitment of slow-twitch and fast-twitch type A muscle fibres.[45] The increased activity of the H form of LDH associated with endurance training[46] should also facilitate recovery by favouring the oxidation of lactate to pyruvate. This adaptation provides ready fuel, in the form of pyruvate, for aerobic metabolism and helps normalise pH by consuming H^+.[47] Thus, it appears that the metabolic and circulatory adaptations associated with high levels of aerobic power should facilitate faster recovery from high intensity exercise.

4. The Relationship Between Aerobic Fitness and Recovery from High Intensity Exercise

Numerous indicators have been used to assess recovery from exercise, including $\dot{V}O_2$, blood lactates, force or power recovery, muscle pH and muscle PCr. When relevant, research on single bouts of submaximal and maximal exercise will be presented, but it is the intention of this review to focus predominantly on the relationship between aerobic fitness and recovery from high intensity intermittent exercise.

4.1 Aerobic Response

Hamilton et al.[48] compared the aerobic response of endurance-trained runners ($\dot{V}O_{2max}$ 60.8 ± 4.1 ml/kg/min) and games players ($\dot{V}O_{2max}$ 52.5 ± 4.9 ml/kg/min) during repeated all-out 6-second treadmill sprints. Although assignment to games players and endurance-trained groups was on the basis of the chosen sport rather than $\dot{V}O_{2max}$, $\dot{V}O_{2max}$ was considerably different ($p < 0.01$), allowing for comparisons on the basis of aerobic power. Both groups attained similar peak (mean 6 seconds) power, but endurance-trained athletes consumed significantly more oxygen during repeated intervals of all-out sprinting and demonstrated a significantly smaller percentage decrement in power over the 10 sprints than did games players. Similar results were reported by Tomlin,[49] who examined the aerobic response of female recreational soccer players to repeated 6-second all-out cycle sprints. Grouped on the basis of $\dot{V}O_{2max}$, both the high (HAP) and low (LAP) aerobic power groups attained similar peak power ($p > 0.05$), yet the HAP group consumed significantly more oxygen over 10 sprint-recovery cycles ($p = 0.02$) and displayed a smaller percentage decrement over the 10 sprints ($p = 0.02$). Perhaps, consuming more oxygen during sprinting results in less reliance on anaerobic glycolysis and thus less lactic acid production, manifesting itself in less lactic acid and H^+ accumulation and thus superior power maintenance.

An increase in $\dot{V}O_{2max}$ has been associated with an increase in $\dot{V}O_2$ during repeated bouts of supramaximal exercise ($r = 0.83$;[48] $r = 0.78$[49]). Additionally, in individuals who completed two 30-second all-out cycle sprints, Bogdanis et al.[50] found a high correlation between $\dot{V}O_{2max}$ and the percentage of energy contributed by aerobic metabolism on sprint 1 ($r = 0.79$) and sprint 2 ($r = 0.87$). Thus, $\dot{V}O_{2max}$ appears to determine the magnitude of the aerobic response to repeated sprints.

Balsom et al.[51] reported that following erythropoietin administration, individuals performing 15 × 6-second treadmill sprints showed reduced accumulations of blood lactate and the adenosine degradation product, hypoxanthine, despite performing the same amount of exercise when compared with the control condition. Additionally, Balsom et al.[52] demonstrated that by decreasing oxygen availability via a hypobaric chamber, individuals consumed less oxygen, accumulated more lactate and experienced larger power decrements during intense intermittent exercise than under normoxic conditions. These findings suggest that increased oxygen availability, as also happens with increases in $\dot{V}O_{2max}$, results in increased aerobic and reduced anaerobic contribution, whereas decreased oxygen availability results in decreased aerobic and increased anaerobic contribution to sprinting.

