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ACADEMIC LITERATURE REVIEW

Anaerobic performance in masters athletes

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Abstract With increasing age, it appears that masters athletes competing in anaerobic events (10–100 s) decline linearly in performance until 70 years of age, after which the rate of decline appears to accelerate. This decline in performance appears strongly related to a decreased anaerobic work capacity, which has been observed in both sedentary and well-trained older individuals. Previously, a number of factors have been suggested to influence anaerobic work capacity including gender, muscle mass, muscle fiber type, muscle fiber size, muscle architecture and strength, substrate availability, efficiency of metabolic pathways, accumulation of reaction products, aerobic energy contribution, heredity, and physical training. The effects of sedentary aging on these factors have been widely discussed within literature. Less data are available on the changes in these factors in masters athletes who have continued to train at high intensities with the aim of participating in competition. The available research has reported that these masters athletes still demonstrate age-related changes in these factors. Specifically, it appears that morphological (decreased muscle mass, type II muscle fiber atrophy), muscle contractile property (decreased rate of force development), and biochemical changes (changes in enzyme activity, decreased lactate production) may explain the decreased anaerobic performance in masters athletes. However, the reduction in anaerobic work capacity and subsequent performance may largely be the result of physiological changes that are an inevitable result of the aging process, although their effects may be minimized by continuing specific high-intensity resistance or sprint training.

Keywords Anaerobic · Masters athletes · Aging

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Introduction

During recent decades, there has been an increase in the number of older individuals engaging in regular physical activity and exercise for the health benefits of decreasing both morbidity and all-cause mortality [118, 143]. An increasing proportion of these active older individuals are becoming recreational or competitive athletes focused on sports performance. For example, the inaugural World Masters Games held in Toronto, Canada had 8,305 participants across 22 sports; the 2002 Melbourne, Australia World Masters Games 24,886 participants across 26 sports, and the upcoming 2009 World Masters Games being held in Sydney, Australia anticipating the biggest mass participation (30,000), multi-sport (28 sports), multi-national, festival in the world (Holm, personal communication, 2008). Each sport's national or international governing body determines the age to define a masters athlete. Internationally, masters' competition in swimming begins at age 25 years, track and field at 35 years, and golf at 50 years. In general, masters athletes are over the age of 40 years and may be either highly conditioned and experienced competitive athletes who have completed their formal competitive careers, the "weekend warriors" who sporadically train and compete, or older competitors

who have resumed physical training after long periods of physical inactivity [104]. For the purposes of this paper, masters athletes are individuals who systematically train for, and compete in, organized forms of competitive sport specifically designed for older adults [127].

While the factors contributing to the age-related reduction in endurance performance have been widely examined in the masters athlete [127, 145, 146], there is a paucity of literature examining the age-related decline in anaerobic performance observed in masters athletes or the factors that contribute to this decline in aging athletes. To our knowledge, the current review is the first to systematically examine anaerobic performance in masters athletes.

For the purposes of this paper, anaerobic performance is defined as the work capacity or sports performance during maximal exercise lasting from about 10 s (e.g., 25 m sprint swim, 100 m sprint run) to approximately 100 s (e.g., 100 m sprint swim, 400 m sprint run). Anaerobic energy production involves the replenishment of adenosine triphosphate (ATP) from both the creatine kinase and adenylate kinase pathways (ATP-PCr system), as well as the glycogenolysis and glycolysis pathways (lactic system). In order to systematically inform the current review, the determinants of anaerobic performance proposed by Bouchard, Taylor, Simoneau, and Dulac [23] will be examined in turn. Specifically, these investigators proposed that anaerobic performance is dependent upon a number of factors including:

1. Gender
2. Muscle mass
3. Muscle fiber type
4. Muscle fiber size
5. Muscle architecture and strength
6. Substrate availability
7. Efficiency of metabolic pathways
8. Accumulation of reaction products
9. Aerobic energy system contribution
10. Heredity
11. Physical Training

Declines in anaerobic work performance

Previous research on nonathletic aging individuals has demonstrated age-related declines in anaerobic work performance using cycle ergometry [102, 103, 105]. Taken together, these studies suggest an age-related decline of 6–8%·decade^{-1} in 30-s work capacity in healthy aging individuals.

Few studies have used cycle ergometry to examine age-related declines in anaerobic work performances in masters athletes using cycle ergometry [126]. While we observed age-related declines in both 10- and 30-s work performance in state–national level track and field athletes using air-braked cycle ergometry, the heterogenous nature of the subject pool (male/female, track/field, power/endurance) and small numbers of subjects per age group prevented statistically based conclusions to be drawn [126].

Numerous studies have conclusively shown a linear age-related decline in actual anaerobic sports performance between the ages of 35 and 65–70 years, followed by a quadratic decline in masters athletes from both track and field [11, 18, 48, 58, 88, 89, 113, 114, 142, 158] and swimming [14, 41, 48, 61, 68, 124, 144]. In support of these age-related declines in both sprint swimming and running performance, the current world and Australian national (25 m swim) records display similar patterns of decline with age (Figs. 1 and 2).

Donato and others [41] conducted a 12-year longitudinal study of high-performance US Masters swimmers and observed a significant linear 3–8%·decade^{-1} increase in 50 m swim time for both men and women until 70 years of age. Following this, the trend became quadratic as a group and greater in women (15–16%·decade^{-1}) than men (13–14%·decade^{-1}). Similarly, Korhonen and others [89] reported that the Records Committee of World Masters Athletics observed 100 m sprint running performance to slow by approximately 7%·decade^{-1} in men and 9%·decade^{-1} in women between the ages of 40 and 80 years. Within their biomechanical analysis of 70 finalists aged 35

