

The [almost] unbelievable effects of a high maximal aerobic capacity on all-cause mortality

PA peterattiamd.com/all-things-vo2-max

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Performance Group by VO₂ max

Age	Low	Below Average	Above Average	High	Elite
Women					
18-19	< 35	35-39	40-45	46-52	≥ 53
20-29	< 28	28-35	36-40	41-50	≥ 51
30-39	< 27	27-33	34-38	39-48	≥ 49
40-49	< 26	26-31	32-36	37-46	≥ 47
50-59	< 25	25-28	29-35	36-45	≥ 46
60-69	< 21	21-24	25-29	30-39	≥ 40
70-79	< 18	18-21	22-24	25-35	≥ 36
≥ 80	< 15	15-19	20-22	23-29	≥ 30
Men					
18-19	< 38	38-45	46-49	50-57	≥ 58
20-29	< 36	36-42	43-48	49-55	≥ 56
30-39	< 35	35-39	40-45	46-52	≥ 53
40-49	< 34	34-38	39-43	44-51	≥ 52
50-59	< 29	29-35	36-40	41-49	≥ 50
60-69	< 25	25-29	30-35	36-45	≥ 46
70-79	< 21	21-24	25-29	30-40	≥ 41
≥ 80	< 18	18-22	23-25	26-35	≥ 36

Table 1. Classification of Cardiorespiratory Fitness by Age and Sex reproduced from Mandsager et al.⁷ VO₂ max (estimated VO₂ peak) in ml/kg/min of oxygen consumption; METS:

metabolic equivalents, with 1 MET equaling 3.5 ml/kg/min of oxygen consumption. Classification (percentile range) is as follows: low (<25th percentile), below average (24th-49th percentile), above average (50th-74th percentile), high (75th-97.6th percentile), and elite (≥97.7th percentile).

A “Goldilocks principle” applies to many health metrics – going either too low or too high can increase the risk of disease and mortality. For instance, when blood pressure is too high, it is a risk factor for heart attacks and strokes, but when too low, it increases the risks of blood clots and fainting. But the Goldilocks rule certainly has exceptions, and with respect to longevity, one of the most notable of these exceptions is maximal aerobic capacity – known as VO₂ max.

Not only has it been shown that increasing VO₂ max is associated with a monotonic risk reduction in cardiovascular mortality, but increasing VO₂ max is also associated with a monotonic risk reduction of *all-cause* mortality (ACM) *at any age*.

What is VO₂ max?

To engage in any aerobic physical activity, whether cycling, hiking up a mountain, or walking around your neighborhood, muscles require the delivery and utilization of oxygen to generate forces that create repetitive movements. This can be measured as the ventilation rate of oxygen, or VO₂, measured in L/min or, when normalized to weight, in ml/kg/min. The *maximum* amount of oxygen that a person can utilize during intense exercise is known as VO₂ max, and it is, generally speaking, the best indicator of aerobic performance and cardiorespiratory fitness (CRF).

VO₂ max is directly related to the body’s ability to deliver oxygen to muscles, which depends very much on cardiac output. Cardiac output, in turn, is highly dependent on maximum heart rate, which is known to decrease with age, so consequently, an individual’s highest achievable VO₂ max also declines with age. While this decline in VO₂ max might mean that you have a lower *absolute* speed that you can briskly walk up a hill in your 70s or 80s than in your 30s or 40s, even beyond exercise and physical activity, we need a certain minimum aerobic capacity to do tasks of daily living, which could include tasks like climbing a set of stairs or grocery shopping.

How is VO₂ max measured?

The most accurate way to measure VO₂ max is in an exercise lab. In this setting, the subject wears a heart rate monitor and a tight-fitting mask to measure oxygen consumption and carbon dioxide (CO₂) production while performing progressively higher-intensity exercise (usually either running on an inclined treadmill or cycling on a stationary bike) until failure.

A VO₂ Master, an at-home device for measuring VO₂, uses the same approach as lab tests and thus represents a highly accurate option for at-home measurement. (After the device was recently recommended to me by Olav Bu, I could not resist getting one, and have found that during a variety of different exercises like Zone 2, hill intervals, and playing with my kids, the VO₂ Master seems to be accurate within 50-100 ml O₂ for absolute VO₂, which is a stunning

technical achievement.) However, this device is mainly for those who are highly interested in *frequently* monitoring their VO_2 , such as coaches or weirdos like me. If you only plan to test your VO_2 max once or twice per year, you're better off finding an exercise lab, as the cost of the device is more than 25 times the cost of a lab test, which runs about \$150-\$250 per test.

While lab testing or the VO_2 Master are direct ways to measure VO_2 max, these testing methods are not always accessible or convenient. Although less precise, several other at-home methods can provide a basic estimate of your VO_2 max with greater convenience. One of the most accurate cycling power tests requires increasing power output on a stationary bike, with each level sustained for 2.5 minutes.¹ In this case, VO_2 max is related to peak power output. Other estimators of VO_2 max use a 12-minute run, a 1.5-mile run, or a 1-mile walk (the submaximal version of the test).²⁻⁴ Several online calculators allow you to determine your results and where you stand relative to others of your age and sex. For all of these at-home methods, the results are entered into an equation that converts your performance into an estimated VO_2 max.

