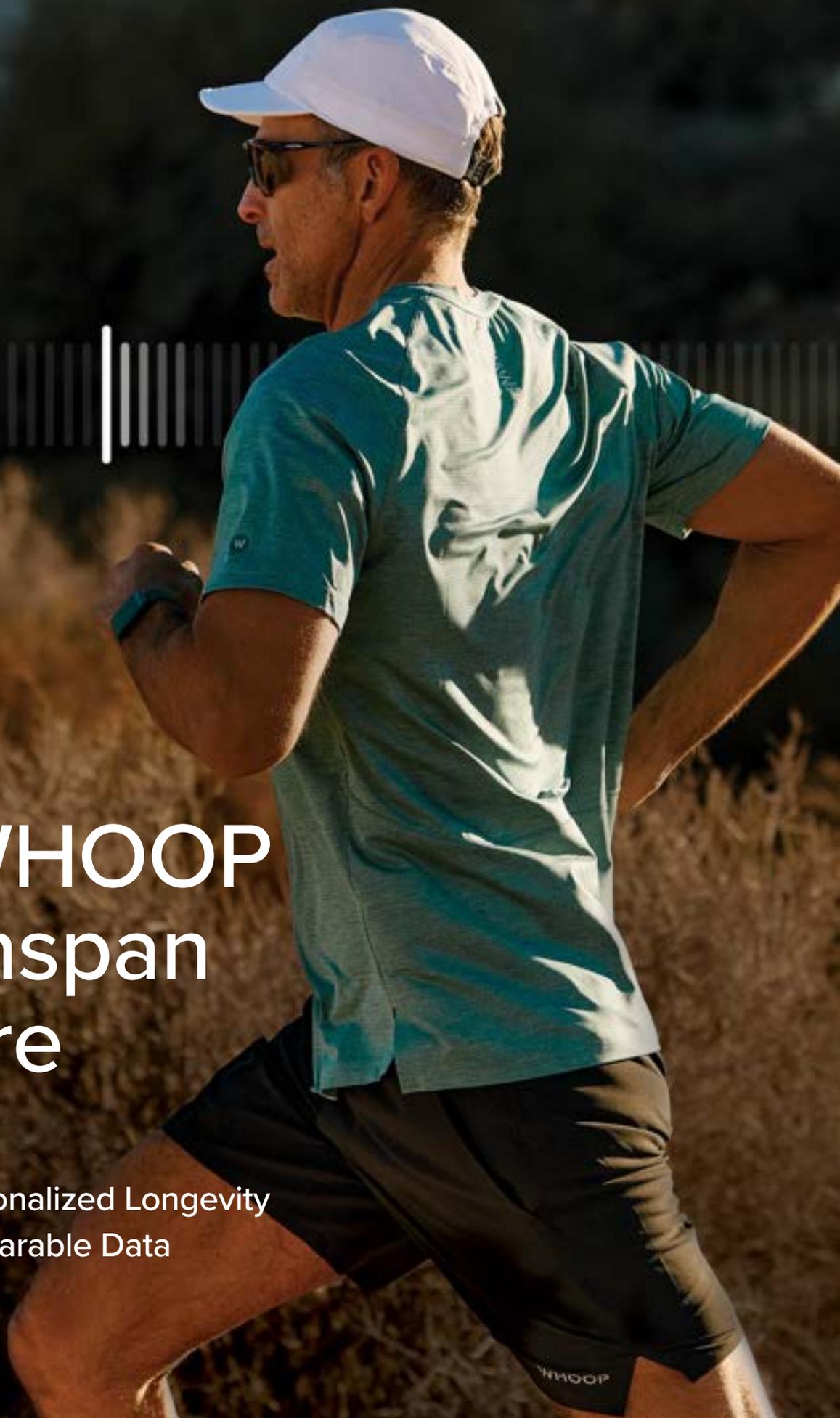


WHOOP®

The WHOOP Healthspan Feature

Advancing Personalized Longevity
Insights with Wearable Data



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Abstract

The WHOOP Healthspan feature provides real-time insights into long-term health risks through WHOOP Age and Pace of Aging, helping individuals understand how their daily behaviors impact their overall well-being. By analyzing key physiological and behavioral metrics — such as sleep, physical activity, and fitness — WHOOP translates complex health data into an actionable framework rooted

in scientific research on all-cause mortality risk. Early findings show that WHOOP Age aligns with self-reported health status and is higher in individuals with chronic conditions, reinforcing its value as a measure of functional health. By offering personalized, data-driven guidance, WHOOP Healthspan empowers individuals to make informed lifestyle choices that optimize longevity and overall well-being.

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WHOOP Age: A New Way to Understand How Today's Choices Shape Tomorrow's Health

We're living through an exciting time in health and wellness. Interest in longevity science is soaring, and new ways to measure biological age are appearing almost daily. But longevity isn't just about living longer — it's about staying healthy and functional for as long as possible. That's healthspan, and it's what truly matters.

WHOOP is taking a major step forward with the launch of the Healthspan feature and a novel, science-based metric: **WHOOP Age**. This is more than just another number — it's a reflection of how your current behaviors are influencing your long-term health and functional capacity.

Built on decades of research in physiology, sleep science, cardiovascular fitness, and aging biology, WHOOP Age gives you a personalized, dynamic picture of how well you're aging — based not on your calendar years, but on

how you live. The core message remains simple and powerful:

Sleep regularly. Move your body. Stay fit. Recover well.

But with WHOOP Healthspan, these principles come alive in a way that's never been possible before. WHOOP gives you real-time feedback on where your daily habits are helping — or hindering — your long-term well-being. In a world full of wellness noise and one-size-fits-all advice, WHOOP Healthspan offers clarity: a personalized, data-driven path to living better, longer.

This isn't about fear of aging. It's about empowerment. It's about giving you the tools to invest in your future — one night of sleep, one workout, one recovery day at a time.



Eric Verdin
CEO of the Buck Institute
for Research on Aging

Introduction

Healthspan, Longevity, and Behavior

A great achievement of the 20th century has been the extension of life expectancy worldwide; however, while people are living longer, they are not necessarily living healthier for longer. Healthspan, the number of years lived without chronic disease or disability, has not kept pace with lifespan (Figure 1). As a result, a growing proportion of life is spent managing illness, Americans can expect to spend 15% of their lives suffering from disease,¹ and by age 65, over 85% of individuals will live with at least one chronic condition.² This growing “healthspan-lifespan gap” is particularly pronounced in countries with the longest life expectancies, including the United States, Australia, and Norway, where chronic conditions such as cardiovascular disease, neurodegenerative disorders, and metabolic syndromes contribute to significant morbidity later in life.¹ As gains in lifespan plateau, the next frontier in longevity science is the extension of healthspan. Recognizing the urgency of this issue, the World Health Organization has proclaimed 2021-2030 as the Decade of Healthy Aging,³ emphasizing the need for interventions that support functional longevity and reduce the burden of age-related diseases.

While genetic predisposition plays a role in disease susceptibility, growing evidence suggests that lifestyle behaviors have an even greater influence on long-term health outcomes. One large-scale epidemiological study demonstrated that individuals who adhere to five low-risk behaviors—healthy diet, regular exercise, avoiding smoking, moderate alcohol intake, and maintaining a healthy weight—live an average of 12-14 years longer than those who practiced none of those habits. Importantly, these behaviors not only extend lifespan but also significantly reduce the risk of chronic disease, such as cancer and cardiovascular disease, thereby prolonging healthspan.⁴ Moreover, environmental and behavioral factors may have a greater impact than genetics on conditions like cardiovascular disease, chronic lung disease, and liver disease. Even for diseases with stronger genetic ties, such as certain cancers and Alzheimer’s disease, premature mortality is still largely influenced by modifiable factors.⁵



Figure 1: Healthspan vs. lifespan



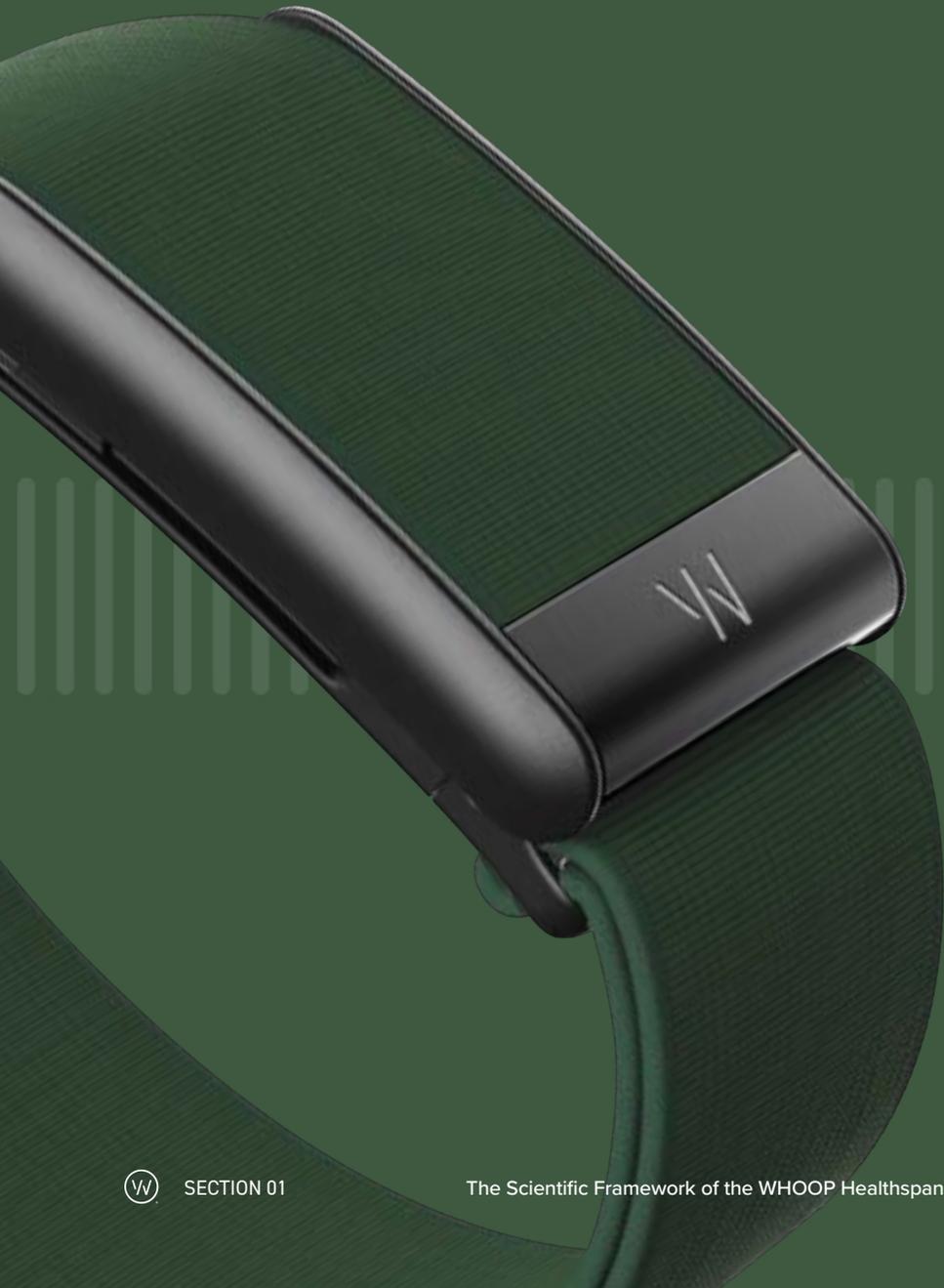
WHOOP Translates Daily Behaviors Into Long-Term Health Insights

Despite strong evidence supporting the role of lifestyle behaviors in longevity, sustained behavior change remains one of the greatest challenges in public health. One reason is that people may struggle to connect daily choices with long-term health outcomes, due to lack of real-time, personalized feedback. Traditional health assessments, such as annual physicals and blood biomarker testing, provide useful clinical insights but offer only intermittent snapshots of health status. These assessments lack the ability to reinforce positive behavior changes in real time, making it difficult for individuals to see the impact of their actions on long-term health risks. For example, high intensity exercise is associated with improved cardiovascular fitness and reduced systemic inflammation, and may manifest in lower levels of inflammatory markers, such as CRP and IL-6.⁶ However, because these biomarkers are measured infrequently in clinical settings, the immediate benefits of increased exercise may not be apparent.

Wearable technology, such as WHOOP, is uniquely positioned to bridge the gap between daily behavior and long-term health by providing continuous, individualized insights. The WHOOP Healthspan feature was designed to advance this mission and help members better understand how their daily choices influence their long-term health. Unlike traditional health assessments that focus on static risk factors, the WHOOP Healthspan feature dynamically tracks and translates daily habits into an ongoing health trajectory. This allows members to see how small, incremental improvements accumulate over time, rather than relying on infrequent clinical assessments. By aligning daily decisions with long-term goals — whether optimizing for athletic performance, overall health, or both — WHOOP Healthspan serves as a data-driven guide to making longevity-focused behavior change more accessible and actionable.

“WHOOP Healthspan serves as a data-driven guide to making longevity-focused behavior change more accessible and actionable.”

