Endurance Exercise Per-



by Lorenzo Pugliese, Gaspare Pavei, Simone Porcelli, Mauro Marzorati, Matteo Bonato and Antonio La Torre

ABSTRACT

Masters athletes are typically characterised as middle-aged and older men and women who continue physical training and sport activities at different levels throughout life. However, regardless of training, a decline in peak athletic performance usually occurs with ageing. In endurance exercise the reduction of performance and its physiological determinants appear to be mediated in large part by a reduction in the exercise training 'stimulus'. This reduction is mainly a result of increased work and family commitments, the inability to follow structured training programmes, increased prevalence of exercise training-associated injuries contributing to reduced training intensity and volume. These concerns highlight the importance for choosing and administering an adequate training stimulus in order to achieve maximum results in the shortest available time for training. Therefore, the first part of this article analyses the factors responsible for the decrease in performance with increased age. It is followed by a presentation different training methodologies that Masters runners in the middle- to long-distance events can use in order to prevent this decrease and, more importantly, increase their performance.

AUTHORS

Lorenzo Pugliese, MSc, PhD, is affiliated with the Institute of Molecular Bioimaging and Physiology, National Research Council, Segrate (Milano), Italy.

Gaspare Pavei, MSc, PhD, is affiliated with the Department of Pathophysiology and Transplantation, Università degli Studi di Milano, Italy.

Simone Porcelli, MD, PhD, is affiliated with the Institute of Molecular Bioimaging and Physiology, National Research Council, Segrate, Italy.

Mauro Marzorati, MD, PhD is affiliated with the Institute of Molecular Bioimaging and Physiology, National Research Council, Segrate (Milano), Italy.

Matteo Bonato, MSc, PhD, is affiliated with the Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milano, Italy

Antonio La Torre, MSc, is affiliated with the Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milano, Italy.

Introduction

ccording to the rules of the International Association of Athletic Federations (IAAF) and World Masters Athletics (WMA), Masters athletes are men and women older than 35 years who continue to physically train, compete recreationally or at organised competitive events.

Since the inaugural World Masters Games in Toronto (Canada) in 1985, where 8,305 participants competed in 22 sports, the number of Masters athletes has been steadily increasing. In 2013, approximately 25,000 athletes competed in the Games in Torino, Italy. Focusing on the marathon, in 1980 the number of U.S. marathon finishers over 40 years old was 37,180 (26% of estimated total of U.S. marathon finishers) and by 2013 this number had grown to 254,270 (47% of estimated total of U.S. marathon finishers)². This huge increase in participation has prompted an interest in issues related to the enhancement of the performance of Masters athletes¹.

Several studies show a decline in peak athletic performance with age across all disciplines³⁻⁵. For example, the age-related decrease in the performance of elite level Masters endurance athletes appears curvilinear from age 35 until approximately 60-70 years, increasing exponential thereafter^{4, 6}. This decrease occurs even if the athletes train for 10 or more hours per week, which they typically do over many decades³. Therefore, the first part of this review will analyse the factors causing the decrease in performance related to ageing while the second part will present the main training methodologies used by Masters runners to enhance performance in the middleand long-distance events.

Physiological Determinants of Endurance Performance and Ageing

Performance in endurance events is dependent upon three main physiological factors: maximal oxygen consumption, the exercise intensity at which a high fraction of the maximal oxygen consumption can be sustained, and exercise economy^{7, 8}.

Maximal oxygen consumption

In Masters athletes the progressive reduction in VO₂max appears to be a key physiological mechanism associated with the decline in endurance performance⁴. Although it is guite clear that it is higher in endurance-trained than in sedentary men of similar age⁹, VO₂max is estimated to decline approximately 10% per decade after the age of 25 in both healthy and sedentary individuals of both sexes¹⁰⁻¹⁶. However, attenuated¹⁷, similar¹⁸, or slightly greater¹⁹ rates of decline have been reported in Masters endurance athletes. Without discussing the reasons of these discrepant results among the studies, reductions in habitual exercise with ageing seems to be the major factor affecting rates of decline in VO max in endurancetrained athletes^{10, 14-16}.

Both central (maximal heart rate and maximal stroke volume) and peripheral (maximal arteriovenous oxygen difference) factors may play a role in age-related declines in VO max. An age-related decrease in maximum heart rate (HRmax) is commonly observed in endurance athletes^{4,11,20}. This decline of HRmax occurs regardless of exercise or gender, at a rate of approximately 3-5% per decade^{11, 21}. A significant age-related decline in maximal stroke volume in endurance-trained athletes has also been observed²². However, compared to age matched sedentary controls, the available research suggests that maximal stroke volume of Masters endurance athletes is elevated²³. This suggests that with this population, long-term physical training maintains a high level of cardiac function and stroke volume. Consequently, the decreased maximal cardiac output and VO_amax observed in Masters athletes appears to be an age-related decrease in HRmax rather than a significant change in stroke volume or cardiac morphology.