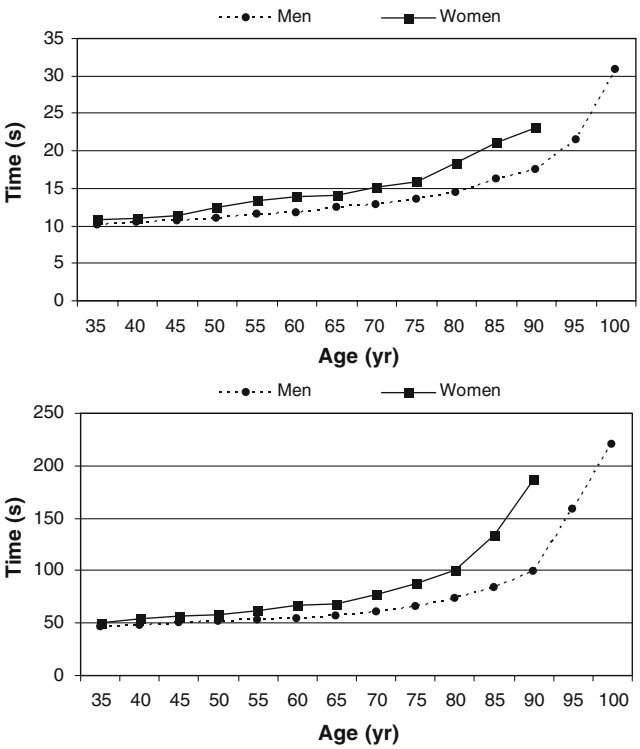

Fig. 1 World records for the 100-m (*top*) and 400-m (*bottom*) track running events in men and women with respect to age

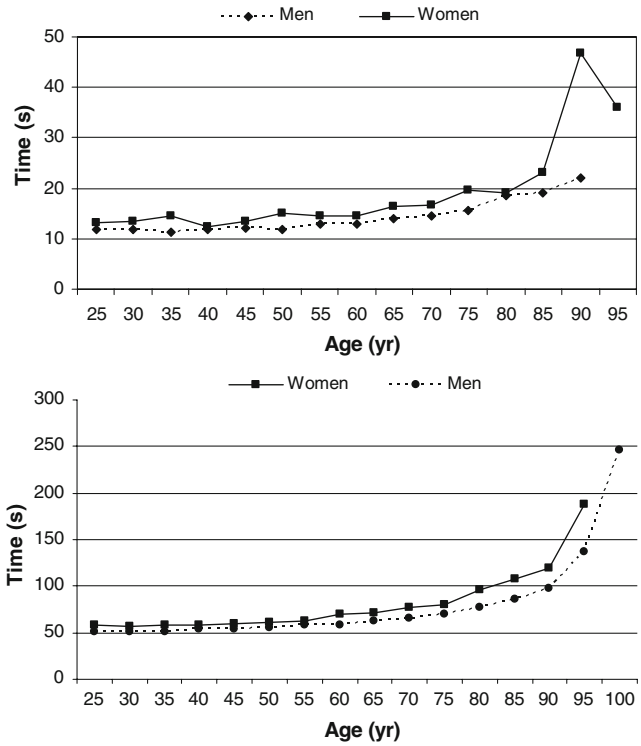

Fig. 2 Australian short-course records for the 25-m (*top*) and world records for the 100-m (*bottom*) freestyle swimming in men and women with respect to age

to 88 years, the same research team observed an exponential 5.8%·decade^{-1} (men) and 6.9%·decade^{-1} (women) decline in 100 m sprint run velocity in masters track athletes competing at the European Veterans Athletics Championships. Similar to findings from sprint swimming [14, 41, 48, 68] and running [11, 48], Korhonen el at. [89] reported that the effect of age was magnified after 65–70 years of age. The age-related decrease in both anaerobic work performance measured using cycle ergometry and sports performance in masters athletes may be due to changes in one or more of the factors that influence anaerobic performance.

Factors affecting anaerobic performance

In younger individuals, Bouchard, Taylor, Simoneau, and Dulac [23] suggested that anaerobic capacity may be influenced by a number of factors, which have since been supported by further research. These include gender [103, 137, 154, 155], total muscle mass [120], fiber type and cross-sectional area [12, 87], physical training [102], substrate availability [107], accumulation of reaction products [40], the oxygen uptake system [69], and heredity [19, 139]. At present, the mechanisms responsible for an age-related decline in anaerobic performance remain to be elucidated but may be due to age-related changes in any or all of the factors above.

Gender

In general, men appear to perform better than women in either accumulated oxygen deficit tests [106, 154, 155] or 10-, 30- and 90-s anaerobic tests [23, 120], with female total work outputs relative to body mass being approximately 25% lower than that of men. In laboratory-based anaerobic tests, older men also perform significantly better than similar-aged women [103, 137]. Similarly, the anaerobic sports performance of elite male masters athletes in both swimming (25 and 100 m) [41, 48, 144] and track and field (100 and 400 m) [11, 48, 89, 158] is consistently 10–20% better than that of elite women of the same age (Figs. 1 and 2) between the ages of 35 and 70 years. Interestingly, the actual rate of age-related decline in sprint performance is similar in both men and women from 35 to 70 years at 5–10%·decade^{-1} [89, 158]. Following this (>70 year), it appears that masters female sprint performances in both track and field [11, 48, 89, 158] and swimming [41, 48, 144] decline significantly faster than men, with the decline reaching 40%·decade^{-1} by 90 years of age.

The major mechanism explaining this gender difference in anaerobic performance appear to be primarily related to gender differences in muscle mass across all age groups but which accelerates in older women after 60 years of age [76, 77, 120]. In both untrained [12, 75, 154, 155] and trained [12, 120, 156] younger individuals, it appears that anaerobic capacity is higher in men when expressed relative to total body mass or to active muscle mass. For example, a recent study observed a 22% higher lean-mass normalized mean power output in young men compared to young female physical education students who undertook a 30-s Wingate test [120]. This would suggest that, apart from a lower active muscle mass, other gender-related factors explain the observed differences in anaerobic performance.