The Scientific Framework of the WHOOP Healthspan Feature



The two new Healthspan metrics, WHOOP Age and Pace of Aging, are calculated by leveraging scientific literature on the association between all-cause mortality and the key contributor metrics, such as Heart Rate Zone Time, Sleep Consistency, and VO₂ Max. The WHOOP Healthspan feature contextualizes WHOOP data, utilizing data derived from the sensors (i.e. daily movement, automatic sleep detection, resting heart rate) as well as data input manually (i.e. biological sex, lean body

mass), within the scientific literature on health outcomes. Decades of research have established clear relationships between lifestyle behaviors and physiological metrics with important health outcomes like morbidity and mortality. The Healthspan feature interprets WHOOP metrics in the context of this research, allowing members to better understand how today's behaviors may impact their healthspan and longevity.

The Dynamics of Long-Term Health Risk over the Human Lifespan

The risk of chronic disease and mortality increases progressively over the human lifespan, driven by a combination of biological aging processes, cumulative lifestyle exposures, and the gradual decline of physiological resilience. In early life, the human body maintains a high capacity for cellular repair, metabolic balance, and immune function. However, over time, these systems become less efficient, increasing susceptibility to disease and reducing overall longevity.

Aging is marked by a series of biological changes that compromise health at a fundamental level.⁷ DNA damage accumulates, leading to mutations that increase cancer risk.⁸ Mitochondrial function declines, reducing the body's ability to generate energy and regulate metabolism.⁹ Aging cells become senescent, secreting inflammatory molecules that contribute to the progression of chronic diseases such as cardiovascular disease, neurodegenerative disorders, and type 2 diabetes.¹⁰ Over decades, these processes accelerate, ultimately driving the exponential rise in mortality risk observed in aging populations.

This steady increase in long-term health risks is captured by all-cause mortality, a key metric in aging research that quantifies the overall likelihood of death from any cause within a population. A principle known as the Gompertz Law of Mortality describes how this risk follows an exponential trajectory, roughly doubling every 8–10 years after early adulthood.¹¹ Beginning around ages 20–25, the probability of death increases by approximately 10% per year (Figure 2).^{11–13} The pattern reflects how chronic diseases that are rare before middle age become dominant drivers of mortality later in life. Cardiovascular disease, for example, remains uncommon at younger

ages but becomes the leading cause of death in the U.S. after age 45.¹⁴ Similarly, the risk of neurodegenerative diseases like Alzheimer's rises dramatically in later decades,¹⁵ reflecting the cumulative impact of cellular, genomic, and metabolic dysfunction.¹⁶



Death rates across ages

National data from the United States between 2018 and 2021.

ANNUAL DEATH RATE, per 100,000 people (log scale)

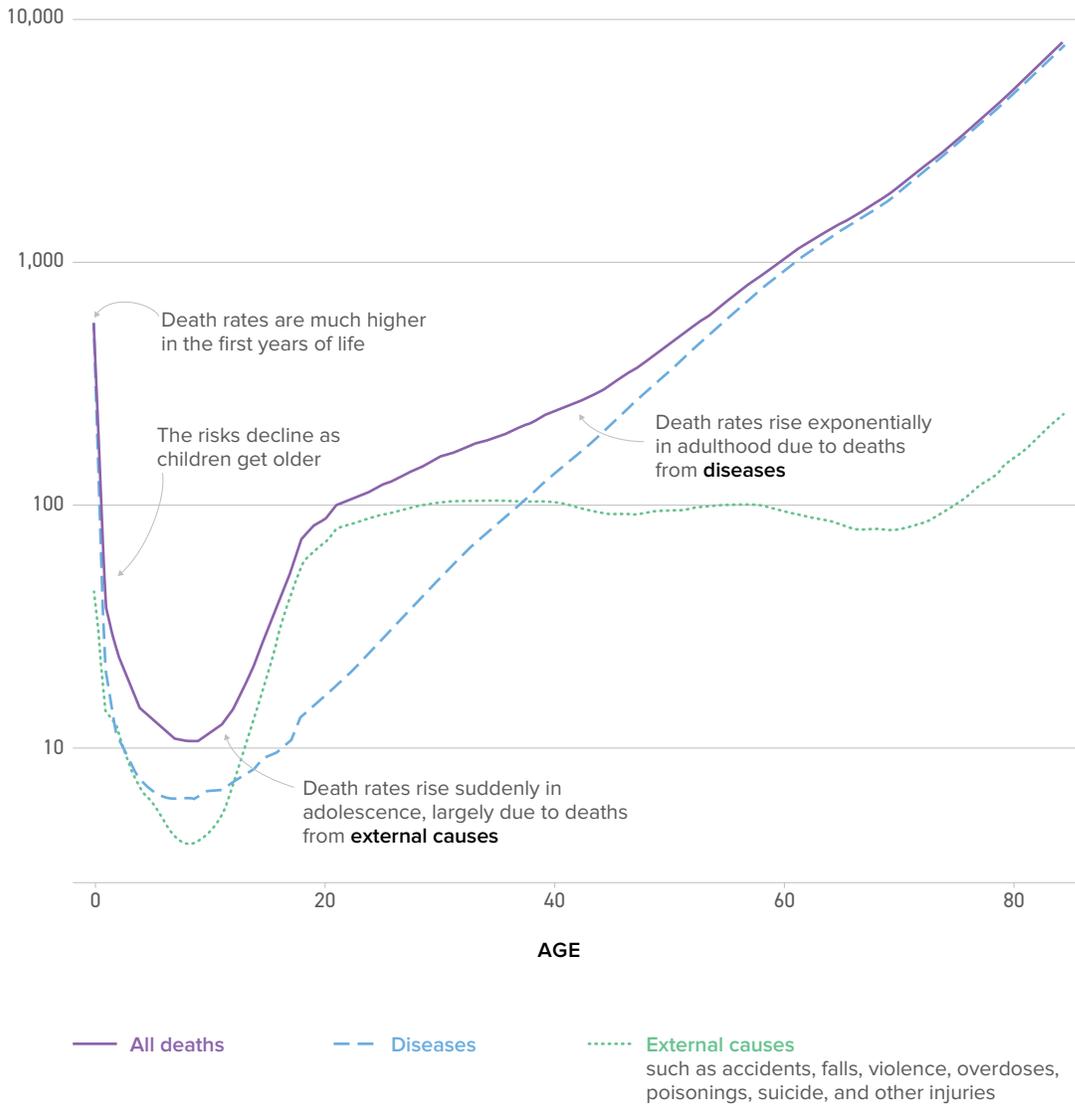


Figure 2: In adulthood, death rates rise exponentially after accounting for deaths due to external causes. Figure adapted from Saloni Dattani, "Our World in Data",¹³ licensed under [CC-BY 4.0](https://creativecommons.org/licenses/by/4.0/).

Using All-Cause Mortality Risk to Calculate an Effective Age

The WHOOP Healthspan metrics rely on the concept of *effective age*, which translates an individual's health risks into an equivalent age based on their all-cause mortality risk profile. This is done by comparing their risk level to that of a "healthy" or "average" individual, termed the referent individual (Figure 3). An individual's effective age is defined as the age of a typical healthy person (the referent individual) who has an equivalent risk profile. For example, if a 50-year-old has an effective age of 60, their risk profile is comparable to that of a 60-year-old. The concept of effective age provides a way to communicate health risks in a manner that is both intuitive and actionable.

The use of effective age to communicate health risk has gained widespread popularity in recent years. Several online tools, including "heart age" and "lung age" calculators, have adopted this method in an effort to better communicate cardiovascular and respiratory health risks.¹⁷ These tools work by estimating an individual's risk for a given condition (e.g., heart disease) and then identifying the age at which the referent individual would have an equivalent risk.¹⁷

While many risk metrics can be used in the calculation of an effective age, the WHOOP Healthspan feature leverages the risk of all-cause mortality, and as described previously, this metric follows a monotonic log-linear curve with respect to age.

Provided two assumptions are met, (1) the change in risk (i.e. hazard ratio) between the exposed and

referent groups remains consistent across ages and (2) the change in the baseline hazard with respect to age is log-linear, the calculation of an effective age is straightforward.¹⁷

1. Estimate the hazard ratio associated with the behavior or condition compared to the "referent" risk.
2. Estimate the risk associated with aging for the outcome of interest. For all-cause mortality, each additional year of age corresponds to a roughly 10% increase in risk.
3. Translate the hazard ratio into an effective age difference. This is done by determining the age, "t", such that $e^{ct} = e^b$, where e^c is the hazard ratio in the outcome of interest associated with a one year increase in age, and e^b is the hazard ratio associated with the exposure. Solving for the effective age delta, "t", yields:

$$t = \frac{\ln(e^b)}{\ln(e^c)} = \frac{\ln(HR)}{0.1} = 10 \times \ln(HR)$$



In many cases, hazard ratios are assumed to be constant across ages, meaning that a given behavior has the same relative impact on risk at any age. However, this is not always true for all health behaviors. For example, similar increases in steps result in larger risk reductions in older individuals compared to younger individuals.¹⁸ As a result, members may notice that the same value for a particular Healthspan metric (e.g., step count or HR zone time) can have a different effect on WHOOP Age depending on the age of the member. This dynamic approach ensures that WHOOP Age accurately reflects how specific behaviors influence health risks over time, rather than assuming a one-size-fits-all relationship across all ages.

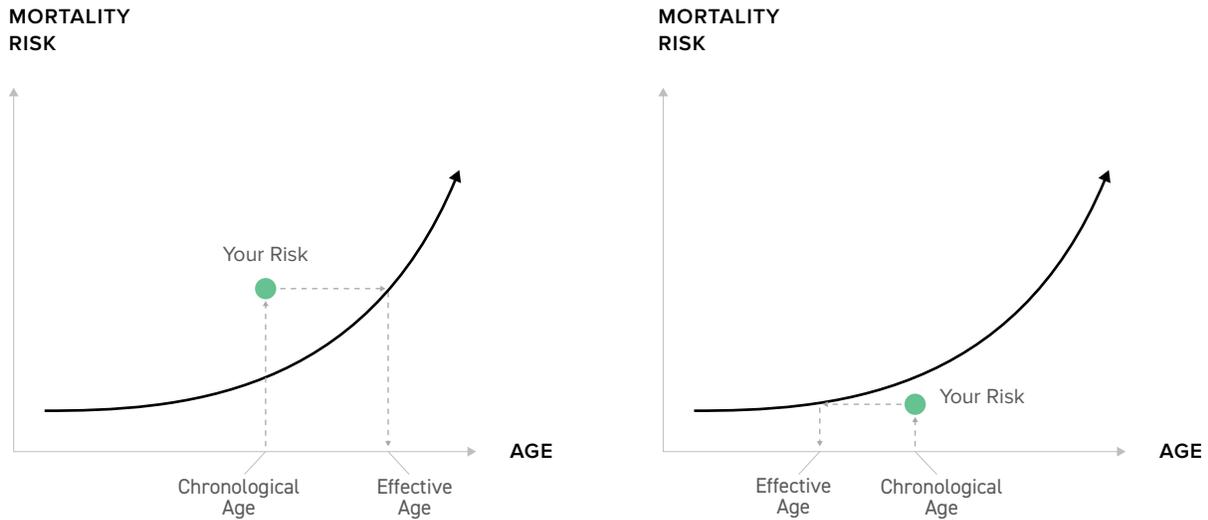


Figure 3: Illustrative depiction of effective age.

(Left) If an individual's mortality risk is greater than the referent risk for their age, then their effective age will be higher than their chronological age.

(Right) if an individual's mortality risk is less than the referent risk for their age, then their effective age will be lower than their chronological age

Determining the “Referent” Individual: How WHOOP Age Compares to Chronological Age

An important aspect of determining an individual’s effective age is the selection of a referent population — the group against which health risk is compared.¹⁷ This decision fundamentally shapes the interpretation of WHOOP Age and what it means for an individual’s WHOOP Age to be equal to their chronological age.

For example, if the referent population were the average American, then a person’s WHOOP Age would match their chronological age if their health risk profile mirrored that of the general U.S. population. However, this approach has significant drawbacks. The average risk profile in many populations is heavily influenced by outliers with poor health, meaning that the statistical “norm” does not necessarily represent an optimal state of health. Some existing effective age calculators have set the reference risk to the mean risk of a given population, and as a result, individuals using these tools often receive an effective age that is much younger than their actual age, simply because the average risk is skewed by those with significant health issues.¹⁷

Rather than defining “normal” based on the general population, WHOOP has deliberately chosen a health-optimized referent risk profile. This means that WHOOP Age does not compare individuals to the average American or even the global average; instead, it is anchored in established public health guidelines and expert recommendations. Each Healthspan component — such as Sleep Duration, Heart Rate Zone Time, and VO₂ Max — is mapped to thresholds that align with optimal health benchmarks.

In cases where clear public health guidelines do not exist, WHOOP relies on expert recommendations to define thresholds for optimal health. These expert-driven benchmarks are informed by scientific literature, clinical expertise, and best practices in health optimization. This ensures that even for emerging or less well-defined health metrics, WHOOP Age remains grounded in evidence-based principles.

The decision to use healthy recommendations for the referent risk profile has important implications for how WHOOP Age should be interpreted relative to other biological aging clocks. A WHOOP Age equal to chronological age means that a member is meeting health-optimized recommendations, not simply aligning with the average population risk. Because many people — particularly in the United States — do not consistently meet these guidelines, the “average” American adult is likely to receive a WHOOP Age that is higher than their chronological age (Table 2). This reflects WHOOP’s commitment to setting an evidence-based standard for long-term health, helping members understand not just where they stand relative to the norm, but how they can improve toward optimal targets.

