TOPICAL REVIEW

Endurance exercise performance in Masters athletes: age-associated changes and underlying physiological mechanisms

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Older ('Masters') athletes strive to maintain or even improve upon the performance they achieved at younger ages, but declines in athletic performance are inevitable with ageing. In this review, we describe changes in peak endurance exercise performance with advancing age as well as physiological factors responsible for those changes. Peak endurance performance is maintained until ∼35 years of age, followed by modest decreases until 50–60 years of age, with progressively steeper declines thereafter. Among the three main physiological determinants of endurance exercise performance (i.e. maximal oxygen consumption ($\dot{V}_{O_2max}$), lactate threshold and exercise economy), a progressive reduction in $\dot{V}_{O_2max}$ appears to be the primary mechanism associated with declines in endurance performance with age. A reduction in lactate threshold, i.e. the exercise intensity at which blood lactate concentration increases significantly above baseline, also contributes to the reduction in endurance performance with ageing, although this may be secondary to decreases in $\dot{V}_{O_2max}$. In contrast, exercise economy (i.e. metabolic cost of sustained submaximal exercise) does not change with age in endurance-trained adults. Decreases in maximal stroke volume, heart rate and arterio-venous O_2 difference all appear to contribute to the age-related reductions in $\dot{V}_{O_2max}$ in endurance-trained athletes. Declines in endurance exercise performance and its physiological determinants with ageing appear to be mediated in large part by a reduction in the intensity (velocity) and volume of the exercise that can be performed during training sessions. Given their impressive peak performance capability and physiological function capacity, Masters athletes remain a fascinating model of 'exceptionally successful ageing' and therefore are highly deserving of our continued scientific attention as physiologists.

The demographics of age are changing dramatically. According to current population forecasts, the number of elderly will increase worldwide from 6.9% of the population in 2000 to a projected 19.3% by 2050. In parallel with this overall increase in older adults, the number of middle-aged and older ('Masters') athletes is expected to increase as well. This is an interesting sub-demographic of adults because many individuals in this group express a highly unique physiological phenotype that could be termed 'exceptionally successful ageing'. Masters athletes strive to maintain and, in some cases, improve upon the performance they have achieved at younger ages. Indeed, the peak exercise performance of Masters athletes continues to increase each year. However, declines in athletic performance are inevitable and the underlying reasons are important to understand. This raises a number of intriguing questions, including: How does peak exercise performance change with advancing age? What physiological mechanisms and modulating factors are responsible for the age-related decline in exercise performance? Why do these mechanisms and factors change with ageing? The aim of the present review is to address these issues in the context of endurance exercise performance, for which a relatively large body of empirical evidence is available.

Table 1. Comparison of 1896 Olympic winning times in running events and current Masters records that surpass those winning times (from ESPN and World Masters Records)

| Running events	1896 Olympic winning time (from the first Olympic games in Athens)	Current age-group records that surpass 1896 Olympic times and age at which these records were achieved
100 m (s)	12.0	11.7 (61 years)
200 m (s)	22.2	22.1 (46 years)
400 m (s)	54.2	53.9 (63 years)
800 m (min:s)	2:11.0	2:10.4 (60 years)
1500 m (min:s)	4:33:2	4:27:7 (60 years)
Marathon (h:min:s)	2:58:50	2:54:5 (73 years)

Endurance exercise performance with ageing

Peak athletic performance has improved dramatically in the past 100 years, although the age at which peak performance is achieved in Olympic track and field (athletics), swimming, baseball, tennis and golf has remained constant over this period (Schulz & Curnow, 1988). However, as the number of older adults participating in competitive events has increased (at a much greater rate than young adults), and training and nutritional practices have evolved, Masters athletes have achieved impressive improvements in peak exercise performance (Ericsson, 1993). For example, in 2005 Kozo Haraguchi of Japan set a new age-group record in the 100 m dash of 21.69 s at the age of 95. In 2003, Ed Whitlock of Canada became the oldest person to break 3 h in marathon at the age of 73. In some athletic events (e.g. marathon running), Masters athletes over 70 years of age have surpassed the winning time at the first Olympic games held in Athens (Table 1). These exceptional individual athletic achievements are fascinating not only to the general public, but also to those of us who study the effects of ageing

on physiological functional capacity. It also highlights the broad question of how endurance exercise performance changes with age in healthy adults.

