

## Effect of Warm up Intensity below the Lactate Threshold on VO<sub>2</sub> Slow Component During Submaximal Exercise in Elite Futsall Players

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**Abstract:** The purpose of this study was to investigate the effects of intensity of warm-up on VO<sub>2</sub> slow component during submaximal exercise in the Iranian national female futsal player's team. The participants of this study were ten female players of Iranian futsal national team (22.5±3.2yrs; weight, 56.04±6.17kg; height, 163±3.68cm; body fat%, 23.5±3.8; BMI, 21.07±2.22kg/m<sup>2</sup>; VO<sub>2</sub>max, 46.05±4.61 ml.kg<sup>-1</sup>.min<sup>-1</sup>). The subjects performed two protocols of warm up (moderate and heavy intensity) and one protocol without warm up before submaximal exercise (80%LT) in three separated sessions after measuring of VO<sub>2</sub> max and lactate threshold. After heavy and moderate warm up, slow component of VO<sub>2</sub> reduced due to without warm-up but it was not significant (36±3.8, 41±5.5 and 46±6.5 ml.min<sup>-1</sup>; respectively). The results suggest that warm up causes reducing the O<sub>2</sub> uptake in third phase or slow component of VO<sub>2</sub> in sub maximal exercise.

**Key words:** Fatigue · VO<sub>2</sub> slow component · Warm up · Sub maximal exercise

### INTRODUCTION

In response to a step transition from rest to constant intensity moderate exercise, below the lactate threshold (LT), pulmonary oxygen uptake (VO<sub>2</sub>) increases to meet the augmented energetic requirement in three characteristic phases [1]. After a short delay of; 20 s, which reflects the transit time from the exercising muscles to the lungs (phase I), pulmonary VO<sub>2</sub> rises in a monoexponential fashion (phase II) to attain a steady state (phase III) within 2–3 min in healthy subjects. The phenomenon has been termed the "VO<sub>2</sub> slow component" or "excess VO<sub>2</sub>" and the extra VO<sub>2</sub> supplement the underlying initial mono exponential response kinetics [2]. Although systemic factors such as increased cardio-respiratory work and hormonal changes may contribute to the slow component and suggested intramuscular causes are as diverse as altered substrate utilization, altered fiber-type recruitment, increased muscle temperature and lactic acidosis [3]. The reduced efficiency could in turn relate to a reduced efficiency of the contractile machinery (increased ATP turn over rate) and a reduced mitochondrial efficiency (decrease ATP/O<sub>2</sub>) [4].

The slow component will reduce exercise tolerance by increasing the metabolic rate [2]. the mechanism underlying the continuous rise in VO<sub>2</sub> during suprathreshold exercise remains poorly understood, Since Poole *et al.* (1991) demonstrated that most of the VO<sub>2</sub> SC (86%) originates from the exercising muscles, It has been proposed that either a progressive increase in the number of motor units recruited and/or the progressive recruitment of less efficient type II motor units contribute predominantly to the appearance of the VO<sub>2</sub> SC during heavy intensity exercise [5]. The findings of several studies provide indirect support for the contention that either the activation of additional motor units or the serial recruitment of less efficient type II motor units results in an increased O<sub>2</sub> cost for a given unit of tension development leading to the appearance of the VO<sub>2</sub> slow component [6].

Several studies have used prior exercise as an experimental tool to alter blood and muscle metabolic status and to assess the resulting effects on VO<sub>2</sub> kinetics [7,8,9].

It seems that duration and intensity of warm up have the potential to enhance exercise performance by altering the VO<sub>2</sub> kinetic response [10]. A warm-up that has enough intensity to elevate blood lactate concentration

(approximately 2-4 mM/lit) can profoundly alter  $\text{VO}_2$  kinetics and has the potential to enhance exercise performance [11]. This phenomenon can be due to accumulation of several by-products from high-intensity exercise, including lactic acid, will increase muscle blood flow and thus make more  $\text{O}_2$  available to muscle [8].

Gerbino *et al.* (1996) were the first to examine the effect of performing prior heavy intensity exercise on the magnitude of the  $\text{VO}_2$  slow component during a subsequent bout of heavy exercise [7]. Since then, other studies have used similar protocols utilizing repeated bouts of heavy intensity exercise as a means of “priming” the metabolic pathways in an attempt to elucidate both the mechanism which limits phase II kinetics and the mechanism responsible for the appearance of the  $\text{VO}_2$  SC [12,13].