4.2 Excess Post-Exercise Oxygen Consumption

When exercising at the same percentage of $\dot{V}O_{2max}$, trained individuals consume more oxygen than untrained individuals because of their higher $\dot{V}O_{2max}$. Therefore, at the start of the recovery period $\dot{V}O_2$ is elevated, resulting in a greater potential magnitude for the fast EPOC. As well, ATP/PCr stores in trained individuals tend to be higher[39] and since PCr replenishment has been coupled to fast EPOC,[4,53] it is not surprising that fast EPOC following submaximal exercise of the same relative intensity is demonstrably higher in endurance-trained individuals.[37,54-56] Given the same percentage of $\dot{V}O_{2max}$, trained individuals have a larger magnitude of fast EPOC whereas total EPOC tends to be about the same and total recovery time shorter.[37,54-56] Hamilton et al.[48] found that following 10 repeated all-out treadmill sprints where endurance-trained athletes consumed significantly more oxygen than games players during the sprint-recovery cycles, endurance-trained athletes appeared to consume more oxygen immediately after exercise, whereas games players consumed more oxygen during the remaining 14 minutes of recovery, resulting in a similar net EPOC, supporting similar patterns of EPOC seen in submaximal studies. With more oxygen consumed sooner, the fit individual should be able to restore more ATP/PCr. The high post-exercise $\dot{V}O_2$ associated with higher aerobic power may be advantageous in priming the aerobic system to consume more oxygen immediately after exercise which, if used to replenish ATP/PCr stores, should be advantageous for repeated exercise, especially when subsequent exercise is primarily dependent on PCr breakdown. This may at least partially explain why the endurance-trained athletes were more successful in maintaining initial power output throughout the 10 repeats. Although the endurance-trained athletes and games players averaged similar power over the 10 sprints (612 *vs* 603W, respectively), and the games players generated higher power outputs on the initial 4 sprints, the endurance-trained athletes' power output on the final 6 sprints exceeded that of the games players.

LeMasurier[57] investigated the relationship between $\dot{V}O_{2max}$ and 3 minute EPOC following high intensity intermittent cycle sprints with 21 active males. He failed to find a significant correlation between relative $\dot{V}O_{2max}$ and 3-minute EPOC following 12 × 5-second sprints ($r = 0.19$; $p = 0.40$), but confirmed moderate relationships between $\dot{V}O_{2max}$ and EPOC following 6 × 10-second sprints ($r = 0.47$; $p = 0.03$) and 3 × 20-second sprints ($r = 0.67$; $p = 0.002$). With a lower volume of post-exercise oxygen consumed following the shorter sprints, the EPOC response of the participants may have been more homogeneous and thereby decreased the likelihood of a significant correlation. Furthermore, with 62% of the total EPOC occurring in the first minute, a $\dot{V}O_{2max}$–1-minute EPOC comparison may have better characterised the relationship.

Bell et al.[58] investigated the relationship between recovery from high intensity intermittent exercise and 2 different measures of aerobic fitness; aerobic capacity and $\dot{V}O_{2max}$. Highly trained endurance athletes performed 3 × 1-minute sprints at 125% $\dot{V}O_{2max}$, after which EPOC was measured. Using ventilatory threshold as the measure of aerobic capacity, the authors failed to find a signifi-

cant relationship between aerobic capacity and recovery or between $\dot{V}O_{2max}$ and recovery.