There is often a learning curve to doing these tests. This is in large part because the first time you do the 12-minute run, you might misjudge the intensity that you can sustain for that time. This could mean either starting too fast and burning out or beginning at a slow pace and not making a true maximal effort. So although not necessarily pleasant if you are doing a maximal effort test, replicating the test a few times on different days is the best way to get the most accurate assessment, as your results will likely converge with repeated efforts.

What evidence links VO_2 max and mortality risk?

We have known for decades based on published studies that high cardiorespiratory fitness as measured by maximal exercise testing is a predictor of both cardiovascular and all-cause mortality. However, many of these studies stratified participants into very broad groups, such as percentiles of the lowest 20%, middle 40%, and highest 40%. Even in these wider-encompassing groups, the group with the lowest CRF had a relative risk of ACM that was 69% higher than the group with the highest CRF, and even as high as a 2.1-fold relative risk increase in ACM in women, with risk increases in cardiovascular death by similar magnitudes.^{5,6} While being in the top 40% of CRF for your age and sex is certainly better than being in the bottom 20%, what these older studies don't indicate is if there was a gradient of risk reduction *within* the highest 40% – that is, does further improvement in CRF lead to further risk reduction, even for those who are already above average?

This question has been answered by two very large studies published in the past few years. In 2018, Mandsager and colleagues published a retrospective study of more than 120,000 adults undergoing treadmill exercise testing, a common method for measuring VO_2 max.⁷ Based on each participant's treadmill grade and speed at peak exercise, a peak energy expenditure in metabolic equivalents (METs) was calculated, which can be converted linearly and directly to VO_2 max by a multiplication factor of 3.5. After a follow-up of over 8.4 years, the authors showed that those who were in the *top 2.3 percentile* (deemed the “elite” category as shown in Table 1 below) for VO_2 max by age and sex had the lowest risk of both cardiorespiratory and all-cause mortality. When compared to individuals in the elite category, the lowest 25% of

cardiorespiratory fitness (the “low” group) had more than a *five-fold* increase in risk of ACM, meaning that someone in the lowest category of fitness was *five times* more likely to die of any cause than someone in the elite category. Likely some of the increase in mortality risk can be attributed to a decreasing prevalence of comorbidities (i.e., hypertension, diabetes) with increasing cardiovascular fitness.

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Even the second highest group (those in the 75th to 97.6 percentiles – deemed the “high” group) had a 29% increase in ACM compared to the elite individuals, though the difference in risk between these two levels of fitness was only significant for those in age groups over 70 years. However, this certainly isn’t to say that there is no benefit of being at a higher fitness level as a younger person! It’s far more likely that the lack of statistical significance in younger age groups in this study can be attributed to the fact that *both* groups of fit, younger individuals were less likely to die in the finite follow-up period (which spanned less than a decade), so the total number of deaths wasn’t great enough to achieve separation between groups.

To appreciate the astounding magnitude of this association between low VO₂ max and mortality risk, we need only look at how it compares with *other* predictors of ACM. From the data shown in Figure 1, what becomes abundantly clear is that being unfit has a *much* greater influence on lifespan than comorbidities like hypertension, diabetes, coronary artery disease, *or even smoking*.

