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# Invited Review: Dynamic exercise performance in Masters athletes: insight into the effects of primary human aging on physiological functional capacity 

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#### Abstract

Tanaka, Hirofumi, and Douglas R. Seals. Invited Review: Dynamic exercise performance in Masters athletes: insight into the effects of primary human aging on physiological functional capacity. J Appl Physiol 95: 2152-2162, 2003;10.1152/japplphysiol.00320.2003.-Physiological functional capacity (PFC) is defined here as the ability to perform the physical tasks of daily life and the ease with which these tasks can be performed. For the past decade, we have sought to determine the effect of primary (healthy) adult human aging on PFC and the potential modulatory influences of gender and habitual aerobic exercise status on this process by studying young adult and Masters athletes. An initial approach to determining the effects of aging on PFC involved investigating changes in peak exercise performance with age in highly trained and competitive athletes. PFC, as assessed by running and swimming performance, decreased only modestly until age $60-70 \mathrm{yr}$ but declined exponentially thereafter. A progressive reduction in maximal $\mathrm{O}_{2}$ consumption ( $\mathrm{V}_{\mathrm{O}_{2} \text { max }}$ ) appears to be the primary physiological mechanism associated with declines in endurance running performance with advancing age, along with a reduction in the exercise velocity at lactate threshold. Because $\dot{V}_{2}$ max is important in mediating age-related reductions in exercise performance and PFC, we then investigated the modulatory influence of habitual aerobic exercise status on the rate of decline in $\mathrm{V}^{2} \mathrm{o}_{\text {max }}$ with age. Surprisingly, as a group, endurance-trained adults appear to undergo greater absolute rates of decline in $\mathrm{V}_{2_{2}}$ max with advancing age compared with healthy sedentary adults. This appears to be mediated by a baseline effect (higher $\dot{\mathrm{V}} \mathrm{O}_{2}$ max as young adults) and/or a marked age-related decline in exercise training volume and intensity (stimulus) in endurance-trained adults. Thus the ability to maintain habitual physical activity levels with advancing age appears to be a critical determinant of changes in PFC in part via modulation of maximal aerobic capacity.


maximal oxygen consumption; sex; physical activity

In the study of the physiology of muscular exercise there is a vast store of accurate information in the records of athletic sports and racing.

$$
\text { A. V. Hill, } 1925 \text { (24) }
$$

Physiological functional capacity (PFC) is defined here as the ability to perform the physical tasks of daily life and the ease with which these tasks can be performed. PFC declines at some point with advancing age even in healthy adults, resulting in a reduced capacity to perform certain physical tasks. This can eventually result

[^0]in increased incidence of functional disability, increased use of health care services, loss of independence, and reduced quality of life ( $3,5,15$ ). Moreover, the decline in PFC provides a serious threat to individuals engaging in physically demanding occupations (59). Because we cannot normally change the physical demands of our daily work with age, a reduction in PFC means that aging workers labor closer to their maximal capacity and that could result in acute cardiovascular events, chronic fatigue, and other health (e.g., orthopedic) problems (59). This situation is expected to worsen in the future given current projections of marked increases in older adults in the United States and other industrialized countries.

For the past decade, we have sought to determine the effects of primary (healthy) adult human aging on PFC and the potential modulatory influences of gender and habitual aerobic exercise status on this process by studying young adult and Masters athletes (12-14, 16, $41,51-55,60$ ). The purpose of this review is to discuss and synthesize our findings from these original research investigations. The focus of the present review is limited to short-term and prolonged large-muscle dynamic exercise performance; therefore, the "expression" of PFC is determined primarily by the capacity to perform this type of activity. Most of the information presented was obtained from cross-sectional observations of athletes differing in age, although the results of longitudinal analyses are also discussed $(12,13)$.