In failing to find significant relationships between aerobic fitness and EPOC (the rate of EPOC recovery and the magnitude of EPOC) following the sprints, Bell et al.[58] concluded that there does not appear to be a relationship between aerobic fitness and recovery from repeated bouts of high intensity exercise in highly trained endurance runners. However, as conceded by the authors, the population studied were highly trained (mean $\dot{V}O_{2max}$ 63.1 ml/kg/min, range 54.4 to 70.3 ml/kg/min), so even individuals with 'low' aerobic power scores were quite fit and may have had an aerobic fitness level sufficient to result in enhanced recovery. The possibility of an aerobic fitness threshold exists, beyond which recovery is enhanced; however, this hypothesis remains to be tested. Unfortunately, the interpretation of the EPOC results may have been hampered by an apparent lack of pre-exercise controls in terms of circadian effects, previous exercise or the thermic effect of food, all of which affect resting $\dot{V}O_2$ [59] and therefore net EPOC. Moreover, conclusions on the rate of recovery were based on the half-time recovery of EPOC, which may not be appropriate. Although it has been shown that the half-time EPOC recovery for submaximal exercise improves with training,[37,60,61] this index may be inappropriate for cross-sectional studies. It may be that rate of recovery is determined somewhat by genetic factors, such as the percentage of slow oxidative fibres, in that while rate of recovery can be improved, $\dot{V}O_{2max}$ is only one of the contributing factors. For example, Colliander et al.[20] demonstrated that individuals in a 'low fast twitch' group were superior to a 'high fast twitch' group in restoring force between sets of concentric contractions. Also, the metabolic profile of muscle fibres can be altered with endurance training through conversion of fast glycolytic fibres to fast oxidative glycolytic fibres,[62] thereby enhancing the oxidative capacity of the muscle, but the enhancements do not always translate into improvements in $\dot{V}O_{2max}$.[31] Furthermore, improvements in half-time recovery may indicate better recovery; however, similar half-time recovery scores do not necessarily suggest that recovery rate was the same for different individuals. If half-time EPOC recovery is the same for 2 individuals who differ in aerobic power, clearly the one with higher $\dot{V}O_2$ at the end of exercise, which is likely to be the one with superior aerobic power,[48,50] will utilise more oxygen in the same period of time. Therefore, use of the half-time recovery rate may not accurately reflect differences in the rate of recovery.

4.3 Lactate Removal

Since the accumulation of H^+ is implicated in fatigue, decreased accumulations create a more favourable contractile environment.[8] If, as well, lactate is removed faster from muscle by an efficient aerobic system, as hypothesised to happen in the aerobically fit, even less lactate will accumulate, resulting in less disruption of baseline pH levels. Unfortunately, all of the studies examining differences in lactate response rely on measurements of blood lactate, which only reflect muscle lactate, providing indirect evidence about lactate production and removal in the muscle from lactate accumulation in blood. Some authors have reported enhanced blood lactate clearance in endurance-trained athletes, but others have failed to find a relationship between blood lactate removal and $\dot{V}O_{2max}$.

When examining post-exercise blood lactate response, methods vary, with some authors using a bi-exponential model to describe lactate clearance[63-66] and others using linear regression, logarithmic or other methods.[67-69] These differences, in conjunction with the difficulties in assigning comparable work bouts to trained and untrained, makes the comparison of results difficult.

It appears that aerobically fit individuals attain peak lactate levels sooner in the post-exercise period with passive[63,64] and active[66] recovery, suggesting a more rapid efflux of lactate from muscle to blood in trained individuals. Some studies fail to support a relationship between improved blood lactate clearance and endurance training[68] or between blood lactate dissipation in well-trained versus untrained individuals;[69] however, in both of these

studies post-exercise blood lactate was not sampled until 3 minutes following the exercise bout. This may have resulted in missing peak lactate, especially in trained individuals,[63,66] which is an important variable in determining the rate of disappearance of blood lactate, regardless of the mathematical model used.

During passive recovery following an incremental procedure to exhaustion, in which endurance athletes worked at higher workloads than sprint-trained athletes, the endurance-trained athletes also had higher blood lactate accumulations than the sprint-trained individuals.[66] Bassett et al.[63] demonstrated similar results, whereby trained individuals cycling for 3 minutes at 85% $\dot{V}O_{2max}$ worked at higher workloads and accumulated more blood lactate sooner than untrained individuals cycling at 80% $\dot{V}O_{2max}$. Although both authors reported similar rates of lactate clearance between their respective groups, it must be emphasised that since both power output and lactate accumulation were higher in the groups with higher aerobic fitness and there is an inverse relationship between the rate of lactate clearance and absolute work,[70] this should be seen as superior recovery ability in the aerobically fit. Taoutaou et al.[66] also found that when the first 20 minutes of recovery was active, post-exercise time to peak lactate and the rate of blood lactate removal was faster in the endurance-trained athletes even though end-exercise blood lactate levels were similar between the groups. It may be that through active recovery some of the enhancements associated with endurance training, such as increased capillary density,[34] may be reflected more readily.