Peripheral adaptations include an arteriovenous oxygen difference - influenced by a variety of factors such as: muscle mass, the capacity of blood to transport and relinquish oxygen (blood volume, hemoglobin) - and the capacity of the working tissues to take up and utilise oxygen (capillarisation, muscle fibre type, aerobic enzyme activity). Reductions in peripheral oxygen extraction during maximal exercise appear to contribute to the decline in VO max with age in endurance exercisetrained adults. In fact, maximal arterio-venous O₂ difference declines modestly (5–10%) in this group over a span of ~30 years²⁴⁻²⁶. It remains to be determined if this reduction in maximal arterio-venous O₂ difference with age reflects reductions in maximal oxygen delivery to or extraction by the active muscles. However, older endurance-trained athletes can oxygenate blood in the lungs to a similar extent as young athletes. Also, their contracting muscles are capable of extracting oxygen as much as their younger counterparts²⁵. Furthermore, a similarity is observed in young and old endurance athletes regarding muscle oxidative enzyme activities and capillarisation (expressed per area or per fibre)²⁷. Thus, it is likely that maximal oxygen delivery, rather than oxygen extraction, is a major contributor to the age-related reduction in maximal arterio-venous O₂ difference in endurance-trained adults. As skeletal muscle mass is closely related to maximal aerobic capacity among healthy humans across the adult age range²⁸, a recent longitudinal investigation demonstrated that maintenance of lean body mass was associated with maintenance of VO, max in male Masters runners¹⁹.

Lactate threshold velocity

A reduction in the ability to sustain a high fraction of maximal oxygen consumption during submaximal exercise, typically evaluated using the blood lactate threshold, also contributes to the reduction in endurance performance with ageing. In older runners, endurance running performance is correlated with both VO max and velocity at lactate threshold^{18, 29-31}. WISWELL et al.31 determined that 60% of the variability in performance for runners aged 23-47 year was explained by the running velocity at which lactate threshold occurred, whereas VO max accounted for 74% of the variability for the runners aged 37-56 years. Absolute work rate or running speed at lactate threshold declines with advancing age in endurance athletes^{29, 31-33}. However, lactate threshold has

been observed not change or even increase with increased age when expressed relative to the percentage of VO₂max^{29, 31, 33, 34}.

Running economy

Running economy is measured as the steady-state oxygen consumption while exercising at a specific submaximal intensity below the anaerobic threshold³⁵. This has been shown to be a stronger predictor of endurance performance than VO_2 max in a homogenous group of endurance athletes^{36, 37}. The few studies focused on Masters endurance athletes^{31, 37} conclude that running economy does not change with age, suggesting that this factor does not contribute significantly to age related decreases in endurance performance.

Ageing

Ageing has been associated with a progressive increase in free radical production, i.e., synthesis of reactive oxygen species, (ROS), which can damage DNA and lipids, and oxidize proteins³⁸⁻⁴⁰, with a concomitant decrease in the enzymatic defense mechanisms (antioxidant). Free radicals promote the development of oxidative stress increasing oxidative damage⁴¹⁻⁴⁴. Optimal higher levels of ROS can improve physical fitness and subsequently delay the ageing process reducing the morbidity or mortality of all-cause diseases. However, an excessive rise in ROS levels may constitute a stress signal damaging antioxidant defenses, ultimately accelerating the ageing process and age-related diseases. On the other hand, lowering ROS levels below the allostatic set point may interrupt the physiological role of ROS in maintaining redox status and cellular adaption to exercise⁴⁵.

Physical exercise is perhaps one of the most characteristic examples demonstrating that ROS are not necessarily harmful, when considering the well-known benefits of regular exercise on the human organism accompanied by repeated episodes of oxidative stress^{46,47}. During exercise, the high energy demanded by muscle contraction causes an increase of oxygen delivery/uptake, leading to an increase of O₂ consumption. The high O₂ flux along the mitochondrial electron transport

chain, in association with an electron leakage, is correlated with an increased production of ROS. However, the chronic repetition of exercise may have the capability to develop a compensation to oxidative stress in skeletal muscle fibres⁴⁸ by means of an adaptation of the antioxidant and repair systems. This may result in a decreased resting level of oxidative damage and an increased resistance to oxidative stress⁴⁹.

Training Recommendations for Masters Runners

Apart from physiological factors, age-related declines in endurance performance have been related to decreased training volumes and intensities resulting from increased work and family commitments and reduced motivation to train (few Masters athletes have coaches and they spend less time training than younger athletes)20. In addition, an increased prevalence of exercise or training-associated injuries among Masters athletes probably contributes to their reduced training intensity and volume⁵⁰. All these factors lead to a reduction in the exercise training 'stimulus' (i.e. exercisetraining intensity, session duration and weekly frequency), which combined with aging and the aforementioned physiological considerations may have a role in the decline of peak performance^{10, 15, 51, 52}.

This highlights the importance of the choice of adequate stimulus to be administered in order to achieve maximum results in the shortest time available for training. Therefore, the second part of this review will focus on the different running training methodologies available that can be used to preserve physiological parameters mentioned above but more importantly increase performance.

Endurance training methods

In general, the main types of endurance training used are: i) continuous training (CON) at moderate intensity or running velocity, characterised by high volumes of training (> 30 min) with intensities between 60% and 80% of VOppeak or below the "anaerobic threshold" (AT) with a nearly constant O_2 consumption and without a "slow component" in O_2 kinetics of oxygen⁵³⁻⁵⁷; and ii) discontinuous high intensity training (DHIT) characterised by repeated exercises performed at running velocities corresponding to VO_2 peak (or slightly lower) or above AT or "all-out". High intensity efforts generally last from a few seconds to several minutes, being interspersed with periods of passive or low intensity recovery, resulting in partial but not complete recovery^{58, 59}.

