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Masters Athletes

Factors Affecting Performance

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Abstract

In recent years there has been an increase in interest in issues related to the enhancement of the performance of the masters athlete. Many of the changes in health status that have been thought to be the normal result of aging have been found to be actually the result of a long-standing sedentary lifestyle. Thus, masters athletes may be able to increase their athletic performance to higher levels than what was once thought. Decreases in muscle strength thought to be the result of aging do not appear to be so. The masters athlete may be able to maintain and increase strength in situations where strength training has not been previously

engaged in. However, the literature lacks longitudinal studies demonstrating improvements in strength with age in masters athletes who have maintained habitual strength training. Studies in the past have shown that aging results in changes in fibre type, with a shift towards a higher percentage of type I fibres. This again may be an adaptation to lack of use. Decreases in heart function and aerobic capacity appear to be immutable, but in the masters athlete the rate of this decrease can be slowed. The masters athlete has certain elevated nutritional needs over younger athletes. Degenerative joint disease, although effecting most persons as they age, is not a certain result of aging and disability as the condition is reduced in the active person. Some orthopaedic conditions are related to decreases in flexibility of soft tissues that appear to accompany the aging process. Performance improvement in the masters athlete requires the same commitment to hard training that it requires from younger athletes, with some modifications for changes that are associated with aging.

For many years the performance of masters athletes was not considered important. The prevailing thought was that older athletes just competed because they were still kids at heart and could not give up their sports. However, as baby boomers began turning 40, they also continued to strive for the maximum performance that they sought when younger. This has resulted in masters athletes winning major events, such as Priscilla Welch (aged 42) winning the 1987 New York City Marathon or insurmountable records falling, such as Eamonn Coghlan breaking the 4-minute mile barrier for a masters runner in 1994. Sports science is catching up with masters athletes and is providing us with an understanding of the factors that affect their performance. This article will discuss some of these factors.

Prior to the recent increases in masters athletic participation, there were many physiological changes seen in the masters age group that were generally believed to be the result of aging. Research comparing masters athletes to their sedentary peers has found that many of these so-called effects of aging are actually the result of a long-standing sedentary lifestyle[1-3] or disuse.[3] Thus, many of these so-called aging effects are not seen or are seen to a lesser degree in the masters athlete.[4,5]

Three studies have found that the older competitive athlete appears to be no more prone to injuries than younger athletes who train a comparable amount.[6-8] However, in their review of the epidemiology of sports injuries in the elderly (which included the aforementioned studies), Kallinen and Markku[9] conclude that older athletes are more prone to injuries than other adults. Compelling evidence for an increased risk of injury in masters athletes is as yet lacking. Some studies have found a greater predilection for overuse injuries amongst the older athletes, while others found a greater incidence of acute injuries. These differences are probably due to sampling methods and the type of treatment facility that performed the studies. Thus, it should be considered equivocal which type of injury is more prevalent.[9]

The masters athlete is more likely to experience sports-related injuries[9] because the very tissues which make up the tendons, ligaments, cartilage and muscle of their bodies break down more easily and heal with greater difficulty, if at all. This is because, as Postlethwaite notes in his review, we repair at a slower rate as we age[10] and often the motivation to comply with a rehabilitation programme may be lower as we age.[11]

1. Muscle Function

Numerous investigators have shown that there is a decrease in muscular strength associated with aging,[1,12-25] and that this decreased strength has a direct and deleterious effect on the ability of the elderly to perform activities of daily living.[12,14,18-20,26-28]

Loss of strength associated with aging is eliminated or almost eliminated when strength is expressed relative to a measure of quantity of muscle.[13,24] Lexell et al.[29] found an average reduction in muscle area of 40% from the age of 20 to 80 years. The quadratic relationship between muscle area and age has its maximum at 23.7 years. There is approximately a 10% loss of muscle area between the age of 25 and 50 years, and thereafter there is an increasing rate of reduction in muscle area.

Frontera et al.[27] found that, in the frail elderly, exercise at 80% of 1 repetition maximum (1RM) 3 times per week results in an average increase in the 1RM of 5% per training day. Frontera noted in their discussion that this result is comparable to that seen in previous studies in the young (average age 28 years) with similar training, which resulted in strength increases of 4.4 to 5.6% per bout of exercise. Likewise, Yarasheski and co-workers[30] found that the untrained elderly (age 63 ± 1 years) will increase their rate of muscle protein synthesis after 2 weeks of daily moderate to high intensity (60 to 90% maximum strength), low repetition[4-10] weightlifting exercise to a rate statistically identical to that of their younger (age 24 ± 1 years) counterparts in this study. The participants in both of these studies were novice exercisers and as such the results may not be applicable to trained masters athletes.

