

A RESEARCH BRIEFING FOR MEN OVER 40

How Men Age Faster at Specific Points in Life

Which ages are real, which are marketing, and what the evidence says you can still change

You have probably read that science has proven men age in sudden bursts, at 44 and again at 60. It is a compelling story and it has travelled a long way. This briefing examines it against the primary research, including a 2026 methods paper co-authored by the very scientists who produced the original finding. The answer turns out to be more useful than the headline: some of your curves genuinely do bend at identifiable ages, those ages are mostly not the ones you have been given, and the sharpest accelerations in a man's life are not triggered by birthdays at all.

Prepared by **Dr. John Spencer Ellis** · Health, Longevity and Aesthetic Optimization Coach for Men Over 40 · johnspencerellis.com

START HERE

Fifteen questions, answered in one line each

Every answer below is defended, sourced and qualified later in the paper. Where the honest answer contradicts something widely published, it says so.

Do men really age in sudden bursts at 44 and 60?

That claim rests on one figure legend in a study of 108 people. The same paper's results text says 45 and 65, and its protein data say 40 and 60. Across six independent studies the reported crest ages run from 34 to 78, and the only one that recurs is about 60.

Has anyone challenged the method behind that finding?

Yes, and the challenge comes partly from inside. A June 2026 methods paper, co-authored by the senior and first authors of the 44-and-60 study, showed that the statistical technique behind it can produce pronounced waves from data in which aging is perfectly linear.

Was the 44 crest just menopause in the female participants?

The authors addressed it, but weakly. They compared clusters separately in roughly 52 men and 56 women, said the clusters looked similar, and concluded the change was *not solely* attributable to menopause. No male-only crest age was ever published. The one study that did stratify by sex found male crests arriving ten to fifteen years later than female ones.

So is there any age at which men genuinely change?

Several, and they are well documented. Brain volume loss begins around 35. Strength and power begin falling at about 40. Measurable decline in reasoning is present in men by 45. Total energy expenditure holds flat until about 63. Hip bone loss speeds up after 65.

Does testosterone fall off a cliff after 40?

No. In 890 men followed longitudinally, total and free testosterone declined in a straight line, and the authors described the age effect as age-invariant — meaning it did not accelerate. No inflection point for male testosterone has ever been demonstrated.

Does my metabolism slow down in my forties?

Not the way you have been told. Across 6,421 people measured with the gold-standard method, energy expenditure adjusted for body composition was statistically flat from 20 to 63. Midlife weight gain is not caused by a slowing metabolism. It is caused by what you eat, how you move, and the muscle you have lost.

What is actually losing me the most, the fastest?

Strength, not muscle. In a longitudinal study of men in their seventies, leg strength fell 3 to 4% a year while leg lean mass fell about 1%. Gaining or maintaining lean mass did not prevent the strength decline. Strength is the curve that predicts function.

When does my face start to change?

Not all at once. In CT scans of 78 men by decade, the maxillary angle changed most in the forties, the palate in the fifties, the eye socket and nasal aperture in the sixties. There is no single decade when a face ages.

Is the 1% a year collagen loss figure real?

Not for men. That number traces back through a review to a two-page editorial, and the only hard per-year collagen figures in the literature come from postmenopausal women, where the driver is estrogen withdrawal — a mechanism men do not have.

What single life event does the most damage to a man?

On the evidence, losing a job in your forties or losing a spouse. Unemployment carries a mortality hazard of 1.78 in men against 1.37 in women, worst between 40 and 50. In the first six months after a spouse's death the risk rises about 41%, and in women the same analysis found no distinguishable effect at all.

Does divorce age a man?

Marital dissolution carries a mortality hazard of 1.37 in men against 1.22 in women, pooled across 104 studies and more than 600 million people, and the male excess is largest in midlife. Whether it causes the aging or selects for men already struggling cannot be separated from this data.

Will a biological age test tell me how fast I'm aging?

Not reliably, as an individual. Technical noise alone produced deviations of up to nine years between replicates of the same sample. In UK Biobank, 68% of people flagged as extreme organ agers were no longer extreme at their next visit.

What about the longevity supplements?

Metformin's flagship aging trial never enrolled a patient. Resveratrol has 104 trials and its own systematic reviewers concluded there is no clinical evidence to recommend it. Rapamycin's one healthspan trial missed its primary endpoint. None of these belongs near the top of your list.

Then what actually works?

In order of evidence: get your blood pressure under 120, stop smoking, have one aneurysm scan at 65, train for strength and for cardiorespiratory fitness, and lose excess weight. The top of the list is cheap, unglamorous and almost entirely unsold.

What is the one thing you would tell a 45-year-old man?

Stop watching your birthdays and start watching your transitions. The best-evidenced accelerations in male aging cluster around job loss, marital breakdown and bereavement — and unlike your chronological age, how you handle those is partly within your control.

ABOUT THIS BRIEFING

Who wrote it, and how it was built



Dr. John Spencer Ellis

Dr. John Spencer Ellis is 57. He has spent more than three decades in health, fitness and human performance — creating certification programs used by tens of thousands of coaches worldwide, founding multiple fitness education organizations, authoring Amazon #1 bestselling books, and producing and directing documentary films on health and human potential. He works today with men over 40 on longevity, health, fitness and aesthetic optimization. He is also living the subject of this paper rather than observing it. Full background and credentials.

What he is not. He is not a physician and not a research scientist. Nothing here diagnoses a condition or replaces medical care. Several of the most valuable items in Part Four — blood pressure control, aneurysm screening, hormone evaluation — require a doctor, and the paper says so at each point.

How the evidence was graded. Every quantitative claim in this paper is traceable to a named primary source, and the principal ones are listed at the back. Where sources disagree, both are printed. Sample sizes and confidence intervals appear wherever they change how much weight a finding deserves. Mechanistic animal work is labeled as animal work. Cross-sectional findings are distinguished from longitudinal ones, because in aging research that distinction routinely reverses conclusions. Where a figure could not be traced to a primary source, the paper says that rather than repeating it.

DISCLOSURE

— Nothing in this paper is sponsored. No supplement company, device manufacturer, clinic, laboratory or pharmaceutical firm paid for, reviewed or influenced any part of it.

— Dr. Ellis sells coaching. That is the commercial interest behind this document, it is disclosed here, and the coaching sections are marked.

— Published 2026. One of the central sources is a June 2026 preprint that has not yet completed peer review, and the paper says so where it is used.

WHAT THIS PAPER DELIBERATELY DOES NOT CLAIM

It does not claim that men age in bursts at 44 and 60. It does not claim testosterone falls off a cliff. It does not claim your metabolism slowed in your thirties. It does not claim you lose 1% of your collagen a year. It does not claim caregiver stress ages you by a decade, that loneliness equals fifteen cigarettes a day, or that any supplement on the market has been shown to extend human life. Each of those appears constantly in content aimed at men your age, each is addressed in the pages that follow, and each fails for a different reason.

PART ONE

The Story You Have Been Told

A single study produced the most widely repeated claim in modern aging content. Here is what it actually found, and what happened to it afterwards.

01

CHAPTER 1

The 44-and-60 claim, examined

Eight studies have looked for bursts of molecular aging. They disagree with each other, and with themselves.

In 2024, a team at Stanford published an analysis of multi-omic profiles — proteins, metabolites, lipids, transcripts, microbes — across the human lifespan, and reported that the molecular changes of aging are not smooth. They cluster. The press coverage settled on two numbers: **44 and 60**. Those two ages have since appeared in more or less every article, podcast and supplement advertisement aimed at men in midlife.

The study is real and the scientists are serious. But the figure deserves four pieces of context that almost never travel with it.

First: the study was small, and barely longitudinal

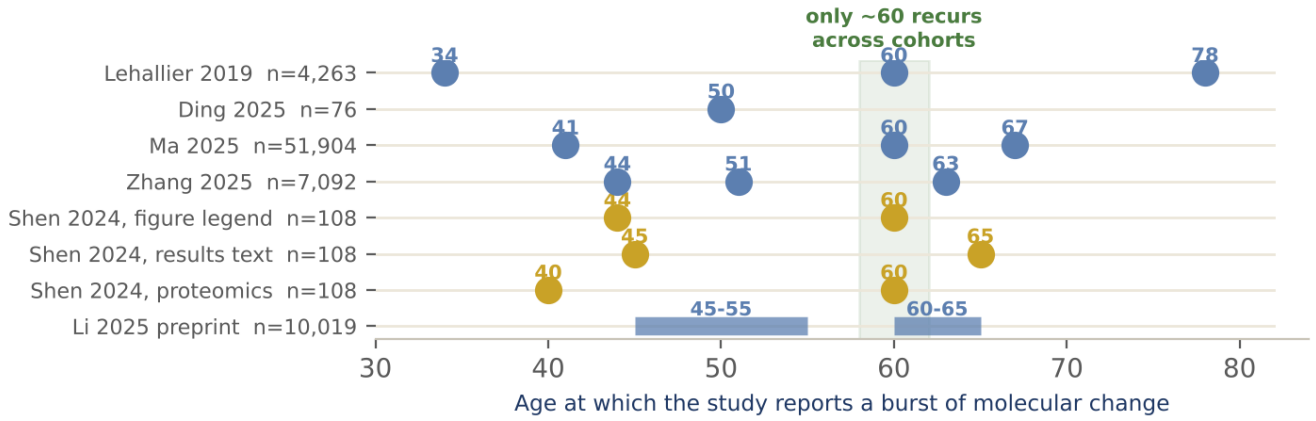
It followed **108 people**, of whom roughly 52 were men, in a single region of California, for a **median of 1.7 years**. A follow-up window of under two years cannot observe a change that happens to a person at age 44. The design is effectively cross-sectional for the question being asked of it. The headline figure of 135,239 biological features is also larger than the analysis: statistical testing ran on **11,305** molecules and microbes.