In younger individuals, it has been observed that, relative to similar-aged men, women display:

- Lower peak blood and muscle lactate following anaerobic performance [62, 154]
- Decreased anaerobic enzyme activity [27, 78]
- Decreased type II fiber size [78, 110]
- Decreased muscular strength [122]
- Decreased VO_2max [157]
- Decreased glycolytic contribution to anaerobic performance [71]
- Decreased peak muscle oxidative capacity [17]

The limited data available suggest that both men and women can both increase their anaerobic capacities signif-

icantly in response to anaerobic training [98, 155]. However, it appears that, at least in younger individuals, men may be able to concurrently develop their aerobic capacity to a greater extent than women when undertaking anaerobic interval training [155].

Few studies have examined gender differences in the factors that affect anaerobic performance of older individuals, particularly masters athletes. However, it appears that relative to similarly aged men, women exhibit:

- Similar rates of decline in muscle size and strength [159–161]
- Greater rates of type II fiber atrophy [32]
- Lower peak blood lactates following anaerobic performance [14, 90]
- Decreased estrogen levels that affect muscular performance [130]

Taken together, the age-related rate of decline in anaerobic performance is similar in both male and female athletes until approximately 70 years of age after which women's performance, possibly for sociological factors, declines at a faster rate. Apart from the well-documented differences (e.g., muscle mass, muscular strength, aerobic capacity) observed between the genders, it appears that both the greater rate of type II fiber atrophy and hormonal changes may explain the gender-related differences in both anaerobic performance and its rate of decline observed in older athletes.

Muscle mass

Muscle mass is highly related to both maximal 30-s cycle ergometry performance and 300 m sprint performance in both young men and women [120]. In an older healthy population, Makrides and others [103] observed that total work performed during a maximal 30-s cycle ergometry test in 100 older sedentary men and women was strongly related (r=0.84) to lean thigh volume estimated anthropometrically. Such data strongly suggest that active muscle mass, at least in older sedentary individuals, is related to maximal short-term anaerobic performance.

An age-related decline in active muscle mass is a consistent finding in aging nonathletic individuals [77, 99, 115], and both endurance-trained [67, 86, 126] and sprint-trained masters athletes [88]. Lexell, Taylor and Sjostrom [100], in their classic cross-sectional autopsy study, observed a modest 10% decrease in muscle size between 24 and 50 years of age that had accelerated to 30% between 50 and 80 years of age. Two recent 9- to 12-year longitudinal studies by Frontera and others [54, 57] used computerized tomography to demonstrate a significant age-related reduction in cross-sectional area of the total anterior muscle compartment of the thigh. These data strongly suggest that the loss of muscle mass with age despite continued training contributes to the decline in anaerobic performance in aging athletes.

In contrast, there exists some evidence to suggest that older strength-trained athletes may be able to maintain muscle mass with age [86, 119], while older endurance athletes appear not to be able to maintain muscle mass [153]. For example, Klitgaard et al. [86] found that elderly men (68±0.8 years) with 12–17 years of heavy resistance training three times a week had muscle fiber sizes similar to young adult (28±1.0 years) sedentary controls. In contrast, Trappe and others [153] observed that older distance runners (68.4±2.7 years), despite having maintained high levels of physical training for 20–25 years, possessed similar muscle cross-sectional areas to age-matched sedentary men. More recently, a group of Scandinavian researchers undertaking extensive cross-sectional, longitudinal, and intervention studies on elite masters athletes observed a general increase in muscle fiber areas and significant increase in type IIA fiber area in elite masters sprinters (66±3 years; 32±7 training years) who undertook a 20-week training program consisting of combined sprint training and heavy and explosive strength exercises [88]. While anaerobic performance was not directly measured using ergometry, significant increases were reported for strength, explosive jump performance, rate of force development, stride length, as well as 10 m and 60 m sprint run performance. These data support another recent Scandinavian study that compared types I, IIA, and IIX muscle fiber areas of elderly (68–78 years) endurance- and strength-trained athletes who were chronically trained for over 50 years [1]. Both muscle fiber size and mechanical muscle performance, especially rate of force development, were elevated in those individuals exposed to lifelong strength training. Taken together, the above data suggest that heavy resistance training combined with high power exercises may help prevent decreases muscle mass through type II fiber atrophy and in turn, losses in explosive muscle force production and decreased anaerobic performance in masters athletes.

Muscle fiber type

The age-related decrease in muscle mass may be due to a reduction in muscle fiber number, a reduction in fiber cross-sectional area, or both [49, 99]. Perhaps the least controversial finding in studies of aging human skeletal muscle is the loss of muscle fiber numbers with increasing age [99]. Direct evidence for loss of muscle fibers has been obtained by counting muscle fibers in autopsied whole *vastus lateralis* muscle in previously healthy individuals aged between 15 to 83 years [99]. These researchers noted a reduction in the total number of muscle fibers with no apparent effect for a particular fiber type. It has been

suggested that the aging process is accompanied by fiber type redistribution with the number of type I fibers increasing at the expense of type II fiber number. This is supported by needle biopsy studies that suggest the ratio of type II to type I fiber areas in the *vastus lateralis* decreases with age in healthy, nonathletic individuals, particularly during the seventh decade of life [5, 45, 94, 96, 97, 99]. More recently, Korhonen et al. [88] observed no age-related differences in fiber type distribution when comparing the types I, IIA, and IIB fiber composition of young (18–33 years) and older (53–77 years) national and international level sprint runners. These data agree with earlier findings from our laboratory where no significant difference in fiber composition was observed between chronically (>35 years) trained sprint- or endurance-trained masters runners and training volume and body mass-matched younger runners [126]. However, when Korhonen et al. [88] combined the fiber composition data with the corresponding fiber area to form the relative fiber type area, there was an age-related increase in type I fiber area and a trend towards a decrease in the area of type IIB fibers with age based on traditional myosin-ATPase histochemical staining techniques. Moreover, using gel electrophoretic techniques, the same study revealed an age-related increase in the single muscle fiber relative content of myosin heavy chain (MHC) I and a decrease in MHC IIx expression with age. Taken together, these finding suggest that highly trained masters sprint runners experience the normal age-related decrease in relative type II fiber area and a shift towards a slower MHC isoform profile that plays a major role in the rate of force development within muscles involved with anaerobic performance.

