Physical Activity, Aging and Physiological function

By

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Abstract

Human evolution suggests that the default position for health is to be physically active. Inactivity, by contrast, has serious negative effects on health across the lifespan. Therefore, only in physically active people can the inherent aging process proceed unaffected by disuse complications. In such individuals, although the relationship between age and physiological function remains complex, function is generally superior with health, well being, and the aging process optimized.
Thanks to improvements in the environment, nutrition, and particularly developments in modern medicine, lifespan has been progressively increasing in the developed world, but this has not been accompanied by concomitant years of healthspan (30, 31). In most developed countries, an insidious and uncontrolled biological experiment is taking place. This experiment involves the effects of inactivity and over-eating on human physiological processes, including aging. Although the experiment has not yet run its full course, it is already evident these two key lifestyle choices are having significant detrimental effects on human health. This can be seen in the form of increased morbidity and mortality resulting from lifestyle diseases including cardiovascular disease, obesity, Type 2 diabetes, and some forms of cancer (9). An unforeseen consequence of this gradual, uncontrolled change is that there has been a tacit acceptance that the sedentary population, because they are in the majority, represent a default state for the provision of physiological norms. As a result, perceptions of the aging process have become distorted. That a decline in physiological function occurs with age is not in doubt, as shown by the decrements in performance of even the most physically active, highly trained masters athletes (3, 4, 22, 57, 77). However, the decline in functional levels of many systems in sedentary individuals is greater, as are the risks of lifestyle and age-associated diseases (83). This article discusses the contention that much of the data being presented as indicating human aging represents a combination of the aging process interacting with the patho-physiological consequences of inactivity. While acknowledging that there are other important lifestyle factors, such as nutrition, smoking, and alcohol consumption, it focuses on issues concerning our understanding of the relationship between aging and physiological function in the context of physical activity. A logical place to start when considering any default state for biological aging and physiological function is with our evolutionary heritage. It is clear that the increased numbers of sedentary and obese people cannot be ascribed to a shift in our genetic makeup (8). Our gene pool was established during the late Palaeolithic period, 40,000 years ago (33). Preceding this, Neanderthals were in Europe 250,000 years ago and were contemporaneous with an archaic form of Homo sapiens (64). Although Neanderthals became extinct 37,000 years ago, interbreeding with Homo sapiens resulted in modern human populations, not indigenous to Africa, showing 2% intrusive Neanderthal DNA in their genomes (80). It is the difference in physiognomy between the two hominoids that provides some clues as to differences in physical lifestyle. The Neanderthals were stocky, and their bone structures too short to allow for endurancetype physical activity. Homo sapiens, being more gracile, were better adapted to hunting for animals on the plains and following migrations.
Whatever the levels of daily physical activity of Homo sapiens may have been, it is clear that evolved human physiological systems are superbly adapted to respond to exercise and particularly long-distance running (53). In short, if we couple what we know of our evolutionary heritage with the overwhelming accumulation of more recent evidence as to the effects of physical inactivity (55), one clear conclusion emerges: being physically active is the body’s biological default position for the maintenance of physiological function throughout the life course.