Freund et al.[64] found that highly trained athletes could clear lactate from working muscles faster than sedentary controls, and Oyono-Enguelle et al.[65] suggested that aerobic training may enhance lactate removal following anaerobic exercise. By comparing blood lactate disappearance rates of trained versus less trained individuals from different studies, Bonen and Belcastro[23] also concluded that trained individuals have faster lactate disappearance rates.

4.4 Power and Force Recovery

Bogdanis et al.[71] found that power recovery on repeated 30-second cycle sprints and resynthesis of PCr proceeds in parallel, confirming the relevance of PCr availability for power recovery. Subsequently, Bogdanis et al.[50] analysed pre- and post-exercise muscle biopsies from individuals who performed 2×30-second sprints separated by 4 minutes of passive recovery. From these results they demonstrated strong relationships between power recovery in the first 10 seconds of the second sprint and the resynthesis of PCr ($r = 0.84$) and between power recovery and endurance fitness ($r = 0.94$), as represented by the percentage of $\dot{V}O_{2max}$ corresponding to a blood lactate concentration of 4 mmol/L. Effectively these results link PCr resynthesis to both power recovery and endurance fitness. Unfortunately, no relationship was reported between $\dot{V}O_{2max}$ and power recovery or power recovery and PCr restoration.

Hakkinen and Myllyla[72] found that power and strength athletes generated higher peak force, whereas endurance athletes were able to maintain a 60% isometric contraction for longer ($p < 0.001$) and exhibited better relative force recovery following a 3-minute rest period. Neither $\dot{V}O_{2max}$ or fibre type were measured but the results imply that differences in force recovery are at least partially the result of differences in aerobic fitness, as aerobic fitness is generally superior in endurance-trained athletes.[73] It is likely that differences in force recovery also reflect peak power differences resulting from the different training regimens of strength, power and endurance athletes.[74]

Gaiga and Docherty[75] found that individuals who participated in 9 weeks of interval training that successfully increased $\dot{V}O_{2max}$ by 6 to 7% displayed increases in peak power and mean power in all 4 repeated 30-second maximal cycling sprints, with slightly greater improvements seen in the final 2 sprints. However, from the results it is difficult to establish if recovery improved or if the interval training merely enhanced the ability to generate peak power, since total work and peak power improved in all 4 repeats, with little change

in the absolute power decrement from sprint 1 to sprint 4.

Caution must be exercised when comparing individuals on relative power recovery such as percentage decrement over trials, as is conventionally done, especially when peak power varies considerably between athletes. Since individuals who produce higher peak power on initial trials have the potential to display greater absolute power decrement, a power recovery index may misrepresent the data. For example, greater percentage decrement trends have been associated with more powerful initial efforts.[48,76] It may be that percentage decrement is more appropriate when comparing athletes that can be matched on initial peak force; however, this is not always possible. A comparison between $\dot{V}O_{2max}$ and percentage decrement by Tomlin[49] was based on the results from 2 groups who generated similar peak power ($p = 0.58$) on the first of 10 cycle sprints; however, the group with higher aerobic power clearly demonstrated a smaller percentage decrement over the 10 sprints when compared with the LAP group ($p = 0.02$).

Other researchers have investigated the relationship between $\dot{V}O_{2max}$ and power maintenance for repeated cycle ergometer sprints. Moderate relationships have been reported between relative $\dot{V}O_{2max}$ and percentage decrement during 6×6-second cycle sprints ($r = –0.56$, $p < 0.05$),[76] 12×5-second cycle sprints ($r = –0.44$, $p = 0.04$)[57] and $6 \times$ all-out 40m treadmill sprints ($r = –0.62$, $p < 0.001$).[76] Using a protocol with 90 seconds active recovery between 6×15-second sprints, McMahon and Wenger[77] verified a significant $\dot{V}O_{2max}$–percentage-decrement relationship ($r = –0.63$; $p = 0.004$). All of these studies appear to support the notion that higher aerobic power may contribute to improving power recovery over repeated intervals.