Gerbino *et al.* (1996) reported that a prior bout of heavy exercise speeds the  $\text{VO}_2$  kinetics during subsequent heavy exercise, but  $\text{VO}_2$  kinetics wasn't affected by the prior bout of heavy-intensity exercise during moderate exercise [7].

However in opposed study Koppo and Bouckaert (2000) showed that a prior sub maximal exercise similar with prior heavy exercise, speeded  $\text{VO}_2$  response as some as heavy exercise. These authors suggested that the prior exercise result in an increase in  $\text{O}_2$  delivery during a second bout and thus speed the kinetics of  $\text{VO}_2$  [14].

Therefore the purpose of this study was to investigate the effect of intensity of warm-up exercise on  $\text{VO}_2$  kinetics during sub maximal exercise in elite female futsal players.

## MATERIALS AND METHODS

**Subjects:** 10 female national team Iranian futsal players (mean± SD: age, 22.5± 3.2yrs; weight, 56.04± 6.17kg; height, 163± 3.68cm), in camping of national team (October, 2009) volunteered and complete written informed consent to participate in this study.

**Experimental Design:** The subject's referred to the national Olympic academy of Iran on four days which was separated by 48h. Exercise testing was performed at approximately the same time of day for each subject and they inhibited of heavy exercise for at least 24h prior to testing.

In the first day subjects performed a graded exercise test (was defined for cosmed treadmill) to volitional fatigue for the determination of maximal oxygen uptake ( $\text{VO}_{2\text{max}}$ ) and lactate threshold. The first stage time of test

was 3min for warm up at work rate 6km/h and then increased incrementally by 1km/h every 1min until the subjects were unable to continue. In three subsequent next days subjects performed two warm up protocols (moderate and heavy intensity) and without warm up randomly before sub maximal exercise training (6 min at 80%  $\text{VO}_2$  at LT). The heavy intensity warm up was running on treadmill by 6min at 50% of the difference between the  $\text{VO}_2$  at LT and  $\text{VO}_{2\text{max}}$  [LT+ 50 % ( $\text{VO}_{2\text{max}} - \text{LT}$ )]. The moderate intensity warm up was running by 6min at 80% the  $\text{VO}_2$  at LT [15]. The both two protocols of warm up were continued by 3min running at 3km/h before the sub maximal exercise is performed. This approach was selected, because restriction effects of increase of heart rate on submaximal exercise.

**Data Collection:** Pulmonary gas exchange was measured breath-by-breath throughout all tests by Gas Analyzer (k4b2, Italy).

The difference in  $\text{VO}_2$  between the 3<sup>rd</sup> min and the 6<sup>th</sup> min of exercise ( $\Delta\text{VO}_{2(6-3)}$ ) has often been used in the literature as an expression of the  $\text{VO}_2$  slow component in this study [14].

**Statistical Analysis:** All statistical analyses were performed with using SPSS 16 soft were (Statistical Package for Social Science). The effect of prior exercise on the  $\text{VO}_2$  slow component responses was compared using a one-way repeated measures analysis of variance (ANOVA). A *P* value of 0.05 was considered statistically significant.

## RESULTS

Their physical characteristics are presented in Table 1.

The  $\text{VO}_2$  in 3<sup>rd</sup> and 6<sup>th</sup> and slow component values were presented in Table 2.

The results (Table 2) showed, the 3<sup>rd</sup>  $\text{VO}_2$  (*p*=0.35), 6<sup>th</sup>  $\text{VO}_2$  (total  $\text{VO}_2$ ) (*p*=0.34) and  $\text{VO}_2$  slow component (*p*=0.18) were not significant difference after three conditions.

Table 1: Values of *Physical characteristics of Participants'* (*M±SD*)

Parameters	Variables
Age (years)	22.5± 3.2
Weight (kg)	56.04± 6.1
Height (cm)	163± 3.6
BMI ( $\text{kg}/\text{m}^2$ )	21.07± 2.2
$\text{VO}_2$ max ( $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ )	46.05± 4.6
Fat percentage (%)	23.5± 3.8

Table 2: Values of Oxygen uptake in 3<sup>rd</sup> and 6<sup>th</sup> min and slow component (Mean ± SD) in different stage of test

	Without warm up	Moderate intensity warm up	Heavy intensity warm up	P value
3 <sup>rd</sup> min VO <sub>2</sub> (ml.min <sup>-1</sup> )	1701.17±145.4	1711±250.3	1616±86.5	0/35
6 <sup>th</sup> min VO <sub>2</sub> (ml.min <sup>-1</sup> )	1747±156	1752.2±203.5	1642±105.4	0/34
ΔVO <sub>2(6-3)</sub> (ml.min <sup>-1</sup> )	46±6.5	41±5.5	36±3.8	0/18

## DISCUSSION

The most important findings of this study was that VO<sub>2</sub> slow component during sub maximal exercise was decreased in both of moderate and heavy intensity warm up. However it was not significant.

