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REVEW ARTICLE

Rethinking Anti-Aging Treatment Planning Through Biological Age Assessment: A Review

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ABSTRACT

Background: Anti-aging clinicians typically stratify patients by chronological age (CA), yet CA captures almost nothing about a patient's actual physiological condition. Two individuals born the same year can carry very different cardiovascular, metabolic, and functional profiles a gap that biological age (BA) assessment is designed to close by comparing a patient's measured physiology against expected norms for their age group.

Purpose: This review considers how a biomarker-driven BA model might reshape anti-aging treatment planning, using a validated statistical approach principal component analysis (PCA) applied to physiological data as a case study for how such assessment could work in practice.

Approach: Drawing on data from 100 healthy adults (51 women, 49 men), an initial pool of 32 physiological measurements covering body composition, metabolic markers, immune activity, lung and heart function, and physical performance was pared down through statistical screening to nine representative biomarkers, which were then mathematically combined into separate BA formulas for men and women.

Key Findings: The resulting equations tracked participants' actual ages closely (correlation coefficients of 0.86 in women and 0.81 in men) without systematic over- or under-prediction, and exposed clear sex-based differences in which biological systems most strongly influence aging insight with real bearing on how anti-aging care might be tailored to the individual.

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Takeaway: Folding BA measurement into anti-aging practice could let clinicians move past CA-based guesswork, pinpoint the physiological systems responsible for a given patient's accelerated aging, direct pre-treatment optimization accordingly, and later verify whether treatment is genuinely working though broader testing is still needed before such tools enter routine practice.

KEYWORDS

• Biological Age • Anti-Aging • Treatment Planning • Epigenetic Clock • Aging Biomarkers • Aesthetic Medicine

INTRODUCTION

For most of its history, anti-aging medicine whether surgical rejuvenation, minimally invasive aesthetic work, or lifestyle-based rejuvenation programs—has organized itself around a single number: the patient's birth year. But CA is a blunt instrument. It says nothing about how well a given 50-year-old's arteries, lungs, or metabolism are actually functioning, and treating patients as interchangeable simply because they share a CA ignores enormous individual variation in physiological reserve and healing capacity.

Biological age was conceived to fill exactly this gap. Rather than counting years lived, it estimates where a person's physiology actually sits relative to same-aged peers, built from measurable biological data rather than a birth certificate. This distinction matters practically, not just academically some BA models have proven better predictors of mortality and disease onset than CA itself, which makes BA a natural candidate for screening tools in anti-aging medicine: flagging patients aging faster than expected, revealing which organ systems are responsible, and giving clinicians a numerical baseline they can revisit after treatment to see whether anything actually changed.

No consensus method for calculating BA currently exists, and multiple competing approaches have been proposed. This review works through one such model in detail—how it was built, what it found, and what a practicing anti-aging clinician might reasonably take from it.

HOW THE MODEL WAS BUILT

Study Population

Researchers recruited 100 healthy Danish adults, split evenly between 51 women and 49 men, spanning ages 18 to 65 and distributed evenly across 5-year age bands. Anyone with diabetes, cardiovascular disease, cancer, thyroid disorders, or relevant medication use was excluded, so that the resulting model would represent a genuinely healthy aging pattern the appropriate comparison point when evaluating whether an individual anti-aging patient's own biology has drifted from that trajectory.

Narrowing 32 Variables to 9

Testing began with 32 physiological measurements spanning body composition, metabolic indicators, immune markers, cardiorespiratory measures, and physical performance tests, all collected under controlled conditions (fasting, no recent exercise or alcohol, no recent smoking).

Two filtering steps reduced this list substantially:

- First, each variable was checked for a statistically meaningful relationship with age; variables with weak or no such relationship were dropped, cutting the list to 15.
- Second, where several remaining variables essentially measured the same underlying trait, only the strongest or most clinically useful representative was kept. Among three lung-function measures, only one (FEV_1) survived; between two cholesterol markers, total cholesterol was chosen over LDL; and rather than keeping systolic and diastolic blood pressure as separate figures, they were merged into a single mean arterial pressure value.

What remained was a **nine-variable panel**: waist circumference, FEV_1, HbA1c, adiponectin, HDL, total cholesterol, suPAR, mean arterial pressure, and VO_2max a set built

almost entirely from tests already common in everyday clinical practice, which matters if a tool like this is ever going to be usable outside a research lab.

Building the Equation

The nine variables were fed into a statistical technique called principal component analysis, run separately for men and women. The resulting "first principal component" essentially a single composite score capturing the shared aging signal across all nine variables was used as the raw material for the age estimate, since it aligned closely with actual age and explained more of the overall pattern in the data than any other component (roughly 31% of total variation in women, 25% in men).

From there, the score was converted into an age figure (in years), then adjusted using a mathematical correction to fix a known distortion: without correction, such models tend to make younger people look artificially older and older people look artificially younger. The corrected figure is what should ultimately inform patient discussions and treatment decisions.

WHAT THE MODEL SHOWED

Accuracy

The corrected biological age tracked real age well in both sexes correlations of 0.86 in women and 0.81 in men and did so evenly across the age range, without the model systematically flattering or penalizing any particular age group.