## CHANGES IN PEAK EXERCISE PERFORMANCE WITH AGE

Experimental model. Determination of the effects of biological aging per se on PFC in humans is difficult because interpretation is confounded by corresponding reductions in physical activity levels (deconditioning), changes in body composition (i.e., increases in body fatness and reductions in fat-free mass), and development of clinical diseases, all of which reduce PFC independent of intrinsic aging processes. Because of such limitations, a novel approach to determining the effects of aging on PFC in humans is to determine changes in peak exercise performance with age in highly trained and competitive athletes ( $11,12,24,29$, $34,55)$. The theory is that these athletes represent an effective experimental model because changes observed with advancing age are thought to reflect mainly the results of primary (physiological) aging. Moreover, 1) the peak performances of these athletes are established under rigorous standardized conditions, particularly at championship-level events; and 2) self motivation, a critical determinant of the ability to precisely determine peak performance, is assumed to be at or near maximal under such conditions. Using athletic records, the Nobel laureate A. V. Hill originally used this experimental strategy to examine the relation between maximal speed and racing distance in various athletic events (24). Since then, a number of investigators have studied age-related changes in PFC primarily by examining endurance running performance.
Age and running performance. Using this approach, we $(11,54)$ and others $(9,29,35)$ have established that endurance running performance decreases with age in a curvilinear fashion (Fig. 1). Specifically, performance is maintained until $\sim 35 \mathrm{yr}$ of age, followed by modest increases in running times until $50-60 \mathrm{yr}$ of age, with progressively steeper increases thereafter. This decrease in performance is up to threefold greater in women compared with men, with the largest differences at $>60 \mathrm{yr}$ of age (11). This apparent faster rate of decline in endurance exercise performance with advancing age in women may have a biological basis. It is also possible, however, that factors independent of


Fig. 1. Ten-kilometer running times with advancing age. (From Ref. 54.)
physiological aging (e.g., sociological influences) may contribute to these differences. For example, the widening of sex differences with advancing age may be explained partly by a smaller total number of female runners in older age groups (i.e., selection bias) (29). At least part of the sex-related differences in performance with aging appears to be attributable to this factor, based on our work on competitive swimmers (see below).
Age and swimming performance. As a next step, we examined swimming performance with advancing age $(12,55)$. We reasoned that one could place greater confidence in the data obtained on running if the results could be confirmed with other types of physical activity. The analyses of swimming performance provide a number of advantages for studies of aging and PFC. First, swimming is a non-weight-bearing activity and has a relatively low incidence of orthopedic injury even among older adults ( 32,33 ). This is particularly important because the interpretation of age-related decrements in exercise performance can be confounded by an increased incidence of orthopedic injuries with age. Second, unlike many athletic events in which male participants outnumber their female counterparts (e.g., running), swimming is known to have an equivalent male-to-female participation ratio (55), thus minimizing the potential "sociological" confound in the interpretation of results on sex-age interactions in PFC. We first performed a retrospective cross-sectional analysis of freestyle swimming performance times collected from the U.S. Masters Swimming Championships (55). Given the inherent limitations of crosssectional comparisons, we then performed a longitudinal study by conducting a follow-up on 321 women and 319 men who participated in the U.S. Masters Swimming Championships over a 12 -year period (12).

Task specificity. Similar to running performance, PFC as assessed by swimming performance declined with advancing age in a curvilinear manner in both men and women (55) (Fig. 2). However, we noted that the pattern of decline was somewhat different from that observed with long-distance running. The magni-


Fig. 2. U.S. Masters swimming: $1,500 \mathrm{~m}$ swimming performance times with advancing age. (From Ref. 55.)
tude of overall reduction in swimming performance with advancing age is as much as $30 \%$ smaller than that observed in running performance. Moreover, the age at which exponential declines in performance begin occurred later with swimming ( $\sim 70 \mathrm{yr}$ ) compared with running ( $\sim 60 \mathrm{yr}$ ) (55). These findings support the idea that the age-associated changes in performance-derived measures of PFC can be influenced by the task involved.

The reasons for these task-specific differences are not clear, but it could be explained by the observation that the swimming performance is relatively more dependent on biomechanical techniques than is running (10). It is not uncommon to see Masters swimmers achieve their personal best time at 40-50 yr of age (9). An alternative explanation is a lower incidence of orthopedic injury during swimming compared with running. The decline in running performance with age has been partly attributed to an increased incidence of orthopedic injuries, which limits the training volume of older runners $(13,29,42)$. Thus swimming performance may be impaired with age to a much lesser extent by this factor. Another possibility is that, because swimming training is interval based, a better maintenance of training intensity with age may have contributed to the smaller decline in PFC in swimmers.

