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Rate and Mechanism of Maximal Oxygen Consumption Decline with Aging

Implications for Exercise Training

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Abstract

Because of the influence of cardiorespiratory fitness on functional independence, quality of life, and cardiovascular disease and all-cause mortality, tremendous interest has been directed towards describing the age-related change in maximal oxygen consumption ($\dot{V}O_{2max}$). Current evidence supports a 10% per decade decline in $\dot{V}O_{2max}$ in men and women regardless of activity level. High-intensity exercise may reduce this loss by up to 50% in young and middle-aged men, but not older men, if maintained long term. Middle-aged and older women do not appear to be able to reduce loss rates in $\dot{V}O_{2max}$ to less than 10% per decade, which may be related to estrogen status. However, maintaining high-intensity training seems limited to approximately one decade at best and to a select few individuals. While the factors limiting the ability to maintain high-intensity training are not completely known, aging most likely plays a role as studies have demonstrated that training maintenance becomes more difficult with advancing age. Age-related loss of $\dot{V}O_{2max}$ seems to occur in a non-linear fashion in association with declines in physical activity. In sedentary individuals, this non-linear decline generally occurs during the twenties and thirties whereas athletic individuals demonstrate a non-linear decline upon decreasing or ceasing training. Non-linear loss rates are also demonstrated in individuals over the age of 70 years. The decline in $\dot{V}O_{2max}$ seems to be due to both central and peripheral adaptations, primarily reductions in maximal heart rate (HR_{max}) and lean body mass (LBM). Exercise training does not influence declines in HR_{max}, while LBM can be maintained to some degree by exercise. Recommendations for exercise training should include aerobic activities utilising guidelines established by the American College of Sports Medicine for improving CV fitness and health, as well as strength training activities for enhancing LBM.

Of the physiological changes associated with advancing age, those occurring in the cardiovascular (CV) system are among the most clinically relevant. These changes, which include morphological, phys-

ical and functional alterations,[1] dramatically limit the functional capacity of the CV system at advanced age. By age 75 years, over half of the functional capacity of the CV system, defined as the maximal oxygen consumption ($\dot{V}O_{2max}$), has been lost,[2] and older adults commonly demonstrate $\dot{V}O_{2max}$ values that are lower than required for many common activities of daily living.[3] $\dot{V}O_{2max}$ represents the functional limit of the body's ability to deliver and extract oxygen to meet the metabolic demands of vigorous exercise, and is recognised as the international reference standard for physical fitness.[4] As well as presenting challenges to physical independence and quality of life, low cardiorespiratory fitness has been consistently associated with CV disease and all-cause mortality.[5-8] More importantly, these studies have demonstrated low cardiorespiratory fitness to be a strong and independent predictor of mortality risk.[5,7]

Because of these issues, tremendous interest has been directed towards describing the age-related change in $\dot{V}O_{2max}$. Beginning with the work of Robinson[9] over 50 years ago, numerous investigations have attempted to describe loss rates in $\dot{V}O_{2max}$ in an attempt to ascertain the influence of age on CV function. However, these efforts are complicated by the multiple factors that influence functional declines in physiological systems, including aging, disuse and disease.[10] It has been proposed that normal, or true, aging rates unaffected by disuse and disease occur in a linear fashion,[11] at a rate of 5% per decade for $\dot{V}O_{2max}$.[10] In addition, early evidence suggested that reductions in $\dot{V}O_{2max}$ with aging were due solely to central adaptations in CV function, in particular reductions in maximal heart rate (HR_{max}).[12,13] This paper will review the current state of the science in relation to aging and $\dot{V}O_{2max}$, focusing on several recent longitudinal investigations. Moreover, the paper will present evidence for proposed mechanisms behind losses in $\dot{V}O_{2max}$ with age, and will conclude with implications for exercise programming.

1. Cross-Sectional Studies

The original work by Robinson[9] produced loss rates in $\dot{V}O_{2max}$ with age in men that approximated 10% per decade. Although the absolute rate of decline in women was lower, relative age-related loss rates in $\dot{V}O_{2max}$ in women were similar to those reported for men.[14] This value (10% decline per decade) has been consistently reproduced in sedentary and physically active men and women on a worldwide basis in cross-sectional studies that have followed (table I).[15-24]

In contrast, early studies of athletic populations generally produced loss rates in $\dot{V}O_{2max}$ that were lower than 10%. Shephard,[15] in a compilation of literature from throughout the world to calculate loss rates with age, suggested that athletic males demonstrated a loss rate of 6% per decade. Although the loss rate for the entire athletic sample was 10% in a study by Pollock et al.,[25] this value was inflated by a 22% per decade rate of loss in athletes over 70 years of age. Those athletes under age 70 years demonstrated a 6% per decade loss rate in $\dot{V}O_{2max}$. Three other early studies suggested reduced loss rates in $\dot{V}O_{2max}$ in athletes similar to those reported by Shephard and Pollock et al. ranging from 5% to 7%.[12,26,32] These data have been widely interpreted to suggest that vigorous exercise reduced age-related loss of $\dot{V}O_{2max}$ by 50%.

