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Longevity and Biological Ageing: Enhancing Health into Advanced Age



SESSION 10

Dr. Denise Furness

Epigenetic Age: From Measurement to Intervention

12:00-12:45pm

An event by:  Nutritional Medicine Institute

Platinum sponsors:



regenerus labs



EPIGENETIC AGE: *From Measurement to Intervention*

DR. DENISE FURNESS (PhD) –
Functional Geneticist & Nutritionist

CSO, The Academy for Precision Health
Founder, Your Genes & Nutrition



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AFFILIATIONS & DECLARATION OF INTEREST

Affiliations

- Founder and clinical practitioner – Your Genes & Nutrition
- Founder and Chief Scientific Officer – Academy for Precision Health
- Board member – Endeavour College of Natural Health
- Faculty member – Australasian College of Nutritional and Environmental Medicine (ACNEM)

Declaration of interest

- Paid advisor/KOL to Pure Encapsulations.
- Presentation content developed independently; Pure Encapsulations has had no involvement in its development.
- No other relevant conflicts of interest to declare.

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LEARNING OBJECTIVES



- Understand the difference between chronological age and biological age.
 - Recognise key DNA methylation based biological age clocks and their relevance in clinical and nutritional settings.
 - Identify practical diet, lifestyle and supplement strategies that may influence biological ageing, and consider their application in longevity-focused practice.
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RETHINKING AGEING IN CLINICAL PRACTICE



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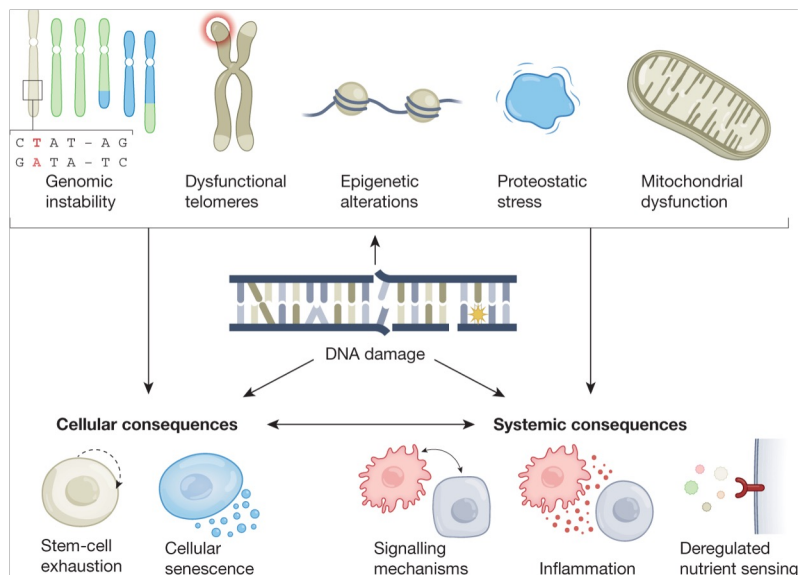
THE REALITY OF AGEING

- Chronological age $\neq$ same biological age.
 - We all age differently
 - We age at different rates across our lifespan
- Ageing is characterised by a gradual and progressive decline in biological systems.
- Visible signs (skin, hair, loss of function) are *later-stage markers*.
- Begins at the **molecular & cellular level**.