Age-sex interactions in exercise performance. In the cross-sectional study, we found that the rate of decline in swimming performance with age was greater in women than in men across all the swimming events analyzed (55) (Fig. 2). However, interpretation of such cross-sectional comparisons of performance times across age must be made carefully. It is possible that factors independent of true physiological aging changes may influence the results. In fact, in the fol-low-up longitudinal study, sex differences in age-related declines in swimming performance were only evident in a short-duration (i.e., sprint) event; no such difference was observed in longer duration (endurance) events (12) (Fig. 3). It is possible that the age-associated declines in the physiological determinants of
sprint and endurance performance may occur at different rates in men and women. In this regard, the relative rates of decline in maximal oxygen consumption ( $\dot{\mathrm{V}}_{2}$ max ; a major determinant of endurance swimming performance) are similar between men and women (26), whereas women appear to experience greater declines in muscular strength and power (primary determinants of sprint performance), particularly in upper extremities, than men $(40,49)$.

Indeed, one of the intriguing patterns we discovered was that sex differences in swimming performance were greatest in the shorter events and became progressively smaller with increasing distance (55) (Fig. 4). We did not see this sort of distance-dependent or exercise duration-dependent trend in running or other sports (55) (Fig. 4). One physiological explanation for a smaller sex difference with increasing swimming distance is that the oxygen cost of swimming has been shown to be lower (i.e., more economical) for women than for men as a result of their smaller body size (resulting in smaller body drag), smaller body density (resulting in greater buoyancy), greater body fat, and shorter legs (resulting in a more horizontal and streamlined position) (31, 39). As such, women may have a greater capability to conserve body energy stores during swimming events of longer duration. Ironically, these are physiological factors that limit the performance of women in other sporting events. In marathon swimming, however, these differences may act in their favor (52).

It is tempting to hypothesize that the sex difference may continue to become smaller as the swim distance increases further. On the basis of the extrapolation of the regression line that we constructed and the reconversion of log scale to normal scale number, the projected swim distance that women may surpass men is $\sim 25 \mathrm{~km}$ (52; Fig. 5). In this context, it is interesting to note three important facts that support our hypothesis. First, in the most recent Ironman triathlon championship, the sex difference in swimming performance was $<1 \%$ (in running and cycling, it was $>10 \%$ ). Second,


Fig. 3. Instantaneous rates of changes in swimming performance times in relation to the present fastest world record time. Women's curves are the top lines for each swimming distance. (From Ref. 12.)


Fig. 4. Gender differences (\%) in performance as a function of event distance. $A$ : running. $B$ : swimming. (From Ref. 55.)
the fastest time to swim across the English Channel had been held by a woman for many years. Third, the Guinness record for the longest nonstop ocean swim currently is owned by an Australian female swimmer who swam 196 km in 38 h and 33 min .
A final, more general, point on the issue of sex differences in performance relates to the possible influence of sport or activity specificity. For example, one reason that age-sex interactions in performance may differ in swimming and running is that women were permitted to swim for many years but discouraged from other sports. It will be interesting to revisit this issue as the Title 9 generation ages.
Factors involved in the decline in exercise performance with aging. With respect to aging per se, it is not entirely clear why there appears to be an accelerated decline in exercise performance after $\sim 60-70 \mathrm{yr}$ of age. This was observed in both running and swimming events. Our findings, however, are consistent with previous studies conducted in the area of physical function and aging. For example, freely chosen walking speed declines relatively little up to $\sim 60-70 \mathrm{yr}$ of age but then is followed by sudden decreases (25). Additionally, muscular strength, as assessed by Olympic weightlifting capacity, declines linearly until $\sim 70 \mathrm{yr}$ of age, after
which the rate of decrease accelerates significantly (34). Thus it is tempting to speculate that fundamental changes in biological aging processes may occur around the age of $60-70 \mathrm{yr}$ that act to substantially impair physical performance, as previously suggested by Joyner (29). Alternatively, this also might be the age at which biobehavioral ("mind-body") changes may occur (e.g., reductions in motivation to train at high levels). In this regard, reduction in spontaneous physical activity with aging is a characteristic of many different animal species ranging from insects (50), to rodents (27), to humans (13, 26, 42).

## PHYSIOLOGICAL MECHANISMS UNDERLYING AGE-RELATED REDUCTIONS IN ENDURANCE EXERCISE PERFORMANCE

The decline in PFC with advancing age can be attributed to collective reductions in the physiological determinants of exercise-work performance. What are the physiological mechanisms that could contribute to age-related reductions in endurance performance? On the basis of the data primarily collected on young men, three primary determinants of endurance performance could be involved (9).
Maximal aerobic capacity. It is generally agreed that $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ is a primary determinant of endurance exercise performance because it establishes the upper limit of maximal energy production through oxidative pathways (47). Endurance performance and $\mathrm{Vo}_{2}$ max are highly correlated in populations heterogeneous with respect to $\dot{\mathrm{V}}_{2}{ }_{\text {max }}(29,53)$. We $(14,53,60)$ and others $(9,18,29)$ have found a tight inverse relation between $\mathrm{V}_{2}{ }_{\text {max }}$ and age in populations of highly trained and competitive distance runners varying in age. Although these observations are consistent with the concept that decreases in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ contribute to the reduction in endurance exercise performance with age, the decline in performance appears to be slightly smaller than the fall in $\dot{\mathrm{V}}_{2}$ max. This could be explained by a smaller rate of age-related decline in the other putative deter-