Interpretation of the Relationship Between Age and Physiological Function

A number of organism models, such as Drosophila, C. elegans, and rodents, are widely used as experimental models for aging research, not only because of ease of manipulations, but also because their short lifespans allow longevity to be a readily attained endpoint for study. However, increasing longevity is not the major concern for humans (83). Of greater importance is the maintenance and prolongation of an independent healthy life: the healthspan and a compression of morbidity (27). Even with small mammals, the extrapolation of aging studies to the human condition can have fundamental limitations (21), which are compounded by the fact that the majority of aging experiments also involve inactive animals. The most informative research about human aging should involve longitudinal studies on humans, but our long lifespan makes any such studies difficult. Furthermore, longitudinal studies of human physiology in particular are beset by logistical, financial, and practical challenges, and also are severely circumscribed by the indexes that can be measured and, with few exceptions, remain rare (41). There is thus a reliance on data from crosssectional studies. Interpretation of these aging studies are recognized to be limited by differences in healthcare, nutrition, socio-economic conditions between generations, as well as genetic differences between individuals. In the context of this review, differences in levels of physical activity between individuals is a particular problem. Despite the acknowledged weaknesses, many assumptions are made about the relationship between age and function based on cross-sectional data. But, if predictions of functional decline in any one system are based on results obtained from an inactive aging cohort, misconceptions about the behavior of this same functional profile in an active population arise. Assessment of lung function may be cited as one widely used example, where data are conventionally presented clinically as a percentage of “age-predicted” norms. However, little is known about the individuals on whom original data sets are collected. Interpretation of cross-sectional data are further complicated by having high-performing young individuals at one end of the age spectrum and low-performing elderly individuals at the
other, with the assumption being that changes follow a linear or curvilinear path through the life course. Yet, close inspection of the middle age ranges, where ends of the spectrum are removed, shows that a relationship between age and function in this middle age group is far from clear across a wide range of physiological functions (51).

Human Aging and Exercise: Toward a Model of Optimal Aging

Since physical activity and exercise are key to maintaining health and thus to the study of inherent aging, it is important to be clear as to what these terms mean. Physical activity has been defined as “any bodily movement produced by the contraction of skeletal muscle that increases energy expenditure above a basal level” (13), whereas exercise has been defined as “a subcategory of physical activity that is planned, structured, repetitive and purposive in the sense that the improvement or maintenance of one or more components of physical fitness is the objective” (13). The key point is that both involve expending energy above the resting state, with the implication being that exercise is a more rigorous and targeted form of physical activity. Replacement of either by inactivity or sedentary behavior will eventually adversely affect the aging process, whatever the age of the individual. It is important to distinguish between acute periods of inactivity and the longerterm effects of chronic sedentary behavior. A good example of the acute effects of inactivity was the landmark Dallas bedrest study performed in the late 1960s by Bengt Saltin and colleagues (78). They undertook a comprehensive physiological evaluation of the cardiovascular system in response to 3 wk of bedrest in a small group of healthy young men. The subjects suffered a marked decline in cardio-respiratory fitness over the 3-wk period, but this was reversible by a subsequent 8-wk period of endurance training. The bedrest model is now widely used as an analog of micro-gravity, inducing deconditioning in other systems such as skeletal muscle. Indeed, both bedrest and spaceflight have been considered as models in which to study accelerated aging, at least for some systems. Importantly, in each case, exercise is used both as a simultaneous countermeasure and to restore function (29). In contrast to acute episodes of disuse, the effects of prolonged sedentary behavior and chronic disuse are more insidious. Returning to the Dallas bedrest study, these same subjects were re-investigated 40 years later in a longitudinal study (56). Interestingly, the deconditioning experienced in the initial 3-wk period of study was comparable to the subsequent 40 years of aging. However, some of these individuals who did not maintain reasonable levels of physical activity had co-morbidities later in life, such that their performance on testing would presumably now have also been influenced by
accompanying pathological processes. Therefore, it can be concluded that, in this instance, neither the acute nor the chronic interventions can be considered to be reflective of the inherent aging process. This is because the acute study reflected a reversible deconditioning, whereas, for the longterm study, aging was now negatively influenced by co-morbidities. It is known that a lack of physical activity is a key risk factor for numerous chronic diseases (9), and even a simple parameter, such as time spent sitting, is an independent predictor of mortality (11). Physical inactivity is thus a significant confounding factor in influencing the trajectory of decline in physiological function as a result of an inherent aging process and must therefore be removed as confounding factor if we are to understand more about the biology of human aging. In this regard, master athletes (sometimes referred to as veteran or senior athletes) have been identified as a population in whom aging, unaffected by many of the complicating factors previously mentioned, can be studied (35, 49, 86, 94). In addition to laboratory investigations of physiological function, there are now vast archives of data on athletic performance (e.g., world records) that cover almost all athletic and sporting disciplines. Indeed, it can be argued that these performance data reflect the ultimate test of physiological integration. These performance curves are effectively cross-sectional studies, with studies on a single master athlete over numerous decades (i.e., a longitudinal study) being extremely rare. Performance curves have been the subject of much analysis (3, 4, 77, 86), and all show a decreasing performance trajectory that is essentially linear until around the eighth decade, at which point the trajectory of decreasing performance accelerates. Because these curves represent the performance profiles of individuals in whom most of the negative factors complicating aging are not present (inactivity, poor nutrition, alcohol use, etc.), it has been proposed that the trajectory of these performance curves reflect the characteristics of the inherent aging process on integrative physiological function (52). Although it is not possible within the constraints of this article to consider in detail the effects of aging and the role of activity on all physiological systems, we will focus briefly on the cardiorespiratory and neuromuscular systems, since these two are particularly important in regard to being physically active and in maintaining health and independence.