By using scientifically backed health standards and expert recommendations as the referent risk profile, WHOOP Age encourages members to strive toward validated health targets rather than simply aiming to be “better than average.” This approach ensures that the metric serves as a meaningful and actionable tool for understanding long-term health risks and optimizing healthspan.

“A WHOOP Age equal to chronological age means that a member is meeting health-optimized recommendations, not simply aligning with the average population risk.”

Accounting for Correlated Behaviors and Metrics

The scientific literature used to estimate hazard ratios for behaviors and physiological markers (e.g., physical activity, VO_2 Max) does not always account for the relationships between multiple health behaviors, which can lead to overlapping effects when mapping these risk associations onto the WHOOP Healthspan framework. Since these certain physiological and behavioral metrics are correlated but not always included as covariates in the referenced studies, their combined influence may result in overestimating a component's impact or duplicating risk attribution.

For example, resting heart rate (RHR) and VO_2 Max are correlated, yet the hazard ratio for VO_2 Max may be derived from studies that do not fully adjust for RHR. Since a lower resting heart rate is often a marker of higher cardiovascular fitness, part of the risk reduction attributed to VO_2 Max may actually be due to lower RHR, rather than VO_2 Max itself. If separate risks are assigned to both RHR and VO_2 Max, the impact of cardiovascular fitness on all-cause mortality may be counted more than once, leading to an exaggerated WHOOP Age estimate.

To correct for these overlapping effects, structural equation modeling was used to quantify the interdependencies between Healthspan components and their collective contribution to WHOOP Age. A structural model was built to define both direct effects (how each component independently influences WHOOP Age) and indirect effects (how components influence one another). Using a large dataset of unadjusted Healthspan data from WHOOP members, an empirical covariance matrix was computed to capture real-world correlations between metrics. The model then derived a theoretical covariance matrix, representing how these components would behave if their effects were independent. The difference between these two matrices was minimized, yielding adjustment factors that correct for interdependencies.

These adjustment factors were applied to the hazard ratios of each Healthspan component, ensuring that WHOOP Age reflects the independent contribution of each behavior without artificial inflation from correlated metrics. This approach prevents double-counting effects, provides a more accurate assessment of long-term risk, and ensures that Healthspan remains scientifically rigorous and personalized to each individual's health profile.



The WHOOP Healthspan Feature



Healthspan Metrics: WHOOP Age and Pace of Aging

The WHOOP Healthspan feature helps members understand how their behaviors and physiology may impact their long-term health. Two main metrics are provided within Healthspan: WHOOP Age and Pace of Aging. WHOOP Age is a novel measure applying the effective age framework to measures of functional capacity. Pace of Aging provides members with an indication as to whether WHOOP Age is likely to increase, decrease, or stay the same based on recent behaviors.





What is WHOOP Age?

WHOOP Age provides members with a snapshot of how their lifestyle behaviors and physiological metrics may impact their long-term health, summarizing complex data points into a single “physiological” or “functional” age based on the last 6 months of WHOOP data. WHOOP uses 9 key metrics spanning 3 domains to calculate your WHOOP Age. Sleep duration and sleep consistency capture a member’s sleep health; steps, heart rate zone time, and strength activity provide a picture of the member’s physical activity levels; and VO_2 Max, resting heart rate, and lean body mass percentage characterize the member’s overall fitness. WHOOP Age is determined by estimating the all-cause mortality risk associated with the member’s data in comparison to expert recommendations within each key contributor metric within Healthspan. The contributors may add or subtract years, and WHOOP Age is the sum of the Age Impacts for each Healthspan contributor. A WHOOP Age equal to your actual age means you’re meeting the WHOOP recommendations for good long-term health

Younger

(WHOOP Age < Actual Age):

You are exceeding expert recommendations for good long-term health.

On Track

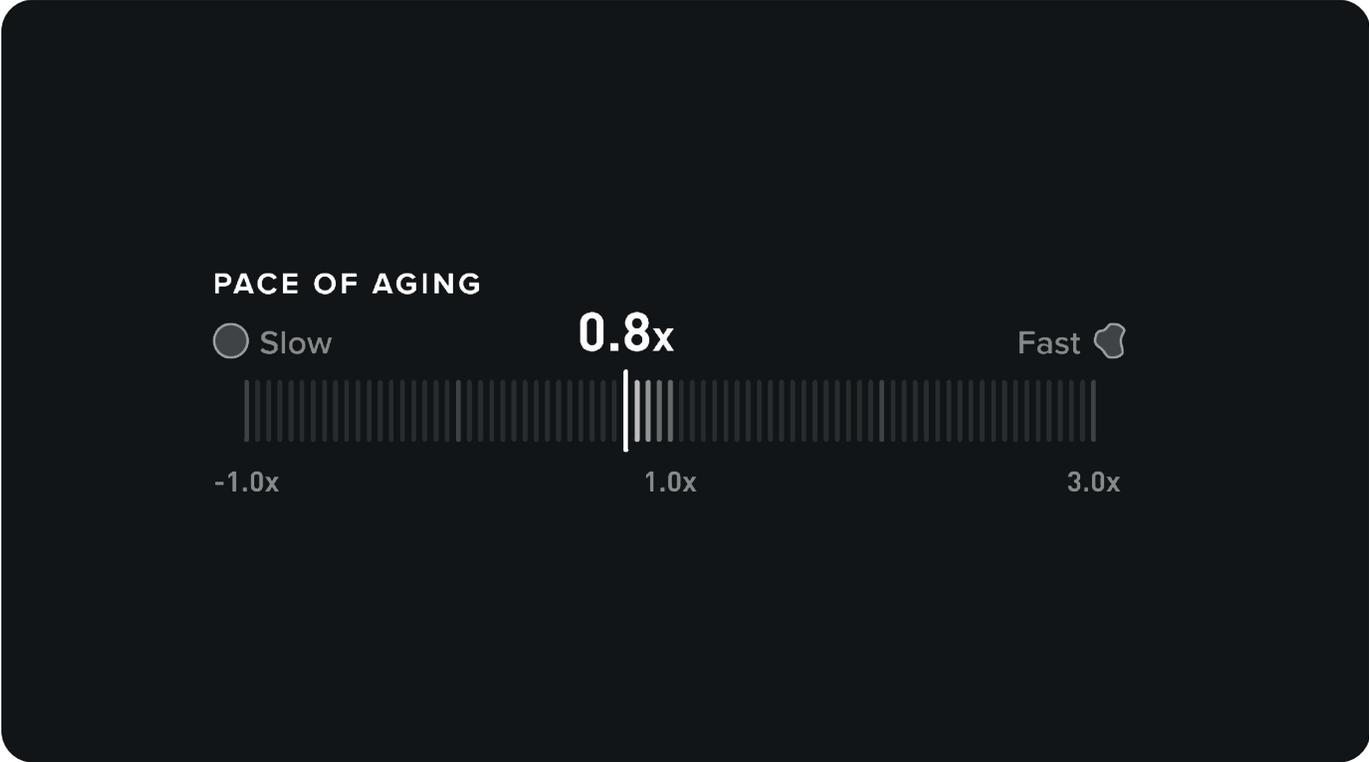
(WHOOP Age = Actual Age):

You’re meeting recommendations for good long-term health.

Older

(WHOOP Age > Actual Age):

You are falling short of recommendations for good long-term health.



What is Pace of Aging?

Pace of Aging reflects how your WHOOP Age will change in the future if recent behaviors and physiology continue. It's measured on a scale from -1.0x to 3.0x, and can act like a speedometer for your WHOOP Age. A projected WHOOP Age is calculated assuming the member's recent 30-day averages for the Healthspan metrics are sustained for the next 6 months. This projected WHOOP Age is then compared to the member's current WHOOP age. If the difference between the projected WHOOP Age and the current WHOOP Age is exactly 6 months, then WHOOP Age is projected to increase at the same rate as chronological age, and the member's Pace of Aging is 1.

Unlike WHOOP Age, which shifts more gradually, Pace of Aging uses your last 30 days of data — making it quicker to respond to changes in your habits.

Accelerated (more than 1.0x):

Behaviors and physiology in the last 30 days are less healthy than in the last 6 months. If sustained, WHOOP age is likely to increase more rapidly than your chronological age.

Steady (1.0x):

Behaviors and physiology in the last 30 days are similar to the last 6 months. If sustained, WHOOP Age is likely to increase at the same rate as chronological age, maintaining their difference.

Slowing (between 0.0x and 1.0x):

Behaviors and physiology in the last 30 days are similar to the last 6 months. If sustained, WHOOP age is likely to rise, but more slowly than time, thereby widening the gap between chronological age and WHOOP Age.

Reversing (less than 0.0x):

Behaviors and physiology in the last 30 days are collectively much healthier than in the last 6 months. If sustained, WHOOP age is likely to decrease.

The WHOOP Healthspan Components



Sleep Consistency



The Impact of Sleep Timing on Physiological Systems

- **Sleep Quality:** Sleep consistency influences your circadian rhythm. More regular sleep schedules improve sleep quality by making it easier to fall asleep and wake up and by promoting deeper and more restorative stages during sleep.^{19,20}
- **Cognitive Function:** Irregular sleep schedules are linked to cognitive function, with irregular sleep patterns associated with decreased alertness, slower reaction times, and impaired executive functioning.²¹
- **Mental Health:** Regular sleep timing has been shown to regulate mood and reduce the risk of mental health disorders such as depression and anxiety.^{22,23}
- **Cardiovascular Health:** Sleep regularity is associated with cardiovascular health, with irregular sleep patterns linked to higher risks of hypertension and cardiovascular disease.^{24,25}

Sleep Timing and Long-Term Health

The National Sleep Foundation emphasizes that consistent sleep-wake schedules are crucial for optimal health and performance, as irregular sleep disrupts circadian rhythms, leading to adverse health outcomes.²¹ Similarly, a prospective cohort study involving over 60,000 participants found that individuals with higher sleep regularity had a 20% to 48% lower risk of all-cause mortality, a 16% to 39% reduction in cancer mortality, and a 22% to 57% decrease in cardiometabolic mortality compared to those with the most irregular sleep patterns.²⁶

The WHOOP Sleep Consistency score evaluates how similar an individual's sleep and wake times are over the last 24 hours compared to the previous 4 days. The metric is scored on a 0-100% scale, with higher percentages indicating more regular sleep patterns. Members averaging below 70% will see years added to their WHOOP Age, reflecting the increased health risks associated with irregular sleep. Conversely, members maintaining Sleep Consistency above 70% will have years subtracted, reinforcing the longevity benefits of stable sleep timing.

Sleep Duration



The Impact of Sleep Duration on Physiological Systems

- **Cognitive Function:** Sufficient sleep duration enhances attention, memory consolidation, and learning by supporting neural plasticity and clearing metabolic waste from the brain. Insufficient sleep has been linked to an increased risk of cognitive decline and neurodegenerative diseases such as Alzheimer's.²⁷⁻²⁹
- **Physical Recovery and Repair:** During deep sleep, the body releases growth hormone and activates cellular repair processes that restore muscles, tissues, and bones.³⁰ Adequate sleep is essential for recovery from physical exertion and injury.
- **Metabolic Health:** Adequate sleep duration plays a vital role in maintaining healthy glucose metabolism and appetite regulation. Sleep deprivation has been linked to insulin resistance, increased hunger hormones, and a higher risk of obesity and type 2 diabetes.³¹
- **Mental Health:** Chronic sleep deprivation as well as excessive sleep is linked to poorer mental health and quality of life, including depression and anxiety.^{32,33}

Sleep Duration and Long-Term Health

Adequate sleep is essential for long-term health, with both insufficient and excessive sleep linked to increased health risks. A meta-analysis with over 5 million participants found that short sleep duration was associated with a

12% higher risk of all-cause mortality, alongside increased risk for conditions such as diabetes, hypertension, cardiovascular disease, coronary heart disease, and obesity.³⁴ Other studies have shown that the risk of all-cause mortality associated with short sleep is even higher when sleep duration is measured objectively using a wearable device instead of relying on self-reported measures.³⁵ Given the elevated risk of long-term health consequences, members averaging less than 7 hours of sleep per night will see an increase in their WHOOP Age.

Some studies suggest that 7 hours is the optimal sleep duration, as sleep durations greater than 7 hours have also been associated with higher mortality risk.³⁵ However, unlike short sleep, the biological mechanisms underlying this association are unclear. Some researchers hypothesize that prolonged sleep may be a symptom rather than a cause of poor health, potentially linked to underlying comorbidities, depressive symptoms, or disease-related fatigue.³⁶ In support of this hypothesis, a study of apparently-healthy individuals found that those reporting 8-9 hours of sleep per night had a slight, though not statistically significant, reduction in all-cause mortality compared to those sleeping 7 hours.³⁷ As a result, those logging 7-9 hours per night may experience a minor reduction in their WHOOP Age, however members who sleep more than 9 hours per night will see no effect on their WHOOP Age, ensuring that the metric remains evidence-based and avoids conflating correlation with causation.