Fig. 5. Relation between sex difference (female swim velocity/male swim velocity) and swimming distance. (Modified from Ref. 52.)
minants of exercise performance: lactate threshold and exercise economy.
Lactate threshold. A second factor is lactate threshold, defined as the exercise intensity at which blood lactate concentrations increase significantly above baseline. The minimal available data suggest that decreases in lactate threshold contribute to the age-related reduction in endurance performance (14, 18). Endurance performance, assessed by 5 -km run time, was closely associated not only with $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ but also with the exercise velocity at lactate threshold in male runners aged 21 to 69 yr (28). More recent data on women distance runners from our laboratory (14) are consistent with this earlier observation in men (Fig. 6). Specifically, we found that a reduction in lactate threshold appears to contribute to the decline in endurance performance from young adulthood to early middle age, whereas reductions in $\dot{\mathrm{V}}_{2}{ }_{2}$ max appear to contribute most to the further decline in performance from early to later middle age (14).
In both male and female trained runners, lactate threshold does not appear to change with age when expressed as the percentage of $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}(2,14)$. For example, Allen et al. (2) showed no differences in the percentage of $\mathrm{VO}_{2}$ max associated with $10-\mathrm{km}$ race pace in highly trained young ( $89 \%$ ) and Masters ( $92 \%$ ) runners. As such, it is plausible that the contribution of lactate threshold to the age-related reduction in endurance performance is mediated, at least to some extent, by the reduction in $\mathrm{Vo}_{2}$ max.
Exercise economy. Finally, exercise economy, defined as the oxygen cost to exercise at a given velocity, is thought to be an important determinant of endurance performance (20, 36). Relatively little is known about the influence of exercise economy on age-related decreases in exercise performance. In a study of male
endurance runners, there was no difference in running economy between young and older runners (2). Similarly, findings on highly trained and competitive female runners aged $35-70 \mathrm{yr}$ indicate that there is no significant relation between running economy and age (58). In a group of highly trained and competitive female distance runners varying in age, we found that running economy explained little additional variance in the age-associated decrease in $10-\mathrm{km}$ running performance after differences in $\dot{\mathrm{V}}_{2}{ }_{2 \text { max }}$ and lactate threshold were considered (14; Fig. 6). Thus the limited experimental evidence in this area suggests that reductions in exercise economy do not significantly contribute to the reduction in endurance performance with advancing age.
Summary. In summary, a progressive reduction in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ appears to be the primary physiological mechanism associated with declines in endurance running performance with advancing age. Given the importance of decreases in $\dot{V}_{2}{ }_{\text {max }}$ in mediating age-associated reductions in exercise performance and PFC, much of our work on this overall topic has focused on reductions in $\dot{V}_{2}$ max with aging. In particular, we have been interested in the potential modulatory influence of habitual aerobic exercise status on the rate of decline in $\mathrm{V}_{\mathrm{O}_{2}}$ max with age.

## HABITUAL EXERCISE STATUS AND MAXIMAL AEROBIC CAPACITY

Background. Maximal aerobic capacity, as measured by $\dot{\mathrm{V}}_{2}$ max, is an important indicator of PFC. Professor Sid Robinson (44) demonstrated in 1938 that $\dot{V o}_{2}$ max declines progressively with advancing age even in healthy adult humans. This decrease in $\mathrm{Vo}_{2}$ max with age has a number of functional and clinical implica-

Fig. 6. Ten-kilometer race pace ( $A$ ) and its physiological determinants. $B$ : maximal $\mathrm{O}_{2}$ consumption ( $\dot{\mathrm{V}}_{2_{\text {max }}}$ ). $C$ : exercise velocity at lactate threshold. $D$ : running economy. (Modified from Ref. 14.)