These results were similar with Gerbino (1996) and Delory (2007) studies [7,16]. These investigators reported that VO<sub>2</sub> slow component during sub maximal exercise was not affected with moderate and heavy intensity warm up. In contrast, Burnely (2000), Koppo and Bouckaert (2000) and findings was inconsistent with this study [1, 14]. These studies have showed that the VO<sub>2</sub> slow component during submaximal exercise was reduced by prior heavy exercise. Difference in the test protocol may explain the discrepancy between our results and the results of the study of Koppo and Bouckaert (2000), in the study of Koppo (2000) the duration of the prior high intensity exercise was 6min however the duration of the prior low- intensity exercise had to be adjusted to 12min, while in this study the prior exercise time was 6min both for high and moderate intensity exercise. Koppo suggested that the total amount of work done during the prior exercise bout must be effect on VO<sub>2</sub> slow component of subsequent exercise [14].

The fundamental difference between the present study and Burntly (2000) was the modeling procedure that used to data analyze. Burnely *et al.* (2000) described the VO<sub>2</sub> slow component of heavy exercise with a mathematical model however in present study has been used different in VO<sub>2</sub> between the 3<sup>rd</sup> min and the 6<sup>th</sup> min of exercise (ΔVO<sub>2(6-3)</sub>) for expression of the VO<sub>2</sub> slow component [1].

Recent studies have argued that the progressive recruitment of less efficient type II muscle fibers may be responsible for the increased O<sub>2</sub> consumption during the VO<sub>2</sub> slow component.

Paterson *et al.* (2005) and Delory (2007) suggest that prior heavy-intensity exercise may improve the matching of muscle O<sub>2</sub> delivery to muscle O<sub>2</sub> utilization during the slow component of subsequent exercise or that a rightward shift of the oxyhemoglobin dissociation curve may facilitate the off loading of O<sub>2</sub> from Hb, thereby reducing the need to increase O<sub>2</sub> delivery [16, 17].

Other finding of this study was, warm up before the sub maximal exercise reduces total oxygen consumption in the end phase of exercise (6<sup>th</sup> VO<sub>2</sub>). Although the reason for this phenomenon wasn't clear, but reduce of total oxygen consumption could be partly due to reduction in VO<sub>2</sub> slow component phase. It is possible that the reduction in amplitude of the VO<sub>2</sub> slow component phase what we observed in the sub maximal exercise reflect a more rapid establishment of intracellular homeostasis in the second exercise bout, leading to the recruitment of fewer type II fibers as the exercise progressed [1, 18]. In support of this hypothesis, an increase in the amplitude of the phase II in VO<sub>2</sub> kinetics and a reduction in the slow component term have been shown in subjects with a high proportion of type I fibers [1]. It has been also suggested that rising muscle temperature might cause the slow component by decreasing the phosphorylation potential and increasing the rate of mitochondrial respiration via Q10 effect (the effect of increased temperature on enzyme-catalyzed reactions) effect [11]. The mechanism for the attenuated slow component response were, when muscle temperature was increased remains to be firmly established, however authors suggest, prior exercise result reduction in muscle viscosity and thus improves mechanical efficiency [10]. However in Gerbino study's (1996), decrease of slow component related to metabolic acidosis stimulation after heavy intensity warm up [7] , but in present study, reducing of slow component after moderate warm up showed metabolic acidosis wasn't necessary or at least couldn't only factor to reduce the slow component.

## CONCLUSION

Therefore the finding of this study showed heavy and moderate intensity warm up were reduced the time constant and slow component of VO<sub>2</sub>, thus reducing O<sub>2</sub> deficit and anaerobic metabolism during sub maximal exercise by increase oxygen available to muscle. The results suggest that warm up notwithstanding to intensity causes reducing the time of reaching to steady state of O<sub>2</sub> uptake in sub maximal exercise.

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