Until a few years ago, it was widely believed that DHIT was a prerogative of elite athletes accustomed to sustain training periods of CON alternating with periods of DHIT, especially during the competitive season⁵⁵. Instead, in sedentary or moderately trained subjects they were prescribed primarily exercises of lowmoderate intensity and high-volume, as it was considered safe and more effective to improving aerobic metabolism⁶⁰⁻⁶². However, different studies indicate that even in sedentary or moderately trained individuals, DHIT might be an efficient strategy inducing adaptations in skeletal muscle and exercise performance, comparable to conventional endurance training^{55, 63-69}.

CON enhances VO₂max and reduces lactate concentration at low intensity by causing an increase in mitochondrial density, efficiency and volume^{8, 35, 70}. This is coupled with a higher capillarisation, which reduces the blood-mitochondria distance and enhances the aerobic pathway. Further, this improved mitochondrial activity shifts substrate oxidation to lipids, sparing carbohydrates for higher intensity exercise⁶⁰.

It is important to remark that the effectiveness of training is strongly related to the training stimulus. For instance, when CON is used to enhance performance over a given distance (let say 10km) the stimulus is mainly determined by the intensity. If athletes continue to train at the same speed, after several sessions the physiological adaptations reach a plateau and the same training will no longer enhance any parameters, including performance.

In studies of DHIT, an improvement in VO₂max was found and, in contrast to CON, running ve-

locity at VO₂max (vVO₂max) possibly mediated by enhanced neuromuscular properties and anaerobic characteristics^{71,72}. Higher muscle oxidative capacity with enhanced mitochondrial activity⁷³, an increased muscular glycogen content at rest and lower glycogen use with lower lactate production during exercise were reported after different DHIT protocols⁷⁴. These improvements may account for improved ventilator and lactate thresholds^{71,75} as well as increased time to exhaustion⁷³. Interestingly, as pointed out by GIBALA & MCGREE⁷³, these changes occur in less time and with lower training volume, which are critical parameters for Masters athletes.

As for oxidative damage, high-intensity discontinuous and continuous moderate-intensity training induced similar beneficial effects in Masters runners, by reducing the resting levels of the oxidative stress biomarkers in plasma and urine⁷⁶. In addition, evidence exists that exercise induced alterations in redox homeostasis are attenuated by both training modalities⁷⁶.

Application of DHIT

Since DHIT is characterised by high intense efforts, it is quite difficult to prescribe work ranges based on HR. Because of its slow on-kinetic HR, cannot properly describe the metabolic demand in short bouts of exercise. In addition, at intensity levels near or above VO₂max, HR changes are minimal due to the plateau near the HRmax, so differences in running velocity in short periods will not be reflected in variations in HR. Thus, incremental test parameters obtained in the laboratory, a a particular velocity at VO₂max or, for short bouts, maximal running speed, are recognised to be more accurate and effective for achieving desired performance outcomes^{58, 77}.

However, since laboratory tests may not be feasible, other approaches have been utilised to prescribe DHIT in endurance athletes. For example, coaches traditionally use a percentage of the 100-400m maximal velocity for short intervals (10-60 sec). For longer intervals, the velocity maintained over 800–1500m to 2000–3000m have been incorporated (2–4 to 6–8 min)^{58, 77}. In addition, the use of the rate of

perceived exertion (RPE) methods is becoming popular because of its simplicity. Using this approach for long intervals, coaches prescribe bouts of duration or distance allowing the athlete to self-regulate intensity, with use of the rate perceived exertion scale. The subjective intensity selected is typically "hard" to "very hard" (i.e. ≥ 6 on a CR-10 Borg scale and ≥ 15 on a 6–20 scale)^{58, 77}.

Unlike CON training, where only two variables (volume and intensity) are manipulated, with DHIT more variables can be incorporated in different sessions. These include, intensity and duration (similar to CON) as well as the type and length of recovery time between sessions^{58, 77}.

Regardless of the DHIT prescribed (long or short interval), training time dedicated to VO₂max is also of great importance. Current evidence suggests that within the total volume during a session, athletes should dedicate at least 10 minutes at an intensity of \geq 90%VO₂max. This can be achieved either as a series of repeated long (\geq 2 min) or short intervals (\leq 45 sec).

The nature of the recovery between efforts could be either active or passive, keeping in mind that during these two antithetic modalities, different physiological mechanisms are at work. When recovery is passive there is a higher rate of phosphocreatine (PCr) resynthesis, compared to an active recovery were PCr resynthesis rate is low. However, with an active recovery, a higher rate of muscular lactate removal occurs since there is a large activity of a lactate shuttle⁷⁸, maximised when the intensity is between 60-70% VO, max. Usually passive recovery is shorter than active. Based on these physiological aspects and the use and benefit of DHIT sessions, coaches can employ either recovery modality.

Minimal delay between the warm-up and the start of a DHIT session is recommended in order to accelerate the time needed to reach VO_2max with a warm-up intensity <60–70 % vVO_2max^{58} .