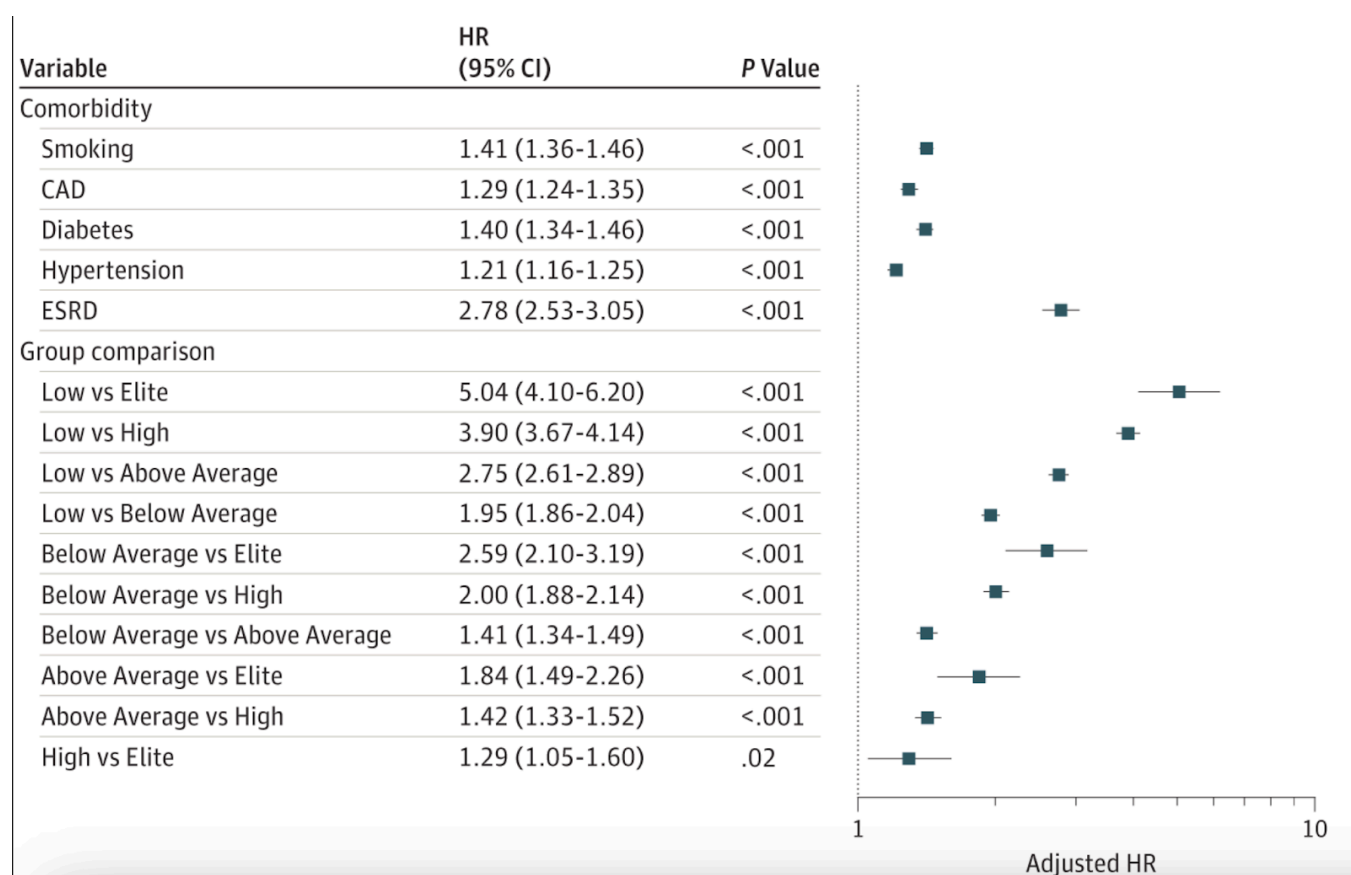


Figure 1. Adjusted hazard ratios (HRs) for comorbidities and between performance groups. Error bars indicate 95% CIs. Performance group classifications by cardiorespiratory fitness are defined in Table 1. Figure adapted from Mandsager et al.⁷

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Such a large study with significant results is compelling on its own, but these findings were further corroborated by an *even larger* study published in 2022 by Kokkinos and colleagues.⁸ In this study (of a non-overlapping population with the Mandsager study), VO₂ max was also estimated from peak METs achieved during a standardized treadmill test, this time in more than 750,000 subjects followed for a median of 10.2 years. Across *all* age groups (including those 80-95 years old) the least fit group – with a VO₂ max in the 20th percentile – had a more than *four-fold increase* in risk of ACM compared to those in the extremely fit group (≥98th percentile), a trend which also persisted across stratifications by race and sex.

In concordance with the findings by Mandsager and colleagues, Kokkinos et al. also found that comorbidities – including smoking, diabetes, and cancer – all increased the risk of ACM *less* than being at or below the 80th percentile for CRF. Even *age* was a worse predictor of ACM than VO₂ max (Figure 2). Let that sink in; *having poor cardiovascular fitness is more strongly associated with the risk of death than being old*. And in contrast to the inevitability of advancing age, we have a degree of control over CRF through aerobic exercise. Even if it is not feasible to reach the highest category of CRF for your age, improving from the 20th to even the 60th percentile will result in an approximately 50% reduction in risk of ACM. In short, if you want to live longer, consistently engaging in aerobic exercise is key.