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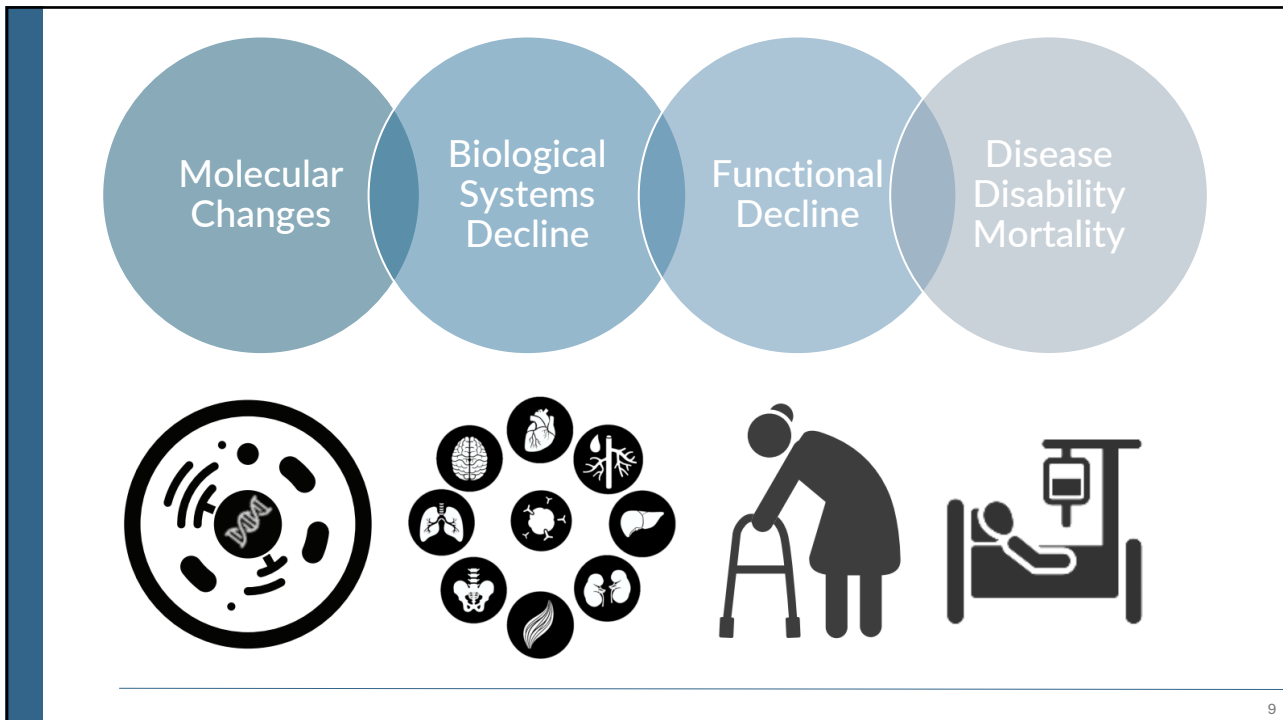
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Schumacher, B. et al. The central role of DNA damage in the ageing process. Nature. 2021

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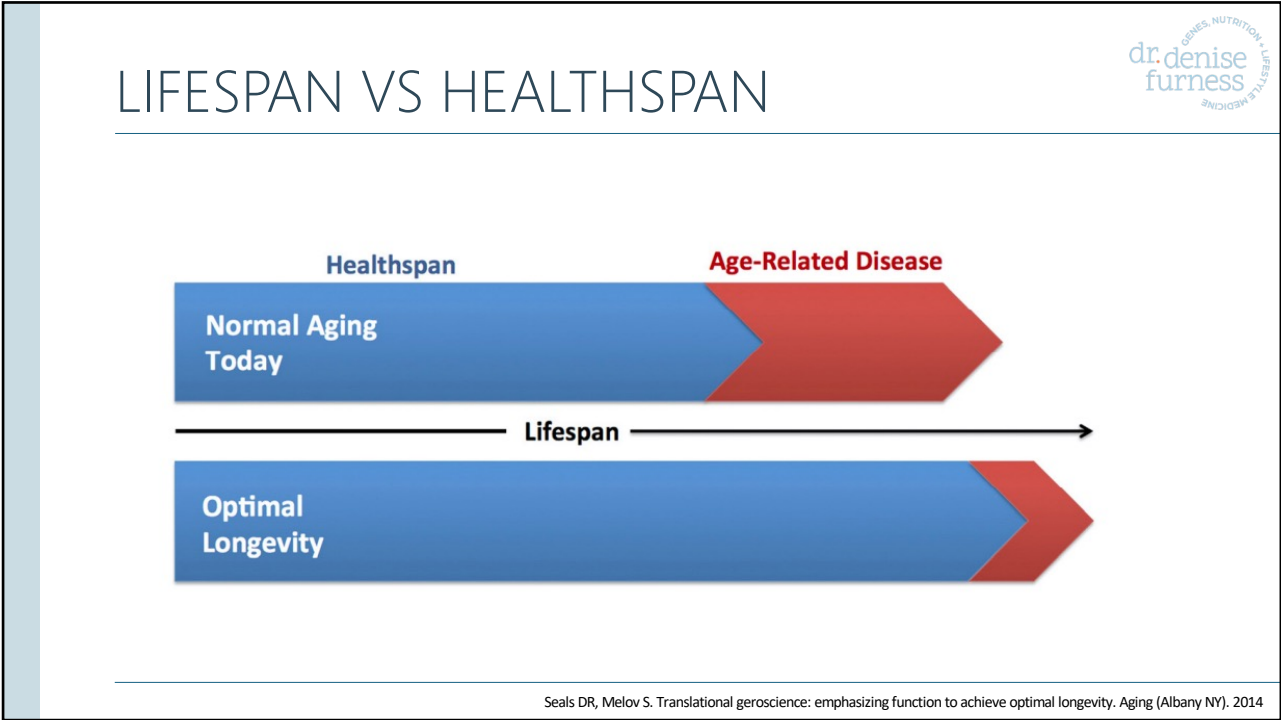
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THE GEROSCIENCE HYPOTHESIS