Second: the paper does not agree with itself on the ages

The famous pairing of 44 and 60 appears in a **figure legend**. The main results text of the same paper says **45 and 65**. The proteomics layer specifically reports **40 and 60**. A separate trajectory analysis in the same paper gives a turning point around **55 to 60**. That is a twenty-year spread inside one document, and the number that escaped into public circulation is the one from the figure caption.

Third: no other cohort agrees either

Six studies, and no two agree on when aging 'accelerates'



The famous 'aging happens at 44 and 60' comes from one figure legend in a 108-person study, which supplies three of the rows below. That same paper's results text says 45 and 65, and its proteomics layer says 40 and 60. Across independent cohorts the only age that recurs is about 60. In June 2026 a methods preprint co-authored by the senior and first first authors of the 44/60 study showed the sliding-window technique behind most of these can manufacture waves from data in which aging is perfectly linear. The Li row is drawn as the ranges its authors published, not as points.

A 2019 analysis of 4,263 people found waves at 34, 60 and 78. A 2025 study of 51,904 people in UK Biobank found peaks at 41, 60 and 67. Another 2025 analysis found 44, 51 and 63. A post-mortem tissue study found an inflection around 50. Taken together, the only age that recurs across independent cohorts is **about 60**.

Fourth, and most seriously: the method itself is now under challenge

THE 2026 REBUTTAL, AND WHO WROTE IT

In June 2026 a methods paper argued that the statistical technique used in all of these studies can manufacture the very thing they report. Three findings: clustering trajectories fitted with a common smoothing method can produce artificial patterns; the waves found in real data are not robust to changes in the age distribution of the sample and fail valid permutation testing; and the technique **alone can produce pronounced nonlinear waves from data in which aging is perfectly linear**. The authors include **Michael Snyder and Xiaotao Shen — the senior and first authors of the 44-and-60 study itself**. This paper is a preprint and has not completed peer review, which is stated here rather than buried. It does not prove that nonlinear aging is unreal. It does mean no honest document can present 44 and 60 as established.

None of this makes the underlying idea wrong. Aging almost certainly is uneven. But the specific, confident, two-number version of that idea — the one being sold to men in their forties — is resting on much less than it appears to.

02

CHAPTER 2

What it means for men specifically

The one study that separated the sexes found the male pattern arriving a decade or more later.

A reasonable objection to the 44 crest is that the age coincides with perimenopause in the female participants, and that a mixed-sex sample could show a crest driven almost entirely by the women in it. The authors anticipated this. What they did was compare the clustering patterns separately in the men and the women, observe that the clusters looked similar, and conclude that the transition was "not *solely* attributed to female menopause."

Read that wording carefully, because it is the authors being careful. What the paper does **not** contain is a male-only crest age, a male-only crest magnitude, or any statistical test that the male crest occurs at the same age as the female one. It is a qualitative comparison of cluster shapes in roughly 52 men, presented in a supplementary figure.

Meanwhile the only study found that did report sex-stratified crest ages — an analysis of 10,019 participants, and itself a preprint with acknowledged inconsistencies — found female crests at 50 to 55, which the authors attributed to menopause, and **male crests arriving ten to fifteen years later**. That is a single unreviewed study and should not be treated as settled either. But it points in the opposite direction to the popular claim.

WHERE THIS LEAVES A MAN IN HIS FORTIES

— There is no published estimate of a male molecular crest age. Not from the Stanford study, and not from any other.
— The ages in circulation come from mixed-sex samples in which the female menopausal transition is a plausible driver.
— The limited sex-stratified evidence suggests the male pattern is later, not simultaneous.
— If you are a man of 44 who has been told the science says this is your year, the science does not say that.

Why John starts here. Men arrive having absorbed a story about their age and bought products built on it. The first useful thing a coach can do is separate what is actually happening in your body from what is being marketed at your birth year, because the effort you spend on the wrong target is effort you do not get back. Request an evaluation

PART TWO

Where Men's Curves Genuinely Bend

Forget the molecular crests. These are the inflection points that longitudinal studies have actually measured in men, with the age attached to each.

Where a man's curves actually bend, by measured inflection age

35	**Brain volume** Steady loss begins, about 0.2% a year	Hedman 2012, 56 pooled longitudinal MRI studies, both sexes
40	**Strength and power** Decline begins. Power falls about 10% faster than strength	Metter 1997, BLSA, men, 9.6-year follow-up
40s	**The maxilla** Largest single decade of change in the mid-face angle	Cotofana 2018, CT scans, 78 men by decade
45	**Reasoning** Already measurable in men: -3.6% per decade	Singh-Manoux 2012, Whitehall II, 5,198 men
46.5	**Basal metabolic rate** Breakpoint, then -0.45% a year	Pontzer 2021, Science, CI 40.6 to 52.4 years
45-50	**Absolute muscle mass** Starts to fall, mostly in the lower body	Janssen 2000, MRI, n=468
60	**Brain volume, again** Loss reaches 0.5% a year and keeps rising	Hedman 2012
60	**Skin thickness** Falls, after holding roughly steady from 20	Hall 1981, n=626
60-70	**Grip strength** Acceleration seen in men and not in women	Fieldsend 2025 preprint, UK Biobank, sex-matched subset
63.0	**Total energy expenditure** The plateau finally ends: -0.7% a year	Pontzer 2021, CI 60.1 to 65.9 years
65	**Hip bone density** Rate of loss increases	Berger 2008, CaMos, 1,935 men, longitudinal

Each row is an age at which a published study found the RATE of change itself alter, not merely continue. Only two of these breakpoints were formally estimated with confidence intervals, both from Pontzer 2021; the rest are read off fitted curves. Testosterone is deliberately absent from this list: in 890 men followed longitudinally its decline was explicitly described as age-invariant, and no inflection point for it has ever been demonstrated.

Two things are worth noticing about that list before reading the chapters behind it. The first is that the ages are spread across thirty years — there is no single cliff. The second is that almost nothing bends at 44.

03

CHAPTER 3

Strength, and the mistake almost everyone makes

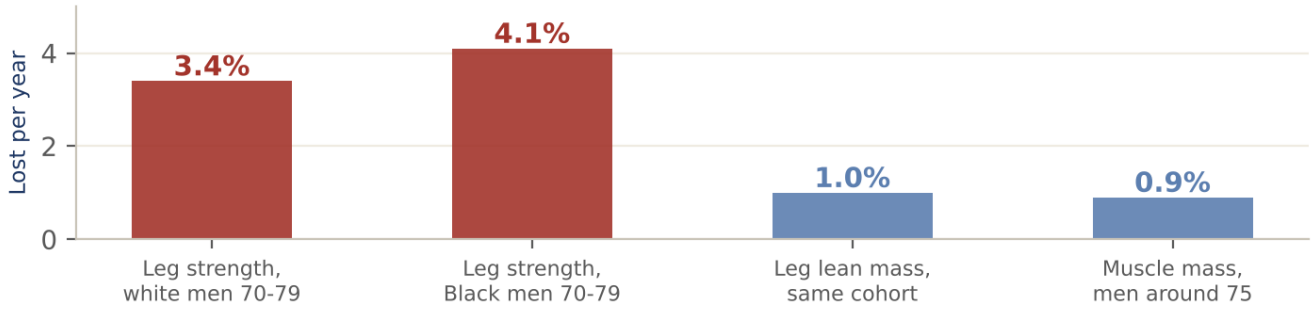
You are not primarily losing muscle. You are losing the ability to use it, three to four times faster.

The familiar line is that men lose 3 to 8% of their muscle mass per decade after 30. The rate is roughly right; the age and the emphasis are both wrong, and the second error is the expensive one.

On the age: MRI measurement in 468 people found that **absolute** muscle mass in men shows no noticeable decrease until the end of the fifth decade — roughly 45 to 50. What falls from 30 is muscle as a *percentage of body weight*, a ratio that drops largely because the denominator is growing. Those are very different statements, and only one of them is about losing muscle.

On the emphasis: the wrong variable is being tracked

Strength disappears three to four times faster than muscle



Health ABC study, Goodpaster 2006, longitudinal and sex-stratified. The finding that matters most: gaining or maintaining lean mass did NOT prevent strength decline. Across the wider literature strength is lost two to five times faster than mass, and in men power falls about 10% faster still. One caveat that is almost always dropped: these rates describe men aged 70 to 79 and must not be applied to a man in his forties. The lean-mass figure was not reported separately by sex, and the muscle-mass figure is the midpoint of a published range of 0.80 to 0.98% a year.