Microscopic histochemical analysis of muscle from biopsy has shown that as humans age there is a loss of both type I (slow oxidative or slow-twitch fibres) and type II (fast oxidative or fast-twitch) muscle fibres, but there is a greater relative loss of type II fibres.[1,15,16,24,29,31-35] However, atrophy due to inactivity may be the cause of small type II fibres.[29]

High intensity strength training in the elderly has been shown to lead to muscle hypertrophy, because of an increase in the size of both type I and type II fibres.[27,36] Strength training in sedentary elderly people (ages 60 to 75 years) has also been shown to increase the percentage of type IIa fibres.[32,37] There is a upward displacement of the torque-velocity curve after strength training where the greatest influence was on the slow velocity, high torque end of the curve (0 to 60°/second). This is indicative of increases in type II fibres.[27]

In a study of elderly people who exercise to maintain fitness, Klitgaard et al.[15] found that elderly runners (n = 5; mean age 70 ± 0.7 SEM years) and swimmers (n = 6; mean age 69 ± 0.5 SEM years) had similar profiles of muscle fibre types as age-matched (n = 8; mean age 68 ± 0.5 SEM years) control individuals. However, strength-trained older participants (n = 7; mean age 68 ± 0.8 SEM years) had a composition of muscle fibres similar to that of the young control group (n = 7; mean age 28 ± 0.1 years), higher in type II fibres than the runners and swimmers.

On the other hand, Coggan et al.[1] found that masters runners (n = 8; mean age 63 ± 6 SD years) had similar muscle fibre type profiles as performance-matched younger runners (n = 8; mean age 26 ± 3 SD years) but had a lower percentage of type I fibres than very competitive younger runners (n = 8; mean age 28 ± 3 SD years). The masters runners trained at a significantly slower pace and shorter distance per week than the competitive runners, but at a statistically comparable pace and distance as the performance-matched control group (table I). Thus, it appears that the muscle fibre type distribution and performance for the masters and matched control group are comparable due to similar training, and differ from the competitive runners because of a lower training volume and not because of any age effects.

In the study by Coggan,[1] it appears that only training intensity and duration but not age affect fibre type distribution. This finding is supported by a 20-year, longitudinal study by Trappe et al.[38] which found no change in fibre type distribution in highly trained, competitive distance athletes (n = 11; mean age 27.4 ± 1.8 years initially, mean age 47.1 ± 2.5 years follow-up). These studies appears to show specificity of training. Thus, the weight-trained participants in study by Klitgaard et al.[15] would be expected to increase their relative amount of type II fibres because of weight training, while on the other hand, Coggan's[1] and Trappe's[38] runners would be expected to maintain an increase

Table I. Comparison of mean values for masters, performance-matched young controls and competitive young runners (adapted from Coggan et al.,[1] with permission)

Characteristic	Masters	Matched controls	Competitive controls
Age (years ± SD)	63 ± 6	26 ± 3[a]	28 ± 3[a]
Years running (± SD)	15 ± 5	6 ± 3[a]	9 ± 5[a]
Training distance (km/wk)	62 ± 26	58 ± 18	96 ± 42[a,b]
Training pace (m/minute)	195 ± 19	203 ± 15	242 ± 16[a,b]
10km time (minutes)	42 : 03 ± 2 : 57	41 : 41 ± 3 : 36	33 : 34 ± 1 : 20[a,b]

a Significantly different from masters ($p < 0.05$).

b Significantly different from matched control group ($p < 0.05$).

SD = standard deviation.

in their relative amount of type I fibres through continued endurance training.

Thus, it appears that much of the loss of muscle strength associated with aging is an effect of reduced muscle mass (i.e. atrophy) and not of the inherent force production capabilities of myofibrils. Older muscle, at least in the untrained, has the same capabilities for hypertrophy as young muscle. Muscle fibre distribution in the trained elderly matches that of the young, with comparable training showing that specificity of adaptation to demand remains as one ages.

The problem may be that a masters athlete does not have a properly designed training regimen that addresses the maintenance of muscle mass in all areas of the body. It would appear that a masters athlete who trains as hard as a younger athlete should expect comparable results, except that the rate of adaptation to such hard training will be slower in the masters athlete.[39] No studies were identified where masters athletes were trained at intensities and durations comparable to elite younger athletes. One can only speculate as to why training intensity and duration decreases with age. It may be due to slowed recovery, decreased motivation or other factors.