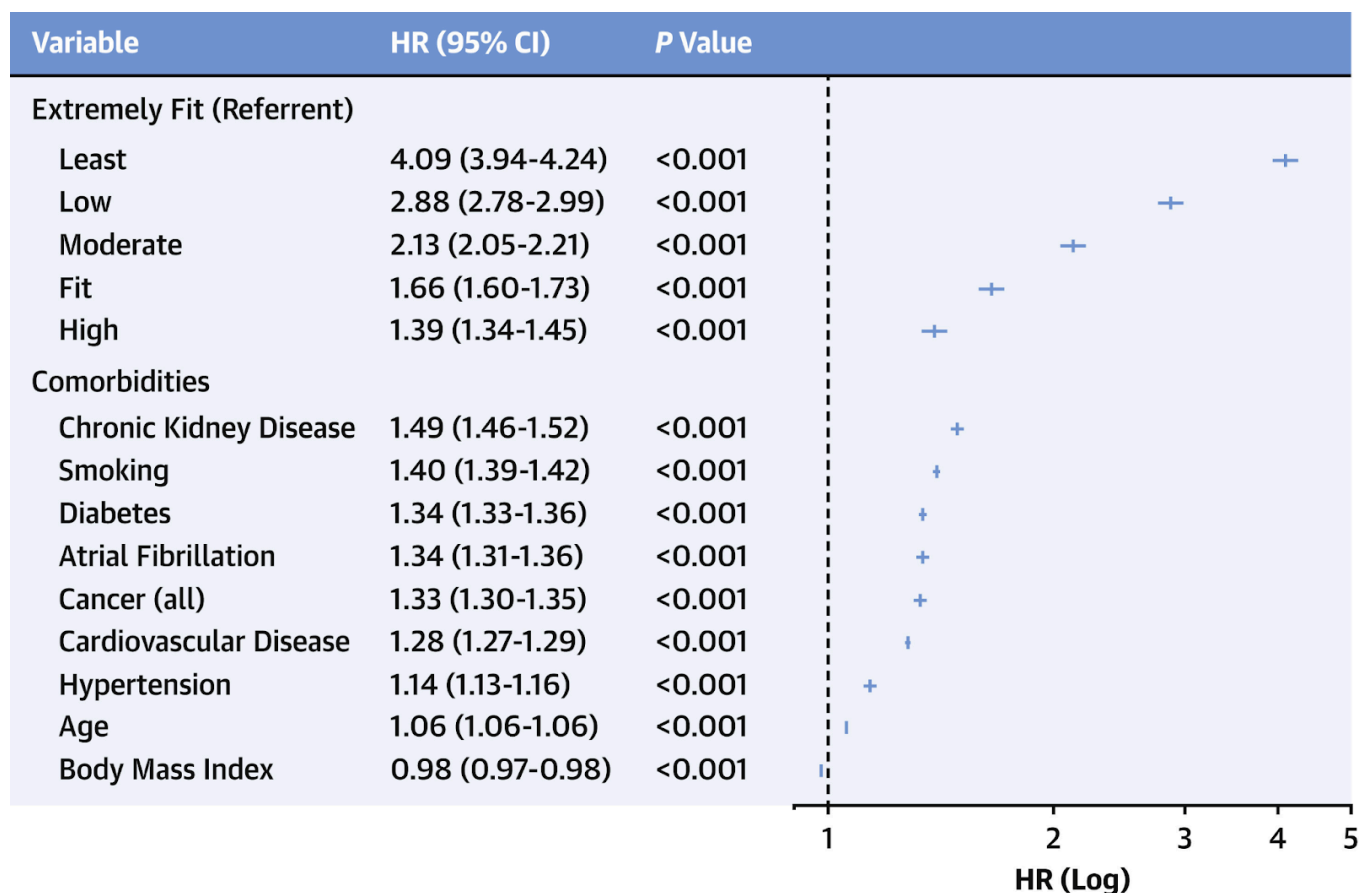


Figure 2. Adjusted hazard ratios (HRs) for comorbidities and between performance groups. Error bars indicate 95% CIs. Performance group classifications by cardiorespiratory fitness are the 20th percentile (Least), 40th percentile (Low), 60th percentile (Moderate), 80th percentile (Fit), 97th percentile (High), and ≥98th percentile (Extremely Fit). Adapted from Kokkinos et al.⁸

Of note, not all studies have reported such a strong, inverse correlation between VO_2 max and mortality risk. Take, as an extreme example, the Copenhagen City Heart observational study of joggers and mortality, in which strenuous joggers (who would likely be equivalent to the higher levels of VO_2 max performers) appear to have a two-fold *increase* in adjusted ACM risk over the *sedentary* reference group.⁹ But looking more closely, we see that the strenuous jogger group only consisted of 36 participants, of which only *two* died in the follow-up period. With such small numbers and no reported cause of death, it is just as possible that these two deaths were anomalies (e.g., a car accident) as it is that they reflect health status. So even though they represent a sizable percentage of the total group, the confidence intervals of this result are gigantic and indicate that this is not a statistically significant finding – and thus, by definition, is not likely to represent a true difference in risk between groups. Indeed, in *any* study, the “elite” category of VO_2 max generally has the smallest number of participants; however, in the studies by Mandsager and Kokkinos, these groups still contain *thousands* of participants. For this reason, we can have higher confidence in their results, as the high number of participants decreases the possibility that any one, anomalous death might skew overall results.

Is the association entirely due to cardiovascular deaths?

The connection between reduced *cardiovascular* mortality and VO_2 max may seem relatively obvious, and since cardiovascular disease is one of the leading causes of death, you might assume that this is the primary reason for the reduction in ACM. However, the influence of VO_2 max on mortality risk likely goes beyond cardiovascular health.

Regular exercise training for increasing VO_2 max results in physiological adaptations that increase one’s capacity to use oxygen to break down and use fuel for energy in a state of stress. Although VO_2 max is measured during an exercise test, the physiologic stress of illness also requires a significant amount of energy to mount an immune response and recover. The higher your VO_2 max, the more reserve or excess energy you have and the smaller percentage of your capacity that has to be used for the process of fighting off pathogens or mounting an inflammatory response. This may be an important contributor to the decreased ACM because those with the highest VO_2 max have a much higher likelihood of tolerating cancer treatments, surviving seasonal viruses or respiratory illnesses, or being better candidates for life-extending surgeries.

Why does VO_2 max seem to be such a reliable metric?