In the Health ABC study, men aged 70 to 79 were followed with objective measurement. Leg strength fell **3.4% a year** in white men and **4.1%** in Black men. Leg lean mass fell about **1%**. And the finding that should change how you train: **gaining or maintaining lean mass did not prevent the strength decline**.

Across the broader literature strength is lost two to five times faster than mass, and in men, power — force produced quickly — declines about 10% more than strength does, beginning around age 40. Sarcopenia, the loss of mass, and dynapenia, the loss of strength, are not the same curve, and the one that predicts whether you can get off the floor at 80 is the steeper one.

40
IS WHEN STRENGTH AND POWER BEGIN

to decline in men, measured longitudinally — five to ten years before absolute muscle mass starts falling at all.

ONE CAVEAT THAT IS ALMOST ALWAYS DROPPED

Those 3.4 and 4.1% figures describe men aged 70 to 79. They are routinely quoted in articles aimed at men in their forties, where they do not apply. A 45-year-old is not losing 4% of his strength a year. He is, however, now on a curve that bends downward, which is a different and more actionable fact.

04

CHAPTER 4

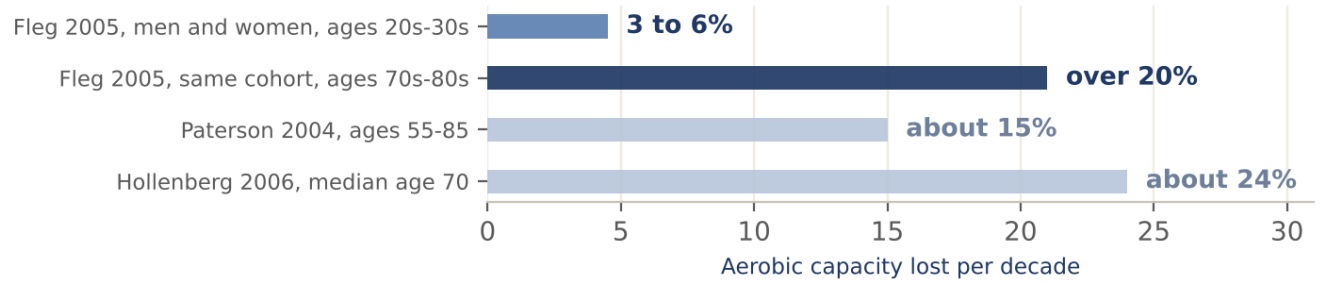
The heart, and the real male midlife divergence

This is the genuine male-specific acceleration, and it has nothing to do with molecular crests.

If you want one well-replicated finding that men hit something in midlife that women do not, it is cardiovascular. Coronary heart disease incidence rises roughly **ten years earlier in men** than in women and runs roughly **double in middle age**. The gap narrows after 60 and is small after 80. This is one of the most robust findings in epidemiology, replicated across countries and decades.

Aerobic capacity tells a related story. In the Baltimore Longitudinal Study of Aging, 810 people including 435 men were followed for seven to nine years. One disclosure before the chart: the figures below could only be confirmed through professional-medical summaries of that study's abstract rather than its full text, and the decades between its two published endpoints were never retrievable.

Why 'VO2 max falls 10% a decade' is wrong at both ends



The two dark bars are the only decade figures the Baltimore Longitudinal Study of Aging published for its 810 participants, 435 of them men: the rate of loss roughly quadruples between the twenties and the eighties, and men decline faster than women from their forties on. The intervening decades were never published in a form this paper could obtain, so the pale bars are estimates from two separate cohorts at the age ranges those studies actually covered, not Fleg's. One disclosure: the Fleg figures could only be confirmed through professional-medical summaries of the abstract rather than the full article. The study's other finding is awkward: decline rates were similar across activity levels, so training lifts the whole curve rather than flattening it.

Two things in that chart matter. The rate of loss **quadruples** across the lifespan, so the commonly quoted flat 10% per decade is wrong at both ends. And **men decline faster than women from their forties onward** — which is the closest thing in a longitudinal dataset to the premise of this paper.

The study's other finding is less comfortable. Rates of decline were similar across levels of self-reported physical activity, while activity predicted better fitness at every age. In other words training appears to raise the whole curve rather than flatten it. That is still an overwhelming argument for building capacity — a fit 50-year-old declining at the same rate from a higher starting point arrives at 75 in a different category of person — but it is not the same as slowing the rate of aging, and this paper will not claim that it is.

05

CHAPTER 5

The brain, measurable earlier than you think

Decline in reasoning is detectable in men at 45, and brain volume has been shrinking since 35.

Pooled across 56 longitudinal MRI studies, brain volume begins a steady decline around **age 35** at roughly 0.2% a year, reaching about **0.5% a year by 60** and continuing to rise from there. That is a clean, quantified, longitudinally measured double inflection — precisely the kind of evidence the molecular crest literature lacks.

On cognition, the Whitehall II study followed **5,198 men** for ten years with repeated testing. Reasoning declined **3.6% per decade in men aged 45 to 49**, rising to **9.6% at 65 to 70** — about 2.7 times the rate. Every cognitive score except vocabulary declined in all five age bands.

Why this contradicts what you may have read. The two study designs disagree about cognition, and they are biased in opposite directions. Cross-sectional studies, comparing different people at one moment, are inflated by cohort effects — younger generations score better for reasons unrelated to aging — and put peak performance as early as the mid-twenties. Longitudinal studies, following the same people, are deflated by practice and retest effects, which can make repeated scores hold steady or even rise. Neither design is simply right. The honest reading is that decline in men is detectable somewhere in the forties, and that Whitehall II is an occupationally selected British civil-service cohort, which is a further limitation.

06

CHAPTER 6

Bone, and the statistic men never hear

Lower prevalence than women. Considerably worse consequences.

Male bone loss is slow and steady from about 35, and then the rate of hip loss **increases after 65** — measured longitudinally in 1,935 men. Men over 65 lose roughly 0.35 to 0.6% of total hip bone density a year, concentrated in the over-75s and the vitamin-D deficient.

A white man's lifetime risk of a clinical osteoporotic fracture is about **13%**, against 40% in women, and roughly **30% of all hip fractures worldwide occur in men**. That is the part men hear, and it reads as reassurance. The next part does not.

AFTER A HIP FRACTURE, MEN DO CONSIDERABLY WORSE

In a meta-analysis of prospective cohorts with the sexes analyzed separately, a man's all-cause mortality hazard in the three months after a hip fracture was **7.95 times** that of an unfractured peer, against 5.75 in women. Excess annual mortality at age 80 ran **18% at one year, 22% at two and 26% at five** in men, against 8%, 11% and 18% in women — more than double at one year. Relative hazards never returned to matched control rates. This is the best-supported under-appreciated statistic in this entire paper.

One practical note. In the same longitudinal cohort, lumbar spine bone density was found to be *rising* in men aged 55 to 59 while hip density in those same men was falling. That is degenerative change inflating the spine reading, not bone being regained. In an aging man, the hip is the site to watch.

07

CHAPTER 7

The face, which ages by structure and not by year

There is no single decade when a man's face changes. There are four, and each one changes something different.

CT scanning of **78 men**, ten per decade, found that different parts of the facial skeleton undergo their largest change in different decades. The **maxillary angle** — the bone supporting the midface — changes most in the **forties**. The palate changes most in the **fifties**. The orbital floor and the nasal aperture change most in the **sixties**. Orbital width and midfacial height change most in a man's **eighties**.

Bone is the foundation under everything a man notices in a mirror. As the maxilla rotates and the orbit widens, the soft tissue above it loses its scaffolding, which is why midface descent and under-eye hollowing read as aging even when skin quality is good.

Three appearance figures that do not survive checking

THE CLAIM	WHAT THE SOURCE ACTUALLY SUPPORTS
Skin collagen falls about 1% a year from your twenties	Unsourceable as applied to men. The most-cited review attributes it to two other reviews, one of which is a two-page editorial, and repeats the figure elsewhere with no citation at all. The only hard per-year collagen rates in that literature are from *postmenopausal women*, where the driver is estrogen withdrawal. Men are being sold a menopause statistic.
Skin thins steadily from early adulthood	Measured skinfold thickness holds roughly steady from 20 to 60 and then falls; that study made no direct collagen measurement and published no annual rate. Male skin is also thicker and oilier than female skin to begin with.
The 50-50-50 rule: half of people are half grey by 50	In 4,192 people the figure for 50% or more grey coverage by age 50 is between 6 and 23%. Between 45 and 65, 74% have *some* grey, at a mean intensity of 27%. Men do grey more than women.
The Norwood rule of decades for hair loss	Not verifiable from Norwood's 1975 primary paper, which is paywalled and whose incidence table could not be retrieved. Treat the tidy decade percentages as folk epidemiology.

08

CHAPTER 8

Two things that do not change the way you were told

Your metabolism and your testosterone are the two most misrepresented curves in male aging.