MODALITY	WORK INTENSITY	WORK DURATION	RECOVERY	RECOVERY DURATION	SERIES & REPS
	≥ 95% vVO₂max or v800m SB	2 – 3 min	Passive	≤ 2 min	6 - 10 x 2 min
DHII Long	≥ 90% vVO₂max or 100 – 105% v3000m SB	≥ 3 mir	≤ 60 – 70% vVO₂max or 60% v3000m	≥ 4 -5 min	5 - 8 x 3 min 4 - 6 x 4 min
DHIT Short	100 – 120% vVO ₂ max or 105 – 120% v800m SB	15 sec ≤ t ≤ 45 sec	Passive or ≤ 60 - 70% wO₂max or 60% v3000m	< 15 sec (passive) ≥ 15 sec	2-3 x ≥ 8 min (total work) Rest between series ≥ 4 – 5 min
CON Long	< vLT or 75 – 85% v10km SB	80 – 120 min			
CON Short	vLT or 90 – 95% v10km SB	30 – 50 min			
DHIT (discontinu v800m (speed c SB (seasonal be	Jous high intensity training); CON (Juring 800m race); v3000m (speec sst). From BUCHHEIT & LAURSEN	continuous low to moderate ir during 3000m race); v10km (; 7 (MODIFIED).	ntensity training); vVO ₂ max (spe speed during 10km race); vLT (;	ed at VO ₂ max); speed at lactate thre	shold);

Table 1: Examples of training modalities for continuous and discontinuous training for Masters runners

Previous training recommendations suggest that on average a ~48h time period separating DHIT will enable the majority of athletes to properly recover and maximise their performance⁷⁷. In fact, when DHIT sessions are separated by less than two days, a progressive overload is likely to occur. Sometimes this "functional" overload condition is sought by athletes during particular short training periods, however, if the overload is not properly controlled (reduced training or tapering) and maximal efforts are repeated over several days or weeks, DHIT can lead to a nonfunctional overreaching condition⁷⁷.

A functional long-term periodisation for Masters runners should begin with predominant low intensity continuous training in order to elicit cardiovascular, muscular and structural adaptations. After that, the high-intensity training stimulus becomes essential in order to obtain further improvements in endurance performance.

For all these reasons, it is recommended that Masters athletes wanting to improve their performance and/or health status must consult experienced coaches and rely on evidencebased training methods.

Examples of DHIT and CON training sessions are shown in Table 1.

Strength training

An age-related decline in strength has been observed in both sedentary individuals and Masters athletes, suggesting that muscles undergo the same physiological changes in both populations79, namely, a loss in type 2 motor units is responsible for the decrease in muscle power⁸⁰. This decline in strength accounts for a reduced capacity for muscle to recover. Therefore, part of the training process should focus on the development of neuromuscular properties of the muscles in order to preserve and/ or possibly improve the function of the muscle fibres. Both CON training and DHIT, associated with repetitions at high intensity, can preserve and enhance the properties of type 1 fibres⁸¹. This results in the neuromuscular improvements in both fibre types⁸². However, resistance training is known to affect type 2 versus type 1 fibres more⁸³.

Being aware of the relationship in young and elite athletes with regard to neuromuscular characteristics and running economy⁸⁴, PIA-CENTINI et al analysed the effect of six weeks of maximal strength training (2 times per week) during the conditioning period in preparation for long endurance events (CON endurance training targeted to increase maximal aerobic power) in Masters athletes. The authors observed that maximal strength training, when performed for a limited amount of time, increased running economy in Masters athletes, similar to younger athletes⁸⁵. This result suggests that resistance training should be integrated in the training programme for older athletes possibly slowing down age-related declines in muscle mass while improving performance. However, further research is needed.

Conclusion

Amongst the main physiological determinants of endurance exercise performance, a progressive reduction in VO₂max appears to be the primary mechanism associated with the decline in endurance performance with increased age. Reductions in lean muscle mass and lactate threshold velocity also contribute to the reduction in endurance performance, although these may be secondary factors. In contrast, exercise economy does not change with age in endurance-trained adults.

High intensity and volume training are important in maintaining or attenuating age-related decreases in VO₂max and endurance performance. However, the greatest challenge to Masters athletes is balancing an adequate physical stimulus for the body while preventing excessive fatigue, which may lead to injury^{50, 86}. Moreover, introducing periods of both high-intensity and volume training at the appropriate time in a training programme, in order to achieve an optimal performance⁵⁵ is imperative.

Please send all correspondence to: Dr Lorenzo Pugliese lorenzo.pugliese@ibfm.cnr.it

REFERENCES

1. LOUIS, J.; HAUSSWIRTH, C.; BIEUZEN, F. & BRISSWAL-TER J. (2009). Muscle Strength and Metabolism in Master Athletes. *International Journal of Sports Medicine*, 30 (10): 754-9.

2. USA R. Running USA Annual Marathon Report (2014) http://www.runningusa.org/running_usa_annual_marathon_report_2014.

3. RITTWEGER, J.; DI PRAMPERO, P.E.; MAFFULLI, N. & NARICI, M.V. (2009). Sprint and endurance power and ageing: an analysis of master athletic world records. *Proceedings of the Royal Society B: Biological Sciences*, 276 (1657): 683-689.