VO_2 max is a uniquely strong predictor of ACM, even relative to other metrics of CRF or metabolism, and this is likely because it provides an integrated metric for fitness and high-intensity aerobic exercise *over a long period of time*. Unlike questionnaires surveying physical activity levels (which can be subject to self-report biases) or measurements of resting heart rate or blood pressure (which can change dramatically in response to acute fluctuations in hydration status, anxiety, etc.), there is no way to “cheat” a VO_2 max test. The only way to achieve a high or elite VO_2 max is to be very aerobically active for many years, which gradually results in denser levels of mitochondria in skeletal muscle, in turn allowing the muscle to use a

greater volume of oxygen to convert into energy. This can only be accomplished with consistent training over time – a few weeks or months of exercise before a VO_2 max test is not going to significantly change mitochondrial function or density.

VO_2 max is also influenced by changes in body composition, which, similar to changes at the cellular level, do not happen on short timescales. From exercise's well-known effects on skeletal muscle, it follows that fat-free mass (FFM) is a strong determinant of VO_2 max. Individuals with a high VO_2 max are indeed likely to have lower amounts of excess fat mass, but either fat mass or percent body fat is generally not a strong predictor of maximal oxygen consumption.¹⁰ This means that if two people have the same total weight, the one with a higher FFM will likely have a higher absolute VO_2 max as well as a higher VO_2 max normalized by body weight (ml/kg/min). Two people with the same amount of lean mass are likely to have similar absolute VO_2 max values, but they will have different normalized VO_2 max levels if they have differing amounts of fat mass. Losing weight usually results in a loss of both lean and fat mass, so increasing your weight-normalized VO_2 max is not as simple as seeing a smaller number on the scale.

Still, though it may not be possible to alter VO_2 max substantially on the timescales of a few days, that is not to say that with consistent training, you can't improve from the lowest levels of CRF relatively quickly. One study in untrained people has shown that only 12 weeks of cycling at 70% of VO_2 max for 45 minutes three times per week can increase VO_2 max by 18-30%.¹¹ This would certainly be enough to move up at least one if not two levels of VO_2 max performance. Regardless, none of these changes are happening in a week or even two, and you're certainly not getting from the lowest to the highest level of fitness without dedicating significant time and effort.

VO_2 max goals and training

Recall that the maximum achievable VO_2 max for any given individual declines with age. Our ability to continue performing the basic activities of daily life into old age (examples shown in Figures 3 and 4) therefore depends on maintaining a significantly above-average VO_2 max throughout our lifetimes. When possible, my goal for myself and my patients is to be in the elite category of VO_2 max for the decade *younger* than you are.