One of the early studies of athletic populations did produce relative loss rates in $\dot{V}O_{2max}$ that were similar to those reported for sedentary and physically active individuals.[2] Additionally, more recent investigations involving athletic populations have demonstrated relative loss rates identical to those seen in sedentary individuals.[27,29-31] Explanations for the conflicting results include small sample sizes, limited age ranges and the lack of a sedentary control group.[27] Many of the early studies did have small sample sizes, which becomes more significant when one considers that cross-sectional designs are particularly vulnerable to producing unrepresentative samples due to selection bias. Therefore, one of the strengths of the more recent investigations[27,29-31] is a significantly greater sample size than previous cross-sectional investigations (table

Table I. Cross-sectional rates of change in $\dot{V}O_{2max}$ and HR_{max} in sedentary, active and athletic individuals

Study	n	Sex	Age (y)	Exercise status	$\dot{V}O_{2max}$ decline/ decade (%)	HR_{max} decline/ decade (%)	Disease excluded
Pollock et al.[25]	25	M	40–75[a]	MA	10	2.5	Yes
Drinkwater et al.[16]	122	F	20–68[a]	Mixed activity	10	NA	Yes
Barnard et al.[2]	13	M	40–78[b]	MA	11.5	5	Yes
Heath et al.[12]	16	M	59 ± 6	MA	4	4	Yes
	16	M	22 ± 2	YA			
Hossack & Bruce[17]	98	M	20–75	Sed	10 M	5 M	Yes
	104	F	[b]		9 F	3 F	
Fuchi et al.[26]	55	M	30–80[a]	MA	7	3	Yes
Inbar et al.[19]	1424	M	20–70[a]	Sed	7	3	Yes
Toth et al.[20]	378	M	17–80	Mixed activity	8 M	NA	Yes
	224	F	18–81		10 F		
Jackson et al.[21]	1499	M	25–70[a]	Mixed activity	10	4	Yes
Jackson et al.[22]	409	F	20–64[a]	Mixed activity	10	4	Yes
Cunningham et al.[23]	124	M	55–86[b]	Mixed activity	11 M	NA	Yes
	97	F			11 F		
Tanaka et al.[27]	84	F	20–75[a]	MA	9.7 MA	3 MA	Yes
	72	F		Sed	9.1 Sed	3 Sed	
Rosen et al.[28]	276	M	45–80[b]	Mixed activity	9 MA	NA	Yes
				Sed	9 Sed		
Wiebe et al.[29]	23	F	20–63[c]	MA	9	5	No
Schiller et al.[24]	146	F	20–75[a]	Mixed activity	9	4.5	Yes
Wiswell et al.[30]	146	M	40–86[b]	MA	12	NA	Yes
	82	F			8		
Pimental et al.[31]	153	M	20–75[b]	MA	11 MA	3.5 MA	Yes
				Sed	11 Sed	4 Sed	

a Grouped by decade.

b Regression.

c Grouped by age.

F = females; **HR_{max}** = maximal heart rate; **M** = males; **MA** = master athletes; **NA** = not available; **sed** = sedentary; **$\dot{V}O_{2max}$** = maximal oxygen consumption; **YA** = young athletes.

I). The dissimilarities noted could also be related to training duration reported as years of training. In the only early study demonstrating ≥10% loss rates per decade in $\dot{V}O_{2max}$ in athletic adults, the authors reported that half of the individuals had trained for more than 25 years consecutively.[2] Similarly, Wiswell et al.[30] reported greater than 15 years as the average training duration with a large range and standard deviation. Therefore, a significant number of those individuals had been training for over 20 years. It has been suggested that maintaining training volume and intensity is extremely difficult for periods well over 10 years.[33] Therefore, the studies demonstrating $\dot{V}O_{2max}$ loss rates of ≥10% in athletic individuals may reflect decreased training in addition to aging.