 TANAKA, H. & SEALS, D.R. (2008). Endurance exercise performance in Masters Athletes: age-associated changes and underlying physiological mechanisms. *Journal of Physiology*, 586 (1): 55-63.

5. ZAMPARO, P.; GATTA, G. & DI PRAMPERO, P. (2012). The determinants of performance in master swimmers: an analysis of master world records. *European Journal of Applied Physiology*, 112 (10): 3511-3518.

6. TANAKA, H. & SEALS, D.R. (1985). Invited Review: Dynamic exercise performance in masters Athletes: insight into the effects of primary human aging on physiological functional capacity. *Journal of Applied Physiology*, 95 (5): 2152-2162.

7. JOYNER, M.J. (1993). Physiological limiting factors and distance running: influence of gender and age on record performances. *Exercise and Sport Sciences Reviews*, 21: 103-133.

8. COYLE, E.F. (1995). Integration of the physiological factors determining endurance performance ability. *Exercise and Sport Sciences Reviews*, 23: 25-63.

9. SHEPHARD, R.J. (1986). Physical training for the elderly. *Clinical Journal of Sport Medicine*, 5: 515–533.

10. TANAKA, H.; DESOUZA, C.A.; JONES, P.P.; STEVEN-SON, E.T.; DAVY, K.P. & SEALS, D.R. (1985). Greater rate of decline in maximal aerobic capacity with age in physically active vs. sedentary healthy women. *Journal of Applied Physiology*, 83 (6): 1947-1953.

11. HAWKINS, S. & WISWELL, R. (2003). Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training. *Sports Medicine*, 33 (12): 877-888.

12. HEATH, G.W.; HAGBERG, J.M.; EHSANI, A.A. & HOL-LOSZY, J.O. (1981). A physiological comparison of young and older endurance athletes. Journal of Applied Physiology: *Respiratory, Environmental and Exercise Physiology*, 51 (3): 634-640.

13. BUSKIRK, E.R. & HODGSON, J.L. (1987). Age and aerobic power: the rate of change in men and women. *Federation Proceedings*, 46 (5): 1824-1829.

14. FITZGERALD, M.D.; TANAKA, H.; TRAN, Z.V. & SEALS, D.R. (1985). Age-related declines in maximal aerobic ca-

pacity in regularly exercising vs. sedentary women: a metaanalysis. *Journal of Applied Physiology*, 83 (1): 160-165.

15. ESKURZA, I.; DONATO, A.J.; MOREAU, K.L.; SEALS, D.R. & TANAKA, H. (1985). Changes in maximal aerobic capacity with age in endurance-trained women: 7-yr follow-up. *Journal of Applied Physiology*, 92 (6): 2303-2308.

16. PIMENTEL, A.E.; GENTILE, C.L.; TANAKA, H.; SEALS, D.R. & GATES, P.E. (1985). Greater rate of decline in maximal aerobic capacity with age in endurance-trained than in sedentary men. *Journal of Applied Physiology*, 94 (6): 2406-2413.

17. KASCH, F.W.; BOYER, J.L.; VAN CAMP, S.; NETTL, F.; VERITY, L.S. & WALLACE, J.P. (1995). Cardiovascular changes with age and exercise. A 28-year longitudinal study. *Scandinavian Journal of Medicine and Science in Sports*, 5: 147–151.

18. WISWELL, R.A.; HAWKINS, S.A.; JAQUE, S.V.; HYS-LOP, D.; CONSTANTINO, N.; TARPENNING, K.; MARCELL, T. & SCHROEDER, E.T. (2001). Relationship between physiological loss, performance decrement, and age in master athletes. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 56 (10): M618-626.

19. HAWKINS, S.A.; MARCELL, T.J.; VICTORIA JAQUE, S. & WISWELL, R.A. (2001). A longitudinal assessment of change in VO₂max and maximal heart rate in master athletes. *Medicine and Science in Sports and Exercise*, 33 (10): 1744-1750

20. REABURN, P. & DASCOMBE, B. (2008). Endurance performance in masters' athletes. *European Review of Aging and Physical Activity*, 5(5):12.

21. TANAKA, H.; MONAHAN, K.D. & SEALS, D.R. (2001). Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*, 37 (1): 153-156.

22. OGAWA, T.; SPINA, R.J.; MARTIN, W.H.; KOHRT, W.M.; SCHECHTMAN, K.B.; HOLLOSZY, J.O. & EHSANI, A.A. (1992). Effects of aging, sex, and physical training on cardiovascular responses to exercise. *Circulation*, 86 (2): 494-503.

23. HAGMAR, M.; HIRSCHBERG, A.L.; LINDHOLM, C.; SCHENCK-GUSTAFSSON, K. & ERIKSSON, M.J. (2005). Athlete's heart in postmenopausal former elite endurance female athletes. *Clinical Journal of Sport Medicine*, 15 (4): 257-262.

24. HAGBERG, J.M.; ALLEN, W.K.; SEALS, D.R.; HURLEY, B.F.; EHSANI, A.A. & HOLLOSZY, J.O. (1985). A hemodynamic comparison of young and older endurance athletes during exercise. *Journal of Applied Physiology*, 58 (6): 2041-2046.

25. SALTIN, B. (1986). The aging endurance athlete. In: RM SJaB, ed. Sports Medicine for the Mature Athlete. Indianapolis, IN: Benchmark Press; 59-80.