Fig. 7. Absolute and relative rates of decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2 \text { max }}}$ (A: in $\mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1} \cdot$ decade ${ }^{-1} ; B$ : in $\% /$ decade) with age in sedentary, active, and endurance-trained populations. (From Ref. 16.)
tions because it is associated with increased risks for cardiovascular and all-cause mortality and disability (8), as well as reductions in physical performance, cognitive function, quality of life, and independence ( 7 , 57). Given this, lifestyle factors that may affect the rate of decline in $\mathrm{V}_{2}$ max with advancing age are of considerable public health interest.

With respect to the influence of habitual aerobic exercise status, early investigations reported that the rate of decline in $\dot{\mathrm{V}}_{2} \mathrm{~m}_{\text {max }}$ with age is smaller (e.g., only $50 \%$ as large) in endurance-trained male athletes than that in sedentary men (19, 22, 30). Based largely on these selective observations in men, the concept that the rate of decline in $\dot{\mathrm{V}}_{2}$ max with age is attenuated in adults who perform regular aerobic exercise has been established and widely promoted. Seemingly in contrast to these early observations in men, our group noted that reported rates of decline in $\dot{V}_{2}{ }_{2}$ max with age in physically active women that were greater than
those generally observed for sedentary women ( 4,14 , 58). Based on this apparent inconsistency, we initiated a series of experimental efforts aimed at determining the relation between habitual aerobic exercise and the rate of decline in $\dot{\mathrm{V}}_{2}$ max with age.
Recent findings in female endurance athletes. As an initial step to investigate this issue, our laboratory (16) used a meta-analytic approach in which mean $\dot{\mathrm{V}}_{2}{ }_{2}$ max values of female subject groups across the adult age range were obtained from the published literature. A total of 239 subject groups from 109 studies involving 4,884 subjects were separated into sedentary, active, and endurance-trained cohorts. In striking contrast to the prevalent belief, the results of this analysis (16) supported the opposite idea, i.e., that the absolute $\left(\mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1} \cdot \mathrm{yr}^{-1}\right)$ rate of decline in $\dot{\mathrm{V}}_{2}$ max with increasing age was greatest in endurance-trained women, next greatest in active women, and lowest in sedentary women (Fig. 7). When expressed as percent or relative decrease from mean levels at $\sim 25 \mathrm{yr}$ of age, however, the rate of decline in $\mathrm{Vo}_{2}$ max was similar in the three groups (16).
A well-recognized limitation of meta-analysis is the lack of experimental control primarily as a result of the heterogeneity of methods used among the individual studies comprising the database. Therefore, we reasoned that a well-controlled laboratory-based study was needed to complement the findings of our metaanalysis. Accordingly, our group (53) studied a total of 156 healthy nonobese women who were either highly endurance trained or sedentary. To establish as well as possible that the endurance-trained women were homogeneous across age with regard to relative "eliteness," subjects were matched across the entire age range for age-adjusted world's best $10-\mathrm{km}$ running times. Moreover, to ensure that voluntary maximal efforts were similar, we made certain that respiratory


Fig. 8. Relations between $\dot{\mathrm{V}}_{\text {2max }^{2}}$ and age in endurance-trained and sedentary women. (From Ref. 53.)


Fig. 9. Absolute ( $A$ ) and relative $(B)$ rates of decline in $\mathrm{V}_{2} \mathrm{max}_{\text {mith }}$ with increasing age in the longitudinal study. (From Ref. 13.) $* P<0.05$, vs. sedentary subjects. $\dagger P<0.05$, vs. sedentary subjects baseline values.
exchange ratio and rating of perceived exertion at $\mathrm{Vo}_{2}$ max were not different across age in both groups. Consistent with our meta-analytic findings, the absolute rate of decline in $\dot{\mathrm{V}}_{0_{2}}$ max was greater in the endurancetrained compared with the sedentary women (53) (Fig. 8). Again, the relative (\%) rate of decline in $\dot{\mathrm{V}}_{2}$ max was similar in the two groups.