Heart Rate Zone Time



The Impact of Heart Rate Zone Time on Physiological Systems

- **Cardiovascular Health:** Regular aerobic activity improves heart efficiency, circulation, and blood pressure regulation, reducing the risk of cardiovascular disease such as coronary heart disease and stroke.³⁸
- **Metabolic Health:** Moderate-to-vigorous intensity exercise enhances insulin sensitivity, regulates blood glucose levels, and lowers the risk of type 2 diabetes and metabolic syndrome.^{39,40}
- **Cognitive Function:** Aerobic exercise increases cerebral blood flow and promotes neurogenesis, and higher cardiovascular fitness has been associated with better memory, executive function, and a lower risk of dementia.^{41,42}
- **Mental Health:** Regular physical activity has been shown to reduce symptoms of depression, anxiety, and psychological distress.⁴³

Heart Rate Zones

WHOOP estimates heart rate zones as a percentage of your heart rate reserve (HRR)—the difference between an individual’s maximum heart rate and resting heart rate.

Heart Rate Zone Time and Long-Term Health

Regular physical activity is one of the most effective ways to improve heart health, metabolic function, and longevity. Research shows that individuals who spend more time in moderate-to-vigorous intensity exercise experience lower

risks of cardiovascular disease, type 2 diabetes, cognitive decline, and all-cause mortality.⁴⁴⁻⁴⁶ Studies have reported a 20% to nearly 50% reduction in all-cause mortality with 5-7 hours of weekly physical activity, with objective activity tracking (e.g. from wearables) showing even stronger associations.^{38,47} Notably, even short bursts of high-intensity activity can provide substantial health benefits. One study found that just 1-2 minutes of vigorous exercise incorporated into daily routines—accumulating to 15-20 minutes per week—was associated with an 18-24% lower risk of all-cause mortality compared to no vigorous activity.⁴⁸

Health organizations, including the CDC and the WHO, recommend at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity exercise per week.^{49,50} WHOOP scientists analyzed members’ data to understand the relationship between general physical activity as defined by these public health guidelines and time spent in HR Zones during dedicated activities. Based on this analysis, WHOOP recommends at least 100 minutes Zone 1-3 Time in activities logged on WHOOP and at least 10 minutes per week of Zone 4-5 Time for young adults to see a decrease in WHOOP age. However, evidence suggests that additional activity provides even greater benefits, with all-cause mortality risk decreasing incrementally up to 600 minutes per week.³⁸

Similar to other metrics, research suggests that the longevity benefits of physical activity become even greater as people age, underscoring the importance of maintaining an active lifestyle throughout the lifespan.⁵¹ As a result, WHOOP recommendations for Zone Time decrease slightly as members age to reflect these evolving benefits.

Strength Activity Time



The Impact of Strength Training on Physiological Systems

- **Musculoskeletal Health:** Strength training helps maintain and improve muscle mass, bone density, and joint stability, all of which are critical for preserving mobility and reducing the risk of injury with age. Regular resistance exercise helps prevent sarcopenia and osteoporosis, two major contributors to frailty and functional decline in older adults.⁵²
- **Metabolic Health:** Regular resistance training boosts resting metabolic rate by increasing lean muscle mass, which raises resting energy expenditure and improves the body's ability to regulate blood glucose levels. These effects contribute to a reduced risk of insulin resistance, type 2 diabetes, and metabolic syndrome.^{53,54}
- **Neuromuscular Function:** Strength training improves balance, coordination, and motor control—particularly important for reducing fall risk and promoting independent living in older adults.⁵⁵

Strength Training and Long-Term Health

Engaging in regular strength training is strongly linked to reduced risks of all-cause mortality and chronic diseases, making it a key component of long-term health. Meta-analyses synthesizing findings from large cohort studies have found that performing 30–60 minutes of muscle-strengthening activities per week is associated with a 10%–30% reduction in all-cause mortality, cardiovascular disease, and cancer.^{56,57} WHOOP measures Strength Activity Time as the total weekly time spent performing strength building activities, such as weightlifting, yoga, and functional fitness. Members with more than 40 minutes per week of strength training will have years subtracted from their WHOOP Age.

Notably, there is a U-shaped association between strength training time and all-cause mortality, with the optimal benefit from strength training time occurring between 30 minutes and 2 hours,^{56,58} though the mechanisms underpinning the attenuated risk reduction with larger amounts of strength training time are not well understood. As such, logging more than 2 hours per week of strength training time will not result in additional benefits for WHOOP Age.

Daily Steps



The Impact of Steps on Physiological Systems

- **Cardiovascular Health:** Daily movement through walking supports cardiovascular health and is associated with a lower risk of heart disease, stroke, and cardiovascular mortality, even when performed at moderate intensity.⁵⁹
- **Metabolic Health:** A higher daily step count is associated with a reduced risk of metabolic syndrome.⁶⁰ Walking also contributes to maintaining a healthy weight by increasing energy expenditure.
- **Mental Health:** Daily walking reduces stress, anxiety, and symptoms of depression through the release of endorphins. It also boosts mood and cognitive function, particularly when done in natural environments.⁶¹

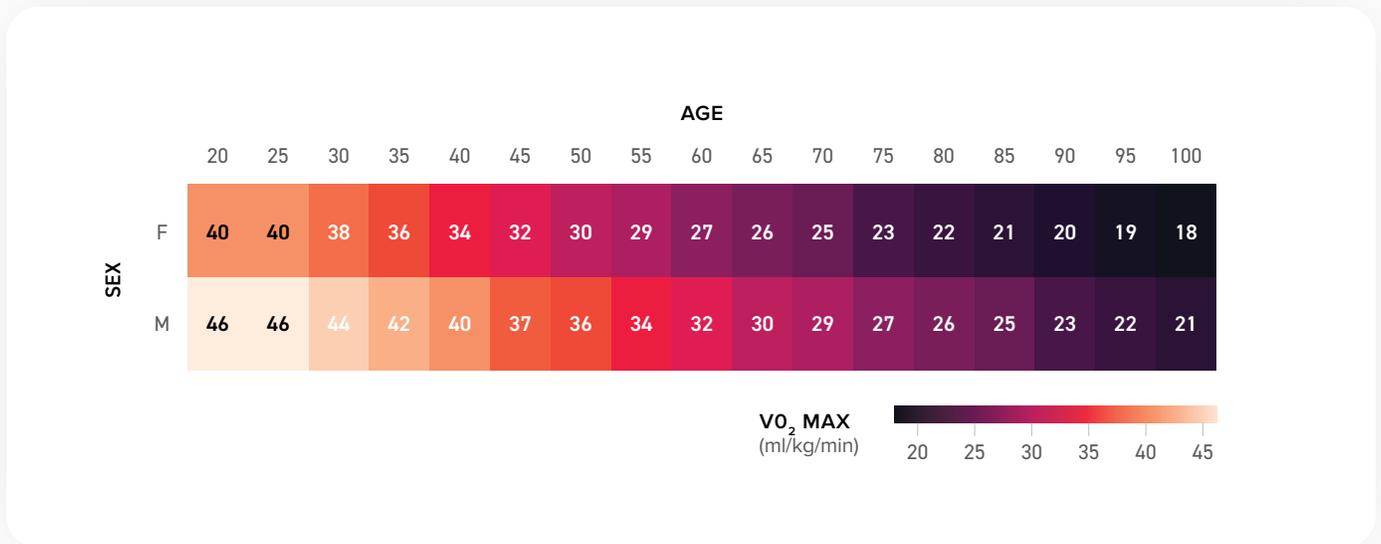
Steps and Long-Term Health

Among various measures of activity, daily step count is a simple yet powerful indicator of movement and long-term health. Research consistently links higher step counts to lower risks of all-cause mortality and cardiovascular disease.⁵⁹ A meta-analysis of 16 publications found that for every additional 1,000 steps per day, the risk of all-cause mortality decreased by 23%, while an increase of 500 steps per day reduced the risk of cardiovascular events by 6%.⁶² Research suggests that the long-term health benefits of daily steps begin to plateau around 8,000–10,000 steps per day.⁵⁹ As a result, younger members who exceed 8,000 steps per day will see modest reductions in their WHOOP Age, while those falling below will have years added to their WHOOP Age, reflecting the health risks associated with lower activity levels.

Additionally, research suggests that a higher daily step count may have an even greater impact in older adults. A study found that for adults over 60, the same number of daily steps resulted in a greater reduction in all-cause mortality compared to those under 60,¹⁸ thus fewer steps are needed to decrease WHOOP age in adults older than 60. This underscores the importance of maintaining regular movement at all ages, particularly later in life when physical activity can provide even greater health benefits.

VO₂ Max

Figure 4: WHOOP minimum recommendations for VO₂ Max.



VO₂ Max is a measure of cardiorespiratory fitness and is the maximum amount of oxygen that an individual can utilize during intense or maximal exercise, measured in milliliters per kilogram per minute (mL/kg/min). This value reflects how efficiently your respiratory and cardiovascular systems can deliver oxygen to muscles during periods of maximum intensity exercise, providing a holistic view of your fitness level. VO₂ Max can range from below 20 mL/kg/min in untrained or older individuals to above 80 mL/kg/min in elite athletes.

The Impact of Cardiorespiratory Fitness on Physiological Systems

- **Cardiovascular Health:** Higher VO₂ Max reflects improvements in cardiac efficiency. Endurance training increases stroke volume, the amount of blood pumped per heartbeat, enhancing overall cardiac output. This increase in cardiac output directly boosts your VO₂ max, enabling longer training periods without fatigue.⁶³
- **Metabolic Health:** Increased VO₂ Max is associated with improved mitochondrial density, enhancing the body’s ability to utilize oxygen for energy production. These adaptations also lower the risk of type 2 diabetes and metabolic syndrome.⁶⁴
- **Respiratory Function:** Improving VO₂ Max enhances lung capacity and respiratory efficiency,⁶⁵ allowing for better oxygen uptake and carbon dioxide removal during exercise. These adaptations support better endurance and reduce the sensation of breathlessness during physical activity.

Cardiorespiratory Fitness and Long-Term Health

VO₂ Max is one of the strongest predictors of long-term health and all-cause mortality, reflecting overall cardiovascular and respiratory fitness more effectively than traditional risk factors like smoking, hypertension, or BMI.⁶⁶ With even modest improvements, such as moving from the bottom quartile for VO₂ Max to the 50th–75th percentile, mortality risk decreases by over 60%.⁶⁷ Similarly, studies show a 13% lower mortality risk for every additional 1 MET (metabolic equivalent, ~3.5 mL/kg/min) increase in VO₂ Max. While VO₂ Max indicates your capacity at maximum intensity, a higher VO₂ Max makes all daily activities easier, underscoring the importance of VO₂ Max for independent daily living.⁶⁸

VO₂ Max naturally declines by 10–12% per decade after age 30, driven by factors like reduced cardiovascular capacity and changes in lean body mass.⁶⁹ Maintaining or improving VO₂ Max through regular activity and exercise mitigates these age-related declines. While there aren’t clear clinical or public health recommendations for VO₂ Max, the WHOOP Healthspan recommendations for VO₂ Max are designed to set members up for independent living at older ages, accounting for the natural decline in VO₂ Max with age.⁷⁰ Research suggests that a minimum of approximately 20 mL/kg/min is required to maintain independent living,⁷¹ with males exhibiting roughly 20% higher VO₂ Max compared to females.⁷² Members with a VO₂ Max greater than their age and sex-adjusted target (Figure 4) will see years subtracted from their WHOOP Age, while a VO₂ Max lower than their target will see years added.

Resting Heart Rate



Resting Heart Rate (RHR) is the number of heartbeats per minute when you're at rest. It reflects how efficiently your heart functions when you are not exercising, and is an important indicator of cardiovascular fitness and overall heart health. A lower RHR typically indicates that a single beat expels a greater volume of blood from the heart, and is generally a sign of better health.

The Impact of Resting Heart Rate on Physiological Systems

- **Cardiovascular Health:** A high resting heart rate indicates that the heart must work harder to circulate blood. With regular aerobic, anaerobic, and resistance training, the heart becomes stronger and more efficient. As a result, resting heart rate decreases, reflecting an improved ability to pump more blood with each beat. This increased efficiency reduces strain on the heart and lowers the risk of cardiovascular disease.⁷³
- **Autonomic Nervous System:** The autonomic nervous system regulates involuntary functions such as heart rate and digestion. Chronic stress and inactivity can lead to an imbalance, with excessive sympathetic nervous system ("fight or flight") activity contributing to an elevated resting heart rate.⁷⁴ Endurance training enhances parasympathetic nervous system activity ("rest and digest"), lowering resting heart rate and improving autonomic balance.⁷⁵

Resting Heart Rate and Long-Term Health

Higher RHR is associated with increased risks of all-cause mortality and cardiovascular diseases like coronary artery disease and stroke.⁷⁶ One study found that men with an RHR of 90 beats per minute (bpm) or higher had a three-fold increase in the risk of premature death compared to those with an RHR of 50 bpm or lower.⁷⁷ While a RHR between 60 and 100 bpm in a clinical setting is considered "normal", even small differences in resting heart rate, particularly when captured during sleep, are associated with meaningful differences in long-term health risk. A 10 bpm increase in RHR has been associated with a 9% increase in mortality risk,⁷³ underscoring the importance of maintaining a lower RHR through lifestyle and physical activity. WHOOP measures RHR during sleep to minimize the influence of movement and physical activity. An average RHR of ~60 bpm for males and ~64 bpm for females will result in neutral impact to WHOOP Age. Higher values will increase WHOOP age and lower values will decrease it.