As illustrated in Fig. 1, endurance performance in running events decreases with age in a curvilinear fashion. In general, peak endurance running performance is maintained until ∼35 years of age, followed by modest decreases until 50–60 years of age, with progressively steeper reductions thereafter (Tanaka & Seals, 2003). The pattern appears to be similar for both non-elite and elite endurance athletes (Joyner, 1993). In general, the magnitude of decline in endurance running performance with age is greater in women than in men (Joyner, 1993; Tanaka & Seals, 1997; Donato *et al.* 2003) (Fig. 1). However, interpretation of this apparent widening of sex differences with advancing age is confounded by the relatively smaller number of female *versus* male runners in the older groups. Indeed, such increasing sex differences with age are absent in the endurance swimming events, where approximately equal number of men and women compete throughout the age range (Donato *et al.* 2003) (Fig. 2).

The overall reduction in peak exercise performance with age tends to be greater in endurance than in 'sprint' events (Tanaka & Seals, 1997, 2003) (Fig. 2). It is not known why or how the duration of the athletic event influences the age-associated decline in peak performance. One possibility is that endurance and sprint events rely on different energy-producing pathways to sustain muscle activity. For example, findings from several cross-sectional studies indicate that the declines in maximal aerobic exercise capacity with age are considerably greater than those observed in 'anaerobic' muscle power (Larsson *et al.* 1979; Grimby & Saltin, 1983; Tanaka & Seals, 1997). This appears contradictory to the well-established preferential loss of type II muscle fibres (one of the determinants of anaerobic muscle power) with advancing age in healthy non-physically conditioned adults (Larsson *et al.* 1979; Grimby & Saltin, 1983; Tanaka & Seals, 1997). However, these fibre type changes are relatively small or non-existent in trained athletes (Coggan *et al.* 1990; Trappe *et al.* 1995).
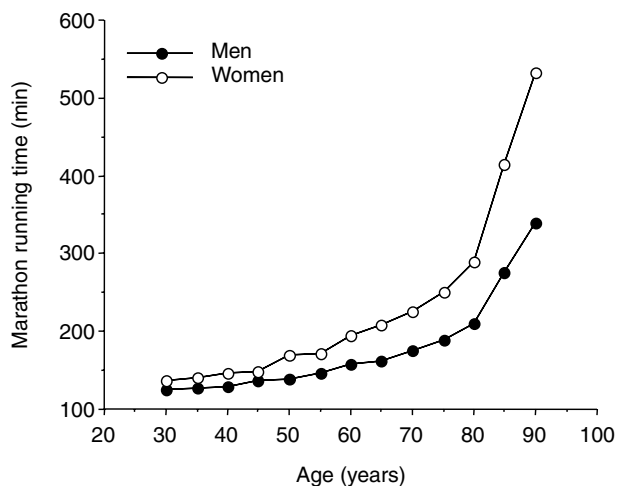
Figure 1

Changes in men's and women's marathon running times with advancing age (from USA Track & Field and World Masters Athletics).

Physiological determinants of endurance exercise performance

Based largely on studies in young endurance-trained athletes, the three main physiological determinants of endurance performance are believed to be maximal oxygen consumption, exercise economy and the exercise intensity at which a high fraction of the maximal oxygen consumption can be sustained, as typically defined by the 'lactate threshold' (Hagberg & Coyle, 1983; Joyner, 1993). In the following section, we review the available information on how changes in these determinants may contribute to age-related declines in endurance exercise performance (Fig. 3).
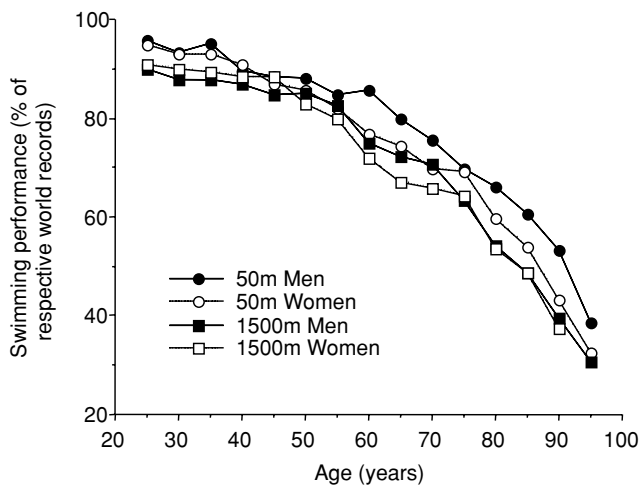