Different Drivers by Sex a Point Worth Building Treatment Around

Which of the nine variables mattered most differed sharply between men and women, a detail with obvious implications for how pre-treatment workups might be tailored:

- **Women's** biological age was shaped mainly by cholesterol levels, blood pressure, and blood sugar control together accounting for well over half the model's weighting pointing toward cardiometabolic management as the priority when a female patient's biological age runs ahead of her actual age.
- **Men's** biological age, by contrast, was driven chiefly by waist size and aerobic fitness, with cholesterol a distant third suggesting that central weight gain and cardiorespiratory conditioning are the more useful levers for male patients.

This divergence likely reflects known biological differences men tend to accumulate fat centrally as they age even without overall weight gain, while women experience shifts in cholesterol handling around menopause and argues against applying identical pre-treatment protocols regardless of sex.

The "Accelerated Agers"

A meaningful minority of the sample roughly 1 in 12 women and 1 in 6 men had a biological age more than a decade above their actual age, and this group skewed toward higher body weight. Interestingly, good cardiorespiratory fitness appeared to soften this gap, echoing other research showing that fitness can blunt many of the health risks otherwise tied to excess weight a nuance worth weighing when deciding whether a patient needs weight loss, fitness training, or both before treatment.

WHAT THIS MEANS FOR ANTI-AGING PRACTICE

A Screening Tool, Not a Gatekeeper

Finding that a patient's biological age significantly exceeds their actual age shouldn't necessarily rule out anti-aging treatment it should prompt a conversation about what's driving the gap and whether addressing it first (blood pressure control, glycemic management, weight and fitness work) would lower procedural risk and improve how well results hold up over time.

Tailoring the Workup

Because the model breaks down exactly which variables are pushing a patient's biological age upward, it opens the door to genuinely individualized pre-treatment planning cholesterol and blood pressure management for a woman whose elevated score traces back to those factors, versus a weight- and fitness-focused program for a man whose score is being driven by waist circumference and low VO_2max.

A Number to Revisit

Because biological age collapses a complex biomarker panel into one intuitive figure,

years, it lends itself naturally to before-and-after comparison. Reassessing it after a prehabilitation program, surgical intervention, or combined regimen gives clinicians (and patients) something more concrete than subjective impressions of "looking younger."

Where this Model Fits Among Alternatives

This isn't the only way to calculate biological age, and it's worth knowing the alternatives:

- **Older regression-based models** were the first generation of this idea but suffer more severely from the over/underestimation problem described above.
- **The Klemera-Doubal model**, a newer statistical method, has outperformed PCA-based approaches like this one in some studies specifically looking at mortality prediction, and may be worth considering alongside or instead of PCA.
- **Narrower, single-domain models**—built around just one marker cluster, like metabolic syndrome severity or waist size alone—trade comprehensiveness for speed and simplicity.
- **Epigenetic clocks**, based on DNA methylation patterns rather than physiological measurements, represent an entirely different, molecular route to the same destination, and are increasingly discussed alongside physiological models in anti-aging circles.

Caveats Before Clinical Adoption

A few limitations deserve emphasis before this kind of tool gets built into routine anti-aging workflows:

- It assumes biomarkers decline in a straight line with age, which likely isn't true across the entire lifespan, especially at older extremes.
- It was built from a single, relatively small (n=100), healthy, ethnically homogeneous population studied at one point in time leaving open how well it would perform in patients with existing health conditions, from different backgrounds, or followed over years rather than assessed once.
- Two of the nine variables suPAR and adiponectin aren't part of standard clinical testing, which could complicate real-world rollout unless the panel is simplified.
- Nobody yet knows whether lowering someone's calculated biological age through intervention actually reduces their real-world disease risk—that link remains to be tested directly.

Where this Research Needs to go Next

Several gaps stand between this model and routine clinical use: testing it in larger and more varied populations, including patients who already have the kinds of health conditions common among anti-aging patients; following people over time rather than just at a single visit, to confirm that a falling biological age score actually corresponds to improving health; formally trialing the model within structured anti-aging programs to see if it responds meaningfully to intervention; and checking whether a trimmed-down version dropping the two less-accessible lab tests still performs well enough to be useful. Longer term, building separate versions for specific populations (pre-versus post-menopausal women, for instance) and eventually pairing physiological scores like this with molecular measures such as epigenetic clocks could sharpen the tool's precision considerably.

CONCLUSION

A biological age figure built from a modest, largely standard panel of physiological tests gives anti-aging clinicians something chronological age never could: a sense of which specific body systems are aging faster than they should in a given patient, and a concrete target for pre-treatment intervention. The model reviewed here performed well against its own reference population and revealed genuinely useful sex-specific patterns, but it remains a first-generation tool built on a small, single-population sample and never yet tested for whether improving the score actually improves outcomes. Bringing this kind of assessment into everyday anti-aging practice will depend on larger studies, longer follow-up, and comparison against competing models like the Klemera-Doubal approach before it can be trusted as more than a promising early step.

Conflict of Interest Disclaimer: The authors declare no conflicts of interest relevant to this review.

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