Your metabolism did not slow down in your thirties

This is the best-verified item in the entire paper. Using doubly labelled water — the gold standard for measuring energy expenditure in free-living humans — across **6,421 people in 29 countries**, total daily energy expenditure adjusted for fat-free mass changed by –**0.02% a year between ages 20 and 60**, with a confidence interval running from –0.07 to +0.03. That is statistically indistinguishable from zero. The breakpoint landed at **63.0 years**, with a confidence interval of 60.1 to 65.9.

The honest footnote is that *basal* expenditure did bend earlier, at about **46.5 years**, so “nothing happens until 60” overstates it too. But the central message stands and it is good news: midlife weight gain is not caused by a failing metabolism. It is caused by intake, activity, and the muscle you have stopped carrying — all three of which you can act on.

Your testosterone is not falling off a cliff

The canonical claim is a 1% annual decline starting at 30. Neither half is right. The Massachusetts Male Aging Study, which is where the figure traces to, enrolled men aged **40 to 70** and therefore says nothing whatsoever about age 30. Its longitudinal total testosterone decline was 1.6% a year. The Baltimore and European studies published absolute rates that work out at roughly 0.7% and 0.6% a year — arithmetic applied here, not figures those papers printed. No primary source reports 1% a year starting at 30.

More importantly, in 890 men followed longitudinally the decline was **linear**, and the authors described the age effect as *age-invariant* — it did not accelerate. The rising prevalence of low testosterone with age is what a straight-line decline looks like when a population crosses a fixed diagnostic threshold at different times. It is not a cliff.

THE NUMBER THAT REFRAMES THIS ENTIRE TOPIC

Applying a rigorous symptomatic definition, the European Male Ageing Study found that **2.1%** of 2,966 men aged 40 to 79 met criteria for symptomatic late-onset hypogonadism — 63 men, of whom 27 were severe. Meanwhile **2.91%** of commercially insured American men over 40 were being prescribed androgen therapy by 2011, up 3.6 times in a decade, and **25.3% of new prescriptions began with no testosterone measurement in the previous year**. These are different populations and the comparison is directional rather than exact. It is still the clearest signal in the literature that prescribing has outrun diagnosis.

None of which means low testosterone is imaginary or that treatment is wrong. Chapter 14 covers what testosterone therapy does and does not do, in detail and without either the enthusiasm or the squeamishness that usually attends the subject. The point here is narrower: **weight, not age, is the main modifiable determinant of a man's testosterone**, and in pooled trials, losing weight raised it more reliably than anything

sold in a bottle.

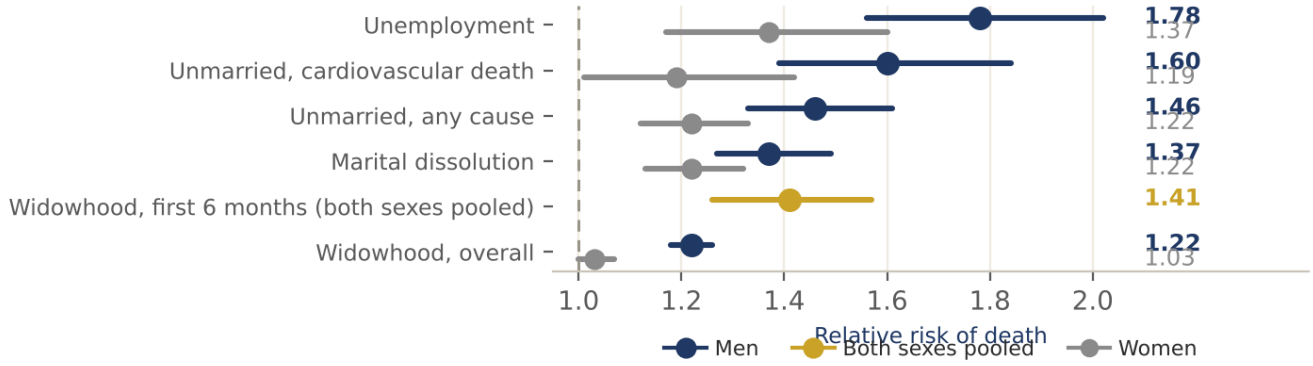
What this part changes in practice. Most men over 40 are training for mass and tracking weight, on the assumption that their metabolism has betrayed them and their hormones are collapsing. The measured curves say the opposite: metabolism is intact until the sixties, hormones decline slowly and largely track body fat, and the thing falling fastest is strength and power — which responds to training better than almost anything else in this paper. See how the program is built around that

PART THREE

The Points That Actually Age Men

The sharpest, best-evidenced accelerations in a man's life are not birthdays. They are events — and they land measurably harder on men than on women.

The events that age men, and how much worse they land on men



Every row is a published meta-analysis with the sexes analyzed separately. Unemployment: Roelfs 2011, 42 studies, over 20 million people. Its by-age breakdown peaks at 1.77 for job loss between 40 and 50, but that breakdown was not split by sex; only the 1.78 against 1.37 figure was. Marital status: Wang 2020, 7.9 million people, with a formal sex-interaction test confirming the male excess. Marital dissolution: Shor 2012, 104 studies. Widowhood: Moon 2011, 2.26 million people, where the female estimate is not distinguishable from no effect at all. These are associations in observational data, not proof that the event caused the death.

Every row in that chart is a published meta-analysis in which the sexes were analyzed separately. Read together they make a claim that the molecular literature cannot: that there are identifiable points in a man's life where his risk changes sharply, that those points are datable, and that in several cases the equivalent effect in women is small or absent.

WHAT THESE NUMBERS ARE, AND ARE NOT

These are associations from observational data. No one has randomized a man to divorce, redundancy or widowhood, and no cohort has measured biological aging in men before and after any of them. Health selection runs the other way too: men who are already unwell, drinking heavily or earning less are more likely to lose a job and less likely to stay married. Throughout this part, read *associated with* and not *causes*.

09

CHAPTER 9

Losing a job, in your forties

The largest effect in the chart, in the narrowest age window, and it hits men far harder than women.

Pooled across 42 studies, 235 risk estimates and more than 20 million people, unemployment carried an all-cause mortality hazard of **1.78 in men** against **1.37 in women**. Meta-regression confirmed the male excess at around 37% greater, with p<0.001.

The age breakdown is the part worth pinning to a wall. Job loss before 40 carried a hazard of 1.73. Between **40 and 50**, **1.77**. Between 50 and 65 it fell to **1.25** — significantly lower, at p=0.0016. The worst window for a man losing his work is not the end of his career. It is the middle of it.

The mechanism matters because it is the part a man can act on. Controlling for health measures did not change the estimates at all. Controlling for **health behaviors** lowered them by 24%. The authors read this as weak support for the idea that sick men lose jobs, and better support for what they call the coping hypothesis — that unemployment drives harmful behavior change. Drinking, inactivity, sleep collapse, and the quiet disappearance of every structure that used to organise a day.

1.77
MORTALITY HAZARD FOR A MAN

who loses his job between 40 and 50 — the worst of any age band, and 42% higher than the same event after 50.

Retirement is a different question, and the answer is mixed

Retirement itself does not appear to be harmful. Pooled across 12 studies, early retirement versus working to retirement gave a fully adjusted hazard of **1.05**, with a confidence interval spanning no effect. The apparent danger of retiring largely dissolves once prior health is accounted for — the older literature was mostly measuring sick people retiring. Worth noting that 23 of those 25 studies were rated high risk of bias.

The exception is specific. A quasi-experimental study exploiting an Austrian policy change, covering 17,590 men, found that one additional year of **early, involuntary, blue-collar** retirement raised the probability of death before 67 by 2.42 percentage points — a 13% relative increase. In 3,283 women the effect was zero. The excess deaths concentrated in heart disease, alcohol-related causes and vehicle injuries, which together accounted for about 78% of the effect. Lost income explained at most 10%.

HOW TO READ THE WORK FINDINGS

— It is not retirement that appears to harm men. It is losing work involuntarily, and it is worst in midlife.
— The damage runs through behavior, not biology: alcohol, inactivity, accidents and the loss of daily structure.
— That is the most actionable finding in this paper, because every one of those is a thing a man can build back deliberately.

10

CHAPTER 10

Losing a spouse, or a marriage

The sharpest short-term risk window in the male literature, and one where the female equivalent is statistically absent.

Bereavement

Pooled across 15 longitudinal studies and **2,263,888 people**, widowhood carried a mortality risk of **1.22 in men** and **1.03 in women** — and that female figure, with a confidence interval of 1.00 to 1.07, is not statistically distinguishable from no effect at all. The sex difference was significant at $p<0.0001$.

The time course is the actionable part. In the **first six months** the risk ran **1.41**; after six months, 1.14. Roughly 40 to 50% excess mortality in the first half-year, settling to about 15% long-term. Adjusting for additional covariates barely moved the estimate, which argues modestly against pure confounding.

Divorce and separation

Pooled across 104 studies in 24 countries and more than 600 million people, marital dissolution carried a mortality hazard of **1.37 in men** against 1.22 in women — and the gender gap **narrows as the sample ages**, meaning the male penalty is largest in midlife. A separate analysis of 7.9 million people found unmarried men at **1.46** for all-cause and **1.60** for cardiovascular mortality, against 1.22 and 1.19 in women, with a formal sex-interaction test confirming the difference.