Figure 2
Men's and women's swimming performance with advancing age (from Federation Internationale de Natation).

(a) Exercise economy. Exercise economy is measured as the steady-state oxygen consumption while exercising at a specific submaximal exercise intensity below the lactate threshold (see below). Among endurance athletes, exercise economy is an important determinant of endurance performance (Morgan *et al.* 1989), particularly in groups that are more homogeneous than heterogeneous in $\dot{V}_{O_2max}$ (Morgan *et al.* 1989). Starting with the findings of the dissertation study of Professor Sid Robinson published in 1938 (Robinson, 1938), cross-sectional observations made by numerous independent laboratories indicate that exercise economy does not change with advancing age (Astrand, 1960; Allen *et al.* 1985; Wells *et al.* 1992; Evans *et al.* 1995). For example, in a cross-sectional study of male endurance runners, there was no difference in running economy between young and older athletes (Allen *et al.* 1985). Similarly, no significant association was found between running economy and age among highly trained and competitive female runners aged 35–70 years (Wells *et al.* 1992). We found that in healthy

female athletes, running economy explained little of the variance in age-related decreases in endurance running performance after accounting for differences in $\dot{V}_{O_2max}$ and lactate threshold, which explained 85% of the variability in 10K performance (Evans *et al.* 1995). The results of several longitudinal studies have confirmed that running economy does not change with age in endurance-trained Masters athletes (Robinson *et al.* 1976; Trappe *et al.* 1996). Thus, available data in this area indicate that reductions in exercise economy do not contribute significantly to the decreases in endurance exercise performance observed with advancing age.

There are a number of physiological factors that determine exercise economy. Among them, the percentage of type I muscle fibres is positively associated with exercise economy in cyclists (Horowitz *et al.* 1994). In this context, well-trained Masters athletes have a similar muscle fibre distribution to performance-matched younger runners (Coggan *et al.* 1990). Consistent with this, a 20 year longitudinal study showed that with maintenance of strenuous endurance training, muscle fibre type distribution did not change with age in highly trained Masters athletes (Trappe *et al.* 1995). Therefore, maintenance of muscle fibre type with ageing could contribute to the preserved exercise economy of Masters athletes.

(b) Lactate threshold. The ability to sustain a high fraction of one's maximal oxygen consumption during submaximal exercise typically is evaluated using the blood lactate threshold. Numerous criteria, techniques and nomenclature for the lactate threshold have been
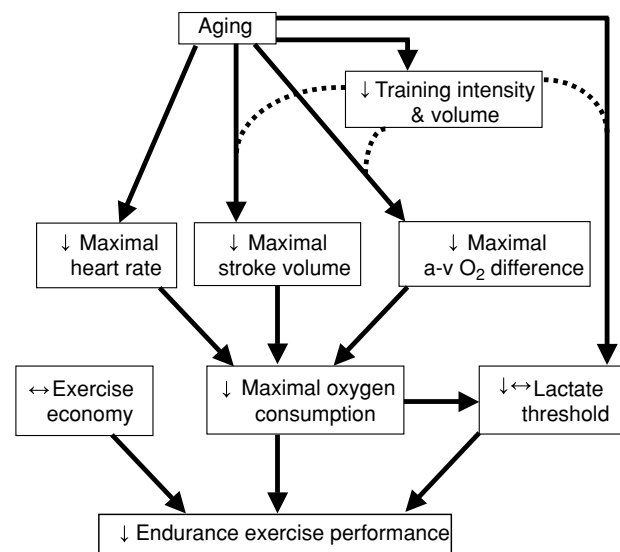