2. Longitudinal Studies

Because of the inherent bias associated with cross-sectional research, longitudinal design studies have been suggested to be a more valid means of assessing age-related changes in physiological function.[34] However, it must be considered that longitudinal studies are susceptible to similar bias due to subject mortality.[35] Therefore, when interpreting the results of longitudinal studies, it is important to consider the number of individuals who dropped out along the way (table II). Despite this limitation,

Table II. Longitudinal rates of change in $\dot{V}O_{2max}$ and HR_{max} in sedentary, active and athletic individuals

Study	n		Sex	Age (y) T1	Exercise status	Follow-up (y)	Exercise status	$\dot{V}O_{2max}$	HR_{max} decline/	Disease
	T1	T2			T1		T2	decline/decade (%)	decade (%)	excluded
Astrand et al.[36]	86	66	M & F	20–33	Active	21	Active	9 M	3 M	Yes
								11 F	4 F	
Kasch & Wallace[37]	NA	16	M	32–56	Active	10	Active	2	4	Yes
Robinson et al.[38]	89	37	M	18–22	Mixed activity	31	Mixed activity	8	3	No
Robinson et al.[39]	16	13	M	20–54	YA	32	Mixed activity	13	1	No
Plowman et al.[35]	122	36	F	18–68	Mixed activity	6.1	Mixed activity	10		Yes
Pollock et al.[40]	25	22	M	40–75	MA	10	MA	1	4	Yes
							Active	12.5	4	
Rogers et al.[41]	29	29	M	47–84	MA	8	MA	5.5 MA	6 MA	Yes
					Sed		Sed	12 Sed	0 Sed	
Marti & Howald[42]	53	50	M	26.7 ± 4.3	YA	15	Athletic	8 Athletic	3 Athletic	Yes
				19.7 ± 3.3	Sed		Sed	11% Sed	5 Sed	
Kasch et al.[43]	NA	30	M	48 ± 7	Active	23	Active	7 Active	NA	No
						18	Sed	21 Sed		
Kasch et al.[44]	NA	24	M	33–57	Active	28	Active	5 Active	5 Active	No
							Sed	19 Sed	8 Sed	
Trappe et al.[45]	>100	53	M	18–55	YA	22	Athletic	6 Athletic	3 Athletic	Yes
					MA		Active	10 Active	3 Active	
							Sed	15 Sed	2 Sed	
							AO	15 AO	5 AO	
Hagerman et al.[46]	9	9	M	23.8 ± 1.5	YA	20	Mixed activity	15	7	Yes
Pollock et al.[33]	25	21	M	40–75	MA	20	MA	15 MA	4 MA	No
							Active	14 Active	4 Active	
							Sed	34 Sed	4 Sed	
Kasch et al.[47]	NA	11	M	33–56	Active	33	Active	7	5	No
Katzel et al.[48]	160	89	M	50–79	MA	9	MA	29 MA	4	No
					Sed		Sed	15 Sed		
Hawkins et al.[49]	228	135	M & F	40–86	MA	8.5	MA	24.5 M	4 M	Yes
								11 F	5 F	
Eskurza et al.[50]	24	24	F	40–78	MA	7	MA	18 MA	5 MA	Yes
					Sed		Sed	15 Sed	5 Sed	

AO = active older; **F** = females; **HR_{max}** = maximal heart rate; **M** = males; **MA** = master athletes; **NA** = not available; **sed** = sedentary; **T1** = test 1; **T2** = test 2; **$\dot{V}O_{2max}$** = maximal oxygen consumption; **YA** = young athletes.

longitudinal investigations offer the advantage of paired observations of not only $\dot{V}O_{2max}$, but also factors that could account for some of the changes observed in $\dot{V}O_{2max}$.

Table II summarises the results of many of the longitudinal studies investigating $\dot{V}O_{2max}$ changes with aging. It has generally been reported that longitudinal rates of loss, while demonstrating significant variance, are greater than cross-sectional rates of loss.[35,51] However, for sedentary and physically active individuals, most longitudinal studies to date report results similar to those produced by cross-sectional data, reflecting loss rates of approximately 10% per decade from age 20 to 60 years.[35,36,41,42,45] Studies that began with older individuals at the initial visit tend to show higher relative loss rates over time of approximately 15% per decade in sedentary individuals,[48,50] as do studies of former elite athletes who reduce training intensity to more recreational levels.[39,46] Interestingly, the study of Plowman et al.[35] showed no significant loss in $\dot{V}O_{2max}$ in women aged in their twenties despite mixed activity levels, whereas beyond 30 years of age loss rates of approximately 10% per decade were uniformly noted. This supports the notion that declines in $\dot{V}O_{2max}$ do not begin until the third decade of life.