2. Cardiovascular

Studies have shown that there is a decrease in cardiovascular function that is associated with aging.[2,40-50] Decreases in maximal cardiac output,[46] stroke volume[46] and maximal heart rate (HRmax),[40-48,51] all contribute to decreased aerobic capacity ($\dot{V}O_{2max}$).[2,40-48,50-52] However, Douglas and O'Toole[49] found that the hearts of ultra-endurance masters athletes who completed the Hawaii Ironman Triathlon (mean age 58 ± 3 years, range 50 to 71 years) exhibited many of the characteristic changes in cardiac structure and function (lower resting heart rate, larger left ventricular diameter at diastole and higher early to late atrial inflow velocities) seen in young athletes (mean age 23 ± 2 years, range 19 to 25 years). The masters athletes did have higher systolic blood pressure, posterior wall thickness and relative wall thickness than matched younger athletes and comparable to age-matched controls individuals. Thus, it was concluded that exercise and aging affect cardiac structure and function of the older athlete.

2.1 Heart Rate

Age predicted HR_{max} decreases 1 beat per year of age after 10 years of age. Tate et al.[53] suggest that in rats, this loss of heart rate is related to 2 factors. One increases the time of the contraction: an age-related shift towards a slower isoform of myosin (β-myosin). The other increases the duration of the relaxation phase: a decrease in sarcoplasmic reticulum (SR) calcium ATPase. The lower content of SR calcium ATPase slows the sequestration of calcium in the SR, thus slowing the relaxation of heart muscle. In rats, exercise initiated when young that is continued can prevent some loss of HR_{max} by preventing the synthesis of the slower isoform of myosin ATPase. However, although exercise started later in life can increase SR calcium

ATPase activity it cannot restore the faster isoform of myosin ATPase. Hence, it appears that physical activity begun while older can slow the loss of HR_{max} in rats but cannot increase heart rate.[53]

After an average 7.5-year follow-up (range 6.0 to 10.5 years), Rogers et al.[50] found that masters male athletes (n = 15; mean age 62.0 ± 2.3 at follow-up) who engaged in regular vigorous endurance exercise (running or cycling) eliminated the decline in HR_{max} that usually occurs, with a HR_{max} of 171 ± 3 beats/min initially and at follow-up.

However, Trappe and co-workers[44] found in a 22-year, longitudinal study of elite male distance runners that those who continued to train and compete at high levels had an average decrease of 11 beats/min in HR_{max} (table II). Likewise, Kasch et al.[2] evaluated cardiovascular function in 15 active men over a 20-year period. The average HR_{max} at age 65 was 166 beats/min. This is 11 beats/min higher than the age predicted maximum. Also, at age 65 these men were found to have peripheral vascular resistance slightly lower than that of 54-year-old normotensive men. The authors concluded that the participants substantially maintained their cardiovascular function as they aged because they adhered to a reasonably consistent training regimen (2300 to 2100 kcal/wk^{-1} energy expended over the 20 years). Even though Trappe and Kasch found a decrease in HR_{max}, it was half of the age predicted decrease.

However, in another longitudinal study lasting 28 years, Kasch et al.[40] did not find that a similar amount of exercise (2294 to 2389 kcal/wk^{-1}) had such a substantial impact on preventing the loss of HR_{max} (180 beats/min initially, 158 beats/min follow-up) in a group of male (mean age 43.2 ± 6.0 initially, 71.3 ± 6.7 at follow-up) exercisers. There was a loss of 22 beats/min in 28 years. These results were not compared to the earlier study by Kasch.[2]

Finally, in the most recent longitudinal study, Pollock et al.[43] followed a group of elite male track athletes for 20 years. For those that continued to train at high intensities (n = 9; mean age 51.2 ± 7.6 initially, 70.4 ± 8.5 at 20-year follow-up) there was a loss of 13 beats/min rather than the predicted 20. Thus, it appears that a hard training regime continued into the masters years should reduce the loss of HR_{max} to 4 to 7 beats/min per decade.[43]

3. Maximum Aerobic Capacity

$\dot{V}O_{2max}$ decreases approximately 10% per decade after the age of 25. The rate of decrease for masters athletes is approximately half of their sedentary peers.[2,40,44,50] This decrease is primarily caused by lower maximal cardiac output. A smaller stroke volume is also significant factor. Finally, a lower HR_{max} contributes to this decrease.[46] Trappe et al.[44] found in their former elite, now competitive, masters runners only a 5.2% decrease in their absolute $\dot{V}O_{2max}$ (L/min) and a 13.4% decrease in their relative $\dot{V}O_{2max}$ (ml • kg^{-1} • min^{-1}) [table II].