Figure 3
Factors and physiological mechanisms contributing to reductions in endurance exercise performance with advancing age in healthy adults.

used in the past. It is generally defined as the exercise intensity at which blood lactate concentration increases significantly above baseline (Allen *et al.* 1985; Coyle, 1995). In young endurance-trained adults, the lactate threshold predicts exercise performance in distance events ranging from 2 miles to the marathon (Hagberg & Coyle, 1983; Allen *et al.* 1985; Joyner, 1993), whereas power output at the lactate threshold is the best laboratory predictor of time-trial performance among competitive female Masters cyclists (Nichols *et al.* 1997). Absolute work rate or running speed at lactate threshold declines with advancing age in endurance athletes (Iwaoka *et al.* 1988; Maffulli *et al.* 1994; Evans *et al.* 1995; Wiswell *et al.* 2000). However, lactate threshold does not appear to change with increasing age when expressed relative to the percentage of $\dot{V}_{O_2max}$ (Iwaoka *et al.* 1988; Maffulli *et al.* 1994; Evans *et al.* 1995). The latter finding suggests that the contribution of decreases in lactate threshold to reductions in endurance exercise performance with ageing may be secondary to decreases in $\dot{V}_{O_2max}$. Indeed, a recent longitudinal study of 51 male and 23 female Masters runners reported that the change in lactate threshold over a mean follow-up period of 6 years was not predictive of a corresponding change in running performance when it was expressed as a percent of $\dot{V}_{O_2max}$ (Marcell *et al.* 2003).

(c) Maximal aerobic capacity. Maximal oxygen consumption establishes the upper limit of maximal energy production through oxidative phosphorylation and is generally considered to be a primary determinant of endurance exercise performance among young endurance-trained athletes (Joyner, 1993; Coyle, 1995). $\dot{V}_{O_2max}$ declines approximately 10% per decade after age
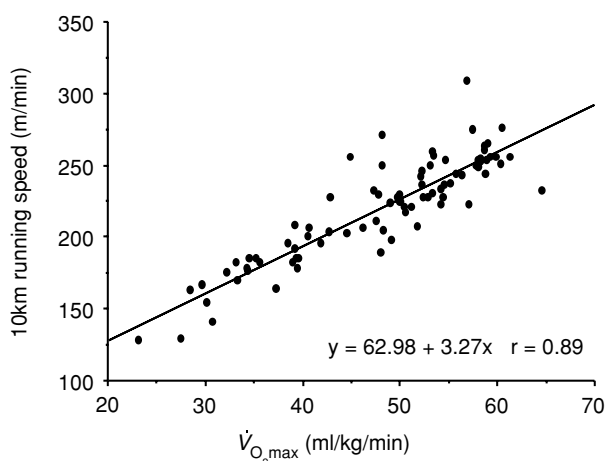

Figure 4
Relation between 10 km running speed and maximal oxygen consumption ($\dot{V}_{O_2max}$) among competitive female distance runners varying widely in age (H. Tanaka and D. R. Seals, unpublished observations).

