

## Factors defining oxygen uptake at peak exercise in aged people

Moran Sagiv · Ehud Goldhammer · David Ben-Sira · Ruthie Amir

Published online: 16 May 2010  
© European Group for Research into Elderly and Physical Activity (EGREPA) 2010

Maximum oxygen uptake ( $\text{VO}_{2\text{max}}$ ) is defined as the highest rates at which oxygen can be taken up and utilized by the body during severe exercise indicating the cardio-pulmonary fitness of the individual. It is one of the main variables in the field of exercise physiology and is frequently used to indicate the cardio-respiratory fitness of an individual [1]. Consequently, there has been great interest in identifying the physiological factors that limit  $\text{VO}_{2\text{max}}$  and determining the role of this variable in endurance and anaerobic performances. Today, it is universally accepted that there is a physiological upper limit to the body's ability to consume oxygen. In the scientific literature, an increase in  $\text{VO}_{2\text{max}}$  is the most common method of demonstrating a training effect. In addition,  $\text{VO}_{2\text{max}}$  is frequently used in the development of an exercise prescription in health and disease. Given these applications of  $\text{VO}_{2\text{max}}$ , there has been great interest in identifying the physiological factors that limit  $\text{VO}_{2\text{max}}$  and determining the role of this variable in endurance performance.

Aging-related changes occur mainly in the cardiopulmonary and skeletal muscles, bringing about a reduction in physical performance [7]. Such myocardial and peripheral functional changes include a decline in the maximum heart rate, stroke volume, and contractility, and an increase in

peripheral vascular resistance. Consequently, the maximal  $\text{VO}_{2\text{max}}$  decreases.

The primary aging process, itself genetically associated, occurs both independently of lifestyle and in the absence of disease [4]. Accordingly, one may expect maximal cardiac output to decrease with aging irrespective of lifestyle because of genetic factors. Reduced arteriovenous oxygen difference at maximal effort [6] is the second factor associated with decrease with aging of  $\text{VO}_{2\text{max}}$ .

Incremental exercise is characterized by exposing the subjects to a high degree of load which may alter the left ventricular contractility and function [5]. This has the effect of placing a large load on the left ventricular which might have significant effects on oxygen delivery to the working muscles. Thus, oxygen delivery to the working muscle may be reduced, and since metabolic demand during incremental exercise is increased over time to maximum, elderly subjects may maintain the energy supply due to the balance between  $\text{O}_2$  delivery and extraction.

Untrained and trained elderly can increase the response of the central factors i.e., cardiopulmonary without a significant reduction in peripheral ability to extract oxygen at the muscle level. It was found that in elderly subjects, skeletal muscle mitochondrial capacity, tissue blood flow capacity, and oxygen exchange capacity appear to be well-matched. It seems that intrinsic mitochondrial function and regulation are not altered significantly.

Values for cardiac output at peak aerobic exercise are low in the untrained compared to the trained at maximal aerobic exercise. The lower cardiac output in the untrained elderly at peak exercise is related to the limited heart rate reserve, stroke volume, contractility, and to the inappropriate adjustment of the circulation [3, 5]. The augmentation in maximal oxygen uptake in the trained elderly in response to

M. Sagiv (✉) · D. Ben-Sira · R. Amir  
Sports Medicine and Rehabilitation Division,  
Zinman College of Physical Education and Sport Sciences Wingate,  
Netanya, Israel 42902  
e-mail: sagiv-moran@wincol.ac.il

E. Goldhammer  
Heart Institute Bnai-Zion Haifa Medical Center,  
Haifa, Israel

exercise training is mainly due to the adaptive increases in cardiac output and to the lesser extent to the arteriovenous oxygen content difference at maximal exercise [2].

This suggests that the differences between the trained and untrained elderly in absolute oxygen uptake of the working muscles and peak power output at peak exercise are due to physical inactivity or physical activity. Although maximal oxygen uptake is lower in the untrained elderly, it seems that intrinsic mitochondrial function and regulation are not altered significantly due to physical inactivity. Thus, untrained subjects can partially compensate for the lower cardiac output by increasing oxygen extraction as the trained elderly.

The higher aerobic capacity in the trained elderly is related to increases in the abilities of cardiovascular factors and to the lesser extent to increases in muscle mitochondria concentration and capillarity.

## References

1. Basset DR Jr, Howley ET (2000) Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc* 32:70–84
2. Blomqvist CG, Saltin B (1983) Cardiovascular adaptations to physical training. *Ann Rev Physiol* 45:169–189
3. Delp MD (1998) Differential effects of training on the control of skeletal muscle perfusion. *Med Sci Sports Exerc* 30:361–374
4. Hawkins S, Wiswell R (2003) Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training. *Sports Med* 33:877–888
5. Sagiv M, Ben-Sira D, Goldhamer E, Soudry M (2000) Left ventricular contractility and function at peak aerobic and anaerobic exercises. *Med Sci Sports Exerc* 32:1197–1201
6. Shephard RJ (1987) Physical activity and aging, 2nd edn. Croom Helm, London
7. Thomas L, Levett K, Boyd A, Leung DY, Schiller NB, Ross DL (2003) Changes in regional left atrial function with aging: evaluation by Doppler tissue imaging. *Eur J Echocardiog* 4:92–100