Table II. Results (mean ± standard error) of a 22-year longitudinal study of elite distance runners who continued to train and compete at high levels (adapted from Trappe et al.[44], with permission)

Characteristic	Initial value	22-year follow-up
Age (years)	25.7 ± 1.1	47.2 ± 1.2[a]
Training distance (km/wk)	125.5 ± 9.4	71.4 ± 7.4
Training pace (km/hr)	14.1 ± 1.2	13.5 ± 0.9
Training frequency (sessions/wk)	6.3 ± 0.2	6.1 ± 0.3
% Fat	7.4 ± 0.2	12.6 ± 0.3[a]
Bodyweight (kg)	66.9 ± 1.7	70.5 ± 1.6[a]
Absolute $\dot{V}O_{2max}$ (L/min)	4.57 ± 0.10	4.14 ± 0.11[a]
Relative $\dot{V}O_{2max}$ (ml • kg^{-1} • min^{-1})	68.8 ± 1.7	59.2 ± 1.3[a]
HR_{max} (beats/min)	191 ± 2.5	180 ± 2.8[a]

a Significantly different ($p < 0.05$).

HR_{max} = maximal heart rate; **$\dot{V}O_{2max}$** = maximum aerobic capacity.

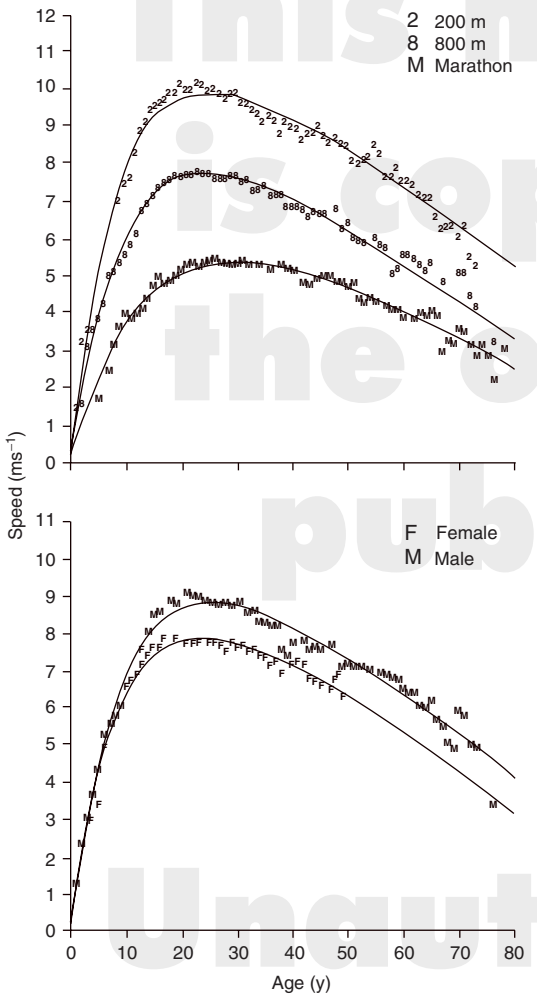

Fig. 1. Running speed records in 1975 by age. **top** Men's records for 200m, 800m and marathon. **bottom** The 400m records for men and women.

This was a loss of 6% per decade. In the study by Rogers et al.,[50] 18 masters athletes were followed for an average of 7.5 years, and $\dot{V}O_{2max}$ decreased by 5.5% per decade. Sedentary age-matched control individuals had a 12% per decade decrease in $\dot{V}O_{2max}$.

Kasch et al.[2] evaluated cardiovascular function in 15 active men over a 20-year period (from age 45 to 65 years). They found only a 3% decline in $\dot{V}O_{2max}$ (which was not statistically significant) over the first 18 years of the study, with a total decrease of 12% over the whole 20 years. Kasch theorises that the participants were able to maintain their $\dot{V}O_{2max}$ because they: (i) followed a consistent training regimen over the 20 years; (ii) were at their optimal bodyweight; (iii) had possible genetic factors; (iv) had normal resting blood pressure and low peripheral vascular resistance and myocardial oxygen uptake ($M\dot{V}O_2$); (v) had relatively high energy output per week (approximately 2100 to 2300 kcal/wk of exercise); and (vi) had above average cardiac reserve. Pollock et al.[42] also found that masters athletes could maintain their $\dot{V}O_{2max}$ if they maintained their high activity level. Thus, it appears that one key to maintaining and possibly improving performance is to train at a very high level. However, as Pollock et al.[43] note in their 20-year, longitudinal study, there have been no reports of maintaining extremely high levels of training for more than 10 years in athletes, whether masters or younger.