25–30 years in healthy sedentary adults of both sexes (Heath *et al.* 1981; Buskirk & Hodgson, 1987; FitzGerald *et al.* 1997; Tanaka *et al.* 1997; Eskurza *et al.* 2002; Pimentel *et al.* 2003). Early investigations suggested that the rate of decline in $\dot{V}_{O_2max}$ with advancing age was as much as 50% smaller in endurance exercise-trained athletes than in sedentary adults (Heath *et al.* 1981; Kasch *et al.* 1990). However, we and others subsequently established that when expressed as per cent decrease from early adulthood, the rate of decline in $\dot{V}_{O_2max}$ with age is not reduced in healthy adults who habitually perform aerobic exercise (Hodgson & Buskirk, 1977; FitzGerald *et al.* 1997; Tanaka *et al.* 1997; Wilson & Tanaka, 2000; Eskurza *et al.* 2002; Pimentel *et al.* 2003; Fleg *et al.* 2005). In fact, endurance exercise-trained men and women demonstrate greater 'absolute' (ml kg^{-1} min^{-1}) rates of decline in $\dot{V}_{O_2max}$ with age than healthy sedentary adults (FitzGerald *et al.* 1997; Tanaka *et al.* 1997; Eskurza *et al.* 2002; Pimentel *et al.* 2003), probably as a result of greater baseline levels of $\dot{V}_{O_2max}$ as young adults and greater reductions in habitual exercise with ageing compared with sedentary adults (FitzGerald *et al.* 1997; Eskurza *et al.* 2002). It is interesting to note that the greater absolute rate of decline in $\dot{V}_{O_2max}$ with age in endurance-trained *vs* sedentary subjects also been observed in rats (Zimmerman *et al.* 1993) and has been reviewed in detail elsewhere (Tanaka & Seals, 2003).

Endurance performance and $\dot{V}_{O_2max}$ are strongly and positively related in groups of highly trained and competitive distance runners varying in age (Fig. 4). Moreover, reductions in endurance exercise performance with age are closely associated with corresponding decreases in $\dot{V}_{O_2max}$ (Fuchi *et al.* 1989; Marcell *et al.* 2003). Indeed, $\dot{V}_{O_2max}$ was the best predictor of age-related changes in endurance exercise performance in 51 male and 23 female Masters runners who were followed longitudinally for a period of 6 years (Marcell *et al.* 2003). Thus, a progressive reduction in $\dot{V}_{O_2max}$ appears to be a key physiological mechanism associated with declines in endurance performance with advancing age.

In spite of the strong association between $\dot{V}_{O_2max}$ and endurance exercise performance, other factors may contribute to age-associated reductions in endurance performance. Although the comparison is not straightforward because of different scaling and units, the fact that the rate of decline in endurance performance with age appears to be smaller than the corresponding fall in $\dot{V}_{O_2max}$ (Joyner, 1993) is consistent with this idea. It may be that other putative determinants of endurance performance (e.g. 'anaerobic' muscular power, muscle capillary density) may decline to a lesser extent with advancing age (Grimby & Saltin, 1983; Coggan *et al.* 1992; Coyle, 1995), thus offsetting the effects of the decrease in $\dot{V}_{O_2max}$. Furthermore, reductions in endurance performance with age are typically curvilinear, whereas the decrease in $\dot{V}_{O_2max}$ typically is presented

as a linear function of age. On the other hand, recent evidence indicates that the rate of decline in $\dot{V}_{O_2max}$ over the entire adult age range actually may be curvilinear, rather than linear, with the rate accelerating with advancing age (Fleg *et al.* 2005). This concept, originally described by Buskirk & Hodgson in 1987 (Buskirk & Hodgson, 1987), would be more consistent with the notion that a decrease in maximal oxygen consumption is the primary mechanism causing age-related reductions in endurance exercise performance.