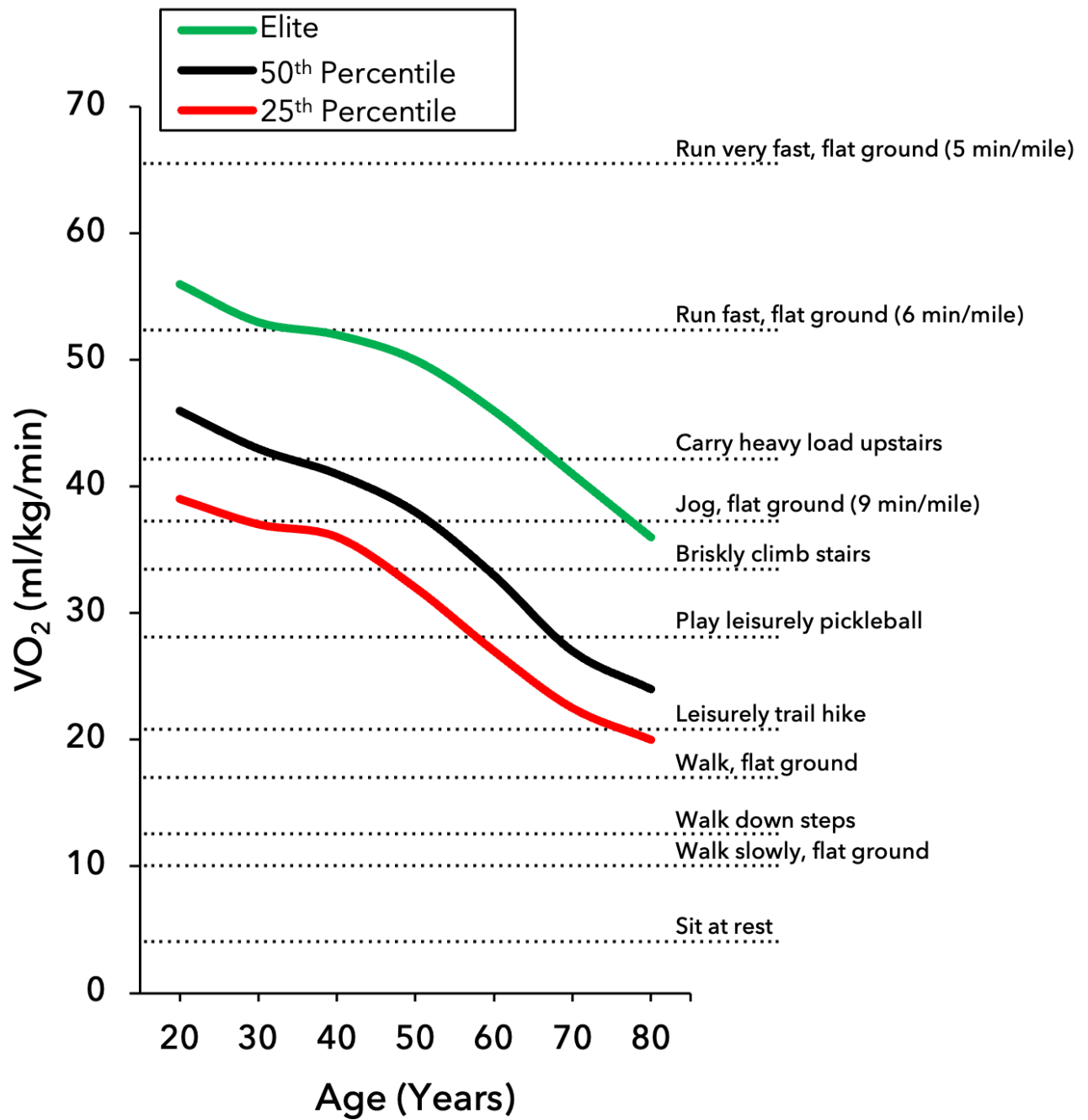


Figure 3. Decline in VO₂ max with advancing age in men. Reproduced from Mandsager et al. and the 2024 Compendium of Physical Activities.^{7,12}

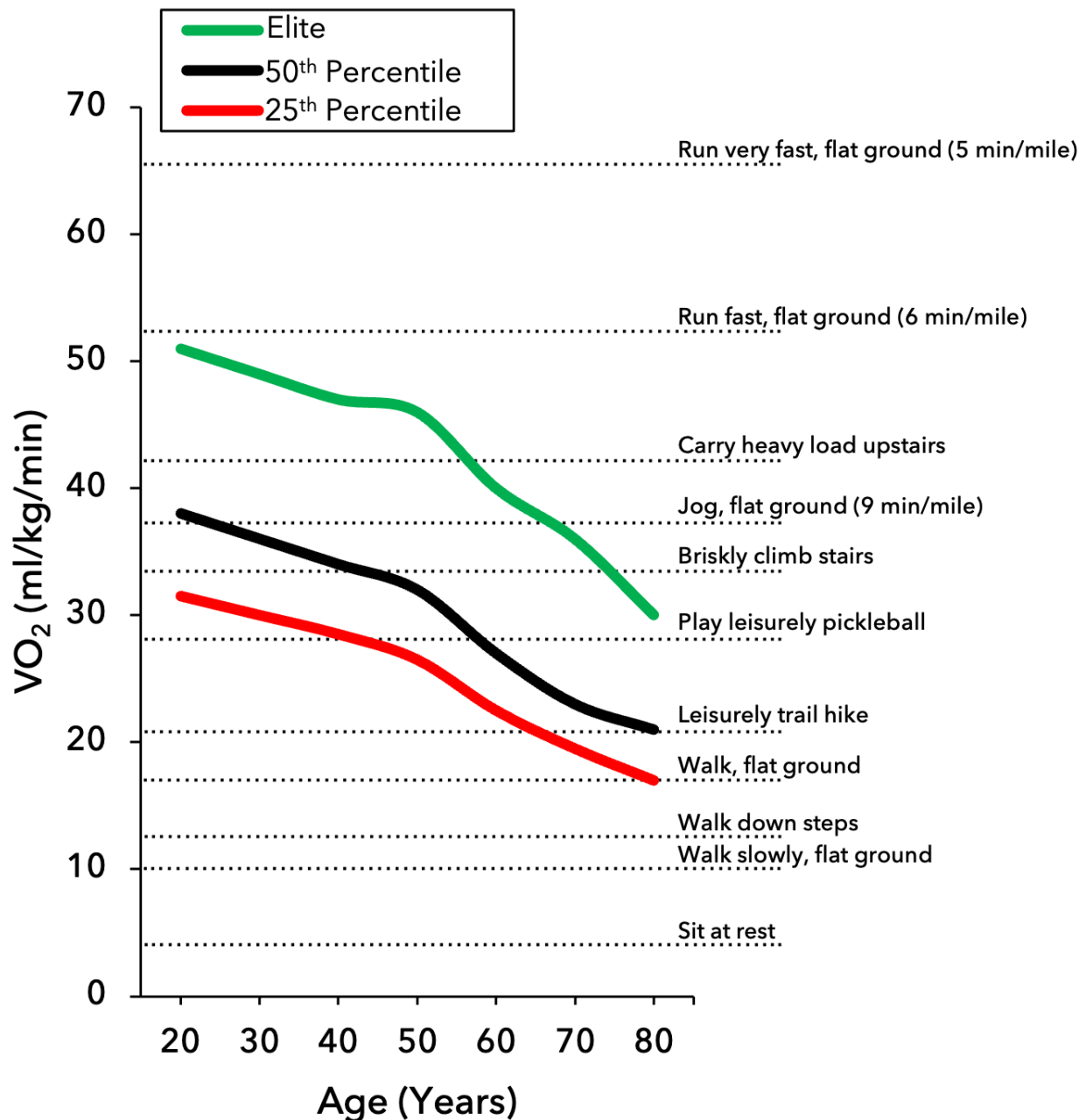


Figure 4. Decline in VO_2 max with advancing age in women. Reproduced from Mandsager et al. and the 2024 Compendium of Physical Activities.^{7,12}