Three explanations compete and the data cannot separate them: health selection, where men already in difficulty are likelier to divorce; the loss of what sociologists call marital resources, which in men typically includes a health-behavior monitor, a prompt to see a doctor, and the bulk of a social network; and acute behavioral shift in the aftermath. The honest position is that all three are probably operating.

ON THE EPIGENETIC EVIDENCE SPECIFICALLY

A 2025 analysis of 2,420 American adults over 50 did find higher biological-age acceleration in never-married men. But it is cross-sectional, only 22 never-married men were in the sample, no adjusted coefficients were published for divorced or separated men, and the paper contradicts itself between its results text and its own table on the direction of the effect in widowed women. The mortality meta-analyses are the finding here. The biology is plausible and unproven.

11

CHAPTER 11

What does not hold up

Four claims routinely used to explain accelerated aging in men, and what the evidence actually shows.

Caregiver stress ages you by a decade

The 2004 study behind this is one of the most cited in stress biology. It compared telomere length in **58 premenopausal mothers** — about 14 per group in the key comparison — using one-tailed tests with no correction for multiple comparisons, and derived the famous “9 to 17 years” figure by dividing a measured difference by literature-average loss rates the authors conceded were never measured in that age band. **Not one man was studied.**

The only prospective test followed 208 new caregivers against 205 matched controls, measured nine years apart, and found **nothing** — with every point estimate favouring the caregivers. Its authors called for a more balanced view. Caregiving is genuinely hard. The telomere number is not evidence.

Loneliness is as bad as fifteen cigarettes a day

This line does not appear in either of the papers it is attributed to. The 2010 meta-analysis of 148 studies reports an odds ratio of 1.50 for survival and compares the magnitude to *quitting smoking*. The 2015 analysis of 3.4 million people reports social isolation at 1.29, loneliness at 1.26 and living alone at 1.32, and compares them to **obesity** — never to cigarettes. For scale, smoking's own mortality hazard is 2.8, roughly six times the loneliness figure. Social connection matters and the effect is real. The cigarette number is a media artefact.

Shift work shortens your life

The largest meta-analysis, covering 34 datasets and 2,011,935 people, found all-cause mortality at **1.04 and not statistically significant**. The real findings are narrower and still worth knowing: myocardial infarction at 1.23, night-shift coronary events at 1.41, ischaemic stroke at 1.05 — that last from only two studies. No sex-stratified analysis was reported at all.

You can reverse your biological age

What a biological age test can and cannot tell you

Technical noise alone, between two runs of the same sample	up to 9 years
People flagged as extreme organ agers who were not flagged at their next visit	68%
Clocks, of 18 tested, achieving excellent biological reliability	none
Two years of caloric restriction, the best randomized trial: DunedinPACE	2 to 3% slower
The same trial's effect on PhenoAge, GrimAge and the Horvath clock	no change

The top three rows are why a single reading means little for one person: Higgins-Chen 2022 in Nature Aging found technical noise alone shifting replicates of one sample by up to nine years; the UK Biobank organ-clock cohort found 68% of extreme agers no longer extreme at their next visit; and a 2025 preprint testing 18 clocks across more than a thousand samples found none achieving excellent biological reliability, with technical precision failing to predict it. The bottom two rows are the CALERIE randomized trial: two years of caloric restriction slowed one clock, which measures a rate rather than a number of years, and left three others unmoved. Note these are different quantities and are deliberately not plotted on a shared scale.

The best randomized trial of a potent intervention — two years of caloric restriction in 220 people — moved a single epigenetic clock by about **2 to 3%** and left three others, including both clocks trained on mortality, completely unmoved. No clinical outcomes were measured. The achieved restriction averaged about 11.9% against a 25% target, and two of the authors hold inventor interest in the one clock that moved.

Against that, technical noise alone produces deviations of up to **nine years** between replicates of the same blood sample. A 2025 analysis of 18 clocks across more than a thousand samples found that while most achieve excellent *technical* precision, **none achieved excellent biological reliability** — and the two were essentially uncorrelated, meaning a laboratory's precision tells you nothing about whether your number is stable. In UK Biobank, **68% of people flagged as extreme organ agers were no longer extreme at their next visit**.

What the clocks are good for. Population research, where they work well: in 44,500 UK Biobank participants, a proteomic brain age gap predicted mortality with a hazard of 1.58 per standard deviation, and people with eight or more aged organs carried a hazard above 8. Those are real and important findings. What they are not is a personal dashboard. The inventor of the original clock has said plainly that the field lacks reference standards and that the clocks were designed for epidemiological studies rather than individuals.

PART FOUR

What Actually Slows It

Ranked by the strength of the evidence rather than by how much it costs or how often you have heard of it.

What actually slows male aging, ranked by the strength of the evidence

Getting blood pressure under 120 All-cause mortality cut 27% in a 9,361-person trial, 64% men	RANDOMIZED
Stopping smoking About 12 years of life lost; roughly 6 regained by quitting at 45-54	COHORT + CAUSAL
One ultrasound scan for aneurysm, at 65 Aneurysm deaths cut 40% in 127,891 men. No benefit in women	RANDOMIZED
Structured exercise, in men with heart disease Cardiovascular death down 42% beyond 3 years, 85 trials, 83% men	RANDOMIZED
Resistance training Large strength gains; about 1.1 kg of lean mass across 49 studies	RANDOMIZED
Strength work, around 40 minutes a week Any strength work: 15% lower mortality. Peak benefit 17% at 40 min	COHORT
Building cardiorespiratory fitness Least fit died at 5x the rate of the fittest, in 122,007 people	COHORT
Losing weight, if you carry excess Raises testosterone more reliably than any supplement sold for it	POOLED TRIALS
Protein at 1.6 g per kg, alongside training Adds about 0.30 kg of lean mass on top of the 1.1 kg from lifting	RANDOMIZED
Testosterone, for confirmed hypogonadism Noninferior for cardiac events; function and vitality both missed	MIXED
NAD+ precursors, NMN and NR They raise NAD+. Functional results inconsistent within single trials	THIN
Rapamycin The one healthspan trial missed its primary endpoint	THIN
Metformin as a longevity drug Its flagship trial never enrolled a patient. Blunts training gains	NONE
Resveratrol 104 trials; reviewers found no clinical evidence to recommend it	NONE

The right-hand column is the kind of evidence behind each row, not a single scale. Everything above the testosterone line is unglamorous, cheap and largely unsold. Everything below it is heavily marketed. That inversion is the most useful thing on this page. Sources for each row appear in the chapter it belongs to and in the reference list.

Look at the shape of that list. Everything with randomized evidence behind it is cheap, old and largely unmarketed. Everything heavily advertised to men over 40 sits at the bottom. That inversion is worth more than any single row.

12

CHAPTER 12

The five things that are actually proven

If a man over 40 did only what is in this chapter, he would be doing more than most.

1. **Get your blood pressure under 120 systolic.** In a randomized trial of 9,361 people, 64% of them men, intensive control cut all-cause mortality by 27% and cardiovascular death by 43%. This is the single

best-evidenced intervention in the entire paper. It also carried real harms — more acute kidney injury and more hypotension — which is exactly why it is a conversation with a physician and not a decision from a PDF.

2. **Stop smoking, at any age.** Across 88,496 men, current smoking carried a mortality hazard of 2.8. Smokers lose about twelve years of life; quitting between 45 and 54 gives back roughly six of them. Survival to 80 was 26% in smokers against 61% in those who never smoked.

3. **Have one ultrasound scan for abdominal aortic aneurysm at 65.** Across four randomized trials and 127,891 men, a single scan cut aneurysm mortality by 40%. There is no equivalent benefit in women. This is a genuinely male-specific intervention and most men have never heard of it.

4. **Train for strength.** Across 49 studies, resistance training produced large strength gains and about 1.1 kg of lean mass. In 263,058 people, any muscle-strengthening activity was associated with 15% lower mortality, and the dose-response peaked at about 17% at **40 minutes a week**. The curve is J-shaped; above 60 minutes a week the data cannot say whether the benefit continues, plateaus or reverses.

5. **Build cardiorespiratory fitness.** In 122,007 people, the least fit died at **five times** the rate of the fittest. That is the largest effect size anywhere in this review. It is observational and from a referred clinical population, so some of that gap is sick people performing badly on a treadmill — but the gradient is too steep to dismiss.

ONE HONEST LIMIT ON THE EXERCISE EVIDENCE

Structured exercise has randomized evidence for reducing cardiovascular death in men who already have coronary disease — 85 trials, about 83% male, a 42% reduction beyond three years. For all-cause mortality in that same analysis, the confidence intervals cross no effect. And as Chapter 4 noted, training appears to raise the whole fitness curve rather than flatten its slope. The case for exercise remains overwhelming. The specific claim that it slows the *rate* of aging is not one this paper can support.

13

CHAPTER 13

Nutrition, weight and the thing that moves testosterone

Less exciting than the supplement aisle, and considerably more effective.