Pollock et al.[43] found higher reductions of $\dot{V}O_{2max}$ during their 20-year, longitudinal study. They found a loss of 8% during the first decade (from a mean age of 51.2 ± 7.6 to 60.4 ± 8.5 years) and an additional loss of 15% for the second decade (from a mean age of 60.4 ± 8.5 to 70.4 ± 8.5 years). This is a similar acceleration in the reduction in $\dot{V}O_{2max}$ in the sixtieth decade of life to that seen by Kasch.[2]

While one might be tempted to ascribe the cause of the decease in $\dot{V}O_{2max}$ to aging, a major confounding factor is that in many of these studies not only did the athletes age, they also reduced the volume and/or intensity of their training.[43,44,50] However, in 2 studies with decreases in $\dot{V}O_{2max}$ of approximately 5% per decade, volunteers did not substantially alter their training.[2,40] Although this would appear to add support to the assertion that a loss of $\dot{V}O_{2max}$ of 5% per decade is an immutable loss due to aging, the athletes in both Kasch studies were not elite competitors. Thus, we are left without compelling evidence of the true cause of the decrease of $\dot{V}O_{2max}$ seen in masters athletes; age or reduced training intensity or duration.

4. Lactate Threshold

It was found in one study[1] that masters athletes have higher lactate thresholds and thus can perform at a higher percentage of their aerobic capacity. Coggan et al.[1] found that the muscle of masters athletes (mean age 63 ± 6 years) has higher succinate dehydrogenase and β-hydroxyacyl-CoA dehydrogenase activities and lower lactate dehydrogenase activity than performance-matched young control individuals (mean age 26 ± 3 years). This difference in enzymatic activity allowed the masters athletes to compete at levels comparable to the younger athletes, even though their $\dot{V}O_{2max}$ was 15% lower (51.1 ± 4.3 and 57.7 ± 3.6, respectively).

5. Performance: Age, Distance and Strength

In 1975, Moore[54] plotted all age group records in track and field and found that speed improves up to the age of approximately 20 years and gradually deteriorates beyond the age of 30 (figure 1). Between the ages of 20 and 30 years, running speed is near maximum and almost constant for all distances included in the records (from 100m to marathon). The age of maximum performance increases with increasing distance. There is also a decrease in the rate of slowing performance as the distance increases. Moore presents the example of the 200m sprint and the marathon. The rate of slowing for the 200m is 0.09 m/s/yr and for the marathon 0.06 m/s/yr. This is consistent with the finding that as one ages there is a shift in muscle fibre distribution towards increase in the relative percent of slow-twitch, type I fibres.[1,15,16,24,29,31-35]

Brooks and Faulkner[55] plotted back, arm and leg strength and 200m sprint world records *versus* age (figure 2). Visually, there is a direct correlation between muscle strength and sprint speed. While Meltzer[56] found predicable decline in bodyweight corrected Olympic weightlifting ability with aging, the decline was not linear. The rate of decline varied by age group (from 40 to 50 years, declines of 7 to 13.5% per decade; from 50 to 60 years, 16 to 21% per decade; from 60 to 70 years, 12 to 14% per decade; over 70 years, 16 to 45% per decade). For all age groups this is a more rapid rate of performance decline than the 0.5% per year decrease that Bortz and Bortz[57] found for non-power sports ($\dot{V}O_{2max}$, 1500m swim, marathon, 100m sprint and 2500m row) after approximately the age of 30 years.

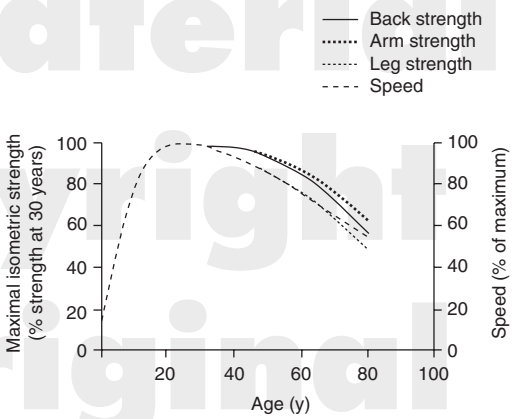

Fig. 2. Data from a cross-sectional study of maximum isometric strength of 3 muscle groups in human beings of different ages are plotted, along with world records for average running speeds of the men's 200m sprint for men of different ages. Strength data are expressed as percentages of the strength measured for 30-year-olds and speed data are presented as percentages of the maximum value. Note that the curve for leg strength and speed almost superimpose.