When first starting to exercise (or returning to exercise after a long period away from training), *any* cardiovascular exercise will start to raise VO_2 max, which is why we typically start our new-to-exercising patients with Zone 2 workouts – or even sometimes Zone 1, depending on their baseline fitness level. This helps build consistency and increases exercise tolerance before jumping into higher-intensity training. After having several months of Zone 2 training under your belt, you may be ready to start adding in a day of VO_2 max training to keep improving your VO_2 max.

It has been repeatedly shown that the best way to increase your VO_2 max is with high-intensity interval training (although not necessarily what is called a “[HIIT](#)” class at your local gym). In my conversation with [Mike Joyner](#), we discussed some common interval programs for cardiovascular exercise (e.g., running, cycling, rowing), which usually consist of 3-8 minute intervals, with a 1:1 ratio of high-intensity to recovery time, repeated 4-5 times. The duration and number of intervals as well as the intensity of exercise during recovery will depend on your level of fitness. An elite athlete might run a 4-minute mile four times, interspersed with a slow jog, whereas someone who is just starting interval training might alternate running and walking for 30 seconds or a minute each, possibly with a higher number of intervals.

That being said, there is no “perfect” exercise for training VO_2 max. In the extreme, if you did a single 5-minute interval at your VO_2 max, your oxygen consumption (recall the units of L/min) for that workout would not be very significant – and so this single interval alone wouldn’t provide much stimulus for adaptation. However, if you did six repetitions of high-intensity 5-minute intervals, you would accumulate 30 minutes of work at a power that is much higher than what you would likely be able to sustain for a continuous thirty minutes. The goal of doing repeated, shorter-duration intervals is to *accumulate a greater total amount of time* at a particular level of work (i.e., at or close to VO_2 max) and thus achieve higher total oxygen consumption within a single training session. This could mean many short intervals or fewer longer intervals, and this flexibility can be a useful part of training to see what works best for you.

Training more than VO_2 max

Given the strength of evidence that VO_2 max is associated with a longer lifespan, many are tempted to think that targeted intervals for VO_2 max are the *only* necessary form of cardio exercise. However, developing a high VO_2 max actually requires targeted intervals *and* an enormous volume of lower-intensity training in Zone 2. There are two reasons for this. The first is efficiency and the second is aerobic base. As an example, compare the amateur cyclist to that of a professional. When the professional rides, they are rock-solid on the bike, with almost no perceivable excess movements. The vast majority of the energy they generate goes into the pedals and translates into forward motion. An amateur, by contrast, is often rocking front-to-back, side-to-side, and wasting lots of energy in the process. The efficiency of a professional is only developed through an incredible volume of low-intensity work, during which motor patterns are developed and honed. This is important because it is much more difficult to think about maintaining form during targeted intervals.

Beyond developing efficient form, Zone 2 work increases your *aerobic base*. Aerobic base is the work (i.e., watts) that you can produce during a longer sustained effort. One of the most precise ways to determine if you are in Zone 2 is by measuring blood lactate concentration, with the aim of achieving a lactate level below 2 mmol/L, usually between 1.7-1.9 mmol/L. Lactate testing is inconvenient for most people, but there are several other ways to estimate that you are in Zone 2. One of the methods for determining the intensity level for Zone 2 is by heart rate, using the [Maffetone](#) heart rate formula: $180 - \text{your age} = \text{estimated heart rate}$, which can also be further modified up or down based on your current level of fitness and health. The

other way to determine your Zone 2 intensity is by rate of perceived exertion (RPE). If you are doing a Zone 2 workout by RPE, you should be able to pass the “talk test,” meaning you should be able to talk, but it should not be comfortable at this level of effort. Just because your starting Zone 2 intensity is at one level does not mean it will remain there indefinitely. With consistent training, the goal is to be able to do more work (i.e., generate more power or run at a faster speed) for a given effort indicated by heart rate or RPE. With training, your oxygen consumption at this lower level increases, which translates to an increase in the amount of work you can do for a fixed effort level, and also a higher peak oxygen consumption, $\text{VO}_2 \text{ max}$.