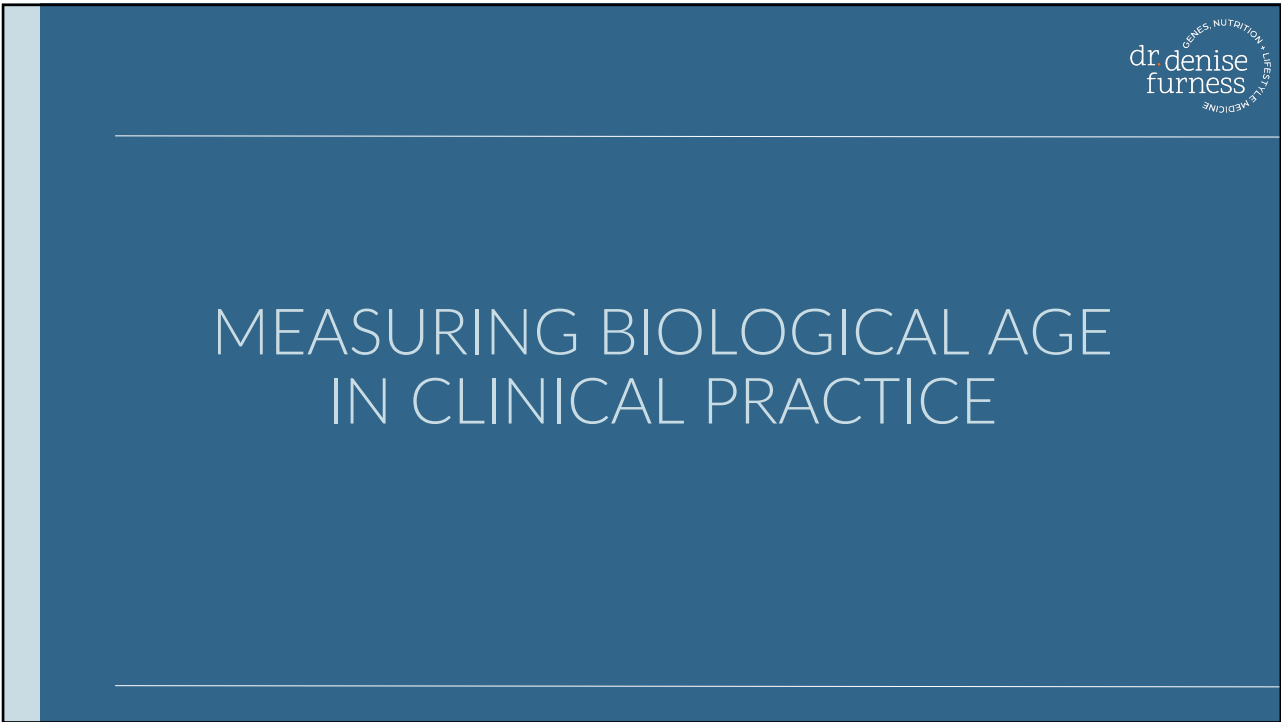
- Ageing drives chronic disease.
- Targeting ageing → impacts age related conditions:
 - Cardiovascular disease
 - Alzheimer's, dementia and neurological conditions
 - Type 2 diabetes and metabolic disorders
- This has led to increased funding in age related research and the rise of longevity clinics.

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BIOLOGICAL AGE TESTS

Anthropometric and physiological measurements

- Body composition, HRV, wearable data.

Blood biomarkers

- Phenotypic Age (more accessible and affordable).

Epigenetic clocks

- DNA methylation – one of the molecular mechanisms and hallmarks of ageing (most of the research and focus is in this area).

Biological age can be different depending on the tool you use

Levine ME, et al. An epigenetic biomarker of aging for lifespan and healthspan. *Aging (Albany NY)*. 2018