Lean Body Mass Percentage



Lean Body Mass Percentage is the relative proportion of lean mass (e.g. bones and muscle) relative to total body mass, expressed as a percentage. Total body mass is the sum of lean mass and fat mass. Most at-home scales measure body composition via a technique called bioelectric impedance analysis. This method is susceptible to changes in hydration, so to best understand changes in your Lean Body Mass, take your measurements when your hydration level is most consistent, such as in the morning after first waking up.

The Impact of Lean Body Mass Percentage on Physiological Systems

- **Metabolic Health:** Increased lean body mass raises basal metabolic rate, aiding in weight management and reducing excess fat storage. The corresponding decrease in proportion of fat mass improves insulin sensitivity and glucose metabolism, lowering the risk of type 2 diabetes and metabolic syndrome.^{53,78}
- **Musculoskeletal and Structural Health:** Higher lean body mass percentage generally means stronger muscles and bones. This helps protect against sarcopenia and osteoporosis, maintain mobility, and reduce the risk of fractures and frailty.^{79,80}
- **Immune Health:** Higher muscle mass supports a robust immune response, lowering systemic inflammation and helping the body fight infections and recover more efficiently.⁸¹

Body Composition and Long-Term Health

Higher lean body mass percentage is strongly associated with lower mortality risk, even after accounting for other factors like body mass index.⁸² A study of nearly one million adults across 35 cohorts found that with each 10% increase in body fat, risk of all-cause mortality increases by 11%.⁸³

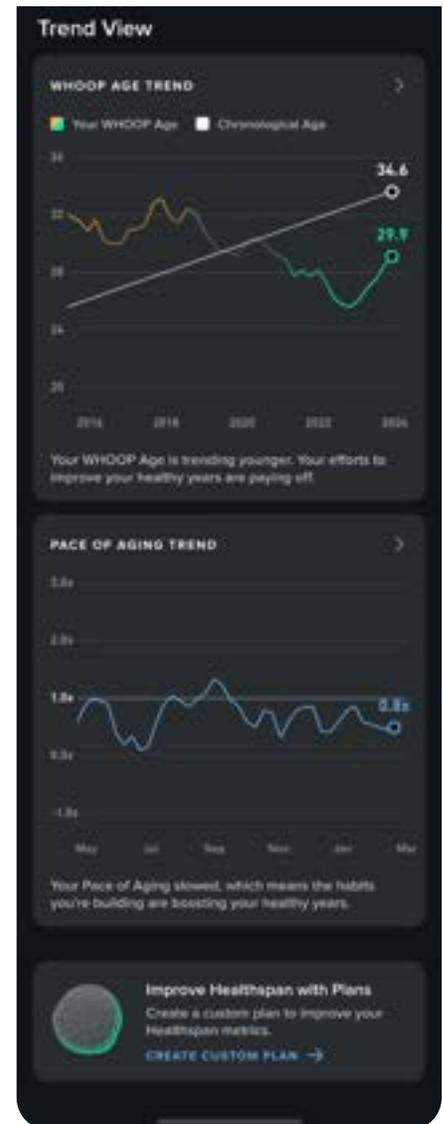
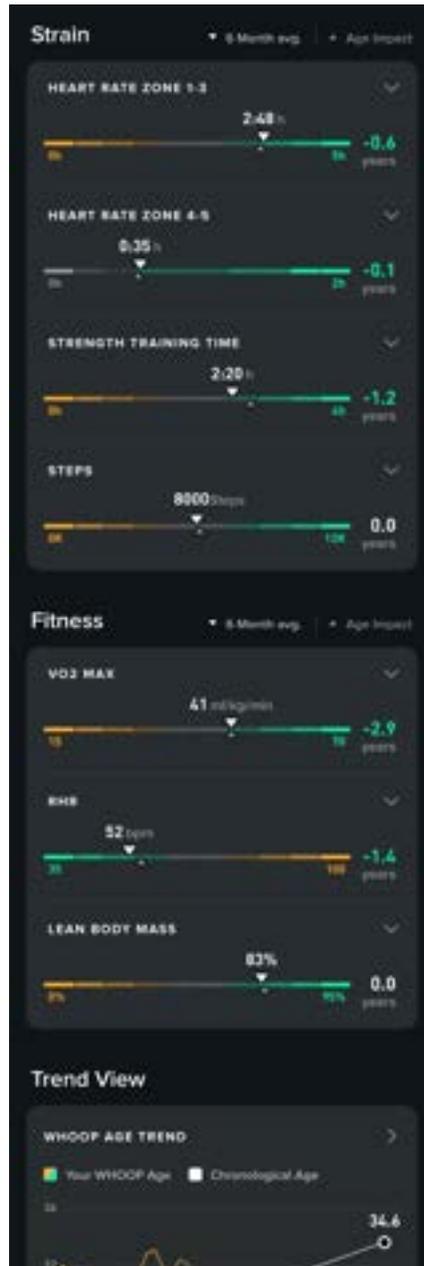
The WHOOP recommendations for lean body mass vary with respect to age and sex – higher lean body mass percentages are recommended for younger individuals and males. The WHOOP recommendation for a 30-year old female is at least 67% and for a 30-year old male is at least 80%. While WHOOP does not directly measure lean body mass, members can connect a smart scale or manually input this metric. Members with lean body mass percentages below these thresholds will see years added to their WHOOP Age and members with values above these thresholds may see small reductions in their WHOOP Age. If lean body mass is not manually entered, there will be no effect on Healthspan metrics.

Component Summary

WHOOP Metric	Healthy Recommendation
Sleep Duration	Aim for 7-9 hours of sleep per night.
Sleep Consistency	At least 70% sleep consistency.
Steps	Daily steps target depends on age; 8,000 for a younger adult and 5,600 steps/day for an older adult.
Heart Rate Zone Time	Zone 1-3: at least 70-100 minutes per week, values decrease with age. Zone 4-5: at least 7-10 minutes per week, values decrease with age
Strength Activity Time	At least 40 minutes of strength activities per week. Includes total weekly minutes logged in the following activities: Strength Trainer, weightlifting, powerlifting, Barre, Barre3, pilates, yoga, hot yoga, functional fitness, Barry's, F45 training, box fitness, HIIT, baby wearing, toddler wearing, rucking, solidcore.
VO₂ Max	Target value decreases with age and is higher for males—for example, a 30-year-old female should aim for greater than 38 ml/kg/min, and a 30-year-old male for greater than 44 ml/kg/min.
Resting Heart Rate (RHR)	Lower than 60 bpm for males and 64 bpm for females.
Lean Body Mass	For young adults, greater than 67% for females and 80% for males. Target value changes with age.

Table 1: Component summary.

Healthspan User Interface



Early Insights with the WHOOP Healthspan Feature



WHOOP Age and Established Measures of Health

To evaluate the utility of WHOOP Age as a meaningful indicator of health status, WHOOP scientists conducted an observational study examining its relationship with self-reported health perception and the presence of chronic conditions. Two hypotheses were tested: (1) WHOOP Age aligns with a member’s perception of their health and (2) members living chronic conditions would have greater WHOOP Age Deltas—the difference between their WHOOP Age and their chronological age—compared to those without chronic conditions.

In the spring of 2025, thousands of WHOOP members participated in the Health History Questionnaire, hosted on the decentralized research platform, Digital WHOOP Labs. WHOOP Scientists compared WHOOP Age at survey submission across survey answers related to health perception and chronic conditions. WHOOP Age was calculated via an automated process without any information from the participants’ survey responses. A positive WHOOP Age Delta indicates that the participant’s WHOOP Age is greater than their chronological age, reflecting behaviors and physiology that do not meet the WHOOP recommendations for long-term health.

Conversely, a negative WHOOP Age Delta indicates that a participant’s WHOOP Age is lower than their chronological age, suggesting that the individual is exceeding the WHOOP recommendations in some capacity.

Analysis revealed a clear stepwise relationship between perceived health and WHOOP Age Delta (Figure 5). Participants who rated their general health as “poor” or “fair” were significantly more likely to have positive WHOOP Age Deltas, whereas those who rated their health as “very good”, or “excellent” tended to have negative WHOOP Age Deltas. Participants who rated their general health as “good” tended to have a WHOOP Age approximately equal to their chronological age. This pattern is consistent with prior research demonstrating that self-reported health measures serve as reliable indicators of both current well-being and long-term health outcomes, including morbidity and mortality.^{84,85} By aligning with individuals’ subjective experiences of health, these results further validate the relevance of WHOOP Age as a meaningful reflection of both physiological and perceived well-being.

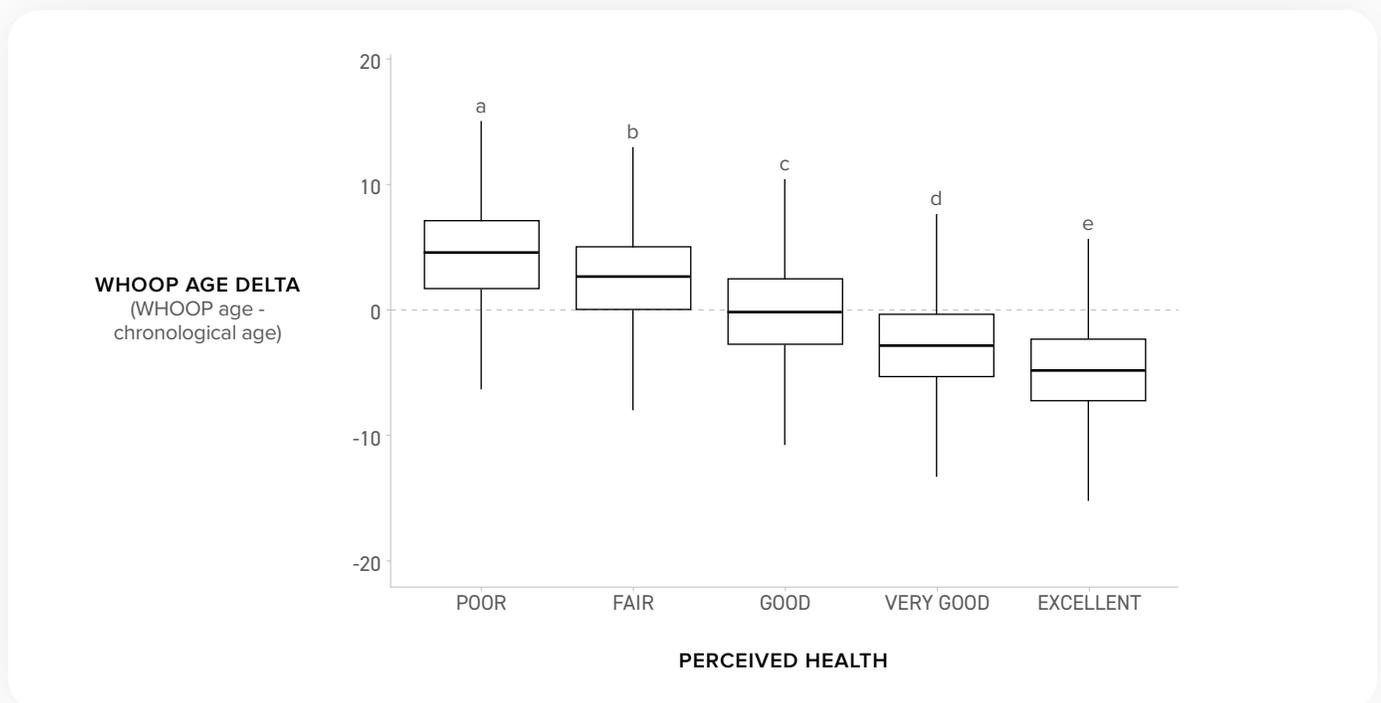


Figure 5: WHOOP Age aligns with perceived health. Sample sizes and mean WHOOP Age Deltas for each answer are: poor (n = 331; \bar{x} = 4.19), fair (n = 2,298; \bar{x} = 2.45), good (n = 13,149; \bar{x} = -0.14), very good (n = 21,782; \bar{x} = -2.80), and excellent (n = 8,287; \bar{x} = -4.64). Data was analyzed using a Welch’s ANOVA and Games-Howell post hoc test, both of which account for unequal sample sizes. Different letters above box-and-whiskers denote significant differences across answers. All p-values < 0.0001.

Participants who reported living with type 2 diabetes or 2 or more condition groups exhibited higher WHOOP Ages relative to their chronological age, suggesting that Healthspan metrics are sensitive to underlying physiological differences beyond the direct inputs used in the model (Figure 6). Though members that reported mental health conditions (ADHD, anxiety, bipolar, borderline personality disorder, burnout, depression, eating disorder, OCD, PTSD, or schizophrenia), cardiovascular conditions (congestive heart failure, coronary artery disease, heart attack, hypertension, stroke), and respiratory conditions (chronic emphysema/COPD, chronic bronchitis) did not tend to have positive WHOOP Ages, they were significantly higher than those that had none of those conditions. Notably, many participants with chronic conditions exhibited a negative WHOOP Age Delta, suggesting they actively engage in behaviors known to support long-term health, despite their diagnoses. This observation aligns with previous research highlighting the significant influence of behavior and environmental factors on longevity regardless of chronic disease state.^{4,5} Given that WHOOP members tend to be a health-conscious population, it is perhaps unsurprising that many individuals with chronic conditions still achieve favorable WHOOP Age estimates. Furthermore, this emphasizes the significant role

of modifiable lifestyle factors in mitigating disease burden.