6. Nutrition

Compared with younger counterparts, the average older individual requires a lower amount of energy for bodyweight maintenance.[58] The 2 principal reasons for this are that the aged generally have a lower amount of lean body mass or skeletal muscle and are less physically active. Lean mass is the major determinant of energy expenditure, and size or mass in the older athlete is most important factor dictating overall energy intake. One benefit of being a masters athlete is the ability to consume greater amounts of energy compared with their peers without causing unwanted bodyweight gain. Overall, the classic predictive equation for determining caloric or energy needs is the Harris-Benedict equation:[59]

Women: BEE = 655 + 9.6(W) + 1.8(h) – 4.7(A)

Table III. Recommended dietary allowances for adults aged 51 years and older (adapted from National Research Council,[63] with permission)

Micronutrient	Males	Females
Retinol (μg/RE/day)	1000	800
Cholecalciferol (μg/day)	5	5
Tocopherol (mg/TE/day)	10	8
Phylloquinone (μg/day)	80	65
Ascorbic acid (mg/day)	60	60
Thiamin (mg/day)	1.2	1.0
Riboflavin (mg/day)	1.4	1.2
Nicotinic acid (mg/day)	15	13
Pyridoxine (mg/day)	2.0	1.6
Folic acid (μg/day)	200	180
Cyanocobalamin (μg/day)	2.0	2.0
Calcium (mg/day)	800	800
Phosphorus (mg/day)	800	800
Magnesium (mg/day)	350	280
Iron (mg/day)	10	10
Zinc (mg/day)	15	12
Iodide (mg/day)	150	150
Selenium (μg/day)	70	55

RE = retinol equivalents; **TE** = tocopherol equivalents.

Men: BEE = 66 + 13.7(W) + 5(H) – 6.8(A)

where BEE = basal energy expenditure (kcal/day), W = bodyweight (kg), H = height (cm), and A = age (years). To estimate the total energy expenditure (TEE), multiply the BEE by 1.3.

6.1 Macronutrient Needs

The current dietary recommendations for macronutrient ratios are: 60 to 65% carbohydrate, where approximately 40% of those carbohydrates come from complex sources, 10 to 20% protein, where lean proteins are included, and less than 30% fat.[60,61] The dietary pattern of the masters athlete should include grain products, vegetables, fruits, low fat dairy products, low fat sources of protein and foods low in saturated fat. Of interest is that it appears that we may need greater amounts of protein as we age. Evans has suggested that a level of protein intake more likely to promote positive nitrogen balance in elderly adults is 1.00 to 1.25 g/kg.[26,58] Maintenance of lean body mass is partially dependent on overall nitrogen balance, although protein metabolism and nitrogen balance are duly dependent on adequate energy intake as well.

6.2 Vitamins and Minerals

Evidence suggests that aging affects the requirements for certain vitamins.[62] Reasons for this include alterations in the capability of absorbing and metabolising these compounds, different baseline requirements and age-related risk factors. Table III shows the US government guidelines of vitamin and mineral intake for people above the age of 51 years. In this section we discuss the following vitamins and minerals where the needs of masters athletes differ from their younger counterparts: cholecalciferol (vitamin D_3), pyridoxine (vitamin B_6), cyanocobalamin (vitamin B_{12}), riboflavin, ascorbic acid (vitamin C), tocopherol (vitamin E), folic acid, calcium and iron.

Vitamin D, a steroid hormone, has been shown to be low in postmenopausal women. This low level was only corrected *via* supplementation.[64] Furthermore, endogenous production of vitamin D has been shown to be less efficient in older *versus* younger individuals.[65] The need for vitamin B_6, a cofactor in protein metabolism, among other functions, may increase as the athlete gets older. Evidence from depletion and repletion studies illustrates that the amount of vitamin B_6 needed to obtain balance is greater than the recommended dietary allowance (RDA)/recommended daily intake (RDI).[66] The requirement for vitamin B_{12} may be higher in people over the age of 65 because of atrophic gastritis (which reduces efficient absorption). Current data suggests that 1 and a half times the RDA is sufficient for preventing B_{12} deficiency symptoms.[62]

Increased amounts of other micronutrients have been documented for older adults, based on disease specific paradigms, such as the ability of vitamin E to reduce oxidised low density lipoprotein, of folate to decrease homocysteine (high homocysteine levels are associated with increased risk for a coronary event), and vitamin C supplementation lowering or reducing the risk of cataracts.[67] It is known that adequate levels of all nutrients are important for promoting overall health. What is not

known is whether specific or different levels of need exist at various times during the life cycle.