Protein is worth getting right and is routinely oversold. Across 49 randomized trials and 1,863 people, protein supplementation added about **0.30 kg** of fat-free mass on top of resistance training, with a breakpoint around **1.6 g per kg of body weight per day** and no detectable benefit above it. The effect is *smaller* in older adults. For context, the training itself delivered about 1.1 kg. The supplement is worth roughly a quarter of the exercise.

Weight is the lever men skip past. Pooled across 24 studies, weight loss through diet raised total testosterone by about 2.87 units and bariatric surgery by 8.73, on a scale the accessible report did not state explicitly. No supplement marketed for testosterone comes close to that, and the obesity-related hormone changes appear to be reversible.

14

CHAPTER 14

Testosterone therapy, assessed honestly

Legitimate and effective where indicated. Not an anti-aging drug, and the trials are unusually clear about which is which.

This section exists because the topic is usually handled by advocates or by sceptics, and a man trying to make a decision deserves both halves.

WHAT TESTOSTERONE THERAPY DOES	WHAT THE TRIALS SHOW
Cardiovascular safety	Reassuring, with a caveat the trial names itself. In 5,246 men, TRT was noninferior to placebo for major cardiac events, hazard 0.96. But about 61% of both arms discontinued treatment, and the authors state this may bias the result toward noninferiority. The trial was funded by a consortium of testosterone manufacturers.
Sexual desire	The best-evidenced benefit. Clear, significant effect in the randomized T Trials.
Anemia	Improved in the same trial program.
Physical function	**Missed.** The T Trials' physical function endpoint was not significant, at p=0.20.
Vitality and energy	**Missed.** Not significant, at p=0.30. This is the single most common reason men ask for it.
Fractures	The only fracture-endpoint trial found *more* fractures on testosterone: 3.50% against 2.46%.
Mortality or lifespan	No trial has shown a benefit. None has been designed to.

The safety trial also found significantly more atrial fibrillation, more arrhythmias requiring intervention, and more acute kidney injury. Noninferior for cardiac events is not the same as harmless.

THE DEFENSIBLE POSITION

— For a man with confirmed, symptomatic hypogonadism, testosterone therapy is legitimate, cardiovascularly safe and effective for the things it has been shown to treat.

— It is not a longevity therapy, and the endpoints men most often want from it — energy and physical function — are the two the trials missed.

— The sequence matters more than the position. Weight, sleep, alcohol, training and medication review all move testosterone and all deserve investigation first — not because therapy is wrong, but because a man who corrects the cause may not need it, and a man who needs it will respond better once the cause is corrected.

— A quarter of new prescriptions in one large analysis began without a testosterone measurement in the prior year. Get the blood work, twice, in the morning.

15

CHAPTER 15

The supplement aisle

Six products marketed to men over 40 on the strength of evidence that mostly does not exist.

PRODUCT	WHAT THE HUMAN EVIDENCE ACTUALLY SHOWS
Metformin	Its flagship aging trial has never enrolled a patient. No randomized trial of metformin for aging or healthspan in non-diabetic humans exists. Worse for this audience: in a trial of 94 older adults, metformin **blunted** the muscle and lean-mass gains from resistance training.
Rapamycin	The one dedicated human healthspan trial, 114 people, **missed its primary endpoint** and found no change in epigenetic age. Its positive results were post-hoc, in a subgroup of roughly eight women.
NMN and NR	They raise NAD+. That is close to all they have been shown to do. In 42 older men, left-hand grip strength and gait speed improved while right-hand grip, muscle mass, insulin sensitivity and cognition did not.
Resveratrol	194 studies and 104 randomized trials, median group size 22, median duration 8 weeks. The systematic reviewers' own conclusion: no conclusive clinical evidence to advocate its recommendation in any healthcare setting. Not one trial used an aging endpoint.
Senolytics	Phase 1 and 2 only. No phase 3, and no aging or mortality endpoint has ever been pre-specified.
Taurine	The 2023 paper that launched it was directly challenged in 2025 by a larger analysis across humans, monkeys and mice finding limited connection between taurine levels and aging.

A note on GLP-1 medications, since they now sit adjacent to this conversation. In a body-composition substudy of 140 people, 76% of them women, fat mass fell 19.3% and lean mass 9.7% — so lean mass as a *proportion* of the body actually rose by 3 percentage points. The “muscle loss” framing needs that context. It also needs the obvious corollary: if you are losing weight rapidly by any means, resistance training and adequate protein are not optional.

Where a coach earns his place in this. Nothing in Part Four is secret and none of it is complicated. It is also, for most men, undone — because the proven items are dull, the marketed ones are exciting, and nobody is watching. What coaching supplies is sequence, accountability and the judgment to send you to a physician for the first three items rather than selling you the fourth. Request an evaluation

PART FIVE

The Transitions, and How to Cross Them

If the sharpest accelerations are events rather than birthdays, then the practical question is what to do when one arrives.

16

CHAPTER 16

A protocol for the windows that matter

Four transitions, with the specific things that collapse during each one.

The evidence in Part Three converges on a single mechanism. In every case where researchers could look, the damage ran through **behavior** — drinking, inactivity, sleep loss, accidents, the disappearance of structure — rather than through some unavoidable biological response to grief or stress. That is the good news buried in a grim set of hazard ratios. Behaviour is the one part of this a man can deliberately defend.

If you lose work between 40 and 55

1. **Protect the schedule before you protect the income.** The mortality signal ran through behavior change, not poverty — lost income explained at most 10% of the effect in the best-identified study. Fix a wake time and keep training days where they were.
2. **Watch alcohol specifically.** Alcohol-related causes accounted for a substantial share of the excess deaths in the early-retirement data. This is the single most predictable failure point.
3. **Keep the medical appointments you would otherwise postpone.** Blood pressure is the top-ranked intervention in this entire paper and is usually the first thing dropped when a man loses employer cover.
4. **Rebuild social contact deliberately.** For most men the workplace is the social network, and it leaves with the job.

If a marriage ends

1. **Assume the health-monitoring function has gone, and replace it.** In most married men, someone else was tracking the appointments, noticing the mole, and prompting the check-up.
2. **Expect the cardiovascular signal, and act on it.** The male excess for unmarried men is largest for cardiovascular death, at 1.60. Blood pressure, lipids, weight, training.
3. **Do not let training be the first casualty.** It is usually the first thing to go and the hardest to restart.

In the first six months after losing a spouse

1. **Recognise this as a defined high-risk window, not a vague bad time.** The pooled risk runs about 41% above baseline in the first six months and settles to about 15% after. It is time-limited and it is known.

2. **Have someone else hold the medical thread for a while.** Medication adherence, appointments, follow-up. This is a reasonable thing to ask of an adult child or a friend.
3. **Eat and sleep on a schedule even when neither is wanted.**
4. **Get help early rather than at the point of crisis.** Grief is not a medical condition, but the six months after bereavement is the best-documented period of elevated risk in a man's life.

Through the forties and fifties, with no event at all

1. **Train for strength and power, not for mass.** Power is the steepest curve and it starts bending at 40.
2. **Get your blood pressure measured properly and treated aggressively.**
3. **Build cardiorespiratory fitness while the returns are cheapest.** Training lifts the whole curve, so the altitude you reach in your forties is what you spend the rest of your life descending from.
4. **Deal with weight, which moves testosterone more than anything in a bottle.**
5. **Book the aneurysm scan at 65.** One scan, male-specific, 40% fewer deaths from aneurysm, and almost nobody knows about it. It did not move all-cause mortality, because aneurysm is a rare cause of death, but it is a cheap one-off win available only to men.

THE WHOLE PAPER IN THREE LINES

— You cannot change your birthdays and, on the current evidence, you cannot change your rate of aging by much either.
— You can change your starting altitude, which determines where the same slope lands you.
— And you can defend the three or four transitions where the evidence says men come apart. That is where the available leverage actually sits.

PART SIX

Questions Men Actually Ask

Direct answers, with the uncertainty left in where it belongs.

I turned 44 this year. Should I be worried?

No more than you were at 43. The 44 figure comes from a figure legend in a 108-person study whose own text says 45 and whose protein data say 40, and whose method is now challenged by its own authors. Nothing in the measured, longitudinal literature bends at 44.

Is there an age when it becomes too late to start?

No age in this literature supports that. Quitting smoking at 45 to 54 recovers about six of the twelve years lost. Resistance training produces large strength gains in men in their seventies and eighties. Blood pressure control works at every age studied. The returns diminish; they do not disappear.

Why do men die younger than women?

Partly behavior, partly biology, and the pattern is strange: men report better health and less disability than women at the same age, yet die earlier. Coronary disease arrives about a decade earlier in men. In the US the

gap widened from 4.8 years in 2010 to 5.8 in 2021, driven by COVID, overdose, accidents and suicide, and has since narrowed to 5.3 years in 2023.

Should I get a biological age test?

Not for decision-making. Technical noise alone can shift a reading by up to nine years, no clock has demonstrated excellent biological reliability within a person, and 68% of people flagged as extreme organ agers in UK Biobank were not flagged at their next visit. If you want a number that predicts your future, a blood pressure cuff and a treadmill test are better and cheaper.

My grip strength is down. Does that matter?

It is one of the better single markers, and in men specifically there is evidence of an acceleration between 60 and 70 that was not seen in women. But that finding is a preprint and the inflection age was read off a curve rather than formally estimated, so treat the direction as solid and the age as approximate.