While avoiding overtraining

An increasing body of evidence also suggests that there is such a thing as “too much” high-intensity exercise, at least within a given period of time. Too much high-intensity training can paradoxically reduce performance – a situation called “nonfunctional overreaching” (NFO) – when done in the short-term, but it can turn into overtraining syndrome in the long term, a state characterized by symptoms such as persistent fatigue, increased irritability, persistent stiff or sore muscles, decreased performance and ability to maintain a training regimen, sleep disturbances, decreased concentration, increased susceptibility to illness and headaches, and loss of appetite and weight loss. While definitive diagnoses of NFO/overtraining syndrome are challenging due to the wide range of non-specific symptoms, it’s clear that increasing recovery is crucial to treating NFO/overtraining syndrome. Usually, this requires more rest through decreasing exercise intensity and increasing focus on improving sleep quality, nutrition, hydration, and addressing non-exercise stressors. Since sleep is one of the most important aspects of recovery, upon waking, a combination of objective measures (e.g., resting heart rate (RHR), heart rate variability (HRV)) and subjective measures (e.g., perceived soreness, desire to train) can predict what the potential “cost” of training is that day. A trend of consistently elevated RHR and decreased HRV compared to your baseline, especially when accompanied by other symptoms of NFO, is an indication that high-intensity training may add significant physical stress without having the desired outcomes of this training.

To avoid NFO/overtraining syndrome, it is important not to increase exercise intensity or volume too quickly and to make sure you have sufficient recovery, especially after intense workouts. A state of overreaching has been shown to occur within a week in one study in which participants were doing only interval training an excessive number of times per week. In this study, the number of sessions ramped up each week. Week 1 (two days of 5×4 min intervals) and Week 2 (two days of 5×8 minute and 1 session of 5×4 minute intervals) improved performance, but performance declined after Week 3 (3 days of 5×8 minute and 2 days of 5×4 minute intervals). In addition to the perceivable drop in performance, mitochondrial impairment and reduced glucose tolerance were also observed. Performing the majority of cardiovascular exercise volume at lower intensity levels causes less physiologic stress on the body and allows adequate recovery from less frequent high-intensity efforts.

The bottom line

The current evidence suggests that the most effective way to increase lifespan and healthspan is through consistent aerobic activity over a long period of time (a lifetime, really) to achieve the highest possible VO₂ max. Although absolute maximal aerobic capacity declines with age, consistent and targeted training may provide the energy reserves you'll need to handle temporary physiologic stresses of illness as well as to participate in activities that you enjoy. This ability, to engage in activities with the people you love, is the essence of what comprises *quality* of life, especially in your marginal decade.

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References

1. Hawley JA, Noakes TD. Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *Eur J Appl Physiol Occup Physiol*. 1992;65(1):79-83. doi:10.1007/BF01466278
2. Cooper KH. A means of assessing maximal oxygen intake. Correlation between field and treadmill testing. *JAMA*. 1968;203(3):201-204. <https://www.ncbi.nlm.nih.gov/pubmed/5694044>
3. Weiglein L, Herrick J, Kirk S, Kirk EP. The 1-mile walk test is a valid predictor of VO₂max and is a reliable alternative fitness test to the 1.5-mile run in U.S. Air Force males. *Mil Med*. 2011;176(6):669-673. doi:10.7205/milmed-d-10-00444
4. Kline GM, Porcari JP, Hintermeister R, et al. Estimation of VO₂max from a one-mile track walk, gender, age, and body weight. *Med Sci Sports Exerc*. 1987;19(3):253-259. <https://www.ncbi.nlm.nih.gov/pubmed/3600239>
5. Sui X, Laditka JN, Hardin JW, Blair SN. Estimated functional capacity predicts mortality in older adults. *J Am Geriatr Soc*. 2007;55(12):1940-1947. doi:10.1111/j.1532-5415.2007.01455.x
6. Blair SN, Kampert JB, Kohl HW 3rd, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *JAMA*. 1996;276(3):205-210. <https://www.ncbi.nlm.nih.gov/pubmed/8667564>
7. Mandsager K, Harb S, Cremer P, Phelan D, Nissen SE, Jaber W. Association of Cardiorespiratory Fitness With Long-term Mortality Among Adults Undergoing Exercise Treadmill Testing. *JAMA Netw Open*. 2018;1(6):e183605. doi:10.1001/jamanetworkopen.2018.3605
8. Kokkinos P, Faselis C, Samuel IBH, et al. Cardiorespiratory Fitness and Mortality Risk Across the Spectra of Age, Race, and Sex. *J Am Coll Cardiol*. 2022;80(6):598-609. doi:10.1016/j.jacc.2022.05.031

9. Schnohr P, O'Keefe JH, Marott JL, Lange P, Jensen GB. Dose of jogging and long-term mortality: the Copenhagen City Heart Study. *J Am Coll Cardiol*. 2015;65(5):411-419. doi:10.1016/j.jacc.2014.11.023
10. Goran M, Fields DA, Hunter GR, Herd SL, Weinsier RL. Total body fat does not influence maximal aerobic capacity. *Int J Obes Relat Metab Disord*. 2000;24(7):841-848. doi:10.1038/sj.ijo.0801241
11. Murias JM, Kowalchuk JM, Paterson DH. Time course and mechanisms of adaptations in cardiorespiratory fitness with endurance training in older and young men. *J Appl Physiol*. 2010;108(3):621-627. doi:10.1152/jappphysiol.01152.2009
12. Herrmann SD, Willis EA, Ainsworth BE, et al. 2024 Adult Compendium of Physical Activities: A third update of the energy costs of human activities. *J Sport Health Sci*. 2024;13(1):6-12. doi:10.1016/j.jshs.2023.10.010