A notable exception to loss rates of approximately 10% per decade in sedentary and active individuals was the series of studies performed by Kasch and colleagues from 1976–99.[37,43,44,47] The authors followed a group of participants involved in a moderate exercise programme for up to 32 years, reporting on subsets of the group at various intervals. At 10 years, Kasch and Wallace[37] reported no significant change in $\dot{V}O_{2max}$ in 16 individuals who were said to maintain training volume and intensity during that time frame. Although the individuals were reported to have been physically active prior to beginning the formal exercise programme, there was clearly a training effect as over half of the individuals demonstrated increases of greater than 10% in $\dot{V}O_{2max}$ during yearly testing that took place throughout the study time period. Therefore, the loss rate reported (2% per decade, not significant) was diluted by the training effect. In subsequent reports, the authors compared individuals who had maintained the exercise programme to those who had dropped out. These comparisons demonstrated 3-fold greater loss rates in those who became sedentary, and suggested loss rates of only 5–7% per decade in the exercisers in relative $\dot{V}O_{2max}$.[43,44,47] However, at all subsequent time points, the exercisers had lower bodyweights, and loss rates in absolute $\dot{V}O_{2max}$ were on the average of 10% per decade in the exercisers. Reduced bodyweight rather than exercise training may have been responsible, either in part or in whole, for the diminished loss in $\dot{V}O_{2max}$. Additionally, it is not clear how many individuals were included in the original training programme, or how the various samples for each study were gathered. For example, no control individuals were included in the 10- or 33-year follow-ups, whereas the 23- and 28-year follow-ups had controls that appeared to be different individuals at the two time points. Therefore, it is difficult to assess the role of selection bias in determining the outcomes that the authors attribute to exercise.

More varied results are seen in longitudinal reports involving athletic individuals. Several studies suggested significantly lower declines in $\dot{V}O_{2max}$ compared with sedentary individuals ranging from 1–6% per decade.[34,40-42,45] One of these studies was a relatively short follow-up (2.3 years) in middle-aged men that demonstrated rates of loss equivalent to 1.4% per decade in active individuals (n = 24) versus 5.1% per decade in inactive individuals (n = 8).[34] The study of Trappe et al.[45] demonstrated reduced loss rates over a significantly longer time frame (22 years), but only in the younger athletes (45.3 ± 2.3 years at follow-up). In contrast, a group of older athletes (68.4 ± 2.7 years at follow-up), classified as 'fit older' because of age over 60 years and training durations of 20–25 years, experienced loss rates of 15% per decade in $\dot{V}O_{2max}$ during the 22 years of follow-up. The athletes in the study by Marti and Howald[42] were also younger (41.7 ± 4.3 years at follow-up), and only those few who had maintained high-intensity training demonstrated a reduced loss in $\dot{V}O_{2max}$ (n = 5, 2% per decade) compared with sedentary loss rates. Therefore, the

lower loss rates reflect only a few young and middle-aged individuals.

Two other early studies demonstrated reduced loss rates in $\dot{V}O_{2max}$ in master athletes followed for relatively long periods of time (8 and 10 years, respectively).[40,41] Rogers et al.[41] compared master athletes to sedentary men and demonstrated a 50% lower loss rate in the athletes. Theirs was a relatively young group at follow-up (62 ± 2.3 years), and two of the oldest from the initial testing were excluded because of heart disease. The study of Pollock et al.[40] involved a follow-up of cross sectional data published in 1974.[25] The authors demonstrated no significant loss of $\dot{V}O_{2max}$ in athletes who maintained training over the 10 years of the study, whereas those who had decreased training experienced a loss averaging 13% per decade.[40] However, in further follow-up after 20 years, the group demonstrated different results.[33] In a subset of athletes that maintained high-intensity training and continued to compete, the total loss in $\dot{V}O_{2max}$ over the 20-year time period was 23%. However, these were not the same individuals that comprised the high-intensity training group at the 10-year follow up, as the high-intensity subset at 20-years had demonstrated an 8% decline in $\dot{V}O_{2max}$ at 10 years and a further 15% decline at 20 years. Athletes who had become sedentary over the previous 10 years experienced a precipitous drop of 34% per decade in $\dot{V}O_{2max}$. Interestingly, the loss rates in this study, and those in Hagerman et al.,[46] were not linear. The master athletes of Pollock et al.[33] experienced dramatic declines in $\dot{V}O_{2max}$ in the second decade in association with reduced training, whereas the elite rowers in Hagerman et al.[46] experienced a 20% decline in $\dot{V}O_{2max}$ in the initial 10 years of the study following cessation of their athletic careers, which normalised to 10% in the second decade during which individuals were involved in mixed activity levels.