Muscle fiber size

In both young nonexercising and athletic individuals, a significant correlation is commonly observed between 30-s maximal exercise performance and both type II fiber composition and cross-sectional area. Thus, an age-related decrease in type II fiber area would be expected to negatively affect anaerobic performance in older individuals. In sedentary aging individuals, one of the most consistent findings is an age-related decrease in the size of type II muscle fibers [4, 26, 39, 100]. Lexell and others [100] observed no decrease in type I fiber size but reported a decrease in the type II muscle fiber cross-sectional area by 26% in individuals aged between 20 and 80 years. However, older athletes appear to slow the rate of this age-related decline in type II fiber area through engaging in high-intensity resistance and sprint training [1, 34, 88, 137].

It appears that there is preferential age-related atrophy among the different type II fibers in sedentary aging individuals [6, 32]. For example, Coggan and others [32] examined ten men and ten women (64±1 years) and ten men and ten women (24±1 years) and observed a greater atrophy in the type IIB (22% male, 30% female) than the type IIA (13% male, 24% female) fiber. Recent evidence from Korhonen and others [88] would suggest that, while aging sprint runners exhibit significant age-related declines in the cross-sectional area of the different type II muscle fibers, the declines were not different for type IIA (6.7%·decade^{-1}) or type IIB (11.3%·decade^{-1}).

In masters athletes, it appears that neither chronic endurance training [67, 86, 123] or sprint training [88] can prevent the age-related loss of muscle mass associated with a decrease in type II fiber size. In contrast, recent studies suggest that strength, explosive power and/or sprint training in masters athletes may prevent the commonly observed age-related decrease in type II fiber size in aging nonathletes [1, 34, 88]. Recent evidence from Korhonen and others [88] demonstrated that, while aging sprint runners exhibited significant declines in the cross-sectional area of type II muscle fibers, the declines were not different for type IIA (6.7%·decade^{-1}) or type IIB (11.3%·decade^{-1}). Taken together, these recent data suggest that high intensity sprint training combined with power and strength training into older age may help reduce the decrease in type II fiber size and thus possibly slow the age-related decline in anaerobic performance commonly observed in masters athletes. Interestingly, beyond 65–70 years of age, a dramatic reduction in fiber size appears evident in all fiber types but particularly type IIA and IIB [6, 32, 45, 94, 99]. While there exists little evidence of similar declines in masters athletes over 70 years of age, Korhonen and others [88] recently observed significantly larger type I fibers than type IIB fibers in 60-plus year-old sprint runners compared to younger runners 18–59 years of age. This finding may help explain the quadratic versus linear rates of decline in anaerobic sports performance in the older age groups (>70 years) within both swimming and track and field cohorts discussed earlier.

Thus, it would appear that the majority of available evidence suggests that the atrophy of skeletal muscle during aging in masters athletes appears to be caused by reduction in both the size and number of the skeletal muscle fibers. It also appears that the reduction in fiber size occurs mainly in the type II fibers. Taken together, these declines may contribute strongly to the age-related decline in anaerobic performance observed in masters athletes.

Muscle architecture and strength

In addition to muscle cross-sectional area, a muscle's geometry (angles and lengths of the muscle's fibers or fascicles) strongly influences force production (strength) and, in turn, anaerobic performance [16, 23, 24]. For

example, both *vastus lateralis* and *gastrocnemius* of highly trained young male sprinters (100 m in <11.0 s) contained longer fascicles attached at smaller angles than in less-performed sprinters (100 m in >11.0 s) [2]. In contrast, it appears that well-trained young endurance-trained runners have shorter fascicles and greater fascicle angles than high-performance sprint runners [2]. Changes in muscle architecture have been proposed to partially explain the decreases in muscular strength associated with aging [16, 149]. The limited data available suggests that normal aging is accompanied by both a decrease in both fascicle angle and length that appear to accompany the well observed decrease in muscle mass in sedentary aging individuals [16]. However, it appears that heavy and long-term weight training may attenuate this decline, at least in fascicle length [16]. A recent study investigated the contribution of muscle architecture to both isometric and isokinetic torque–velocity and power–velocity relationships between older (69–82 years) and younger (19–35 years) men [149]. Decreases in muscle cross-sectional area, fiber fascile length, together with a change towards slower MHC composition accounted for age-related decreases in both muscle strength and power in the older men. Therefore, together with changes in fiber number and area, changes in muscle architecture may also contribute to strength declines with age and thus anaerobic performance.

Muscular strength in both young athletes [8] and older strength-trained individuals [137] has been reported to be significantly correlated with performance in the Wingate 30-s anaerobic test, suggesting that muscular strength is important to anaerobic performance. Cross-sectional studies suggest muscular strength peaks at approximately 30 years of age and is well maintained to 50 years of age [96], declines gradually between 50 and 60 years, and rapidly declines thereafter [96]. This rapid decline in muscular strength of up to 2.5% per year after 60 years of age is strongly supported by a number of longitudinal studies on men aged over 60 years [6, 53, 56].

A similar observation has also been made by Alway and others [3] who observed a loss of muscle strength in older endurance-trained athletes relative to untrained younger men. Results from our laboratory also suggest a decrease in knee extensor isokinetic strength in aged sprint and endurance runners (>60 year) compared to younger (20–25 years) runners matched for body mass, training status, and training volume [126]. The age-related decrease in lower body muscular strength of older athletes may therefore contribute to the age-related declines in anaerobic performance observed in masters athletes.

Neural factors may also play a role in the age-related decline in both muscular strength and anaerobic performance in masters athletes. The specific tension (force per unit cross-sectional area) of whole muscle appears to decline with age in sedentary aging individuals [35, 66, 161] but is maintained in older sprint athletes [88]. However, in elite masters sprint runners, the rate of force development (RFD) has recently been shown to decrease significantly with age at approximately 9.7%·decade^{-1}, with the time taken to reach peak RFD almost doubling between the 18–33 years group and the >70 years group in the same cohort [88]. Crucially, both the RFD and time taken to reach peak RFD were significantly related to MHC II isoform content, which decreased significantly with age while MHC I isoform content significantly increased with age. Thus, it appears that in masters sprint runners there is a slowing of muscle contractile velocity due to age-related increases in muscle cells expressing the slow MHC isoform, a finding commonly observed in sedentary aging individuals [35, 91].