26. RIVERA, A.M.; PELS, A.E.; SADY, S.P.; SADY, M.A.; CULLINANE, E.M. & THOMPSON, P.D. (1985). Physiological factors associated with the lower maximal oxygen consumption of master runners. *Journal of Applied Physiology*, 66 (2): 949-954. 27. PROCTOR, D.N.; SINNING, W.E.; WALRO, J.M.; SIECK, G.C. & LEMON, P.W. (1985). Oxidative capacity of human muscle fiber types: effects of age and training status. Journal of Applied Physiology, 78 (6): 2033-2038.

28. FLEG, J.L. & LAKATTA, E.G. (1985). Role of muscle loss in the age-associated reduction in VO₂max. *Journal of Applied Physiology*, 65 (3): 1147-1151.

29. IWAOKA, K.; FUCHI, T.; HIGUCHI, M. & KOBAYASHI, S. (1988). Blood lactate accumulation during exercise in older endurance runners. *International Journal of Sports Medicine*, 9 (4): 253-256.

30. TANAKA, K.; TAKESHIMA, N.; KATO, T.; NIIHATA, S. & UEDA, K. (1990). Critical determinants of endurance performance in middle-aged and elderly endurance runners with heterogeneous training habits. *European Journal of Applied Physiology and Occupational Physiology*, 59 (6): 443-449.

31. EVANS, S.L.; DAVY, K.P.; STEVENSON, E.T. & SEALS, D.R. (1985). Physiological determinants of 10-km performance in highly trained female runners of different ages. *Journal of Applied Physiology*, 78 (5): 1931-1941.

32. WISWELL, R.A.; JAQUE, S.V.; MARCELL, T.J.; HAWKINS, S.A.; TARPENNING, K.M.; CONSTANTINO, N. & HYSLOP, D.M. (2000). Maximal aerobic power, lactate threshold, and running performance in master athletes. *Medicine and Science in Sports and Exercise*, 32 (6): 1165-1170.

33. MAFFULLI, N.; TESTA, V. & CAPASSO, G. (1994). Anaerobic threshold determination in master endurance runners. *Journal of Sports Medicine and Physical Fitness*, 34 (3): 242-249.

34. MARCELL, T.J.; HAWKINS, S.A.; TARPENNING, K.M.; HYSLOP, D.M. & WISWELL, R.A. (2003). Longitudinal analysis of lactate threshold in male and female master athletes. *Medicine and Science in Sports and Exercise*, 35 (5): 810-817.

35. JONES, A.M. & CARTER, H. (2000). The effect of endurance training on parameters of aerobic fitness. *Sports Medicine*, 29 (6): 373-386.

36. CONLEY, D.L. & KRAHENBUHL, G.S. (1980). Running economy and distance running performance of highly trained athletes. *Medicine and Science in Sports and Exercise*, 12 (5):357-360.

37. ALLEN, W.K.; SEALS, D.R.; HURLEY, B.F.; EHSANI, A.A. & HAGBERG, J.M. (1985). Lactate threshold and distance-running performance in young and older endurance athletes. *Journal of Applied Physiology*, 58 (4): 1281- 1284.

38. TAPPEL, A.L. (1973). Lipid peroxidation damage to cell components. *Federation Proceedings*, 32 (8): 1870-1874.

39. ESTERBAUER, H. & ZOLLNER, H. (1989). Methods for determination of aldehydic lipid peroxidation products. *Free Radical Biology and Medicine*, 7(2):197-203.

40. STADTMAN, E.R. & OLIVER, C.N. (1991). Metal-catalyzed oxidation of proteins. Physiological consequences. *Journal Biol Chem*, 266 (4): 2005-2008. 41. WEI, Y.H.; LU, C.Y.; WEI, C.Y.; MA, Y.S. & LEE, H.C. (2001). Oxidative stress in human aging and mitochondrial disease- consequences of defective mitochondrial respiration and impaired antioxidant enzyme system. *Chinese Journal of Physiology*, 44 (1): 1-11.

42. SQUIER, T.C. (2001). Oxidative stress and protein aggregation during biological aging. *Experimental Gerontology*, 36 (9): 1539-1550.

43. BRISSWALTER, J. & LOUIS, J. (2014). Vitamin supplementation benefits in master athletes. *Sports Medicine*, 44 (3): 311-318.

44. RADAK, Z.; ZHAO, Z.; GOTO, S. & KOLTAI, E. (2011). Age-associated neurodegeneration and oxidative damage to lipids, proteins and DNA. *Molecular Aspects of Medicine*, 32 (4-6): 305-315.

45. GUOLIN, L.I. (2013). The Positive and Negative Aspects of Reactive Oxygen Species in Sports Performance. Current Issues in Sports and Exercise Medicine, Associate Prof. Michael Hamlin (Ed.).

46. WANG, J.S.; LEE, T. & CHOW, S.E. (1985). Role of exercise intensities in oxidized low-density lipoprotein-mediated redox status of monocyte in men. *Journal of Applied Physiology*, 101 (3): 740-744.