Dietary calcium plays an intrinsic role in age-related bone loss. Adequate calcium intake is important for the older athlete. Beneficial aspects of calcium supplementation may include lower risk of colon cancer and age-related bone loss. Evidence suggests that the RDA (800 mg/day) is lower than the level needed to facilitate calcium balance (1000 to 1500 mg/day).[68,69]

Iron is another mineral of interest. Issues of both inadequacy and excessive intake are of concern. A recent Finnish study[70] where excessive iron intakes were associated with heart disease (presumably by enhanced oxidation of low density lipoprotein) raised concern in the athletic world. Runners of both genders may have increased iron losses, and such research has suggested that an increased intake to 18 mg/day is appropriate.[71] Similarly to vitamins, an inadequate intake of essential minerals may occur as a result of dietary inadequacy, medical reasons, physiological reasons or aging itself. Excessive intake of some vitamins or minerals may cause adverse and toxic effects, including birth defects. For older athletes who choose to use a dietary supplement or have dietary inadequacies, using a low dose multivitamin and multimineral at the RDA levels appears to be a low risk approach to ensuring that they are attempting to obtain all of the vitamins and minerals.

6.3 Timing of Meals

In terms of performance goals, masters athletes should consider some very basic strategies to help keep performance at a consistent level. These include:

- Eating throughout the day and snacking regularly to maintain adequate caloric intake and glucose control.[72]
- Having some carbohydrate within 1 hour of a training session/competition to help endurance.
- Experimenting with low and high glycaemic foods.[73]
- Paying attention to the recovery snack/meal after an exhaustive training session.
- Consuming some protein with adequate carbohydrate (from 0.7 up to 2 g/kg of bodyweight) to help restock muscles for the next training session.

6.4 Hydration

Although it is always a challenge, athletes should stay well hydrated. With age the thirst mechanism becomes even less sensitive and water output by the kidneys is greater, so athletes should create a hydration strategy and stick to it. Masters athletes should be encouraged to weigh in before and after training sessions and to consume 2 cups of water for each pound lost. Additionally, check urine: if of great volume and pale, then the athlete is probably well hydrated; if dense and a dark yellow, they should start drinking water. Remember that hydration levels also effect the toxicity of medications.[74,75]

6.5 Quality of Intake

The nutritional quality of food eaten is important, and athletes should choose carbohydrates that are rich in nutrients like whole grains (phytochemicals, fibre, B vitamins, vitamin E, chromium), fruit and vegetables (phytochemicals, flavinoids, fibre, retinol (vitamin A), phylloquinone (vitamin K), vitamins C and E, iron, calcium, potassium, magnesium), low fat milk and yogurt (calcium, vitamin D in milk, vitamin B_{12} and B_2, protein) and beans (phytochemicals, protein, B vitamins).[75]

Masters athletes have the nutritional edge if, simply by continuing to expend energy, they are able to consume enough calories to keep nutrient intakes high. The metabolic adaptations associated with exercise, socialising and being out of doors also contributes positively to nutrient intake. Resistance training should be encouraged to help delay decrease in muscle mass and maintenance of strength and neural pathways, as well as delay the decrease in bone density. Differentiating the young masters athlete from the older masters athlete, differentiating the health status from performance goals and individualising all recommendations will help masters athletes perform at their peak for as long as is humanly possible.

7. Orthopaedics

As a result of aging, there is a decrease in the amount of insoluble collagen and total collagen. Both of these have been correlated with a decrease in the tensile strength of tendons[76] and an increase in the stiffness of tendons.[77,78]

Flexibility and joint range of motion commonly decrease with aging.[79] While a decrease in flexibility is often viewed as a negative, Craib et al.[80] found that decreased flexibility in hip horizontal abduction and dorsiflexion correlated ($r = 0.65$ and 0.53, respectively, $p < 0.05$) with increased efficiency of running in middle-distance runners.[80] The increased efficiency of running due to decrease of range of motion may explain why older athletes, who are less flexible, may provide another explanation why masters athletes can compete at a higher level than one would predict given their $\dot{V}O_{2max}$.[1]

7.1 Degenerative Joint Disease

Aging also adversely effects the articular cartilage, and therefore degenerative joint disease (DJD) or osteoarthrosis occurs more frequently with aging. The amount of mucopolysaccharides decreases in response to aging. This leads to an increase in the water content of the articular cartilage matrix, which weakens the cartilage. The entire process appears to be mediated by proteinases and lysosomal enzymes.[81-83] The aging process and weakening of the articular cartilage tend to lead to fibrillation, thinning and ultimately to the loss of the articular cartilage and DJD.