Three very recent longitudinal studies have also suggested higher rates of decline in older athletes compared with sedentary individuals.[48-50] Reported loss rates in these studies ranged from 18–29% per decade for the entire sample, rates two to three times greater than experienced by sedentary individuals. However, these studies also subdivided the sample into various groups to further examine age-related losses in $\dot{V}O_{2max}$. Two of the studies,[48,50] which included sedentary comparison groups, identified athletes who had not decreased training volume and intensity over the duration of the study (7–9 years). Katzel et al.[48] demonstrated that men who maintained training volume and intensity experienced a decline in $\dot{V}O_{2max}$ of 5.8% per decade, significantly lower than athletes who reduced training and sedentary individuals. However, only seven of the 42 individuals were included in this subgroup. In contrast, Eskurza et al.[50] demonstrated similar loss rates in $\dot{V}O_{2max}$ between sedentary individuals and athletic women who had not decreased training (n = 6 of 16 total). Women athletes who had significantly reduced exercise training experienced the most dramatic losses that were greater than those seen in sedentary individuals.

Hawkins et al.[49] also identified a subgroup of both men (n = 28 of 86 total) and women (n = 16 of 49 total) who did not experience significant decrements in $\dot{V}O_{2max}$ over the time-frame of the study. While these athletes did reduce training volume (kilometres of running per week), the authors suggested no reduction in training intensity as reflected by maximal ventilation. Moreover, men who best maintained lean body mass (LBM) experienced no change in $\dot{V}O_{2max}$, whereas women who maintained training volume and estrogen were more likely to maintain $\dot{V}O_{2max}$. This latter point may aid in understanding the contrasting findings from Katzel et al.[48] and Eskurza et al.[50] regarding sex-related changes in $\dot{V}O_{2max}$. While Eskurza et al.[50] reported that women on hormone replacement therapy (HRT) were not excluded, they did not report the numbers of women taking HRT nor did they perform any subanalyses based on HRT. Hawkins et al.[49] demonstrated that women not replacing estrogen following menopause experienced the greatest decrements in $\dot{V}O_{2max}$ over the 8.5-year study, so perhaps the result of Eskurza et al.[50] was influenced by HRT. Hawkins et al.[49] also subdivided the sample of master athletes by age group, and found that loss rates in $\dot{V}O_{2max}$ increased with age, from rates simi-

lar to those reported for sedentary in the younger master athletes to rates four times greater than sedentary in the older master athletes (figure 1). The older athletes also demonstrated the greatest reductions in training volume and intensity. These findings suggest that aging either accelerates the reduction in $\dot{V}O_{2max}$ or increases the difficulty of maintaining training.

Several observations can be made concerning age-related loss in $\dot{V}O_{2max}$. Current results support the hypothesis proposed by Buskirk and Hodgson[51] of a curvilinear loss rate in $\dot{V}O_{2max}$ related primarily to reductions in physical activity and exercise. The authors proposed that $\dot{V}O_{2max}$ in sedentary individuals declines rapidly in the twenties and thirties related to decreasing physical activity and increasing bodyweight, followed by a slower rate of decline as they age. In active and athletic individuals, the authors proposed that $\dot{V}O_{2max}$ declines slowly as long as exercise is maintained, and accelerates when exercise is reduced or ceased. The studies that report declines of 20–30% per decade most likely reflect the periods of rapid decline associated with reductions in physical activity and exercise. The more recent longitudinal studies reflecting these large loss rates have all been for less than 10 years,[48-50] and likely coincide with a period of declining training and rapid loss. This is supported by the two 20-year studies that included testing at 10 years.[33,46] Both demonstrate loss rates that differ in the two decades studied in association with reduced training. The slower decline proposed in the model of Buskirk and Hodgson[51] is likely reflected by the 10% per decade value demonstrated in many of the reviewed studies, which appears to be similar between active and sedentary individuals. However, at a similar relative loss rate, absolute loss rates will be greater in athletic and active individuals due to greater absolute values of $\dot{V}O_{2max}$. In addition, there appears to be a non-linear increase in $\dot{V}O_{2max}$ loss around 70 years of age.[33,49] As there is evidence of accelerated decrease in training, LBM and other physiological factors beyond 70 years of age, this non-linear increase at old age is likely multifactorial in nature.