47. SUREDA, A.; FERRER, M.D.; TAULER, P.; ROMA-GUERA, D.; DROBNIC, F.; PUJOL, P.; TUR, J.A. & PONS, A. (2009). Effects of exercise intensity on lymphocyte H2O2 production and antioxidant defences in soccer players. *British Journal of Sports Medicine*, 43 (3): 186-190.

48. POWERS, S.K.; JI, L.L. & LEEUWENBURGH, C. (1999). Exercise training-induced alterations in skeletal muscle antioxidant capacity: a brief review. *Medicine and Science in Sports and Exercise*, 31 (7): 987-997.

49. ALESSIO, H.M. (1993). Exercise-induced oxidative stress. *Medicine and Science in Sports and Exercise*, 25 (2): 218-224.

50. KALLINEN, M. & MARKKU, A. (1995). Aging, physical activity and sports injuries. An overview of common sports injuries in the elderly. *Sports Medicine*, 20 (1): 41-52.

51. POLLOCK, M.L.; MENGELKOCH, L.J.; GRAVES, J.E.; LOWENTHAL, D.T.; LIMACHER, M.C.; FOSTER, C. & WILM-ORE, J.H. (1985). Twenty-year follow-up of aerobic power and body composition of older track athletes. *Journal of Applied Physiology*, 82 (5):1508-1516.

52. MCGUIRE, D.K.; LEVINE, B.D.; WILLIAMSON, J.W.; SNELL, P.G.; BLOMQVIST, C.G.; SALTIN, B. & MITCHELL, J.H. (2001). A 30-year follow-up of the Dallas Bedrest and Training Study: I. Effect of age on the cardiovascular response to exercise. *Circulation*, 104 (12): 1350-1357.

53. MIDGLEY, A.W.; MCNAUGHTON, L.R. & JONES, A.M. (2007). Training to enhance the physiological determinants of long-distance running performance: can valid recommendations be given to runners and coaches based on current scientific knowledge? *Sports Medicine*, 37 (10): 857-880.

54. LAURSEN, P.B. & JENKINS, D.G. (2002). The scientific basis for high-intensity interval training: optimising training

programmes and maximising performance in highly trained endurance athletes. *Sports Medicine*, 32 (1): 53-73.

55. LAURSEN, P.B. (2010). Training for intense exercise performance: high-intensity or high-volume training? Scandinavian *Journal of Medicine and Science in Sports*, 20 Suppl 2: 1-10.

56. SEILER, K.S. & KJERLAND, G. (2006). Quantifying training intensity distribution in elite endurance athletes: is there evidence for an "optimal" distribution? *Scandinavian Journal of Medicine and Science in Sports*, 16 (1): 49-56.

57. KUBUKELI, Z.N.; NOAKES, T.D. & DENNIS, S.C. (2002). Training techniques to improve endurance exercise performances. *Sports Medicine*, 32 (8): 489-509.

58. BUCHHEIT, M. & LAURSEN, P.B. (2013). High-intensity interval training, solutions to the programming puzzle: Part I: cardiopulmonary emphasis. *Sports Medicine*, 43 (5): 313-338.

59. GIBALA, M.J.; LITTLE, J.P.; VAN ESSEN, M.; WILKIN, G.P.; BURGOMASTER, K.A.; SAFDAR, A.; RAHA, S. & TARNOPOLSKY, M.A. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol*, 575 (Pt 3): 901-911.

60. HOLLOSZY, J.O. & COYLE, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 56 (4): 831-838.

61. BERGER, N.J.; TOLFREY, K.; WILLIAMS, A.G. & JONES, A.M. (2006). Influence of continuous and interval training on oxygen uptake on-kinetics. *Medicine and Science in Sports and Exercise*, 38 (3): 504-512.

62. ROSE, A.J.; FRØSIG, C.; KIENS, B.; WOJTASZEWSKI, J.F. & RICHTER E.A. (2007). Effect of endurance exercise training on Ca2+ calmodulin-dependent protein kinase II expression and signalling in skeletal muscle of humans. *Journal of Physiology*, 583 (Pt 2): 785-795.

63. GIBALA, M.J. & MCGEE, S.L. (2008). Metabolic adaptations to short-term high-intensity interval training: a little pain for a lot of gain? *Exercise and Sport Sciences Reviews*, 36 (2): 58-63.

64. DAUSSIN, F.N.; PONSOT, E; DUFOUR, S.P.; LON-SDORFER-WOLF, E; DOUTRELEAU, S.; GENY, B.; PI-QUARD, F. & RICHARD, R. (2007). Improvement of VO₂max by cardiac output and oxygen extraction adaptation during intermittent versus continuous endurance training. *European Journal of Applied Physiology*, 101 (3): 377-383.

65. BURGOMASTER, K.A.; HUGHES, S.C.; HEIGENHAUS-ER, G.J.; BRADWELL, S.N. & GIBALA, M.J. (1985). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal* of Applied Physiology, 98 (6): 1985-1990.

66. GOROSTIAGA, E.M.; WALTER, C.B.; FOSTER, C. & HICKSON, R.C. (1991). Uniqueness of interval and continuous training at the same maintained exercise intensity. *European Journal of Applied Physiology and Occupational Physiology*, 63 (2): 101-107. 67. HICKSON, R.C.; BOMZE, H.A. & HOLLOSZY, J.O. (1977). Linear increase in aerobic power induced by a strenuous program of endurance exercise. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 42 (3): 372- 376.