These findings provide early validation of the WHOOP Healthspan metrics as meaningful indicators of both perceived and physiological health. The observed association between WHOOP Age Delta and self-reported health perception suggests that the framework effectively captures real-world differences in well-being, reinforcing its relevance as a measure of functional health. Additionally, the relationship between WHOOP Age and chronic disease status highlights the sensitivity of Healthspan metrics in reflecting underlying physiological differences beyond the direct model inputs. The variability observed among individuals with chronic conditions suggests that WHOOP Age is not simply a proxy for disease diagnosis, but rather a dynamic reflection of the intersection between physiological function and health behaviors. These insights further demonstrate the potential of Healthspan metrics to contribute to broader longevity research, providing a continuous, real-world dataset for understanding how behavioral and physiological factors interact to shape health trajectories over time. Future research will focus on longitudinal validation of WHOOP Age, assessing its ability to track changes in health status over time.

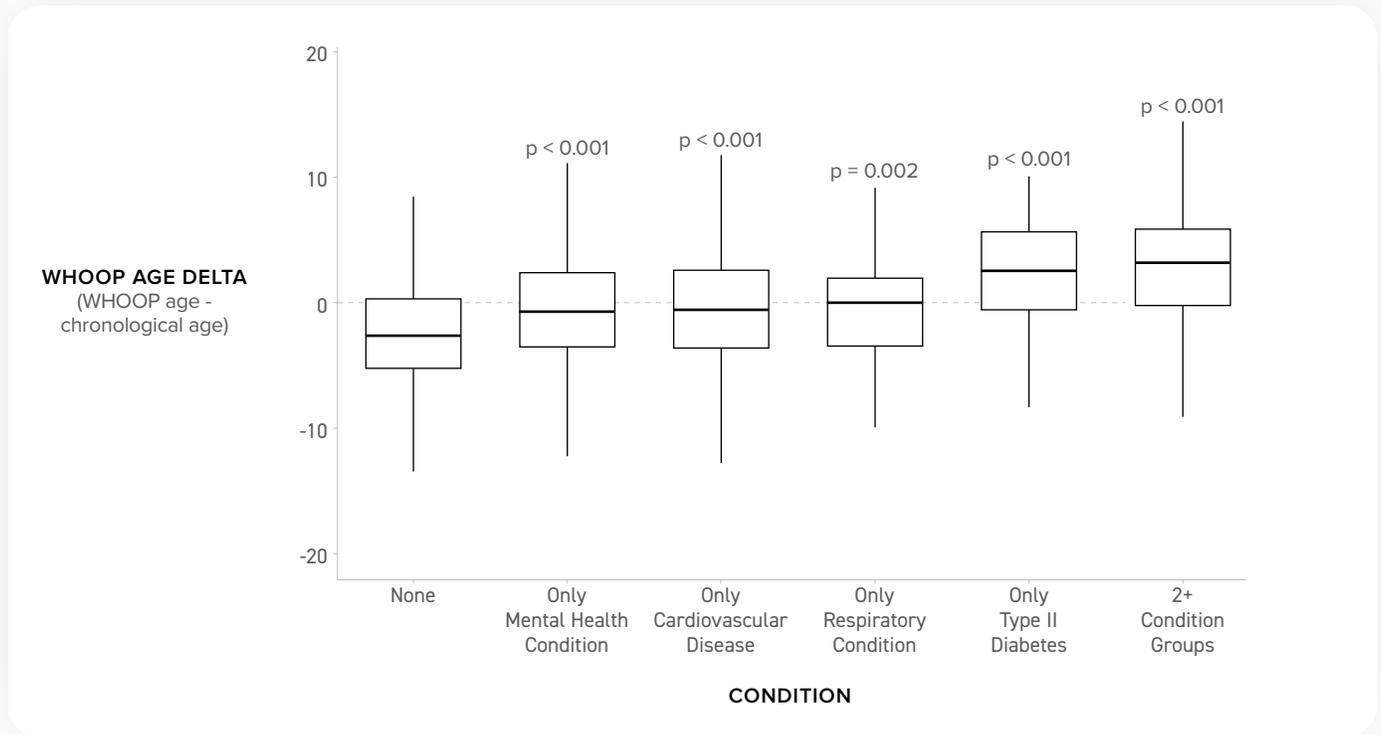


Figure 6: Participants living with one or more chronic diseases have a higher WHOOP Age Delta than participants living with no health conditions. Sample sizes and mean WHOOP Age Deltas for groups are: none ($n = 37,410$; $\bar{x} = -2.42$), only mental health condition ($n = 4,425$; $\bar{x} = -0.57$), only respiratory condition ($n = 62$; $\bar{x} = -0.34$), only cardiovascular disease ($n = 2,348$; $\bar{x} = -0.50$), only type 2 diabetes ($n = 199$; $\bar{x} = 2.39$), 2 or more conditions ($n = 512$; $\bar{x} = 3.83$). Data was analyzed using a Welch's ANOVA and Games-Howell post hoc test, both of which account for unequal sample sizes. P-values above each box-and-whisker plot were calculated for each condition compared to the group with no significant medical conditions.

WHOOP Age: Normative Values

To illustrate how WHOOP Age varies across different populations, typical values for 30-year-old males and females were compiled for two groups: the general U.S. adult population and WHOOP members (Table 2). The U.S. population estimates were derived from published literature and national health databases, with metrics selected to align with the definitions used in the WHOOP Healthspan framework. Where multiple sources existed or estimates varied, ranges were reported. Median values for WHOOP members were calculated using de-identified data from the month of April 2025, representing a snapshot of behavior and physiology among wearable users. This side-by-side comparison helps contextualize WHOOP Age values by showing how WHOOP members' metrics differ from national averages and how these differences translate into long-term health risk, as reflected by WHOOP Age.

The comparison between U.S. population averages and WHOOP member data (Table 2) underscores a critical challenge in public health: a substantial proportion of U.S. adults are not meeting basic recommendations for sleep and physical activity. In contrast, wearable device users — including WHOOP members — tend to be more health-conscious and engaged in tracking their behaviors, often representing a more active and healthier subset of the population.⁹⁸ These differences highlight the importance of WHOOP's decision to use health-optimized referent values rather than comparisons to population averages. By anchoring WHOOP Age to healthy recommendations — rather than norms defined by an increasingly sedentary and sleep-deprived population — the Healthspan feature provides feedback that is both aspirational and grounded in scientific guidelines for healthy living, ensuring that members receive meaningful and actionable insights even if the majority of the population falls short of these standards.

“The Healthspan feature provides feedback that is both aspirational and grounded in scientific guidelines for healthy living, ensuring that members receive meaningful and actionable insights even if the majority of the population falls short of these standards.”

30-Year Old Male

30-Year Old Female

	U.S.	WHOOP	U.S	WHOOP
Sleep Duration Hours / Day	6.1-6.6 ^a	7.0	6.1-6.6 ^a	7.3
Sleep Consistency Percent	60-70% ^b	67	60-70% ^b	68
Daily Steps Steps / Day	5200 ^c	10,900	5000 ^c	11,500
HRZ 1-3 Minutes / Week	69 ^d	141	46 ^d	124
HRZ 4-5 Minutes / Week	N/A ^e	6	N/A ^e	7
Strength Activity Time Minutes / Week	0 ^f	46	0 ^f	56
VO₂ Max mL/kg/min	42.4 ^g	46.8	30.2 ^g	40.0
Resting Heart Rate BPM	67 ^h	58	72 ^h	63
Lean Body Mass Percentage	73.9% ⁱ	81%	62.2% ⁱ	73%
Expected WHOOP Age	~+6 years	-1.6 years	~+7.5 years	-1.6 years

Table 2: Comparison of U.S. Adult and WHOOP member typical values across Healthspan component metrics and resulting WHOOP Age.

^a The average U.S. adult reports approximately 7.1 hours of sleep per night.⁸⁶ Given that individuals tend to self-report 30 minutes to 1 hour more sleep than objective estimates from actigraphy,^{87,88} we estimate adult Americans would record 6.1-6.5 hours of sleep.

^b The irregularity of sleep timing can be captured in various ways: sleep regularity index (SRI), sleep onset / wake time variability, and sleep duration SD are common methods. The literature on typical sleep patterns in Americans is varied: one population-level study suggested typical SRI to be 61.3,⁸⁹ whereas another reported 74.7.⁹⁰ The metric used by WHOOP for sleep timing, Sleep Consistency, tends to be slightly lower than SRI due to the use of a longer baseline period, and therefore we estimate a range of 60-70% for average adult Americans.

^c A study of ~2500 individuals, weighted to reflect the demographics of the general U.S. population, reported that adults averaged 5,117 steps per day, with slight differences in gender and age.⁹¹

^d An examination of the accelerometer-derived physical activity time in bouts of 10 minutes or greater reported 9.9 minutes/day in males aged 30-39 and 6.5 minutes per day in females aged 30-39.⁹²

^e Research on typical amounts of very high intensity (HR Zone 4-5) for U.S. adults could not be found.

^f In a study of nearly 400,000 U.S. adults, across all sex and age cohorts, the majority of individuals reported no muscle-strengthening exercise.⁹³

^g Average VO₂ Max for males and females aged 30-39 from the Fitness Registry and the Importance of Exercise: A National Database (FRIEND) cohort were used.⁹⁴

^h The average day-time heart rate estimate for males and females from nearly 70,000 participants were 73.8 and 79.3 beats per minute, respectively.⁹⁵ These values were adjusted to account for the differences between day-time and night-time heart rate.⁹⁶

ⁱ Data from the National Health and Nutrition Examination Surveys (NHANES) 1999–2004 that leveraged dual energy x-ray absorptiometry (DXA) to estimate body fat percentage were used for lean body mass.⁹⁷

WHOOP Healthspan: Scientific and Practical Implications



The WHOOP Healthspan feature represents an innovative approach to quantifying long-term health risk by integrating real-time physiological data with established longevity research. By leveraging a behavior-based framework, the feature provides a dynamic and personalized assessment of how daily choices influence long-term health outcomes. However, while the methodology is firmly rooted in epidemiological and physiological research, there are inherent strengths and limitations that should be acknowledged when interpreting WHOOP Age.

Strengths of the WHOOP Healthspan Feature

The primary strength of the WHOOP Healthspan feature lies in its ability to serve as both an educational and motivational tool for individuals seeking to improve their long-term health. By translating complex epidemiological data into a format that is easily understood, WHOOP Healthspan facilitates greater awareness of the long-term implications of daily behaviors.

Additionally, the feature democratizes access to longevity science by making personalized health risk assessments more accessible to the general population. Although decades of research have established strong relationships between lifestyle factors such as sleep, physical activity, and cardiovascular fitness with long-term health, these

findings are often difficult for individuals to apply to their own lives. WHOOP Healthspan bridges this gap by providing a framework that contextualizes an individual's physiological data within the broader landscape of longevity research.

A key distinguishing feature of WHOOP Healthspan is its grounding in modifiable behaviors. Unlike other age-based health markers that may be influenced by genetic predisposition or unmodifiable physiological traits, WHOOP Age is directly linked to lifestyle factors such as sleep duration, heart rate zone time, and VO_2 Max. This ensures that the feature not only quantifies risk but also provides clear, evidence-based pathways for improvement. By aligning WHOOP Age calculations with established public health guidelines and expert recommendations, the feature offers an individualized roadmap for optimizing healthspan.

Limitations of the WHOOP Healthspan Feature

Despite its strengths, WHOOP Healthspan has inherent limitations that must be considered when interpreting its outputs. One limitation is the availability of empirical data for individuals at the extreme ends of the Healthspan components. The hazard ratios used to develop the feature are derived from large-scale epidemiological studies, which primarily focus on population averages.

“Although decades of research have established strong relationships between lifestyle factors such as sleep, physical activity, and cardiovascular fitness with long-term health, these findings are often difficult for individuals to apply to their own lives. WHOOP Healthspan bridges this gap by providing a framework that contextualizes an individual’s physiological data within the broader landscape of longevity research.”

While the feature applies rigorous statistical modeling to estimate risk across a wide range of values, there are regions where limited data availability necessitates the use of well-established physiological principles to make reasonable approximations. The WHOOP Healthspan algorithms have been designed to minimize extrapolation beyond the bounds of available evidence, but estimates may be less precise for individuals with exceptionally high or low values in certain metrics. Continued refinement of these models, informed by real-world WHOOP data and emerging scientific research, will be critical for enhancing accuracy in future iterations.

The observational nature of the underlying scientific literature also introduces limitations related to causal inference. The hazard ratios used to calculate WHOOP Age are primarily derived from prospective cohort and cross-sectional studies, which, while robust in identifying associations between behaviors and mortality risk, do not establish direct causality. Although some studies have provided evidence for the causal relationship between many of the components included in WHOOP Age and overall health,^{99,100} the majority of the literature on these behaviors and long-term health outcomes remains observational. While efforts have been made to prioritize risk estimates from studies with strong methodological rigor, there are cases where residual confounding or unmeasured variables may influence the reported associations. However, the consistency of these findings across diverse populations, combined with mechanistic insights from physiological research, suggests that many of these relationships may be causally related.

Further, WHOOP Healthspan is inherently constrained by the selection of metrics included in its framework. While the feature provides a comprehensive assessment of key physiological and behavioral factors that influence longevity — sleep, physical activity, and fitness — it does not capture all domains of healthspan. Notably, factors such as nutrition, social connection, and mental well-being, which have been shown to play critical roles in long-term health, are not currently integrated into the model. Future expansions of the feature may incorporate these additional domains to provide a more holistic representation of healthspan.