Declines in the exercise training 'stimulus' with advancing age

The factors that contribute to reductions in $\dot{V}_{O_2max}$ with age in Masters endurance athletes are incompletely understood. Available evidence points to the seemingly inevitable consequence of an overall reduction in the exercise training 'stimulus' (i.e. exercise-training intensity, session duration and weekly frequency) with advancing age (Pollock *et al.* 1997; Tanaka *et al.* 1997; McGuire *et al.* 2001; Eskurza *et al.* 2002) (Fig. 5). As early as 1967, Dill *et al.* suggested that highly trained distance runners who become sedentary exhibit a greater than normal decrease in maximal aerobic capacity with advancing age (Dill *et al.* 1967). Similarly, the results of longitudinal studies suggest that $\dot{V}_{O_2max}$ can be fairly well maintained over phases of middle-age lasting up to 10 years in men and women who continue to train vigorously (Kasch & Wallace, 1976; Pollock *et al.* 1987). However, there is no evidence that exercise training intensity and volume (and $\dot{V}_{O_2max}$) can be maintained for longer periods, especially at older ages (Dill *et al.* 1967; Pollock *et al.* 1997). Increases in job- and family-related responsibilities may impinge on the availability of time and energy for the intensive training required to remain competitive. Increased prevalence of exercise training-associated injuries among Masters athletes also probably contributes to their reduced training intensity and volume (Kallinen & Markku, 1995). Moreover, the motivation to train may be reduced with advancing age among Masters athletes similar to the declines in compliance observed in older patients participating in cardiac rehabilitation programs (Cooper *et al.* 2002). The goals underlying the motivation to train also may shift somewhat from achieving personal records in younger athletes to health benefits in older athletes (Ogles & Masters, 2000); the latter would probably accommodate reductions in exercise intensity with age. The 'intrinsic drive' to exercise or be physically active may decline with ageing as well, as rodents given life-long access to running wheels demonstrate marked reductions in noctural running behaviour with advancing age (Valentinuzzi *et al.* 1997). In summary, it appears that at least in healthy adults, the ability to maintain the overall

exercise-training stimulus contributes to the rate of decline in $\dot{V}_{O_2max}$ and therefore endurance exercise performance with advancing age.

Physiological mechanisms responsible for age-related declines in $\dot{V}_{O_2max}$ in the endurance-trained athletes

$\dot{V}_{O_2max}$ has an exact physiological definition that is expressed by the Fick equation: maximal cardiac output × maximal arterio-venous O_2 difference. Within this physiological context, there is some controversy regarding the mechanisms responsible for the reduction in $\dot{V}_{O_2max}$ with age in endurance athletes. In particular, the exact contribution of central (i.e. cardiac) and peripheral (i.e. oxygen extraction) factors to the reduced $\dot{V}_{O_2max}$ in Masters compared with young adult endurance athletes is unclear. However, it appears that decreases in both maximal cardiac output and maximal arterio-venous O_2 difference may play a role (Table 2 and Fig. 3). Attempts to determine the effects of ageing on the Fick determinants of $\dot{V}_{O_2max}$ in endurance athletes have relied almost exclusively on cross-sectional studies comparing young and Masters
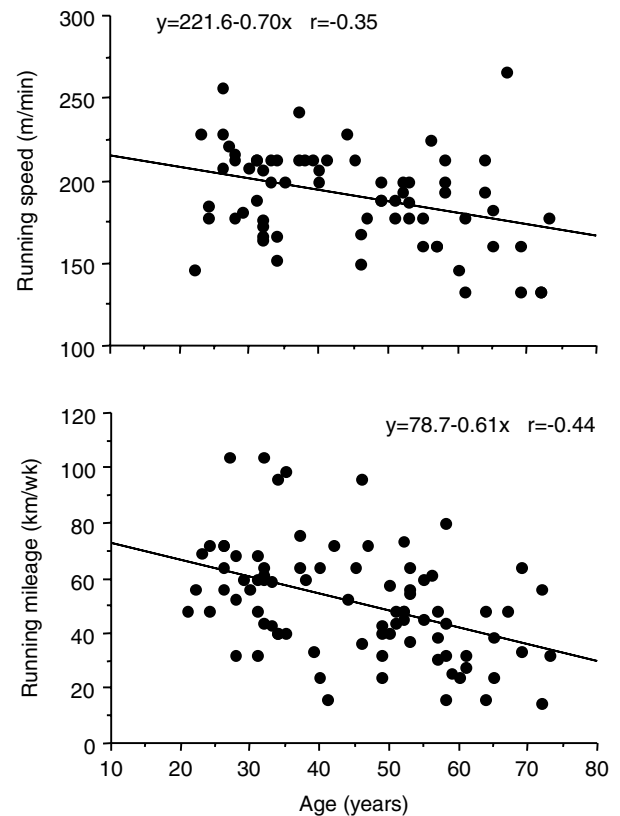
Figure 5

Progressive declines in running (training) mileage and speed with age in female endurance-trained runners. (Modified from Tanaka *et al.* (1997); used with permission).