68. HELGERUD, J.; HØYDAL, K.; WANG, E.; KARLSEN, T.; BERG, P.; BJERKAAS, M.; SIMONSEN, T.; HELGESEN, C.; HJORTH, N.; BACH, R. & HOFF, J. (2007). Aerobic highintensity intervals improve VO₂max more than moderate training. *Medicine and Science in Sports and Exercise*, 39 (4): 665-671.

69. IAIA, F.M.; HELLSTEN, Y.; NIELSEN, J.J.; FERNSTRÖM, M.; SAHLIN, K. & BANGSBO, J. (1985). Four weeks of speed endurance training reduces energy expenditure during exercise and maintains muscle oxidative capacity despite a reduction in training volume. *Journal of Applied Physiology*, 106 (1): 73-80.

70. BASSETT, D.R. & HOWLEY, E.T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and Science in Sports and Exercise*, 32: 70–84.

71. ESFARJANI, F. & LAURSEN, P.B. (2007). Manipulating high-intensity interval training: effects on VO₂max, the lactate threshold and 3000 m running performance in moderately trained males. *Journal of Science and Medicine in Sport*, 10 (1): 27-35.

72. MARLES, A.R.; LEGRAND, R.; BLONDEL, N.; MUCCI, P.; BETBEDER, D. & PRIEUR, F. (2007). Effect of high-intensity interval and detraining on extra VO₂max and on the VO2 slow component. *European Journal of Applied Physiology*, 99 (6): 633-640.

73. GIBALA, M.J. & MCGEE, S.L. (2008). Metabolic adaptations to short-term high intensity interval training: a little pain for a lot of gain? *Exercise and Sport Sciences Reviews*, 36 (2):58-63.

74. BURGOMASTER, K.A.; HEIGENHAUSER, G.J.F. & GIBALA, M.J. (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time trial performance. *Journal of Applied Physiology*, 100: 2041-2047

75. EDGE, J.; BISHOP, D.; GOODMAN, C. & DAWSON, B. (2005). Effects of high- and moderate- intensity training on metabolism and repeated sprints. *Medicine and Science in Sports and Exercise*, 37:1975–1982;

76. VEZZOLI, A.; PUGLIESE, L.; MARZORATI, M.; SER-PIELLO, F.R.; LA TORRE, A. & PORCELLI, S. (2014). Time-course changes of oxidative stress response to high-intensity discontinuous training versus moderate-intensity continuous training in masters' runners. PLoS One, 9(1):e87506.

77. BUCHHEIT, M. & LARSEN P.B. (2013). High-Intensity Interval Training, Solutions to the Programming Puzzle. Part II: Anaerobic Energy, Neuromuscular Load and Practical Applications. *Sports Medicine*, 43: 927-954.

78. BERGMAN, B.C.; WOLFEL, E.E.; BUTTERFIELD, G.E.; LOPASCHUK, G.D.; CASAZZA, G.A.; HORNING, M.A. & BROOKS, G.A. (1999). Active muscle and whole body lactate kinetics after endurance training in men. *Journal of Applied Physiology*, 87: 1684–1696.

79. BRISSWALTER, J. & NOSAKA, K. (2013). Neuromuscular factors associated with decline in long-distance running performance in master athletes. *Sports Medicine*, 43 (1): 51-63.

80. BROOKS, S.V. & FAULKNER, J.A. (1994). Skeletal muscle weakness in old age: underlying mechanisms. *Medicine and Science in Sports and Exercise*, 26: 432–9.

81. TRAPPE, S.W.; COSTILL, D.L.; FINK, W.J. & PEARSON, D.R. (1995). Skeletal muscle characteristics among distance runners: a 20-yr follow-up study. *Journal of Applied Physiology*, 78:823–9.

82. CREER, A.R.; RICARD, M.D.; CONLEE, R.K.; HOYT, G.L. & PARCELL, A.C. (2004). Neural, metabolic, and performance adaptations to four weeks of high intensity sprintinterval training in trained cyclists. *International Journal of Sports Medicine*, 25 (2): 92-98. 83. KLITGAARD, H.; MANTONI, M.; SCHIAFFINO, S.; AU-SONI, S.; GORZA, L.; LAURENT-WINTER, C.; SCHNOHR, P. & SALTIN, B. (1990). Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. *Acta Physiologica Scandinavica*, 140: 41–54

84. ARAMPATZIS, A.; DE MONTE, G.; KARAMANIDIS, K.; MOREY-KLAPSING, G.; STAFILIDIS, S. & BRUGGEMANN, G.P. (2006). Influence of the muscle-tendon unit's mechanical and morphological properties on running economy. *Journal of Experimental Biology*, 209: 3345–3357.

85. PIACENTINI, M.F.; DE IOANNON, G.; COMOTTO, S.; SPEDICATO, A.; VERNILLO, G. & LA TORRE, A. (2013). Concurrent strength and endurance training effects on running economy in master endurance runners. *Journal of Strength and Conditioning Research*, 27 (8): 2295-2303.

86. MAHARAM, L.G.; BAUMAN, P.A.; KALMAN, D.; SKOL-NIK, H. & PERLE, S.M. (1999). Masters athletes: factors affecting performance. *Sports Medicine*, 28 (4): 273-285.