Inherent Aging and Cardiorespiratory Fitness

The maximum rate at which the body can utilize oxygen (V˙ O2 max) is a reflection of the workings of the cardiovascular, respiratory, and neuromuscular systems during maximal whole body exercise. As well as being one of the key requirements for a high level of endurance performance,
it is also viewed as the gold-standard measure of health and physical fitness (5). Lower levels of
fitness are associated with an increasing risk of both morbidity and mortality (7, 47). Studies in
exercising individuals have consistently found clear associations between age and \( V' \) \( \text{O}_2 \) max (93).
Importantly, this association remains even when the ends of the age spectrum are not included
(71). Since the age-\( V' \) \( \text{O}_2 \) max relationship occurs in populations of highly trained individuals, the
decline with age cannot be ascribed to a disuse phenomenon, although in older master athletes it
could also relate to a decline in training volume and intensity (70, 87), a point we consider later
on. The data assembled by Wilson and Tanaka in their meta-analysis of 242 cross-sectional studies
on \( V' \) \( \text{O}_2 \) max (93) provides one of the starkest contrasts of the differences between populations
of people classified as sedentary, active, and endurance trained. These data highlight that the
response of the trained to aging is essentially a slope that is shifted upward and to the right of
sedentary people. FIGURE 1A illustrates this point schematically and highlights another important
point that relates to health and well being. For any given age, \( V' \) \( \text{O}_2 \) max values are higher in
exercisers, or, viewed another way, for a given \( V' \) \( \text{O}_2 \) max, the age at which this occurs in
exercisers is older. In practical terms, this equates to a “buying back” of years of function
compared with sedentary people. This pattern of functional advantage in exercisers is also evident
in other systems, particularly in muscle function (39, 66). Furthermore, when scatter plots of data
are expressed in a different way, such that band widths of functional equivalence are plotted
against age (FIGURE 1B), the data show more clearly two additional points. The first is the almost
complete lack of overlap in \( V' \) \( \text{O}_2 \) max between exercisers and the sedentary, and the second is
that, even for the endurance-trained individuals, in the middle age ranges, a band of functional
equivalence can incorporate a very large age range (49). The differences in \( V' \) \( \text{O}_2 \) max values
between the sedentary and exercisers clearly show that interpretations of the effects of the aging
process on physiological function are fundamentally dependent on the physical activity status, as
well as the inherent fitness levels of the individuals selected for study. Since disuse is not a
confounding factor in both cross-sectional and longitudinal studies of exercisers (34, 69, 70), this
decline in \( V' \) \( \text{O}_2 \) max can be ascribed to an inherent aging process. In which case, the physiological
mechanisms relating to aging per se can now legitimately be considered. At sea level in adult
healthy populations, \( V' \) \( \text{O}_2 \) max is determined primarily by an individual’s maximum cardiac output
(79). Thus the decline in \( V' \) \( \text{O}_2 \) max might simply be attributable to a reduction in maximal heart
rate, maximal stroke volume, or both (59). However, this must also be seen in the context of any
age-related loss of muscle mass (26). Interestingly, maximal heart rate is one function of age that
appears unaffected by fitness status and seems to be an inherent aging effect in all individuals, irrespective of lifestyle. The mechanism underlying this inherent aging effect seems to relate to changes in both pacemaker function of the sinoatrial node (intrinsic heart rate), and decreases in chronotropic responsiveness to -adrenergic stimulation (15). Furthermore, in vitro patch-clamp recordings from acutely isolated individual sinoatrial node murine myocytes suggest that aging causes an alteration in action potential ion channels, which depresses the excitability of these cells and contributes to the decline in maximal heart rate (46). If we accept exercising individuals as a model in whom to study human aging, we can conclude that a decline in cardiorespiratory fitness is an inherent biological process. An interesting corollary to this is the observation that the slope of decline in cardiorespiratory fitness has been shown, in some instances, to be greater in athletes compared with sedentary individuals, even though athletes still have far superior values at every age. How might this be explained in the light of athletes being considered to be optimally aging compared with sedentary individuals? In all athletes, V’O_2 max is highly dependent on the level of training, and there is also a well known age-related reduction in training volume (87). Thus it might logically be viewed that an absolute change in activity levels from being sedentary at a young age to being sedentary in later life might be small compared with the change in absolute training load that might occur when comparing a highly trained young athlete with an older master athlete (52). Why this reduction in training load occurs is another issue. We have suggested that, although there is a reduction in absolute training levels (volume and intensity), older athletes are still training at the same relative level, normalized to an inherent age-related decline in sustainable training load (52). In addition to changes in maximum cardiac output, there are other changes to the cardiovascular system that are traditionally attributed to the aging process, such as a general stiffening of the arterial tree (88). However, the extent to which this reflects an inherent aging process, or the effects of inactivity, which is well known to be an independent and significant risk factor for cardiovascular disease, is unknown. In a study using pulmonary artery cathererization to define Starling and left ventricular pressure-volume curves, left ventricular stiffness was compared in sedentary older people with master athletes. No age-related effect was seen in the master athletes (2). Furthermore, in a subsequent study from this group (6), a crosssectional study of older individuals aged over 64 years divided into four groups based on habitual physical activity levels (sedentary, casual exercisers, committed exercisers, and competitive masters athletes) was performed, and a dose-response exercise-stiffness relationship was observed, such that a lesser decrease in left ventricular stiffness was associated with more exercise (FIGURE 2). This
observation raises fundamental questions as to the dose-response effects of exercise. How much and what type of exercise is sufficient to avoid the negative influence of inactivity affecting the inherent aging process? In this regard, our laboratory (51) has recently proposed a Set Point theory of aging, which hypothesizes that, for each individual, there is a threshold for exercise that offsets inactivity so that decreases in physiological function are solely the result of the inherent aging process. The hypothesis states that, below this set point, decreases in function are due to a combination of the aging- and inactivity-mediated processes with a compromised healthspan and extended morbidity. Exercise above this Set Point level would result in the potential for enhanced function and improved athletic performance, but there are no further gains in healthspan or an optimal aging trajectory. This hypothesis might be applied to the data in FIGURE 2 such that the master athletes may not age any more optimally, even with their more compliant left ventricles, than the committed exercisers, since both groups might reasonably be considered to be above their Set Point levels. Casual exercisers were not significantly different in terms of ventricular stiffness compared with the sedentary, suggesting that both groups might be below their Set Point for optimal aging and be predicted to have a compromised healthspan.