Given the inherent limitations of cross-sectional comparisons, we deemed that these observations should be confirmed with a longitudinal study design to provide more definitive insight. Recently, our group (13) studied 8 sedentary and 16 endurance-trained women before and after a mean follow-up period of 7 yr . At follow-up, body mass, fat-free mass, maximal respiratory exchange ratio, and maximal rating of perceived exertion were not different from baseline in either group. Consistent with the results of our cross-sectional studies, the absolute rate of decline in $\dot{V}_{2}{ }_{2}$ max was twice as great in endurance-trained compared with sedentary group, with no differences in the relative rates of decline (13) (Fig. 9).
Recent findings in male endurance athletes. In contrast to these findings in women, a meta-analysis of the literature in men indicated no significant difference in the absolute rate of decline in $\mathrm{V}_{2}$ max between endur-ance-trained and sedentary men (60). Given the limitations of meta-analysis and the contradictory findings between men and women, we recently completed a
well-controlled, cross-sectional laboratory-based investigation to obtain further insight into this possible inconsistency. We demonstrated that the absolute, but not relative, rate of decline in $\dot{\mathrm{V}}_{2}$ max with age is greater in endurance-trained compared with sedentary men (41). These results are consistent with recent findings from longitudinal studies that have reported rates of decline in $\mathrm{VO}_{2}$ max with age in endurancetrained men that are greater than those generally reported for sedentary men ( $21,42,56$ ). Interestingly, a similar trend has been observed in an animal study that has reported a greater absolute rate of decline in $\dot{\mathrm{V}}_{2}{ }_{\text {max }}$ with age in trained compared with sedentary rats (61).
Summary. Together, these results indicate that en-durance-trained men and women appear to demonstrate greater absolute rates of decline in $\mathrm{Vo}_{2}$ max with advancing age compared with healthy sedentary adults. However, when expressed as percent reductions from early adulthood, the rates of decline in $\mathrm{Vo}_{2}$ max with aging are similar between endurancetrained and sedentary humans.

## MECHANISMS UNDERLYING THE GREATER ABSOLUTE DECLINE IN $\dot{\mathrm{V}}_{2_{2} \text { max }}$ WITH AGE IN ENDURANCE EXERCISE-TRAINED ADULTS

The prevailing concept of a smaller rate of decline in maximal aerobic capacity with age in endurancetrained adults is logical, based on our understanding of the physiological adaptations to regular aerobic exercise, and is certainly attractive from a preventive gerontology point of view (6). However, several arguments also can be made for hypothesizing greater rates of decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ with age in endurance-trained adults.
"Baseline effect." The first argument involves a baseline effect. That is, individuals with higher levels of $\dot{\mathrm{V}}_{2_{2} \text { max }}$ as young adults demonstrate a greater rate of decline with advancing age. This argument is supported by the observation that, when the baseline effect was removed by expressing the data as relative (percent) changes, the rate of decline in $\dot{\mathrm{V}}_{2}$ max with age was similar in endurance exercise-trained and


Fig. 10. Absolute rates of decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2 \text { max }}}$ in endurance-trained women who reduced and who maintained and/or increased their baseline training volume. (From Ref. 13.) $* P<0.05$ vs. other 2 groups.
sedentary groups $(13,16,41,53)$. An analogy is apparent in the relation between baseline values and changes in $\dot{V}_{2}{ }_{2}$ max with age in men compared with women. Men have higher absolute values of $\dot{\mathrm{V}}_{2}$ max $^{\text {as }}$ young adults compared with women but demonstrate a greater absolute rate of decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ with age compared with women (26). When expressed as percent reductions from young to older adulthood, however, gender-related differences are no longer evident (26). Thus a baseline effect stemming from their markedly higher absolute levels of $\dot{\mathrm{V}}_{2_{2}}$ max as young adults could contribute to the greater absolute rates of reduction in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ with age in endurance-trained adults compared with their sedentary peers.
Decline in the exercise-training stimulus. A second argument involves declines in the intensity and volume of habitual aerobic exercise with advancing age, i.e., the "exercise stimulus." Because sedentary adults, by definition, are not performing regular aerobic exercise, it follows that the magnitude of decline in physical activity with age and, therefore the absolute decrease in the exercise stimulus, would be much greater in regularly exercising individuals.
Indeed, the results of our investigations and others support this idea. In the first meta-analysis study, our group (16) reported a progressive age-related decline in training volume among the available sample of groups of endurance-trained women. However, many studies failed to report training mileage; thus the limited database precluded us from drawing any definite conclusions. In the subsequent laboratory-based cross-sectional study, weekly running mileage, frequency, and training speed all declined significantly with age, and these declines were associated with the corresponding decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ (53). However, the strongest evidence for the influence of declines in the training stimulus comes from the results of a recent longitudinal study from our laboratory (13). Specifically, we found that the greater rate of decline in $\dot{\mathrm{V}}_{2}$ max over a 7 -yr follow-up period in the overall sample of endurancetrained compared with sedentary women was associated with significant reductions in training volume in some of the endurance-trained women. The endurancetrained women who maintained their training volume over the follow-up period demonstrated reductions in $\dot{\mathrm{V}}_{2}{ }_{2 \text { max }}$ similar to those observed in healthy sedentary women (Fig. 10).