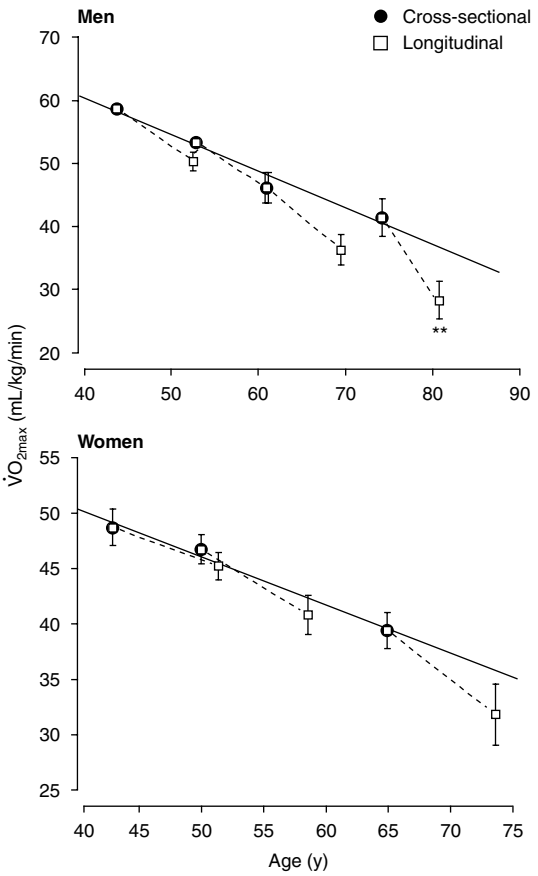

Fig. 1. Cross-sectional versus longitudinal comparison of loss rates in maximal oxygen consumption ($\dot{V}O_{2max}$) [mL/kg/min] in men and women master athletes. ** indicates significantly different rate of loss compared with cross sectional (reproduced from Hawkins et al.,[49] with permission).

Moreover, while there is evidence that high-intensity exercise training, such as that undertaken by master athletes, can reduce loss rates in $\dot{V}O_{2max}$ to below 10% per decade during young and middle age,[40,41,48,49,52] there is no evidence that this type of training can reduce loss rates of older athletes, nor that it can be maintained for periods much longer than 10 years. Explanations for why training maintenance is limited are not available, but they could include aging itself. Therefore, aging may present an indirect as well as direct influence on $\dot{V}O_{2max}$. There is also evidence that age-related loss of $\dot{V}O_{2max}$ may differ by sex, with athletic women less

able to limit decrements than athletic men. This sex difference may be related to HRT. Additionally, it is not completely clear that reduced loss rates in $\dot{V}O_{2max}$ demonstrated by several studies do not reflect a training effect that diluted the true age-related loss.[37,40,43,44,47] Only one longitudinal study actually reported a training effect in their results,[38] but it remains possible that training effects related to increased physical activity/exercise and seasonal variations in testing confound other studies. Therefore, while the 5% per decade value suggested to represent the true age-related decline in $\dot{V}O_{2max}$ may be correct,[10] a value that better reflects the majority of people, including athletic, active and sedentary, may well be closer to 10% per decade (figure 2).

3. Proposed Mechanisms

Aging and alterations in exercise training clearly contribute to reductions in $\dot{V}O_{2max}$. Many studies have attempted to identify the mechanism for the age-related decline, focusing on central and peripheral adaptations to aging that explain the loss of $\dot{V}O_{2max}$. Centrally, HR_{max} declines at a rate uninfluenced by exercise training or sex of approximately 3–5% per decade.[12,21,22,29,36,40,42,45,49,50] While maximal stroke volume (SV_{max}) clearly declines with age in sedentary individuals,[13] the role of exercise in preventing this decline is unclear, as several researchers have demonstrated no change in SV_{max} with age in athletic individuals[12,13] while others have.[29,53] However, older adults, whether sedentary or athletic, rely on the Frank-Starling mechanism to increase maximal cardiac output through increases in SV_{max}.[53] Despite this positive adaptation, maximal cardiac output is reduced in athletic older adults and contributes to age-related decrements in $\dot{V}O_{2max}$.[12,13,29] The relative contribution of the reduction in HR_{max} to reduced maximal cardiac output ranges from 40–100% in various investigations.[12,13,53] Therefore, it is likely that reduced HR_{max} plays a major role in the central adaptations to aging that contribute to reduced $\dot{V}O_{2max}$.