**Inherent Aging and Neuromuscular Function**

Like the cardiorespiratory system, declines in muscle function occur even in highly trained master sprinters, power-/weightlifting athletes (32, 39, 66). Here, age-related declines in strength and power are reflected in slower sprinting speeds, shorter jumps and throws, and lighter weights lifted. As for endurance events, the decline in world-record sprint performances shows an accelerated decline after the eighth decade (3, 76). One of the major contributing factors to the loss of strength and power is a loss of muscle mass. An age-related loss of tissue mass has been termed sarcopenia (“loss of flesh”) (24) and is used to differentiate it from the loss in muscle mass that occurs as result of disuse atrophy or a disease-induced cachexia. Like osteoporosis, arbitrary criteria have been applied to define a disease (18), blurring its relationship with an underlying aging process. The sarcopenic muscle is characterized by a loss of muscle mass and by infiltrations of fat and connective tissue (1, 58, 65). The consequence of this loss of mass and tissue quality are a decline in both contractile and metabolic function (12). Sarcopenia has been associated with numerous factors, many of which are related to purported fundamental mechanisms of aging, such as lowgrade chronic inflammation (“inflamaging”) (25), oxidative stress (38), impaired regenerative capacity (satellite cells dysfunctioning in an aged milieu) (14), and anabolic resistance
(reduced muscle protein synthesis in response to feeding and exercise) (75). At the anatomical level, muscle loss in older people is associated with atrophy of fast-contracting, type II muscle fibers (84). Indeed, it has been suggested that type II fiber atrophy might completely explain the loss of muscle bulk in later life (61). However, a prevailing view is that skeletal muscle loss also relates to a loss of muscle fibers secondary to a loss of motor units (37). Partial re-innervation of abandoned fibers is believed to occur, resulting in an increased size of remaining motor units (68), with knock-on effects for fine motor control. This hypothesis is supported with histological evidence of fiber-type grouping and through electromyographic estimates of motor unit number. Whereas evidence of streaming of Z line and rod formation, and angulated fibers further point to dysregulation of skeletal muscle in later life (81). Studies of master athletes paint a very different picture (as illustrated in FIGURE 3), which once more challenges the assertion that these changes can be explained by an inherent aging process and are inevitable consequences of getting older. Cross-sectional studies of master athletes who are strength and power athletes show superior contractile strength and power compared with age-matched sedentary individuals and to endurance runners (34, 39, 66). In a cross-sectional study of Finnish sprinters encompassing an age range of 18 – 82 years (40), a progressive decline in type II fiber size with increasing age was demonstrated, suggesting that at least a part of the type II fiber atrophy is an inherent aging effect. But is continued exercise able to counter age-related motor unit loss? A study using spike-triggered averaging to estimate motor unit number in the tibialis anterior and biceps brachii muscles of endurance runners and sedentary individuals in their mid-sixties showed that, relative to young individuals, sedentary individuals had a lower number of motor units in both muscles. However, in the master runners, although a similar low number of motor units were observed in the biceps brachii, motor unit number was similar to the young individuals in the tibialis anterior (73, 74). This was taken as evidence that maintained activity of a muscle would prevent motor unit loss, suggesting that this is also an aging phenomenon influenced by activity status. A similar study (23) was undertaken on the hypothenar muscle on both endurance- and power-trained master athletes up to 89 years of age. Here, the power-trained athletes, rather than the endurance-trained athletes, were the ones who had significantly more motor units compared with the non-trained age-matched individuals in this muscle. The loss of muscle strength and power is explained in large part, but not completely, by the loss of muscle size. Contractile quality can be defined as the force generated per unit cross-sectional area (specific force) or power per unit volume but is complex to determine from in vivo measurements (62). The study of segments of single fibers