Table 2. Oxygen consumption and its determinants at maximal exercise in endurance exercise-trained men

	Young men (28 years)	Older men (60 years)	Age-related change (%)
Oxygen consumption (ml kg^{-1} min^{-1})	68.2	49.4	28
Cardiac output (l min^{-1})	27.0	21.7	20
Stroke volume (ml beat^{-1})	147	132	10
Heart rate (beats min^{-1})	184	165	10
a–v O$_2$ difference (ml (100 ml)$^{-1}$)	16.7	15.2	8

The data were compiled from four studies in which values for all of the variables were reported in groups of young and older groups (Grimby *et al.* 1966; Hagberg *et al.* 1985; Rivera *et al.* 1989; Ogawa *et al.* 1992).

endurance-trained athletes. Longitudinal studies clearly are needed to fully address this question.

(a) Central factors. *Maximal cardiac output.* Although it has been reported that maximal cardiac output is maintained with advancing age (Rodeheffer *et al.* 1984), the majority of the evidence supports the idea that maximal cardiac output decreases with advancing age in healthy sedentary adults (Julius *et al.* 1967; Saltin, 1986; Rivera *et al.* 1989; Ogawa *et al.* 1992; Hunt *et al.* 1998) in proportion to the decline in maximal oxygen consumption (Grimby *et al.* 1966; Proctor *et al.* 1998). Maximal cardiac output also is reduced in older Masters (60–70 years) compared with young (20–30 years) endurance-trained athletes as a result of reductions in both maximal stroke volume and maximal heart rate (Rivera *et al.* 1989; Ogawa *et al.* 1992) (Table 2).

Maximal heart rate. Historically, maximal heart rate has been viewed as the primary mechanism mediating age-related reductions in maximal cardiac output and $\dot{V}_{O_2max}$, particularly in endurance exercise-trained athletes (Heath *et al.* 1981; Hagberg *et al.* 1985). Starting from early adulthood, maximal heart rate declines with age at a rate of ~0.7 beats min^{-1} year^{-1} in healthy sedentary, recreationally active and endurance exercise-trained adults (Tanaka *et al.* 2001). A slower conduction velocity, a reduced responsiveness of the sinoatrial node to β-adrenergic stimulation (Fleg *et al.* 1994) and a decreased intrinsic heart rate (Jose & Collison, 1970) are among the mechanisms believed to contribute to the reduction in maximal heart rate with ageing.

Maximal stroke volume. In older endurance exercise-trained adults, maximal stroke volume is reduced modestly to 80–90% of that observed in young endurance-trained adults (Ogawa *et al.* 1992) (Table 2). There is very limited information as to how changes in the major determinants of stroke volume (e.g. preload, afterload and intrinsic contractility of the heart) contribute to

the age-related reduction in maximal stroke volume in endurance-trained adults. It is unclear if a reduction in left ventricular filling is involved. Results of studies indicating that left ventricular preload, as expressed as left ventricular end-diastolic dimension, area or volume, is not related to age in healthy relatively active adults (Fleg, 1986) do not support such a role. In young adults, total blood volume exerts an important influence on maximal stroke volume and maximal oxygen consumption (Convertino, 1991). However, total blood volume appears to be preserved in older endurance-trained athletes (Jones *et al.* 1997). We cannot exclude the possibility that other determinants of the cardiac preload, including LV end-diastolic pressure, diastolic filling time, venomotor tone, myocardial compliance and/or a combination of these factors, contribute to the age-related decline in maximal stroke volume in endurance exercise-trained adults (Schulman *et al.* 1992; Arbab-Zadeh *et al.* 2004).

The large elastic arteries stiffen with advancing age and lead to an increase in aortic input impedance as well as vascular afterload, thereby impeding the ejection of blood from the left ventricle during systole and, consequently, stroke volume during exercise (Chen *et al.* 1999). Although the degree of arterial stiffening with advancing age is attenuated in endurance-trained compared with sedentary adults, endurance athletes nevertheless demonstrate large elastic artery stiffening with age (Tanaka *et al.* 2000). This could contribute to the reduction in maximal stroke volume seen in older endurance-trained adults via increases in the left ventricular afterload and aortic input impedance (Mazzaro *et al.* 2005). On the other hand, no differences in left ventricular afterload, as indirectly assessed by mean arterial pressure or total peripheral resistance, have been reported in young and older endurance-trained men (Rivera *et al.* 1989).