These findings are in agreement with longitudinal studies in men, which showed a significant association between expressions of the endurance exercise training stimulus and the rate of decline in $\dot{\mathrm{V}}_{2}$ max with age (42, 56). Together, these results suggest that the greater rate of decline in $\dot{\mathrm{V}}_{2_{2}}$ max with age in the endurancetrained adults may be mediated, at least in part, by a marked age-related decline in their level of training.

Changes in body weight. Because $\mathrm{VO}_{2}$ max is traditionally expressed in units corrected for differences in body weight, it is possible that greater increases in body weight with age in the endurance-trained groups contribute to their greater rates of decline in $\dot{V O}_{2}$ max. However, this does not appear to be the case. Body
mass and fat-free mass are maintained across age in the endurance-trained adults, whereas the sedentary adults demonstrate a significant increase in fat and total body mass across age (53). The age-related increase in fat and total body mass in the sedentary adults should act to increase their rate of reduction in $\dot{\mathrm{V}}_{2}$ max (when expressed in $\mathrm{ml} \cdot \mathrm{kg}^{-1} \cdot \mathrm{~min}^{-1}$ ) compared with the endurance-trained adults.

Physiological determinants. Based on the Fick equation, $\mathrm{VO}_{2}$ max is the product of maximal heart rate, maximal stroke volume, and maximal arteriovenous $\mathrm{O}_{2}$ difference (46). It has been hypothesized that the decline in $\dot{\mathrm{V}}_{2}{ }_{2}$ max with age in trained and untrained adults may be influenced by the corresponding reduction in maximal heart rate (19, 22). However, our laboratory has repeatedly demonstrated ( $13,16,41,53$, 60 ) that there is no relation between reductions in maximal heart rate and habitual exercise status. This indicates that other factors, such as declines in maximal stroke volume or skeletal muscle oxidative capacity, are responsible for differences in the absolute rate of decline in $\dot{V}_{0_{2}}$ max observed in regularly exercising compared with the sedentary adults.

Calculations based on the data of Ogawa and colleagues (37) indicate that their endurance-trained women demonstrated $\sim 50 \%$ greater absolute rate of decline in $\dot{\mathrm{V}}_{2}{ }_{\text {max }}$ compared with their sedentary peers. This was closely associated with $\sim 60 \%$ greater rate of decline in maximal stroke volume in endurancetrained vs. sedentary women (37). In contrast, the age-associated differences in maximal arteriovenous $\mathrm{O}_{2}$ difference were similar in the endurance-trained and sedentary groups. A similarly greater rate of decline in maximal stroke volume was also observed in male cohorts of endurance-trained athletes that they studied (37). These results are consistent with the hypothesis that the greater absolute rate of decline in $\dot{\mathrm{V}}_{2}{ }_{\text {max }}$ in endurance-trained adults is mediated by the greater decline in maximal stroke volume and, therefore, maximal cardiac output. Definitive mechanistic insight into this issue will need to be obtained from future longitudinal studies.

## PERSPECTIVES

It is widely appreciated that the demographics of age in the United States and other industrialized societies are changing dramatically. The percentage of adults aged 65 yr and older will continue to increase for the foreseeable future. Older adults have the highest rates of morbidity, functional disability, loss of independence, and mortality. Per capita, they also utilize by far the greatest percentage of overall health care resources. As such, it is critical that we identify effective strategies to extend the health, functional capacity, and independence of older adults to as close to the age of natural death as possible, as so elegantly argued by Professor Fries (17) in his emphasis of the concept of compression of morbidity. Rowe and Kahn (45) have made similarly important contributions to this conversation through their stimulating discussions promot-
ing the idea of "successful aging." We believe that our work and those of others presented here have several important implications regarding our capacity for maintaining physical function with advancing age and some key physiological determinants of our ability to do so.

First, we found that, although large-muscle dynamic performance declines modestly after $\sim 35-40$ yr of age, performance is relatively well maintained until $\sim 60-$ 70 yr on average ( $11,12,54,55$ ). This suggests that, from a purely physiological perspective, most adults should be able to sustain PFC until this age. When significant loss of physical function is observed before this age, it likely is attributable to the effects of disease, negative lifestyle behaviors (e.g., extreme sedentary lifestyle, marked weight gain, smoking), and/or unfavorable genetics. In contrast, after 60-70 yr of age, exercise performance declines exponentially (11, 12 , $54,55)$. These observations support the hypothesis that significant reductions in physical function during and after the seventh decade of life are mediated, at least in part, by decreases in the intrinsic physiological capacity (reserve) to perform such tasks. Comorbidities and other factors could of course further accelerate loss of PFC during these ages.