From these studies, age-related changes in muscle architecture, muscular strength, and neural activation may help to explain the observed decline in anaerobic performance with age, even in trained masters athletes.

Substrate availability

The availability of the substrates phosphocreatine (PCr) and glycogen has also been suggested to influence anaerobic performance, as they both significantly decrease during maximal exercise lasting between 10 and 30 s and beyond [23]. Intramuscular concentrations of PCr in the *vastus lateralis* have been shown to be 5% lower in older sedentary men and women (52–79 years) compared with younger adults (18–36 years) [111]. Furthermore, the same researchers reported similar trends in the total nucleotide pool, despite no such age effect in resting ATP or adenosine monophosphate concentrations. In support, McCully and others [108] reported a small but significant age-related decrease in the ratio of PCr to inorganic phosphate using magnetic resonance spectroscopy. Interestingly, Moller and Brandt [112] exercised seven 61- to 80-year-old, apparently healthy male subjects on a cycle ergometer twice weekly for 6 weeks. Following the training period, the ratio between PCr and total creatine increased, and a small but significant rise was observed for the ATP/adenosine diphosphate (ADP) ratio. The researchers concluded that age-related changes of intramuscular phosphagens in elderly subjects may in part be due to physical inactivity. While these data suggest that these changes in high energy substrates may also contribute to the age-related decrease in anaerobic performance observed in a sedentary aging population, no data have detailed the effects of age on the PCr availability in well-trained older athletes. Limited research has examined the effects of age on glycogen concentrations within the musculature of aging sedentary populations or masters athletes. However, glycogen, the

substrate of anaerobic glycolysis, has been shown to also significantly decline with age in sedentary populations [30, 109] with muscle glycogen levels being 61% higher in sedentary 24-year-old men compared to sedentary 65-year-old men [109]. Previous research has shown that glycogen content significantly correlated with the glycogen synthase protein content that in turn correlated inversely with age [133]. In summary, declines in the availability of the major substrates used in anaerobic performance may contribute to age-related declines in anaerobic performance observed in masters athletes. However, the possible contribution of the reduction in these substrates to the decline in anaerobic performance observed in masters athletes remains unknown.

Efficiency of metabolic pathways

The anaerobic energy systems refer to the breakdown of stored PCr and ATP, as well as the breakdown of glucose and glycogen to lactic acid via glycolysis without consuming oxygen [157]. These processes are reliant upon several enzyme-catalyzed reactions in order to occur, and thus any age-related changes in the activity of these enzymes may limit the rate at which energy can be produced anaerobically.

The enzyme creatine kinase (CK) catalyzes the reaction in which the phosphate group from PCr is transferred to ADP, with the formation of adenosine triphosphate in a reversible reaction. The amount of PCr stored in the muscle only permits maximal exercise of 5 to 10 s and is thus used in events such as the 100-m sprint run or 25-m sprint swim [136]. Single-muscle-fiber studies have demonstrated that fast-twitch fibers are favorable for rapid energy production during high-intensity sprint exercise. These fibers possess higher basal PCr content and consume more of their PCr stores during sprinting than slow-twitch fibers [63, 148]. Moreover, fast-twitch fibers are rich in key glycolytic enzymes and produce more lactic acid than slow-twitch fibers [47, 147].

CK has recently been shown to be 21% lower in the *obliquus internus abdominis* muscle of older (61–74 years, $n=11$) men compared to middle-aged (29–054 years, $n=13$) men [80]. This finding supports previous data that observed age-related decreases in CK in human skeletal muscle [141]. In contrast, a number of previous studies have observed no age-related change in CK activity in weight-bearing muscles such as *vastus lateralis* [17], *gastrocnemius* [32], or ankle dorsi flexors measured using phosphorous magnetic resonance spectroscopy [93]. Of interest in the Lanza and others [93] study is that both the rate of PCr hydrolysis during the first 6 s of a 60-s maximal isometric contraction of the ankle dorsi flexors and the overall and peak glycolytic rate were significantly faster in young (20–35 years) versus older (65–80 years) men. Within the same study, the older subjects were more reliant on oxidative ATP production more than young subjects and derived a smaller proportion of their ATP from anaerobic glycolysis during maximal voluntary isometric contractions [93].

In summary, the available data suggest that, in aging sedentary individuals, skeletal muscle CK and PCr concentrations and PCr hydrolysis rates are lower than those observed younger sedentary individuals. This might suggest that the capacity of the ATP-PCr system is compromised in masters athletes and that this may compromise anaerobic performance in this cohort. However, research has yet to examine this proposition.

The second energy system contributing to anaerobic performance is anaerobic glycolysis that leads to the formation of lactic acid and ATP in the cytosol and is regulated by the enzymes phosphofructokinase (PFK) and lactate dehydrogenase (LDH) [136]. Anaerobic glycolysis is the major energy system involved with events such as the 400-m sprint run or 100-m swim [157]. LDH exists in two main forms within skeletal muscle—heart-specific (H-LDH), which is preferentially involved in lactate oxidation to pyruvate and predominating in type I fibers, and muscle-specific (M-LDH), which mainly catalyzes the reduction of pyruvate to lactate and predominates in type II fibers [136].