It is, especially difficult to evaluate the contractility of the left ventricle in intact human subjects during exercise because of the complex and concurrent actions of multiple influencing factors. Moreover, there is no satisfactory index of contractility that is completely independent of left

ventricular preload and afterload (Fleg, 1986; Giada *et al.* 1998). Animal studies using the isolated perfused heart preparation find that contractility declines significantly with advancing age in endurance-trained rats and that the magnitude and the rate of the decline is similar to sedentary rats (Starnes & Rumsey, 1988). The similarly lower ejection fractions at maximal exercise observed in both older sedentary and older endurance-trained athletes compared with young men are consistent with these observations in experimental animals (Schulman *et al.* 1992).

(b) Peripheral factors. Maximal arterio-venous O_2 difference reflects the capacity of (primarily) active skeletal muscles and the respiratory muscles to extract and consume oxygen from the blood for ATP production during maximal exercise. In sedentary adults, maximal arterio-venous O_2 difference clearly declines with advancing age, consistent with the marked reductions in capillary density and mitochondrial enzyme activities observed with ageing in this group (Coggan *et al.* 1992). Reductions in peripheral oxygen extraction during maximal exercise also appears to contribute to the decline in $\dot{V}_{O_2max}$ with age in endurance exercise-trained adults (Table 2), as maximal arterio-venous O_2 difference declines modestly (5–10%) over a span of ~30 years in this group (Hagberg *et al.* 1985; Saltin, 1986; Rivera *et al.* 1989). It remains to be determined if the reduction in maximal arterio-venous O_2 difference with ageing in endurance athletes 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, and their contracting muscles are capable of extracting oxygen as much as their younger counterparts (Saltin, 1986). Furthermore, muscle oxidative enzyme activities and capillarization (expressed per area or per fibre) are similar between young and older endurance-trained adults (Coggan *et al.* 1992; Proctor *et al.* 1995). 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 O_2 difference in endurance-trained adults.

As skeletal muscle mass is closely related to maximal aerobic capacity among healthy humans across the adult age range (Fleg & Lakatta, 1988), a decline in maximal arterio-venous O_2 difference may be secondary to an age-related loss of muscle mass. However, $\dot{V}_{O_2max}$ remains lower in older compared with young endurance-trained athletes after correcting for muscle mass (Proctor & Joyner, 1997). Rather, we find that among healthy men varying in age, fat-free mass exerts its permissive influence on $\dot{V}_{O_2max}$ via an effect on central circulatory function involving blood volume, stroke volume and cardiac output (Hunt *et al.* 1998).

Summary and conclusions

In summary, Masters endurance athletes are capable of remarkable athletic and physiological functional performance, thereby representing a uniquely positive example of 'exceptional ageing'. Nevertheless, endurance exercise performance decreases during middle-age and declines at an even more rapid rate in older age. The available data indicate that decreases in $\dot{V}_{O_2max}$ are the most clear and consistent contributor to these declines in performance. Reductions in the lactate threshold also may contribute, whereas submaximal exercise economy is preserved with ageing in endurance athletes. The age-associated decreases in $\dot{V}_{O_2max}$ in endurance exercise-trained adults are mediated by reductions in maximal cardiac output and maximal arterio-venous O_2 difference, with reductions in both maximal stroke volume and heart rate contributing to the former. The decreases in endurance exercise performance and $\dot{V}_{O_2max}$ with ageing in endurance exercise-trained athletes are associated most closely with reductions in exercise training intensity and volume, probably as a consequence of changes in a number of physical and behavioural factors (e.g. increased prevalence of injuries, and reductions in energy, time and motivation to train). The 'Masters athlete model' continues to be a rich source of insight into our ability (or lack thereof) to maintain peak physical performance and physiological function with human ageing.

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