Finally, while effective age serves as a valuable heuristic for understanding long-term health risk, it should not be misconstrued as a predictor of life expectancy. Effective age is derived from all-cause mortality risk estimates, which do not directly translate into estimates of remaining lifespan. The relationship between all-cause mortality and remaining life expectancy is complex, and attempts to extrapolate WHOOP Age into predictions of longevity would be mathematically flawed. Research has

highlighted the limitations of using effective age metrics for lifespan estimation, particularly in older populations where discrepancies between effective age and chronological age may not correspond proportionally to differences in remaining life expectancy.¹⁰¹

Healthspan in Practice

The WHOOP Healthspan feature represents a significant advancement in translating longevity science into a practical and accessible tool for individuals seeking to optimize their long-term health. By bridging real-time physiological data with established mortality risk models, the feature offers a novel approach to understanding how daily behaviors influence healthspan.

The WHOOP metrics included in Healthspan — Sleep Consistency, Sleep Duration, Heart Rate Zone Time (1-3 and 4-5), Strength Activity Time, Daily Steps, VO₂ Max, Resting Heart Rate, and Lean Body Mass Percentage — were selected based on their strong associations with longevity in epidemiological and public health research, as well as their physiological basis, which provide potential biological mechanisms for these associations. WHOOP Age was shown to reflect both subjective health perception and the presence of chronic conditions, reinforcing its relevance as a measure of functional health. Individuals who reported better overall health tended to have lower WHOOP Age Deltas, while those with chronic conditions exhibited higher deltas on average — suggesting that Healthspan metrics effectively capture real-world differences in well-being and physiological status. As WHOOP continues to refine its approach to longevity tracking, future research will explore how these insights can be further leveraged to drive meaningful behavior change, empowering individuals to take proactive steps toward optimizing their long-term health.

Through continuous feedback, personalized accountability, and a strong scientific foundation, WHOOP Healthspan serves as a powerful tool for understanding and improving long-term health. More than just tracking data, Healthspan actively guides members on their path to a healthier, longer, and more fulfilling life, providing rewarding reinforcement when they succeed and a clear understanding of where to focus when challenges arise.

Reference list

1. Garmany A, Terzic A. Global Healthspan-Lifespan Gaps Among 183 World Health Organization Member States. *JAMA Netw Open*. Dec 2024;7(12):e2450241. doi:10.1001/jamanetworkopen.2024.50241
2. Boersma P, Black LI, Ward BW. Prevalence of Multiple Chronic Conditions Among US Adults, 2018. *Prev Chronic Dis*. Sep 17 2020;17:E106. doi:10.5888/pcd17.200130
3. Garmany A, Yamada S, Terzic A. Longevity leap: mind the healthspan gap. *NPJ Regen Med*. Sep 23 2021;6(1):57. doi:10.1038/s41536-021-00169-5
4. Li Y, Pan A, Wang DD, et al. Impact of Healthy Lifestyle Factors on Life Expectancies in the US Population. *Circulation*. Jul 24 2018;138(4):345-355. doi:10.1161/CIRCULATIONAHA.117.032047
5. Argentieri MA, Amin N, Nevado-Holgado AJ, et al. Integrating the environmental and genetic architectures of aging and mortality. *Nat Med*. Mar 2025;31(3):1016-1025. doi:10.1038/s41591-024-03483-9
6. Mehta NN, deGoma E, Shapiro MD. IL-6 and Cardiovascular Risk: A Narrative Review. *Curr Atheroscler Rep*. Nov 26 2024;27(1):12. doi:10.1007/s11883-024-01259-7
7. Lopez-Otin C, Blasco MA, Partridge L, Serrano M, Kroemer G. The hallmarks of aging. *Cell*. Jun 6 2013;153(6):1194-217. doi:10.1016/j.cell.2013.05.039
8. Basu AK. DNA Damage, Mutagenesis and Cancer. *Int J Mol Sci*. Mar 23 2018;19(4)doi:10.3390/ijms19040970
9. Bhatti JS, Bhatti GK, Reddy PH. Mitochondrial dysfunction and oxidative stress in metabolic disorders - A step towards mitochondria based therapeutic strategies. *Biochim Biophys Acta Mol Basis Dis*. May 2017;1863(5):1066-1077. doi:10.1016/j.bbadis.2016.11.010
10. Zhu Y, Armstrong JL, Tchkonja T, Kirkland JL. Cellular senescence and the senescent secretory phenotype in age-related chronic diseases. *Curr Opin Clin Nutr Metab Care*. Jul 2014;17(4):324-8. doi:10.1097/MCO.000000000000065
11. Olshansky SJ, Carnes BA. Ever since Gompertz. *Demography*. Feb 1997;34(1):1-15.
12. Kirkwood TB. Deciphering death: a commentary on Gompertz (1825) 'On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies'. *Philos Trans R Soc Lond B Biol Sci*. Apr 19 2015;370(1666)doi:10.1098/rstb.2014.0379
13. Dattani S. How does the risk of death change as we age – and how has this changed over time? Our World in Data. <https://ourworldindata.org/how-do-the-risks-of-death-change-as-people-age>
14. Heart Disease Prevalence. CDC. Accessed April 12, 2025. <https://www.cdc.gov/nchs/has/topics/heart-disease-prevalence.htm>
15. Alzheimer's Disease Facts and Figures. Alzheimer's Association. 2025. <https://www.alz.org/alzheimers-dementia/facts-figures>
16. Bellelli F, Angioni D, Arosio B, Vellas B, De Souto Barreto P. Hallmarks of aging and Alzheimer's Disease pathogenesis: Paving the route for new therapeutic targets. *Ageing Res Rev*. Apr 2025;106:102699. doi:10.1016/j.arr.2025.102699
17. Spiegelhalter D. How old are you, really? Communicating chronic risk through 'effective age' of your body and organs. *BMC Med Inform Decis Mak*. Aug 5 2016;16:104. doi:10.1186/s12911-016-0342-z
18. Paluch AE, Bajpai S, Bassett DR, et al. Daily steps and all-cause mortality: a meta-analysis of 15 international cohorts. *Lancet Public Health*. Mar 2022;7(3):e219-e228. doi:10.1016/S2468-2667(21)00302-9
19. Taub JM. Behavioral and psychophysiological correlates of irregularity in chronic sleep routines. *Biol Psychol*. Sep 1978;7(1-2):37-53. doi:10.1016/0301-0511(78)90041-8
20. Soehner AM, Kennedy KS, Monk TH. Circadian preference and sleep-wake regularity: associations with self-report sleep parameters in daytime-working adults. *Chronobiol Int*. Nov 2011;28(9):802-9. doi:10.3109/07420528.2011.613137
21. Sletten TL, Weaver MD, Foster RG, et al. The importance of sleep regularity: a consensus statement of the National Sleep Foundation sleep timing and variability panel. *Sleep Health*. Dec 2023;9(6):801-820. doi:10.1016/j.sleh.2023.07.016
22. Sano A, Yu AZ, McHill AW, et al. Prediction of Happy-Sad mood from daily behaviors and previous sleep history. *Annu Int Conf IEEE Eng Med Biol Soc*. 2015;2015:6796-9. doi:10.1109/EMBC.2015.7319954
23. Han KT, Kim SJ. Instability in daily life and depression: The impact of sleep variance between weekday and weekend in South Korean workers. *Health Soc Care Community*. May 2020;28(3):874-882. doi:10.1111/hsc.12918
24. Huang T, Mariani S, Redline S. Sleep Irregularity and Risk of Cardiovascular Events: The Multi-Ethnic Study of Atherosclerosis. *J Am Coll Cardiol*. Mar 10 2020;75(9):991-999. doi:10.1016/j.jacc.2019.12.054
25. Scott H, Lechat B, Guyett A, et al. Sleep Irregularity Is Associated With Hypertension: Findings From Over 2 Million Nights With a Large Global Population Sample. *Hypertension*. May 2023;80(5):1117-1126. doi:10.1161/HYPERTENSIONAHA.122.20513
26. Windred DP, Burns AC, Lane JM, et al. Sleep regularity is a stronger predictor of mortality risk than sleep duration: A prospective cohort study. *Sleep*. Jan 11 2024;47(1)doi:10.1093/sleep/zsad253
27. Walker MP, Stickgold R. Sleep, memory, and plasticity. *Annu Rev Psychol*. 2006;57:139-66. doi:10.1146/annurev.psych.56.091103.070307
28. Goldstein AN, Walker MP. The role of sleep in emotional brain function. *Annu Rev Clin Psychol*. 2014;10:679-708. doi:10.1146/annurev-clinpsy-032813-153716

29. Lv YN, Cui Y, Zhang B, Huang SM. Sleep deficiency promotes Alzheimer's disease development and progression. *Front Neurol*. 2022;13:1053942. doi:10.3389/fneur.2022.1053942
30. Adam K, Oswald I. Sleep is for tissue restoration. *J R Coll Physicians Lond*. 1977;11(4):376-88.
31. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. *Ann N Y Acad Sci*. 2008;1129:287-304. doi:10.1196/annals.1417.033
32. Ohayon MM, Reynolds CF, 3rd, Dauvilliers Y. Excessive sleep duration and quality of life. *Ann Neurol*. Jun 2013;73(6):785-94. doi:10.1002/ana.23818
33. Harvey AG. Sleep and circadian functioning: critical mechanisms in the mood disorders? *Annu Rev Clin Psychol*. 2011;7:297-319. doi:10.1146/annurev-clinpsy-032210-104550
34. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes: a systematic review, meta-analysis, and meta-regression. *Sleep Med*. Apr 2017;32:246-256. doi:10.1016/j.sleep.2016.08.006
35. Saint-Maurice PF, Freeman JR, Russ D, et al. Associations between actigraphy-measured sleep duration, continuity, and timing with mortality in the UK Biobank. *Sleep*. Mar 11 2024;47(3)doi:10.1093/sleep/zsad312
36. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep*. May 2010;33(5):585-92. doi:10.1093/sleep/33.5.585
37. Magee CA, Holliday EG, Attia J, Kritharides L, Banks E. Investigation of the relationship between sleep duration, all-cause mortality, and preexisting disease. *Sleep Med*. Jul 2013;14(7):591-6. doi:10.1016/j.sleep.2013.02.002
38. Lee DH, Rezende LFM, Joh HK, et al. Long-Term Leisure-Time Physical Activity Intensity and All-Cause and Cause-Specific Mortality: A Prospective Cohort of US Adults. *Circulation*. Aug 16 2022;146(7):523-534. doi:10.1161/CIRCULATIONAHA.121.058162
39. Colberg SR, Sigal RJ, Yardley JE, et al. Physical Activity/Exercise and Diabetes: A Position Statement of the American Diabetes Association. *Diabetes Care*. Nov 2016;39(11):2065-2079. doi:10.2337/dc16-1728
40. Chomiuk T, Niezgodna N, Mamcarz A, Sliz D. Physical activity in metabolic syndrome. *Front Physiol*. 2024;15:1365761. doi:10.3389/fphys.2024.1365761
41. Erickson KI, Voss MW, Prakash RS, et al. Exercise training increases size of hippocampus and improves memory. *Proc Natl Acad Sci U S A*. Feb 15 2011;108(7):3017-22. doi:10.1073/pnas.1015950108
42. Ahlskog JE, Geda YE, Graff-Radford NR, Petersen RC. Physical exercise as a preventive or disease-modifying treatment of dementia and brain aging. *Mayo Clin Proc*. Sep 2011;86(9):876-84. doi:10.4065/mcp.2011.0252
43. Singh B, Olds T, Curtis R, et al. Effectiveness of physical activity interventions for improving depression, anxiety and distress: an overview of systematic reviews. *Br J Sports Med*. Sep 2023;57(18):1203-1209. doi:10.1136/bjsports-2022-106195
44. Kraus WE, Powell KE, Haskell WL, et al. Physical Activity, All-Cause and Cardiovascular Mortality, and Cardiovascular Disease. *Med Sci Sports Exerc*. Jun 2019;51(6):1270-1281. doi:10.1249/MSS.0000000000001939
45. Lavie CJ, Johannsen N, Swift D, et al. Exercise is Medicine - The Importance of Physical Activity, Exercise Training, Cardiorespiratory Fitness and Obesity in the Prevention and Treatment of Type 2 Diabetes. *Eur Endocrinol*. Feb 2014;10(1):18-22. doi:10.17925/EE.2014.10.0118
46. Mandolesi L, Polverino A, Montuori S, et al. Effects of Physical Exercise on Cognitive Functioning and Wellbeing: Biological and Psychological Benefits. *Front Psychol*. 2018;9:509. doi:10.3389/fpsyg.2018.00509
47. Matthews CE, Keadle SK, Troiano RP, et al. Accelerometer-measured dose-response for physical activity, sedentary time, and mortality in US adults. *Am J Clin Nutr*. Nov 2016;104(5):1424-1432. doi:10.3945/ajcn.116.135129
48. Ahmadi MN, Clare PJ, Katzmarzyk PT, Del Pozo Cruz B, Lee IM, Stamatakis E. Vigorous physical activity, incident heart disease, and cancer: how little is enough? *Eur Heart J*. Dec 7 2022;43(46):4801-4814. doi:10.1093/eurheartj/ehac572
49. Adult Activity: An Overview. U.S. Centers for Disease Control and Prevention. <https://www.cdc.gov/physical-activity-basics/guidelines/adults.html>
50. Physical Activity. World Health Organization. <https://www.who.int/news-room/fact-sheets/detail/physical-activity>
51. Martinez-Gomez D, Luo M, Huang Y, et al. Physical Activity and All-Cause Mortality by Age in 4 Multinational Megacohorts. *JAMA Netw Open*. Nov 4 2024;7(11):e2446802. doi:10.1001/jamanetworkopen.2024.46802
52. D'Onofrio G, Kirschner J, Prather H, Goldman D, Rozanski A. Musculoskeletal exercise: Its role in promoting health and longevity. *Prog Cardiovasc Dis*. Mar-Apr 2023;77:25-36. doi:10.1016/j.pcad.2023.02.006
53. Atlantis E, Martin SA, Haren MT, Taylor AW, Wittert GA, Members of the Florey Adelaide Male Ageing S. Inverse associations between muscle mass, strength, and the metabolic syndrome. *Metabolism*. Jul 2009;58(7):1013-22. doi:10.1016/j.metabol.2009.02.027
54. Kim G, Kim JH. Impact of Skeletal Muscle Mass on Metabolic Health. *Endocrinol Metab (Seoul)*. Mar 2020;35(1):1-6. doi:10.3803/EnM.2020.35.11
55. Keating CJ, Cabrera-Linares JC, Parraga-Montilla JA, Latorre-Roman PA, Del Castillo RM, Garcia-Pinillos F. Influence of Resistance Training on Gait & Balance Parameters in Older Adults: A Systematic Review. *Int J Environ Res Public Health*. Feb 11 2021;18(4)doi:10.3390/ijerph18041759
56. Momma H, Kawakami R, Honda T, Sawada SS. Muscle-strengthening activities are associated with lower risk and mortality in major non-communicable diseases: a systematic review and meta-analysis of cohort studies. *Br J Sports Med*. Jul 2022;56(13):755-763. doi:10.1136/bjsports-2021-105061
57. Shailendra P, Baldock KL, Li LSK, Bennie JA, Boyle T. Resistance Training and Mortality Risk: A Systematic Review and Meta-Analysis. *Am J Prev Med*. Aug 2022;63(2):277-285. doi:10.1016/j.amepre.2022.03.020