It is well known that type I fibers exhibit lower activities of glycolytic enzymes and produce less lactic acid than type II fibers [47, 148]. It is also known that aging in adults is accompanied by a loss of muscle mass and a shift toward a more oxidative muscle profile mediated by the atrophy of the more anaerobic type II fibers [26, 39, 122]. Indeed, Korhonen and others [88] recently observed elite masters sprint runners (53–77 years) with similar fibre composition to young (18–33 years) sprinters to demonstrate an increase in their muscle area occupied by type I fibers and decrease in the area occupied by type II fibers. These changes in the muscle characteristics of masters sprint athletes would suggest both a reduced PCr metabolic capacity and a decreased rate of glycolysis and lactic acid formation in masters sprint athletes.. These changes in muscle characteristics would suggest both a reduced PCr metabolic capacity and a decreased rate of glycolysis and lactic acid formation. It is thus also possible that anaerobic energy production decreases with age due to reductions in key glycolytic enzymes, particularly PFK and phosphorylase which appear to be more highly concentrated in type II muscle fibers [73, 81]. The morphologic changes in type II fibers described above suggest the possibility that a decline in glycolytic enzyme activity with increasing age may contribute to the age-related decline in anaerobic performance observed in both aging sedentary individuals and masters athletes.

Data on age-related changes in the glycolytic pathway enzymes of aging sedentary individuals are equivocal [6, 17,

32, 33, 64, 115]. The cross-sectional study by Coggan and others [32] observed no differences in LDH, PFK, or phosphorylase activity in the *gastrocnemius* muscle of sedentary young (26±1 years) and older (64±1 years) individuals. In contrast, a 5-year longitudinal study by Aniansson and others [6] observed a significant decrease in total LDH activity of *vastus lateralis* of men from the age of from 76 to 80 years of age. However, many of the subjects within this study not only increased activity levels over the 5-year period but were already physically training and not a sedentary population. However, more recent research also suggests that LDH activity is markedly decreased in older adults, at least from studies that have examined total LDH activity in abdominal muscles [80, 117]. Kaczor et al. [80] compared the LDH activity in the *obliquus internus abdominis* muscle of middle-aged (29–54 years, $n=13$) and older (61–74 years, $n=11$) men and observed a highly significant ($p<0.0003$) almost two-fold decrease in LDH activity expressed per gram of wet weight or relative to total protein content in the older group. However, while Pastoris and others [117] support this finding of decreased LDH activity in abdominal muscles, the same research team found no changes LDH activity in *vastus lateralis* or *gluteus maximus* muscles of 76 sedentary subjects (32 men, 44 women) between 15 and 91 years of age. This may suggest that the rate of change in glycolytic enzymes is significantly greater in muscle groups that are not load-bearing or prime movers with increasing age. Similarly, Coggan and others [33] obtained gastrocnemius muscle biopsy samples from eight young endurance runners (26±3 years) and eight master athletes (63±6 years) matched for volume, pace, and type of training as well as 10-km running performance. While no difference was observed in fiber type distribution, LDH activity was significantly lower in the master athletes.

While total LDH activity does not appear to change with aging, there appears to be a shift in the relative proportion of the LDH isoenzymes. For example, Larsson [97] evaluated muscle biopsy tissue from 55 men (22–65 years), measuring total LDH and the components LDH-M and LDH-H. There was an age-related shift in the relative proportions of the two forms of the enzyme; LDH-M decreased significantly with LDH-H showing no significant change. An increase in relative activity of the LDH-H isoform may allow for greater oxidation of lactate, a function of type I muscle fiber, and may reflect a relative increase in type I fiber area with age [97] and/or an increase in the proportion of type I fiber number [97]. Thus, the majority of evidence supports maintenance of total LDH activity with a decrease in the M form and increase in the H form of LDH activity with aging in sedentary individuals.

In summary, the limited available data suggest that a decline in some glycolytic enzymes or changes in LDH isoform activity may contribute to the age-related decline in anaerobic performance observed in masters athletes.

Accumulation of reaction products

Anaerobic glycolysis is initiated at the beginning of maximal exercise when there is a need for greater reliance on ATP regeneration from both glycolysis and the phosphagen system than can be provided from mitochondrial sources [157]. During anaerobic exercise, there is an increase in both lactate and hydrogen ion (H^+) concentration, and thus a decrease in pH within both the muscle cell and the blood. Thus, proton release from both the breakdown of ATP and lactic acid causes the acidosis of intense exercise [131]. The decrease in muscle pH causes both an increased need for Ca^{++} for tension development, an inhibition of effective cross bridge cycling [40, 116], as well as an inhibition of enzymes involved with maximizing glycolytic flux [116], thus leading to a decrease in anaerobic performance.

Peak blood lactate concentration is widely used as an indirect measure of anaerobic glycolytic activity or capacity to discriminate between athletes of different glycolytic capacity and performance levels [92] and correlates with muscle lactate following maximal anaerobic performance [31, 72]. In older sedentary men aged 60–70 years, peak blood lactate has been shown to be lower than young man aged 20–30 years following a maximal 30-s cycle ergometer test [102, 105].

In masters athletes, there appears to be an age-related decrease in peak blood lactate following both maximal sprint running [90] and swimming [14]. We have previously compared peak blood lactate values in male masters swimmers (28–80 years) following maximal 100 m freestyle swimming and found no effect of age on peak blood lactate [129]. However, we only had a total of 16 state-level masters swimmers, four subjects per 10-year age group, and one swimmer above 70 years of age and collected our lactates in a controlled noncompetitive pool environment. More recent studies on large numbers of high-performance masters swimmers (52 men and 56 women aged 40–79 years) [14] and track sprinters (81 men and 75 women aged 35–88 years) [90] competing in international level competition have conclusively shown that peak blood lactate following maximal anaerobic exercise decreases with age. Both studies demonstrated that the peak blood lactate values observed were much higher than values reported for aged-matched sedentary men and women. Of interest is that in both male and female masters track sprinters [90] and male masters swimmers [14], the age-related decline in both performance and blood lactate values was most evident after 70 years of age. Taken together, these data lend support to the theory that

anaerobic glycolytic energy production declines with age, and this may be a contributing factor to the age-related decreases in anaerobic performance seen in many sports. Moreover, it appears that after 70 years of age, anaerobic glycolytic capacity is significantly reduced, secondary to decreases in muscle mass and type II fiber area, thus contributing to the curvilinear decrease in sports performance observed after that age.