obtained from biopsy samples, permeabilized and activated chemically, allow the contractile machinery to be studied under controlled conditions in vitro. Studies on the effects of aging on specific force in single fibers have resulted in mixed findings, with some studies reporting a decline in specific force in both main fiber types (I and IIa) (28) and others showing no decline in either (89). A similar lack of consistency is also observed for maximal shortening velocity. One of the reasons for the conflicting results may well relate to the physical activity status of the individuals studied. D’Antona and colleagues (20) examined fibers from older individuals (aged 70 years) with a wide range of activity backgrounds and master runners. The study showed that specific force was lower in inactive and immobilized individuals, but there was no difference in type I- or IIa-specific force in fibers taken from the masters runners compared with the young controls. This suggests that the mechanisms related to changes in cellular function attributed to aging, such as loss of myosin concentration (19) and posttranslational modifications of myosin (54), are heavily influenced by inactivity and thus reflect an aging-inactivity interaction rather than inherent aging per se. Muscle is exquisitely sensitive to use and disuse, such that exercise enhances the metabolic potential of muscle through increasing oxidative capacity and insulin sensitivity (76). Sedentary behavior coupled with high energy intake is known to decrease this oxidative potential and is a high risk factor for Type 2 diabetes. In sedentary aging, there are reduced enzyme activities, mutations in mitochondrial DNA, and a decreased expression of some mitochondrial proteins. However, it is clear that exercise remains a potent stimulus for mitochondrial biogenesis in later life (43, 67). In a study on young and old trained and sedentary people, the effects of aging and activity on insulin sensitivity and mitochondrial function were explored to determine whether mitochondrial dysfunction and lowering of insulin sensitivity were inevitable consequences of aging (44). The main findings were that both young and older trained subjects had substantially higher insulin sensitivity compared with the sedentary old and young subjects. Thus exercise status and not age was the prime factor differentiating the groups. There were also age-related declines in muscle mitochondrial oxidative capacity in both groups, such that capacities in old trained individuals were lower than in young trained individuals, but importantly were still superior compared with young sedentary individuals. This study also showed that endurance exercise may exert similar potentially lifespan-enhancing effects to those of caloric restriction through elevated SIRT3 expression in both young and older adults. Taken together, both the superior skeletal muscle and the cardiovascular health profiles of even octogenarian athletes provides a large functional reserve above thresholds associated with disability and mortality in
older people (90). Two further points are worth emphasizing at this juncture. The first relates to the age range over which we consider maintenance of function by activity to be effective. Studies in master athletes rarely consider those most old and usually up to 80 years of age (90), and the extent to which these benefits are maintained as function and activity are compressed toward the end of life remains to be determined. The second is that the exercise modality needed to optimize aging, will be different for different physiological systems. Cardiorespiratory function is best maintained by aerobic exercise, whereas muscle strength is best maintained by resistance training. One exercise modality will not be sufficient to optimize all systems. For example, although cycling provides great benefits to both cardiorespiratory and muscular systems, it does not protect against loss of bone mineral density (60, 71).