Second, age-associated reductions in performance generally appear to be greater in women (11, 54, 55 ). Our data indicate that such gender-related differences may be confined to certain types of large-muscle dynamic activities (e.g., distance running, sprint swimming), whereas reductions in performance with age appear to be similar in men and women in other tasks (endurance swimming performance) (12). These observations support the concept that a greater decline in physical function with aging in females, when observed, may have an underlying physiological basis, depending on the task involved. On the other hand, excessive reductions in functional capacity in women in certain types of activities may be linked to nonbiological influences (e.g., sociological factors that act to reduce habitual physical activity).

A third novel concept supported by our findings is the importance of maximal aerobic capacity in determining age-related changes in large-muscle endurance performance. Indeed, results of investigations in both women (14) and men (2) indicate that even the apparent contribution of reductions in the so-called "lactate threshold" to declines in endurance exercise performance with aging may be secondary to decreases in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$. These findings emphasize the need to identify and apply strategies for optimally maintaining maximal aerobic capacity with advancing age to preserve PFC. They also raise the question of whether higherintensity aerobic exercise, which is known to provide a more effective stimulus for maintaining or augmenting $\dot{\mathrm{V}}_{2}{ }_{\text {max }}(23,48)$, should be considered in developing optimal exercise prescriptions for older adults. This would represent somewhat of a paradigm shift from current recommendations that emphasize daily participation in low- to moderate-intensity aerobic activities for middle-aged and older adults (1, 38).

A fourth new concept advanced by our work is the finding that endurance athletes do not demonstrate attenuated absolute reductions in $\dot{\mathrm{V}}_{2}{ }_{2}$ max with age compared with their sedentary peers but, rather, at least as a group, just the opposite ( $13,16,41,53$ ). However, perhaps the most important observation from this series of recent studies is the fundamental importance of maintaining habitual exercise levels ("training stimulus") in the age-associated decline in $\dot{\mathrm{V}}_{\mathrm{O}_{2} \text { max }}$ in endurance-trained adults. Those who are able to best maintain vigorous and frequent training demonstrated reductions in maximal aerobic capacity with age similar to those of sedentary adults, whereas those who underwent greater reductions in training intensity and volume demonstrate exacerbated declines in $\dot{\mathrm{V}}_{2}{ }_{\text {max }}$ (13). Stated more broadly, it appears that the rate of decrease in maximal aerobic capacity and, therefore, PFC, with age is not inherently different among populations or groups (e.g., sedentary adults vs. endurance athletes). Rather, at least in healthy adults, the rate of decline in $\dot{\mathrm{V}}_{2}$ max with advancing age will be determined largely by the corresponding reduction in the overall habitual exercise stimulus among individuals. Those who undergo the greatest decreases in their exercise volume and/or intensity will demonstrate the greatest age-associated reductions in maximal aerobic capacity and PFC.
Finally, it is important to emphasize that, although the rate of decline in $\dot{\mathrm{V}}_{2}{ }_{\text {max }}$ with age on average is greater in endurance-trained vs. sedentary adults, the former possess higher absolute levels of maximal aerobic capacity and PFC compared with sedentary adults at any age. Thus those who engage in regular exercise are capable of performing physical tasks that cannot be performed by their sedentary peers, at least with the same degree of exertion or effort (59). Moreover, mid-dle-aged and older adults who engage in regular aero-bic-endurance exercise demonstrate much lower prevalence of many chronic degenerative diseases, including cardiovascular disorders, diabetes, and certain types of cancer, than sedentary adults of the same gender and similar age (1, 43). As a result, physically active adults are at lower risk of premature mortality and functional disability compared with their sedentary peers (8). Accordingly, despite the fact that the rate of decline in $\dot{\mathrm{V}}_{2}{ }_{2}$ max with aging is greater in endurance-trained adults who experience significant reductions in their physical training than in the average sedentary healthy adult, overall, individuals who are habitually physically active enjoy a greater PFC and a reduced risk of chronic disease, supporting current recommendations for the importance of exercise in maintaining general health and quality of life (38).

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