It is well known that type II fibers exhibit higher activities of glycolytic enzymes and produce more lactic acid than type I fibers [46, 147] and that post-exercise lactate concentration depends on the total muscle mass engaged in the activity [79, 140]. Thus, the well-observed age-related loss of muscle mass and shift toward a more oxidative muscle profile mediated by the atrophy of fast-twitch fibers in both aging sedentary [26, 39, 122] and masters athletes [67, 84, 88, 123] may explain age-related decreases in blood lactate and as a result partly explain age-related decreases in anaerobic performance in masters athletes.

The H^+ ions from anaerobic glycolysis are partly buffered by the muscle buffer system including bicarbonate that is also present within the bloodstream [157]. Sprint training that stresses the anaerobic glycolytic system has been shown to increase buffering capacity and thus glycolytic capacity and sports performance in young athletes [44, 116, 134]. The limited data related to buffering capacity in older individuals suggest that there is a 6–7% increase in blood acidity and 12–16% reduction in blood bicarbonate concentration when comparing 80-year-old with 20-year-old sedentary individuals [51]. Within muscle, a recent study observed similar buffering capacity within young (20–35 years) and old (65–80 years) sedentary man following a 60-s maximal voluntary isometric contraction of the ankle dorsiflexor muscles, despite a lower pH and greater glycolytic rate in the younger men [93]. These data again suggest that older individuals rely more on oxidative pathways for ATP resynthesis during anaerobic performance, while younger persons rely more on the PCr and glycolytic pathways, possibly as a result of common finding of younger populations possessing a greater type II muscle fiber area compared to older individuals [32, 117], a common finding in both anaerobic [88, 126] and endurance [67, 85, 126] masters athletes.

In summary, the limited available data suggest that both peak blood lactate and blood buffering capacity are compromised with aging in sedentary aging individuals. In anaerobic masters athletes, it appears that there is an age-related reduction in blood lactate production, possibly related to an age-related reduction in the type II muscle fiber area.

Aerobic energy system contribution

Age-related differences in the response of the aerobic energy system to the initiation of maximal exercise may also play a significant role in the observed age-related decline in maximal short-term work performance. In highly trained young athletes, the aerobic energy system contributes an increasingly greater percentage of overall energy demand the longer the anaerobic performance [42, 43, 59, 69, 70, 138]. In young highly trained athletes, the aerobic contribution to 100 m sprint run events range between 3% and 25% of overall energy demand and for the 200 m event between 14% and 33% [42, 59]. For 400 m track running events that stress the anaerobic glycolytic system, research suggests the aerobic contribution ranges between 28% and 64% depending on the method of measuring the aerobic contribution (accumulated oxygen deficit, blood lactate, PCr degradation, direct measurement of oxygen uptake, or mathematical modeling) [43, 59]. During all-out cycling exercise (e.g., Wingate test), early studies using breath-by-breath gas analysis estimated the aerobic contribution to 30-s Wingate tests at between 14% and 29% [69, 70]. Separate work by Gastin and Lawson [60] used the measurement of accumulated oxygen deficit to estimate the aerobic system contribution to 30-s all-out cycling at 30%. The aerobic contribution increased to 73% and 91% for 60- and 90-s all-out cycling, respectively. Thus, it appears that the longer the anaerobic performance, the greater the aerobic contribution to overall energy demands. Few studies have examined the aerobic contribution to anaerobic performance in older sedentary or masters athlete populations. Early work by Makrides and others [103] investigated 50 men and 50 women aged 15–71 years who performed a standard 30-s cycle test. Total work performed in 30 s declined linearly for both men and women by about 6%·decade^{-1} and was significantly related to both lean thigh volume estimated anthropometrically (r=0.84) and VO_2max (r=0.86). This latter finding strongly suggests that aerobic capacity is related to anaerobic performance in older individuals. In support of the suggestion that the aerobic system strongly contributes to anaerobic performance in older individuals is the finding that high-intensity endurance training in older sedentary individuals has been shown to significantly improve 30-s maximal exercise capacity by 12.5% [102].

Importantly, the proportion of aerobic contribution to anaerobic energy metabolism is largely influenced by the initial speed of the VO_2 response at the onset of exercise. Previously, Babcock and colleagues [9] reported that the speed of the VO_2 response was slowed with aging in sedentary individuals but later demonstrated that physical training may speed the VO_2 response in older populations [10]. More recently, DeLorey and colleagues [37, 38] reported that sedentary older individuals (68±3 years) demonstrated a slower VO_2 response to both moderate and high-intensity (exercise compared to a younger (26±3 years) cohort. However, concurrent training into older age

has been shown to ameliorate the moderate-intensity VO_2 response speed in sprint and endurance-trained athletes between the ages of 45–85 years [15]. From their work, Berger and colleagues [15] reported that sprint-trained masters athletes demonstrated slower VO_2 responses than aged-matched endurance trained cohorts between 46–55 years, 56–65 years, 66–75 years, and >76 years of age. Interestingly, the sprint-trained veteran athletes developed increasingly slowed VO_2 responses as age increased between 56–65 and 66–75 years, while the endurance-trained athletes showed no effect of age. These findings suggest that aging sprint-trained athletes are likely to possess slowed VO_2 response to exercise, which most likely reflects changes in muscle fiber characteristics and enzyme activities as discussed above. This slowed VO_2 response may allow a smaller aerobic contribution to anaerobic exercise, which may either require greater anaerobic contribution to meet the energetic demands of the effort, or alternatively, decrease the power output to match the available energy resources, and in turn decrease performance.

In summary, given the importance of the aerobic contribution to anaerobic performance, it is possible that the lower VO_2max of masters athletes [25, 121] and slower oxygen uptake responses [15] may contribute to the age-related decreases in anaerobic performance observed in laboratory-based studies and sprint events from a range of sports.