Several studies have also implicated peripheral adaptations with aging that contribute to reduced $\dot{V}O_{2max}$. These clearly involve alterations in body composition, including decreased LBM and increased fat mass.[20-22,49,54,55] The cross-sectional data of Toth et al.[20] demonstrated loss rates in $\dot{V}O_{2max}$ of approximately 9% per decade for both men and women that were reduced to 4% per decade when controlling for changes in LBM and fat mass. Similarly, a recent longitudinal investigation demonstrated that maintenance of LBM was associated with maintenance of $\dot{V}O_{2max}$ in men master runners.[49] Rosen et al.[28] utilised statistical modelling to suggest that 35% of the decline in $\dot{V}O_{2max}$ with age was due to age-associated declines in LBM. Peripheral adaptations with aging also include reductions in maximal arteriovenous oxygen difference, reflecting less oxygen utilisation by skeletal muscle.[29,53] While this could simply be an artifact of age-related reductions in LBM, as previously described, there is evidence that aged skeletal muscle has a reduced aerobic capacity independent of changes in LBM.[55] Whether this reflects less oxygen delivery to the muscle, or an inability of muscle machinery to utilise the oxygen, is at present unclear.

4. Implications for Exercise Programming

While it is not clear that aerobic exercise training provides any long-term benefit in regards to reducing age-related losses in $\dot{V}O_{2max}$, an important point is that in all the studies cited, the $\dot{V}O_{2max}$ of active and athletic individuals was significantly greater than sedentary individuals of similar age.[39,41,48,50] Moreover, the CV system remains fully adaptable to training at any age,[56,57] with relative increases in $\dot{V}O_{2max}$ in adults of any age equivalent to those seen in young individuals. Given the effect of CV exercise training and greater CV fitness on CV disease risk factors,[58-60] CV disease mortality, and all-cause mortality,[5-8] recommending aerobic activities to adults of all ages would seem prudent.[61]

While the minimum effective dosage for intensity and volume that will result in improved $\dot{V}O_{2max}$ and reduced disease and mortality outcomes is not known, current recommendations for improving CV fitness and disease risk are certainly effective.[62]