Hoffman[78] estimated the aerobic fitness of 197 infantry soldiers on a 2000m run, then compared this to their performance on a field test of anaerobic ability, which consisted of 3 repeats of a 143m line drill sprint. He found that aerobic fitness had a low but significant correlation with the fatigue index based on the increase in sprint times over the 3 sprints ($r = – 0.33$, $p < 0.05$). Unfortunately, with 7 directional changes in the field test, skill may have had a larger impact on performance than the ability to recover between sprints and this may have contributed to the low correlation coefficient. However, when the participants were divided into 5 fitness categories based on their 2000m run times, clearly the fatigue index of the low fitness group was inferior ($p < 0.05$) to the fatigue index of the 3 fittest groups, supporting aerobic fitness as a contributor to power maintenance.

In a subsequent study, Hoffman et al.[79] investigated the influence of directly measured $\dot{V}O_{2max}$ on the ability of 20 basketball players to recover during the same 143m field test and failed to establish a significant relationship ($r = 0.01$, nonsignificant). It may be that the impact of skill on performance is even greater in such a small group, and that the $\dot{V}O_{2max}$ of the basketball players was too similar (mean 50.2 ± 3.8 ml/kg/min) to show differences in recovery.

4.5 Phosphocreatine Restoration

Increased rates of PCr resynthesis following submaximal exercise have been documented in endurance-trained athletes versus individuals in a control group[80] and in endurance-trained athletes versus sprinters, middle-distance runners and individuals in the control group.[81] Furthermore, McCully and Posner[82] demonstrated enhanced PCr resynthesis following only 2 weeks of muscle-specific aerobic training. Unfortunately, $\dot{V}O_{2max}$ was not measured in any of these studies, so extrapolation of the findings to $\dot{V}O_{2max}$ is somewhat limited. Even though most endurance-trained athletes possess high levels of aerobic power, enhanced aerobic power is not necessarily the only feature associated with endurance training.

Few studies have investigated the relationship between aerobic fitness and PCr resynthesis following a single bout of high intensity exercise[83-85] or high intensity intermittent exercise.[16] ^{31}P magnetic resonance spectroscopy (^{31}P-MRS) was utilised to investigate differences in PCr resynthesis in the gastrocnemius between HAP and LAP groups[83]

and between endurance-trained and sedentary individuals[84] following single 2-minute bouts of high intensity exercise. Neither study demonstrated differences in PCr recovery between the groups when PCr recovery was expressed as a percentage of resting levels or by using nonlinear regression model results, so it was concluded that $\dot{V}O_{2max}$ was a poor predictor of PCr recovery. It may be more appropriate to interpret the lack of difference in the percentage PCr recovery as a better recovery rate in the endurance-trained individuals and HAP groups since they would probably have had higher initial PCr levels[39] and would therefore have replenished more PCr in the same time. Furthermore, as a weight-bearing muscle the gastrocnemius may have been a poor choice for comparison where groups not only differed in $\dot{V}O_{2max}$ but also in bodyweight, with the sedentary and LAP groups significantly heavier than their aerobically fit counterparts. It may also be unrealistic to expect a whole body measure of $\dot{V}O_{2max}$ to correlate well with PCr recovery in a small muscle such as the gastrocnemius. In contrast to the results from Petersen and Cooke[84] and Cooke et al.,[83] Takahashi et al.[85] found $\dot{V}O_{2max}$ to be significantly correlated to the rate of PCr recovery in the quadriceps muscle following exhaustive exercise in endurance-trained and untrained individuals.

It may also be that the impact of aerobic power becomes more obvious with repeated bouts of high intensity exercise. Using ^{31}P-MRS to examine PCr resynthesis following 4 repeats of 2 minutes of moderately high intensity exercise, Yoshida and Watari[16] clearly demonstrated that endurance-trained individuals ($\dot{V}O_{2max}$ 73.6 ml/kg/min) had significantly faster PCr recovery than control individuals ($\dot{V}O_{2max}$ 46.6 ml/kg/min), which became increasingly apparent after the first bout of exercise.

5. Conclusion

The literature suggests that aerobic fitness probably enhances recovery from high intensity intermittent exercise through enhanced aerobic contribution, increased post-exercise $\dot{V}O_2$, and possibly by increased lactate removal and increased PCr restoration, which has been linked to improved power recovery. Now the challenge is to determine which of these relationships are cause and effect and which variables are coincidental.

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