Heredity

Athletic performance has long been recognized as having a strong heredity component [36, 101, 125]. Early genetic studies using young monozygotic (MZ) and dizygotic (DZ) twins suggested that up to 93% of aerobic capacity and 81% of anaerobic capacity may be genetically determined [82]. Later muscle biopsy studies on MZ and DZ twins identified that a major contributor to anaerobic performance, the relative percentage of fiber type, was 99.5% (men) and 93% (women) genetically determined [87]. Similarly, other factors that may affect anaerobic performance including muscular strength and power [28, 50], peak blood lactate [82], and the anaerobic enzymes PFK and LDH [22] appear to have considerable genetic significance. Later work using different methodologies has suggested that many of the above factors that contribute to anaerobic performance are highly heritable including aerobic capacity (47%) [20], 10- and 90-s maximal work outputs (44–92%) [19, 135], 30-s work capacity (86%) [83], muscular strength and mass [74], sprint running performance lasting less than 20 s [21], as well as the trainability of the anaerobic energy systems [19]. There is limited research examining the heritability of muscle strength and power in older populations [7, 150]. Arden and Spector [7] estimated that 46% of the variance in leg extensor power was accounted for by genetics in 45- to 70-year-old female twins. Recent data from the Finnish Twin Study on Aging examined both the leg extensor power and isometric strength of 101 MZ and 116 DZ female twin pairs aged 63–76 years. Leg extensor power and strength shared a genetic component that explained 32% of the variance in leg power and 47% of the leg strength. Moreover, both leg power and strength had a nonshared environmental influence in common that accounted for 4% of the variance in power and 52% of the strength variance [151]. While no longitudinal studies to date have measured muscle power, the age effect of heritability of hand grip strength has been shown to be 52% [52]. Moreover, during a 10-year follow-up of isometric hand grip strength in men, Carmelli and others [29] estimated the heritability to decrease from 35% to 22% from the ages of 63 to 73 years of age. In contrast, the shared environmental effects increased from 39% to 45%. A more recent study by the same Finnish team [150] examined maximal walking speed and maximal leg extensor power and isometric strength in 217 twin pairs aged 63–76 years. Their genetic model suggested that strength, power, and walking speed had a common genetic effect which accounted for 52% of the variance in strength, 35% in leg power, and 34% in walking speed. Taken together, the above data suggest that many of the factors contributing to anaerobic performance in older persons are genetically predetermined but that exercise training can have a major influence on these factors into older age.

Physical training

Physical training for maximal anaerobic performance significantly enhances the capacity of both the ATP-PCr and glycolytic energy systems in younger individuals by influencing many of the factors above that effect anaerobic performance [13, 98, 132]. While the training adaptations that occur with sprint training have been less well documented than those of endurance training, it would appear that the following adaptations may occur: increased anaerobic capacity, muscle fiber hypertrophy of both the type I and II fibers, increased CK and glycolytic enzyme activity (phosphorylase, PFK, LDH), and improved anaerobic performance [13, 98, 132].

While the effects of endurance training on previously sedentary older individuals have been well documented, there are few investigations examining the effects of anaerobic training in the same population. To date, only one study has examined changes in anaerobic capacity with high-intensity endurance training in the aged [102]. In 60 to 70-year-old men that participated in interval training on a cycle ergometer at 50–85% of VO_2max three times per

week for 12 weeks, there was a significant 12.5% increase in total work output and significant decrease in fatigue during a 30-s anaerobic capacity test. Since the 30-s test also has a significant aerobic contribution [70], it is possible that the observed increase in anaerobic capacity may have been partly attributable to a significant increase in VO_2max in the same group following training.

In addition to the positive effects of high intensity endurance training on anaerobic performance, many studies have examined the effects of resistance training in previously sedentary older individuals. Adaptations to such training are likely to include muscle hypertrophy with an increase in the size of both type I and II muscle fibers [56, 66, 95], increased muscle shortening velocity [152], increased muscular force and specific muscle tension [55], muscular power [152], and increased neural activity as evidenced by large increases in electromyographic activity with minimal changes in muscle size [65].

To our knowledge, only two studies have specifically examined the effects of anaerobic or resistance training on anaerobic performance in masters athletes [34, 128]. We examined the effects of an 8-week hypertrophy resistance training program on muscular strength, thigh girth measured anthropometrically, and both 100 and 300 m sprint performance in eight high-performance male sprint runners aged 45–79 years who maintained their normal sprint training over the study period. Significant increases in both isoinertial and isokinetic strength, thigh girth, and 100- and 300-m track performance following resistance training were observed [126]. More recently, Cristea and others [34] examined the effects of a 20-week progressive and combined sprint and strength training program on muscle morphology and contractile characteristics in seven elite sprint runners (66±3 years) with no prior strength training experience. The researchers observed significant increases in isometric torque of the knee extensors (21%) and flexors (40%), 1-RM squat (27%), squat jump (10%), triple jump (4%), reactive jump test power (29%), rate of force development of braking (12%) and propulsion (14%), together with significant increases in 10 m sprint velocity (4%) and 60 m sprint time (2%). These performance changes were accompanied by a 9% increase in neural activity (iEMG) in the jump squat and hypertrophy of the type II (17%) and IIA (20%) fibers. The investigators concluded that the major adaptation to combined sprint and resistance training appears to be improved speed and power performance as a result of muscle hypertrophy, primarily of the muscle fibers expressing type II myosin heavy chain isoforms. Taken together, the results of the two studies above strongly suggest that resistance training combined with speed training enhances anaerobic performance in well-trained and motivated masters athletes.

Conclusion

There is limited research that has specifically examined anaerobic capacity and performance in masters athletes. From the data available, there appears no doubt that anaerobic performance declines linearly in older athletes to around 65–70 years of age, after which the decline in anaerobic performance accelerates. While the actual mechanisms explaining these declines remain to be elucidated, any number of factors contributing to anaerobic performance appears to contribute. The consensus of research suggests that decreased muscle mass, type II muscle fiber atrophy, and a shift towards a greater expression of the MHC I isoform in older age, together with the effect these changes have on many other factors (i.e. fiber contractile velocity, enzyme activity, lactate production) are the major contributors to the age-related decline in anaerobic performance in masters athletes. It also appears that high-intensity sprint training, combined with hypertrophy resistance training, may potentially slow the decline in anaerobic performance of masters athletes into older age.

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