58. Liu Y, Lee DC, Li Y, et al. Associations of Resistance Exercise with Cardiovascular Disease Morbidity and Mortality. *Med Sci Sports Exerc.* Mar 2019;51(3):499-508. doi:10.1249/MSS.0000000000001822
59. Stens NA, Bakker EA, Manas A, et al. Relationship of Daily Step Counts to All-Cause Mortality and Cardiovascular Events. *J Am Coll Cardiol.* Oct 10 2023;82(15):1483-1494. doi:10.1016/j.jacc.2023.07.029
60. Sisson SB, Camhi SM, Church TS, Tudor-Locke C, Johnson WD, Katzmarzyk PT. Accelerometer-determined steps/day and metabolic syndrome. *Am J Prev Med.* Jun 2010;38(6):575-82. doi:10.1016/j.amepre.2010.02.015
61. Mikkelsen K, Stojanovska L, Polenakovic M, Bosevski M, Apostolopoulos V. Exercise and mental health. *Maturitas.* Dec 2017;106:48-56. doi:10.1016/j.maturitas.2017.09.003
62. Sheng M, Yang J, Bao M, et al. The relationships between step count and all-cause mortality and cardiovascular events: A dose-response meta-analysis. *J Sport Health Sci.* Dec 2021;10(6):620-628. doi:10.1016/j.jshs.2021.09.004
63. Levine BD. $\dot{V}O_2$ max: what do we know, and what do we still need to know? *J Physiol.* Jan 1 2008;586(1):25-34. doi:10.1113/jphysiol.2007.147629
64. Najafipour F, Mobasser M, Yavari A, et al. Effect of regular exercise training on changes in HbA1c, BMI and VO_2 max among patients with type 2 diabetes mellitus: an 8-year trial. *BMJ Open Diabetes Res Care.* 2017;5(1):e000414. doi:10.1136/bmjdr-2017-000414
65. Rasch-Halvorsen O, Hassel E, Langhammer A, Brumpton BM, Steinshamn S. The association between dynamic lung volume and peak oxygen uptake in a healthy general population: the HUNT study. *BMC Pulm Med.* Jan 6 2019;19(1):2. doi:10.1186/s12890-018-0762-x
66. Weeldreyer NR, De Guzman JC, Paterson C, Allen JD, Gaesser GA, Angadi SS. Cardiorespiratory fitness, body mass index and mortality: a systematic review and meta-analysis. *Br J Sports Med.* Feb 20 2025;59(5):339-346. doi:10.1136/bjsports-2024-108748
67. 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.* Oct 5 2018;1(6):e183605. doi:10.1001/jamanetworkopen.2018.3605
68. Sorensen L, Honkalehto S, Kallinen M, et al. Are cardiorespiratory fitness and walking performance associated with self-reported quality of life and work ability? *Int J Occup Med Environ Health.* 2007;20(3):257-64. doi:10.2478/M10001-007-0023-3
69. Hawkins S, Wiswell R. Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training. *Sports Med.* 2003;33(12):877-88. doi:10.2165/00007256-200333120-00002
70. Strasser B, Burtscher M. Survival of the fittest: VO_2 max, a key predictor of longevity? *Front Biosci (Landmark Ed).* Mar 1 2018;23(8):1505-1516. doi:10.2741/4657
71. Cress ME, Meyer M. Maximal voluntary and functional performance levels needed for independence in adults aged 65 to 97 years. *Phys Ther.* Jan 2003;83(1):37-48.
72. Martins HA, Barbosa JG, Seffrin A, et al. Sex Differences in Maximal Oxygen Uptake Adjusted for Skeletal Muscle Mass in Amateur Endurance Athletes: A Cross Sectional Study. *Healthcare (Basel).* May 22 2023;11(10)doi:10.3390/healthcare11101502
73. Zhang D, Shen X, Qi X. Resting heart rate and all-cause and cardiovascular mortality in the general population: a meta-analysis. *CMAJ.* Feb 16 2016;188(3):E53-E63. doi:10.1503/cmaj.150535
74. Lutin E, Schiweck C, Cornelis J, et al. The cumulative effect of chronic stress and depressive symptoms affects heart rate in a working population. *Front Psychiatry.* 2022;13:1022298. doi:10.3389/fpsy.2022.1022298
75. Carter JB, Banister EW, Blaber AP. Effect of endurance exercise on autonomic control of heart rate. *Sports Med.* 2003;33(1):33-46. doi:10.2165/00007256-200333010-00003
76. Zhang D, Wang W, Li F. Association between resting heart rate and coronary artery disease, stroke, sudden death and noncardiovascular diseases: a meta-analysis. *CMAJ.* Oct 18 2016;188(15):E384-E392. doi:10.1503/cmaj.160050
77. Jensen MT, Suadicani P, Hein HO, Gyntelberg F. Elevated resting heart rate, physical fitness and all-cause mortality: a 16-year follow-up in the Copenhagen Male Study. *Heart.* Jun 2013;99(12):882-7. doi:10.1136/heartjnl-2012-303375
78. Gomez-Ambrosi J, Silva C, Galofre JC, et al. Body adiposity and type 2 diabetes: increased risk with a high body fat percentage even having a normal BMI. *Obesity (Silver Spring).* Jul 2011;19(7):1439-44. doi:10.1038/oby.2011.36
79. Gielen E, Dupont J, Dejaeger M, Laurent MR. Sarcopenia, osteoporosis and frailty. *Metabolism.* Aug 2023;145:155638. doi:10.1016/j.metabol.2023.155638
80. Landi F, Calvani R, Cesari M, et al. Sarcopenia as the Biological Substrate of Physical Frailty. *Clin Geriatr Med.* Aug 2015;31(3):367-74. doi:10.1016/j.cger.2015.04.005
81. Duggal NA, Niemi G, Harridge SDR, Simpson RJ, Lord JM. Can physical activity ameliorate immunosenescence and thereby reduce age-related multi-morbidity? *Nat Rev Immunol.* Sep 2019;19(9):563-572. doi:10.1038/s41577-019-0177-9
82. Padwal R, Leslie WD, Lix LM, Majumdar SR. Relationship Among Body Fat Percentage, Body Mass Index, and All-Cause Mortality: A Cohort Study. *Ann Intern Med.* Apr 19 2016;164(8):532-41. doi:10.7326/M15-1181
83. Jayedi A, Khan TA, Aune D, Emadi A, Shab-Bidar S. Body fat and risk of all-cause mortality: a systematic review and dose-response meta-analysis of prospective cohort studies. *Int J Obes (Lond).* Sep 2022;46(9):1573-1581. doi:10.1038/s41366-022-01165-5
84. Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. *J Health Soc Behav.* Mar 1997;38(1):21-37.
85. DeSalvo KB, Bloser N, Reynolds K, He J, Muntner P. Mortality prediction with a single general self-rated health question. A meta-analysis. *J Gen Intern Med.* Mar 2006;21(3):267-75. doi:10.1111/j.1525-1497.2005.00291.x

86. Ford ES, Cunningham TJ, Croft JB. Trends in Self-Reported Sleep Duration among US Adults from 1985 to 2012. *Sleep*. May 1 2015;38(5):829-32. doi:10.5665/sleep.4684
87. Lee PH. Validation of the National Health And Nutritional Survey (NHANES) single-item self-reported sleep duration against wrist-worn accelerometer. *Sleep Breath*. Dec 2022;26(4):2069-2075. doi:10.1007/s11325-021-02542-6
88. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? *Epidemiology*. Nov 2008;19(6):838-45. doi:10.1097/EDE.0b013e318187a7b0
89. Degenhard SM, Farmer N, Yang L, Barb JJ, Maki KA, Wallen GR. Specific Nutrients Mediate the Association of Food Insecurity and Sleep Regularity Index (SRI) in U.S. Adults: NHANES 2011-2014. *Nutrients*. Jan 18 2025;17(2)doi:10.3390/nu17020340
90. Lunsford-Avery JR, Engelhard MM, Navar AM, Kollins SH. Validation of the Sleep Regularity Index in Older Adults and Associations with Cardiometabolic Risk. *Sci Rep*. Sep 21 2018;8(1):14158. doi:10.1038/s41598-018-32402-5
91. Bassett DR, Jr., Wyatt HR, Thompson H, Peters JC, Hill JO. Pedometer-measured physical activity and health behaviors in U.S. adults. *Med Sci Sports Exerc*. Oct 2010;42(10):1819-25. doi:10.1249/MSS.0b013e3181dc2e54
92. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. *Med Sci Sports Exerc*. Jan 2008;40(1):181-8. doi:10.1249/mss.0b013e31815a51b3
93. Bennie JA, Lee DC, Khan A, et al. Muscle-Strengthening Exercise Among 397,423 U.S. Adults: Prevalence, Correlates, and Associations With Health Conditions. *Am J Prev Med*. Dec 2018;55(6):864-874. doi:10.1016/j.amepre.2018.07.022
94. Kaminsky LA, Arena R, Myers J. Reference Standards for Cardiorespiratory Fitness Measured With Cardiopulmonary Exercise Testing: Data From the Fitness Registry and the Importance of Exercise National Database. *Mayo Clin Proc*. Nov 2015;90(11):1515-23. doi:10.1016/j.mayocp.2015.07.026
95. Avram R, Tison GH, Aschbacher K, et al. Real-world heart rate norms in the Health eHeart study. *NPJ Digit Med*. 2019;2:58. doi:10.1038/s41746-019-0134-9
96. Johansen CD, Olsen RH, Pedersen LR, et al. Resting, night-time, and 24 h heart rate as markers of cardiovascular risk in middle-aged and elderly men and women with no apparent heart disease. *Eur Heart J*. Jun 2013;34(23):1732-9. doi:10.1093/eurheartj/ehs449
97. Borrud LG, Flegal KM, Looker AC, Everhart JE, Harris TB, Shepherd JA. Body composition data for individuals 8 years of age and older: U.S. population, 1999-2004. *Vital Health Stat 11*. Apr 2010;(250):1-87.
98. Chandrasekaran R, Katthula V, Moustakas E. Patterns of Use and Key Predictors for the Use of Wearable Health Care Devices by US Adults: Insights from a National Survey. *J Med Internet Res*. Oct 16 2020;22(10):e22443. doi:10.2196/22443
99. Posadzki P, Pieper D, Bajpai R, et al. Exercise/physical activity and health outcomes: an overview of Cochrane systematic reviews. *BMC Public Health*. Nov 16 2020;20(1):1724. doi:10.1186/s12889-020-09855-3
100. Zhu B, Shi C, Park CG, Zhao X, Reutrakul S. Effects of sleep restriction on metabolism-related parameters in healthy adults: A comprehensive review and meta-analysis of randomized controlled trials. *Sleep Med Rev*. Jun 2019;45:18-30. doi:10.1016/j.smr.2019.02.002
101. Pang M, Hanley JA. "Translating" All-Cause Mortality Rate Ratios or Hazard Ratios to Age-, Longevity-, and Probability-Based Measures. *Am J Epidemiol*. Dec 1 2021;190(12):2664-2670. doi:10.1093/aje/kwab178