Global Impact of Physical Activity on Aging Processes

The two systems discussed above demonstrate the close interaction of physical activity, function, and aging. Important facts emerge from this analysis, such as the consistently superior function of active individuals relative to sedentary individuals. Yet one component of this function, maximum heart rate, highlights the fact that there are some physiological indexes that are not influenced by activity but are under the control of the aging process. Furthermore, there are others factors that, in the absence of pathology, are seemingly unaffected by either age or activity, such as renal and hepatic function and the regulation of blood glucose by insulin (63, 82). However, there is emerging evidence of the role of physical activity during aging that spreads beyond the cardiorespiratory, neuromuscular, and metabolic systems. The decline in cognitive function is a serious issue for many older people, as white and grey matter volumes fall and there is impaired cerebral perfusion coupled to oxidative stress and inflammation. However, prospective and retrospective studies have shown a lower incidence of cognitive impairment, depression, and dementia in people who have maintained regular physical activity (48, 85). Other studies in both animals and humans show that physical exercise can alter mechanisms of neuronal plasticity involved with learning and memory (16, 17) and may prevent age-related deterioration of cognitive function and reduce age-related brain atrophy (92). It is beyond the scope of this review to cover all systems, but the immune system (90) can be added to those systems where assumptions about aging need to be seen firmly in the context of the level of physical activity. A weakness of aging research, even when studying exercisers, is the fact that often studies do not focus on more than one or two systems. In an investigation that undertook a deep phenotyping by
measuring a plethora of indexes in a cohort of healthy exercisers (aged 55–79 yr), the relationship between age and function was poor (70). This made it hard to make any prediction as to the age of a given individual from any of the physiological indexes measured. A more successful attempt to find a biological “aging clock” was made using DNA methylation in a range of tissues (36), but this was not coupled to markers of physiological function and not undertaken in exercisers. Telomere shortening, another purported marker of aging, is also subject to influence by exercise and not just by chronological age (45).