Is it true that men lose deep sleep and testosterone together?

Both decline, and one controlled study found a week of five-hour nights lowering daytime testosterone 10 to 15% in healthy young men. But that study had ten participants and a separate controlled study of fourteen men found no significant change, so the effect is not robust across small trials. The testosterone decline with age is in any case linear and modest, and much of what is attributed to age tracks body fat instead. Fix the sleep and the weight before concluding anything about your hormones.

Does stress literally age you?

The honest answer is that we do not know at the individual level. The famous caregiver-telomere finding came from about 14 women per group, studied no men, and failed to replicate in the only prospective test. What is well established is that stressful life events are associated with higher mortality in men, and that the association runs through behavior.

Is TRT going to make me feel like I did at 30?

The trials say probably not. Testosterone therapy has clear evidence for sexual desire and anemia, and it is cardiovascularly safe in the indicated population. But the randomized trials *missed* their physical function and vitality endpoints — the two things men most often want from it.

What about NAD+, rapamycin, all of that?

Interesting science, thin human evidence. Rapamycin's one healthspan trial missed its primary endpoint. NAD+ precursors reliably raise NAD+ and have not reliably done anything else. None belongs above blood pressure, training or weight on your list.

My face has changed more than my body. Is that normal?

Yes, and the structure underneath is part of it. Facial bone changes by decade: the maxilla in the forties, the palate in the fifties, the orbit and nasal aperture in the sixties. Skin thickness holds roughly steady until 60 and then falls. The 1%-a-year collagen figure you have read is not sourceable for men.

Can I slow the rate, or only change where I start?

On the current evidence, mostly the latter, and this paper will not pretend otherwise. Aerobic decline rates were similar across activity levels while fitness itself differed enormously at every age. That is an intercept effect. It is also still the best argument for training hard in your forties, because the whole curve moves up with you.

What is the single highest-yield thing I can do this month?

Get your blood pressure measured properly — not once in a pharmacy, but repeatedly — and if it is above 130, start the conversation about getting it under 120. It is the best-evidenced intervention in this paper, it is cheap, and most men over 40 who have it do not know.

PART SEVEN

How Dr. John Spencer Ellis Helps

What a coach adds to a body of evidence that is, for the most part, already public.

17

CHAPTER 17

Knowing is not the problem

Almost nothing in this paper is secret. Almost all of it is undone.



Dr. John Spencer Ellis

John is 57. He is inside the age range this paper describes, not observing it from outside, and he has spent three decades watching men arrive at the same four or five moments and handle them badly — not from ignorance, but because the fix is behavioral, the timing is terrible, and there is nobody watching.

What a coaching relationship supplies is the part no document can. Sequencing, so you are not attempting six changes at once. Accountability through the specific windows Part Three identifies, which is exactly when self-directed effort collapses. Integration, so that training, nutrition, sleep and medical follow-up are one plan rather than four competing ones. And the judgment to stop and send you to a physician, which for the top three items in Part Four is the correct answer.

What the longevity program covers

— **Establishing where you actually are.** Which curves have bent, which have not, and which of the measurable markers in Part Two are worth tracking for you specifically rather than in general.

— **Strength and power first.** Programming built around the finding in Chapter 3 — that strength falls three to four times faster than mass and that maintaining mass does not protect it.

— **Cardiorespiratory capacity.** Raising the whole curve while the returns are still cheap, which on the Fleg data is the main thing training is demonstrably good for.

— **Weight and nutrition.** Including the testosterone route that works, which is body composition rather than a bottle.

— **Transition planning.** The explicit point of difference. If a job ends, a marriage ends, or a spouse dies, the plan for those weeks is built before they arrive rather than improvised during them.

— **Appearance and aesthetics, treated as legitimate.** Most men over 40 care how they look and are embarrassed to say so. It is a reasonable goal and it responds to the same inputs as everything else in this paper.

WHAT JOHN DOES NOT DO

He does not diagnose, prescribe, manage medication, or treat disease. He does not sell supplements, and this paper should make clear why. Blood pressure control, aneurysm screening and any hormone decision belong with a physician, and the referral is made early rather than late.

Reading from Dr. Ellis on related topics

— USA Today: men's health and longevity coaching program launch — the program this paper's coaching sections refer to.

— USA Today: Blue Zones evidence review — the same approach to grading evidence that this paper uses, applied to longevity claims.

— Yahoo Finance: program launch coverage.

— AP News: press announcement.

— Background, credentials and career history.

— One-to-one strategy sessions — for men who want a single focused conversation rather than an ongoing program.

Start with an evaluation. Before any program begins, John reviews where you actually are — what has changed, what has not, what you have tried, and which of the items in Part Four needs a physician rather than a coach. Request an evaluation · Contact · johnspencerellis.com

REFERENCES

Primary sources

These are the principal sources behind the figures in this paper. Where a claim in the text is marked as unsupported, unsourceable or overstated, the source listed is the one that fails to support it.

Shen X, Wang C, Zhou X, et al. Nonlinear dynamics of multi-omics profiles during human aging. *Nat Aging*. 2024. doi:10.1038/s43587-024-00692-2.

Carbonneau M, Shutta KH, Miller JW, Snyder MP, Shen X, Quackenbush J. LOESS and DE-SWAN can induce artifactual 'waves' of molecular aging. *bioRxiv*. 2026. doi:10.64898/2026.06.24.734079. *(Preprint; not peer reviewed.)*

Lehallier B, Gate D, Schaum N, et al. Undulating changes in human plasma proteome profiles across the lifespan. *Nat Med*. 2019;25(12):1843–1850.

Ma LZ, Liu WS, He Y, et al. Plasma proteomics identify novel biomarkers and dynamic patterns of biological aging. *J Adv Res*. 2025.

Ding et al. Comprehensive human proteome profiles across a 50-year lifespan reveal aging trajectories and signatures. *Cell*. 2025. doi:10.1016/j.cell.2025.06.047.

Li H, Tang F, Xue H, et al. Phenome-wide multi-omics integration uncovers distinct archetypes of human aging. *arXiv* preprint 2510.12384. *(Not peer reviewed.)*

Oh HS, Rutledge J, Nachun D, et al. Organ aging signatures in the plasma proteome track health and disease. *Nature*. 2023;624(7990):164–172.

Oh HS, Le Guen Y, Rappoport N, et al. Plasma proteomics links brain and immune system aging with healthspan and longevity. *Nat Med*. 2025.

Higgins-Chen AT, Thrush KL, Wang Y, et al. A computational solution for bolstering reliability of epigenetic clocks. *Nat Aging*. 2022;2(7):644–661.

Sehgal R, Higgins-Chen AT, et al. Biological versus technical reliability of epigenetic clocks. *bioRxiv*. 2025. doi:10.1101/2025.10.13.682176. *(Preprint.)*

Bell CG, Lowe R, Adams PD, et al. DNA methylation aging clocks: challenges and recommendations. *Genome Biol*. 2019;20:249.

Waziry R, Ryan CP, Corcoran DL, et al. Effect of long-term caloric restriction on DNA methylation measures of biological aging in healthy adults from the CALERIE trial. *Nat Aging*. 2023;3:248–257.

Hedman AM, van Haren NEM, Schnack HG, Kahn RS, Hulshoff Pol HE. Human brain changes across the life span: a review of 56 longitudinal magnetic resonance imaging studies. *Hum Brain Mapp*. 2012. doi:10.1002/hbm.21334.

Singh-Manoux A, Kivimäki M, Glymour MM, et al. Timing of onset of cognitive decline: results from Whitehall II prospective cohort study. *BMJ*. 2012;344:d7622.

Pontzer H, Yamada Y, Sagayama H, et al. Daily energy expenditure through the human life course. *Science*. 2021;373(6556). doi:10.1126/science.abe5017.

Janssen I, Heymsfield SB, Wang Z, Ross R. Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. *J Appl Physiol*. 2000;89:81–88.

Metter EJ, Conwit R, Tobin J, Fozard JL. Age-associated loss of power and strength in the upper extremities in women and men. *J Gerontol A Biol Sci Med Sci*. 1997;52(5):B267.

Goodpaster BH, Park SW, Harris TB, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the Health ABC study. *J Gerontol A Biol Sci Med Sci*. 2006;61(10):1059.

Mitchell WK, Williams J, Atherton P, et al. Sarcopenia, dynapenia, and the impact of advancing age on human skeletal muscle size and strength. *Front Physiol*. 2012;3:260.

Fleg JL, Morrell CH, Bos AG, et al. Accelerated longitudinal decline of aerobic capacity in healthy older adults. *Circulation*. 2005;112:674–682.

Berger C, Langsetmo L, Joseph L, et al. Change in bone mineral density as a function of age in women and men and association with the use of antiresorptive agents. *CMAJ*. 2008;178(13):1660.

Haentjens P, Magaziner J, Colón-Emeric CS, et al. Meta-analysis: excess mortality after hip fracture among older women and men. *Ann Intern Med*. 2010;152(6):380–390.