However, DJD may only be an incidental finding on an x-ray and may not really contribute to an athlete's joint discomfort. This is because there is no statistical correlation between the severity of the joint degeneration and the amount of pain, if any, that a person experiences.[84,85] It is commonly believed that DJD is the result of wear and tear of joints.[86,87] In fact, DJD may actually be the result of a lack of movement of a joint.[88,89] Studies have shown that there is no correlation between long distance running and the later development of DJD,[84,86,90-94] and that the degree of pain and/or disability associated with DJD may be reduced by regular activity.[94-96]

7.2 Osteoporosis

Aging is also associated with osteoporosis. Although osteoporosis is seen in males, this is an especially common problem in older, postmenopausal, female athletes. A decrease in estrogen levels seen after menopause has been correlated with osteoporosis and stress fractures.[97,98] Males generally do not have osteoporotic problems until their 80s, losing bone mass at a rate of 0.4% per year after the age of 50 years. Women, on the other hand, loose 0.75 to 1% of their bone mass per year beginning in their early 30s. After menopause this rate may triple, with many women losing 30% of their bone mass before their 70s.[79]

Female masters athletes who have trained and competed since childhood in sports such as swimming, track, long distance running and gymnastics face an especially high risk of osteoporosis and stress fractures, since many of these athletic young females have a delay in menarche, or develop secondary amenorrhoea related to their training and diet, which predisposes them to osteoporosis and stress fractures.[99]

7.3 Common Injuries

Rotator cuff injuries and complete tears are much more common in athletes over 40 years of age. Impingement syndrome, as originally described by Neer,[100] constitutes the primary cause of most rotator cuff injuries and pathology, but intrinsic changes which result from aging of the tendon also play a significant role in the ultimate failure or tear of the rotator cuff.[101] Various studies have shown that the incidence of rotator cuff tears increases with aging and may in part be related to an area of hypovascularity in the supraspinatus tendon, near its insertion.[101,102]

Achilles tendon ruptures are also much more common over the age of 30 years. Jozsa et al.[103] reported a series of 292 athletes with Achilles ruptures and only 15 of the athletes were under the age of 20 years. The overall incidence of sports-related

Achilles ruptures was 59% in the series, and those between 41 and 50 years old had the highest incidence of sports-related Achilles ruptures.

Meniscal cartilage in the knee is also subject to the same type of aging process and changes which occur in the articular cartilage. These changes have lead Buckwalter and Mow[104] to conclude that the meniscus can tear by shear failure at least occasionally and that these tears result from age-related changes in the collagen proteoglycan matrix rather than from acute trauma.

In summary, orthopaedic injuries in the masters athlete often result from the effects of aging on tendons, cartilage and bone. Orthopaedic injuries which are more common in the masters athlete include rotator cuff tears, quadriceps tendon ruptures, Achilles tendon ruptures, degenerative meniscus tears, focal articular cartilage defects and injuries, and stress fractures. Attention to proper training, with a gradual increase in the amount and level of exercise, is especially important in this older population of competitive athletes in order to avoid serious injury. When injuries occur, the healing process is more often prolonged and complete recovery can take up to a full year.

8. Conclusion

The available evidence at this time suggests that for the masters athlete to improve or maintain athletic performance they need to do just what a younger athlete would do: train and train hard. There are of course certain limitations within which the masters athlete must live. There appears to be an immutable and progressive decrease in HR_{max} and VO_{2max}. Given that limitations, like the sub 4-minute mile for a masters athlete, have fallen, the absolute rate of that decrease, if there is an absolute rate, is not known. Changes in nutritional requirements as one ages must be accommodated. There are changes in connective tissue that predispose the masters athlete to injury. Since a chain breaks at the weak link, the masters athlete must do his or her best to minimise the stress on the known weak links.

In particular, one should keep in mind the rule of thumb of no more than a 10% increase in volume or intensity of exercise per week. When young, the body's ability to adapt to training stress may allow one to break this rule with an impunity that the more slowly-adapting body of the masters athlete will not allow. Optimising athletic performance can be seen as trying to create a balance between a stimulus that promotes fitness (the training effect) and fatigue.[105] The greatest challenge to the masters athlete is to maintain a high level of training that will provide adequate stimulus to the body to promote high performance while preventing fatigue that leads to injury or overtraining.

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