Summary

Human evolution suggests that the default position for health is to be physically active. Inactivity, by contrast, has serious negative effects on health across the lifespan. Although other lifestyle factors such as nutrition, smoking, and excessive alcohol consumption also distort physiological function during aging, this review has focused on physical activity. This is because, from the available data, we conclude that being physically active is the human biological default condition, counteracting the distorting effects of inactivity and allowing aging to proceed optimally. Thus only in the physically active can the inherent biological human aging process be studied.

References


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Figure legends

Figure 1. Depiction of the trajectory of agerelated decline in physiological function

A: depiction of the trajectory of age-related decline in physiological function (such as V’ O2 max or muscle power) in master athletes, active individuals, and sedentary people. The graph depicts that the age-function relationship is moved upward and to the right as levels of activity increase. This results in a higher age for the same level of physiological function. The graph also depicts the highest values for any age occurring in the master athletes but also a relatively greater loss in V’ O2 max in athletes, associated with a decline in training load. Linear declines in function are shown for illustrative purposes but should not be assumed. B: V’ O2 max values taken from Wilson and Tanaka (Ref. 93; men, bottom) and Tanaka and Seals (Ref. 86: women, top) have been allocated to bandwidths of functional equivalence (5 ml·kg⁻¹ ·min⁻¹). The data for men represent mean values from studies used in the meta-analysis, and for women each data point represents the raw value for a given individual. The graphs separate vigorous exercisers and sedentary people, and show that bandwidths representing different functional equivalence encompass a wide range of ages. Note that the data points plotted are only those that could be discerned from the original publication and that a number of points are overlaid (50).

Figure 2: Group-averaged left ventricular pressure-volume relationships

Group-averaged left ventricular pressure-volume relationships determined by right heart catheterization (pulmonary capillary wedge pressure) and echocardiography (left ventricular volume) for sedentary subjects (blue), casual exercisers (red), committed exercisers (purple), and competitive exercisers (green) aged 70 yr. The sedentary and casual exercisers have a leftward shift and steeper slope of curve, reflecting a stiffer, less distensible ventricle compared with the rightward shift and flatter slope of curve for the committed and competitive exercisers, reflecting a more distensible and compliant ventricle. Committed exercisers demonstrated improved ventricular compliance and distensibility compared with casual exercisers and sedentary subjects, and almost matched values achieved by competitive exercisers (6).

Figure 3: MRIs taken across the mid region of the thigh

Images taken across the mid region of the thigh, showing a sarcopenic muscle (middle) from a sedentary individual with reduced contractile mass, more subcutaneous fat, and infiltration of fat and connective tissue. By contrast, the muscles of a similarly aged master triathlete (bottom) shows little difference to that of a younger athlete (top) (95).
Figure 2

[Graph showing relationship between Pulmonary Capillary Wedge Pressure (mmHg) and Left Ventricular End Diastolic Volume Index (mL/m²).]

- Sedentary Subjects (n=27)
- Casual Exercisers (n=20)
- Dedicated Exercisers (n=25)
- Competitive Exercisers (n=35)
Figure 3