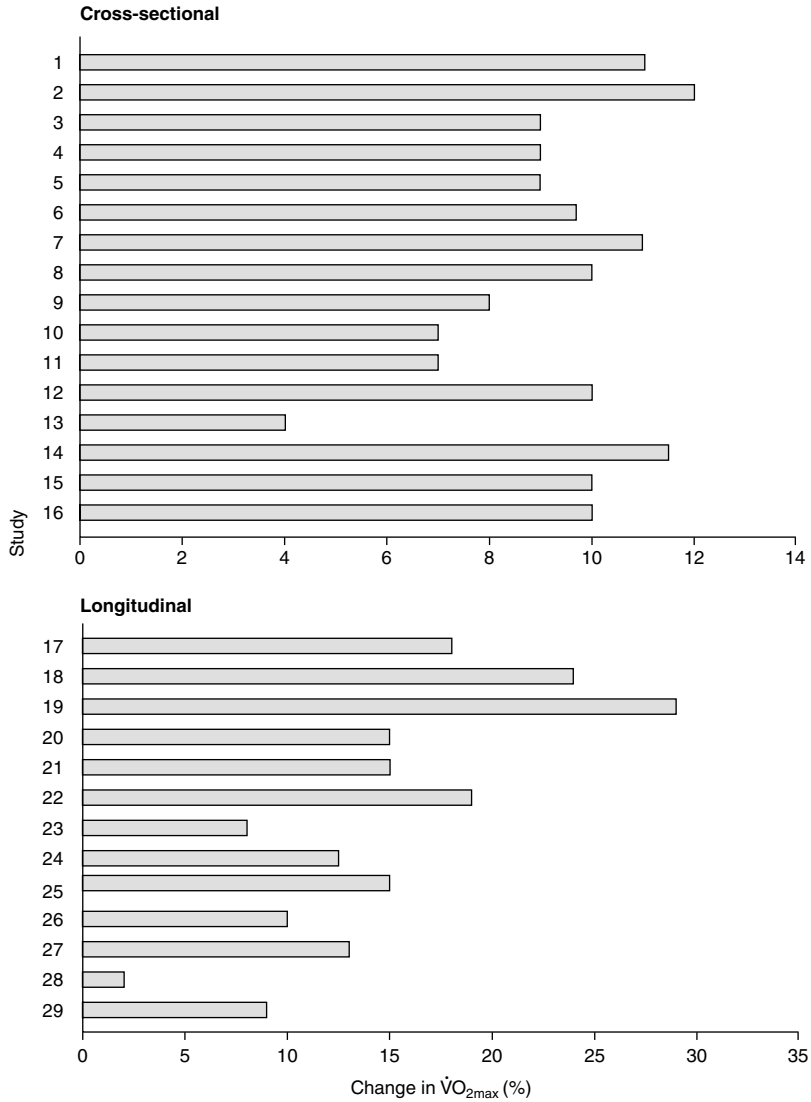

Fig. 2. Cross-sectional and longitudinal studies reporting age-related loss of maximal oxygen consumption ($\dot{V}O_{2max}$) in various populations. **1** = Pimental et al.;[31] **2** = Wiswell et al.;[30] **3** = Schiller et al.;[24] **4** = Wiebe et al.;[29] **5** = Rosen et al.;[28] **6** = Tanaka et al.;[27] **7** = Cunningham et al.;[23] **8** = Jackson et al.;[21] **9** = Toth et al.;[20] **10** = Inbar et al.;[19] **11** = Fuchi et al.;[26] **12** = Hossack and Bruce;[17] **13** = Heath et al.;[12] **14** = Barnard et al.;[2] **15** = Drinkwater et al.;[16] **16** = Pollock et al.;[25] **17** = Eskurza et al.;[50] **18** = Hawkins et al.;[49] **19** = Katzel et al.;[48] **20** = Hagerman et al.;[46] **21** = Trappe et al.;[45] **22** = Kasch et al.;[47] **23** = Marti and Howald;[42] **24** = Rogers et al.;[41] **25** = Pollock et al.;[33] **26** = Plowman et al.;[35] **27** = Robinson et al.;[39] **28** = Kasch and Wallace;[37] **29** = Astrand et al.[29]

These recommendations call for 15–60 minutes of aerobic activities that require large muscle, rhythmic movement, 3–5 days per week, at an intensity equivalent to 40–85% of $\dot{V}O_{2max}$. The implication is that exercise of a very high intensity and volume, such as would be undertaken by athletes, is not a requirement for significant improvements in CV performance and health. However, in light of more recent

guidelines issued by the Centers for Disease Control and Prevention/American College of Sports Medicine (ACSM) recommending light- to moderate-intensity physical activity on a more frequent basis to optimise health,[63] it should be pointed out that the authors clearly indicate this guideline is insufficient to stimulate improvements in $\dot{V}O_{2max}$, which should provide a major focus of aerobic exercise prescription for adults of all ages.[61] The ACSM guideline[62] should therefore provide the basis for exercise prescription for most adults.

Additionally, given the contribution of declines in LBM to age-related losses in $\dot{V}O_{2max}$, it would be advisable to recommend some form of strength training. Strength training leads to increased muscle strength and muscle mass, and has been shown to be effective even at older ages in both men and women.[64,65] Endurance training does not appear to provide a sufficient stimulus for increasing muscle mass and muscle strength,[65] and results for the ability of endurance training to maintain muscle mass and strength are mixed.[66-68] Recommendations for strength training for general fitness are similar regardless of age, and include 1–3 sets of 8–15 repetitions at 70–80% of one repetition maximum (the maximum amount of resistance an individual can control during one repetition of an exercise) 2–3 times per week.[69] One repetition maximum may be extremely low in older adults, and for frail elders beginning levels of intensity and frequency may need to be reduced. Resistance exercise bands, dumbbells and strength exercise machines can all be used effectively for strength training in adults.

5. Conclusions

Age-related loss of $\dot{V}O_{2max}$ seems to occur in a non-linear fashion in association with declines in physical activity/exercise. In sedentary individuals, this non-linear decline generally occurs during the twenties and thirties, whereas athletic individuals demonstrate a non-linear decline upon decreasing or ceasing training. Non-linear loss rates are also demonstrated in individuals beyond the age of 70 years. The normal, age-related decline in $\dot{V}O_{2max}$ is reported at approximately 10% per decade regardless of activity level, although there is some indication that maintenance of high-intensity training in middle age can reduce this somewhat in men. Current evidence suggests that middle-aged and older women are unable to limit the rate of loss in $\dot{V}O_{2max}$ to lower than that experienced by sedentary individuals, which may be related to estrogen status. However, maintenance of high-intensity training appears difficult to manage with advancing age or for much longer than 10 years. The decline in $\dot{V}O_{2max}$ seems due to both central and peripheral adaptations, primarily reductions in HR_{max} and LBM. Training does not influence declines in HR_{max}, while LBM can be maintained to some degree by exercise training. Recommendations for exercise training should include aerobic activities utilising the guidelines established by the ACSM for improving CV fitness and health, as well as strength training activities for enhancing LBM.

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