Gennari L, Bilezikian JP. Idiopathic osteoporosis in men. *Curr Osteoporos Rep*. 2013. doi:10.1007/s11914-013-0164-1.

Cotofana S, Gotkin RH, Frank K, et al. The relationship between bone remodeling and the clockwise rotation of the facial skeleton: a computed tomographic imaging-based evaluation. *Plast Reconstr Surg*. 2018. doi:10.1097/PRS.0000000000004976.

Hall DA, et al. Changes in skinfold thickness with increasing age. *Age Ageing*. 1981;10(1):19.

Rahrovan S, Fanian F, Mehryan P, Humbert P, Firooz A. Male versus female skin: what dermatologists and cosmeticians should know. *Int J Womens Dermatol*. 2018;4(3):122–130.

Panhard S, Lozano I, Loussouarn G. Greying of the human hair: a worldwide survey. *Br J Dermatol*. 2012;167(4):865–873.

Feldman HA, Longcope C, Derby CA, et al. Age trends in the level of serum testosterone and other hormones in middle-aged men: longitudinal results from the Massachusetts Male Aging Study. *J Clin Endocrinol Metab*. 2002;87(2):589–598.

Harman SM, Metter EJ, Tobin JD, Pearson J, Blackman MR. Longitudinal effects of aging on serum total and free testosterone levels in healthy men. *J Clin Endocrinol Metab*. 2001;86(2):724–731.

Tajar A, Huhtaniemi IT, O'Neill TW, et al. Characteristics of androgen deficiency in late-onset hypogonadism: results from the European Male Aging Study. *J Clin Endocrinol Metab*. 2012. doi:10.1210/jc.2011-2513.

Baillargeon J, Urban RJ, Ottenbacher KJ, Pierson KS, Goodwin JS. Trends in androgen prescribing in the United States, 2001 to 2011. *JAMA Intern Med*. 2013. doi:10.1001/jamainternmed.2013.6895.

Corona G, Rastrelli G, Monami M, et al. Body weight loss reverts obesity-associated hypogonadotropic hypogonadism: a systematic review and meta-analysis. *Eur J Endocrinol*. 2013.

Lincoff AM, Bhasin S, Flevaris P, et al. Cardiovascular safety of testosterone-replacement therapy (TRAVERSE). *N Engl J Med*. 2023;389(2):107–117.

Snyder PJ, Bhasin S, Cunningham GR, et al. Effects of testosterone treatment in older men (The Testosterone Trials). *N Engl J Med*. 2016;374(7):611–624.

Shor E, Roelfs DJ, Bugyi P, Schwartz JE. Meta-analysis of marital dissolution and mortality. *Soc Sci Med*. 2012;75(1):46–59.

Wang Y, Jiao Y, Nie J, et al. Sex differences in the association between marital status and the risk of cardiovascular, cancer, and all-cause mortality. *Glob Health Res Policy*. 2020;5(1):1–16.

Moon JR, Kondo N, Glymour MM, Subramanian SV. Widowhood and mortality: a meta-analysis. *PLoS ONE*. 2011;6(8):e23465.

Roelfs DJ, Shor E, Davidson KW, Schwartz JE. Losing life and livelihood: a systematic review and meta-analysis of unemployment and all-cause mortality. *Soc Sci Med*. 2011;72(6):840–854.

Sewdas R, de Wind A, Stenholm S, et al. Association between retirement and mortality: working longer, living longer? *J Epidemiol Community Health*. 2020;74(5):473–480.

Kuhn A, Wuellrich JP, Zweimüller J. Fatal attraction? Access to early retirement and mortality. IZA working paper, 2014. *(Working paper.)*

Cui X, Huo J, Xu Y, et al. Association of marital status with DNA methylation-based biological age among US older adults. *Front Public Health*. 2025;13:1535524.

Epel ES, Blackburn EH, Lin J, et al. Accelerated telomere shortening in response to life stress. *Proc Natl Acad Sci USA*. 2004;101(49):17312–17315.

Armstrong ND, et al. Telomere shortening and the transition to family caregiving in the REGARDS study. *PLoS ONE*. 2022;17:e0268689.

Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. *PLoS Med*. 2010.

Holt-Lunstad J, Smith TB, Baker M, Harris T, Stephenson D. Loneliness and social isolation as risk factors for mortality: a meta-analytic review. *Perspect Psychol Sci*. 2015.

Vyas MV, Garg AX, Iansavichus AV, et al. Shift work and vascular events: systematic review and meta-analysis. *BMJ*. 2012;345:e4800.

SPRINT Research Group. A randomized trial of intensive versus standard blood-pressure control. *N Engl J Med*. 2015;373(22):2103–2116.

Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med*. 2013.

Cosford PA, Leng GC, Thomas J. Screening for abdominal aortic aneurysm. *Cochrane Database Syst Rev*. 2007;(2):CD002945.

Dibben G, Faulkner J, Oldridge N, et al. Exercise-based cardiac rehabilitation for coronary heart disease. *Cochrane Database Syst Rev*. CD001800.pub5.

Borde R, Hortobágyi T, Granacher U. Dose-response relationships of resistance training in healthy old adults. *Sports Med*. 2015.

Peterson MD, Sen A, Gordon PM. Influence of resistance exercise on lean body mass in aging adults. *Med Sci Sports Exerc*. 2011.

Momma H, Kawakami R, Honda T, Sawada SS. Muscle-strengthening activities are associated with lower risk and mortality in major non-communicable diseases. *Br J Sports Med*. 2022.

Mandsager K, Harb S, Cremer P, et al. Association of cardiorespiratory fitness with long-term mortality among adults undergoing exercise treadmill testing. *JAMA Netw Open*. 2018;1(6):e183605.

Morton RW, Murphy KT, McKellar SR, et al. A systematic review, meta-analysis and meta-regression of the effect of protein supplementation on resistance training-induced gains. *Br J Sports Med*. 2018.

Oksuzyan A, Juel K, Vaupel JW, Christensen K. Men: good health and high mortality. Sex differences in health and aging. *Aging Clin Exp Res*. 2008;20(2):91–102.

Yan BW, Erias E, Geller AC, et al. Widening gender gap in life expectancy in the US, 2010–2021. *JAMA Intern Med*. 2023. doi:10.1001/jamainternmed.2023.6041.

Murphy SL, Kochanek KD, Xu JQ, Arias E. Mortality in the United States, 2023. *NCHS Data Brief No. 521*. National Center for Health Statistics; 2024.

Fieldsend TW, O'Neill CR, Shrivastava A, et al. Sexual dimorphism in human skeletal muscle ageing. *medRxiv*. 2025. doi:10.1101/2025.01.06.25319958. *(Preprint.)*

Leproult R, Van Cauter E. Effect of 1 week of sleep restriction on testosterone levels in young healthy men. *JAMA*. 2011;305(21):2173–2174.

Walton RG, Dungan CM, Long DE, et al. Metformin blunts muscle hypertrophy in response to progressive resistance exercise training in older adults (MASTERS). *Aging Cell*. 2019.

Mannick JB, et al. Rapamycin and healthspan in normative aging adults (PEARL trial, NCT04488601). *medRxiv*. 2024. doi:10.1101/2024.08.21.24312372. *(Preprint.)*

Brown K, Theofanous D, Britton RG, et al. Resveratrol for the management of human health: how far have we come? A systematic review of resveratrol clinical trials. *Int J Mol Sci*. 2024;25(2):747. doi:10.3390/ijms25020747.

Ensrud KE, Taylor BC, Paudel ML, et al. Serum 25-hydroxyvitamin D levels and rate of hip bone loss in older men. *J Clin Endocrinol Metab*. 2009. doi:10.1210/jc.2008-2786.

Reilly DM, Lozano J. Skin collagen through the lifestages: importance for skin health and beauty. *Plast Aesthet Res*. 2021. doi:10.20517/2347-9264.2020.153. *(The review from which the 1%-per-year collagen figure is usually taken.)*

Norwood OT. Male pattern baldness: classification and incidence. *South Med J*. 1975;68(11):1359–1365. *(Primary incidence table not retrievable.)*

Salthouse TA. When does age-related cognitive decline begin? *Neurobiol Aging*. 2009. doi:10.1016/j.neurobiolaging.2008.09.023.

Reynolds AC, Dorrian J, Liu PY, et al. Impact of five nights of sleep restriction on glucose metabolism, leptin and testosterone in young adult men. *PLoS ONE*. 2012.

Horvath S, Gurven M, Levine ME, et al. An epigenetic clock analysis of race/ethnicity, sex, and coronary heart disease. *Genome Biol*. 2016;17:171.

Kankaanpää A, Tolvanen A, Saikkonen P, et al. Do epigenetic clocks provide explanations for sex differences in life span? *J Gerontol A Biol Sci Med Sci*. 2022;77(9):1898–1906.



Medical disclaimer. This document is educational and does not constitute medical advice, diagnosis or treatment. Dr. John Spencer Ellis is not a physician. Decisions about blood pressure medication, hormone therapy, screening and any other medical intervention discussed here belong with a qualified clinician who knows your history. Do not start, stop or change any medication on the basis of this document.

© 2026 Dr. John Spencer Ellis. Published 2026. johnspencerellis.com