Methylation, Epigenetics & Biological Aging

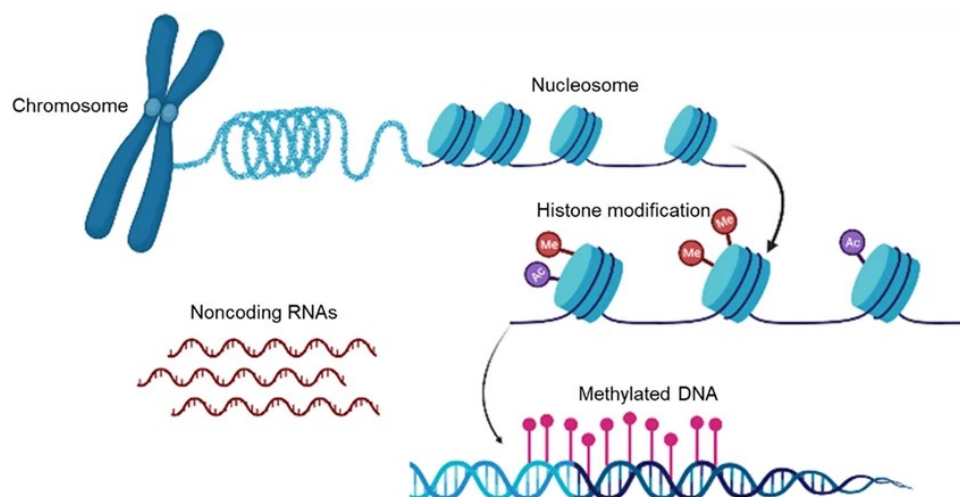
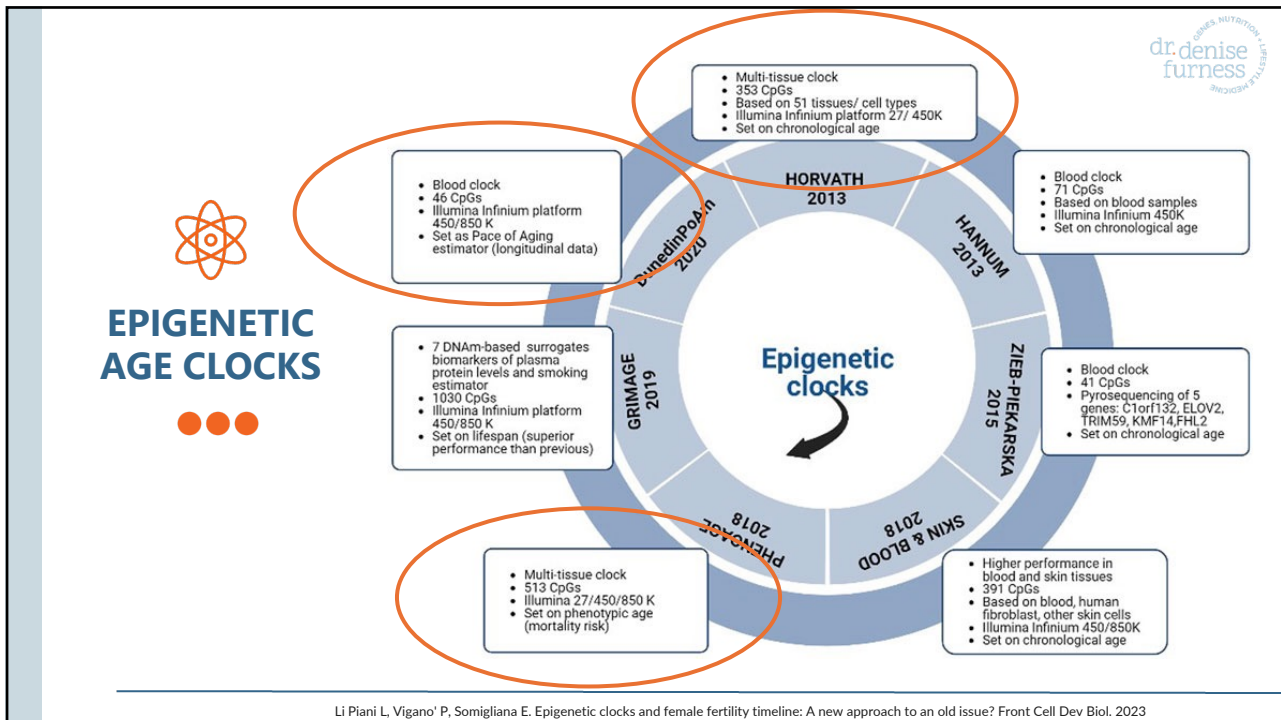


Image: <https://www.londonosteoporosisclinic.com/the-role-of-genetic-factors-in-bone-health/>



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PHENOTYPIC AGE
Biological Age Clock

Blood biomarker & DNA methylation Clock

- Developed to estimate biological age linked to age-related disease risk and mortality.
- Combines **chronological age + 9 standard blood markers**.
- Accessible, cost-effective, and clinically practical compared to epigenetic testing.
- Biomarkers reflect key physiological systems, including:
 - immune/inflammatory activity, metabolic health, liver & kidney function, and haematological (RBC width) markers.
- Interpretation:
 - Higher than chronological age → accelerated biological ageing

Levine M, et al. An epigenetic biomarker of aging for lifespan and healthspan. Aging (Albany NY). 2018; Liu Z, et al. A new aging measure captures morbidity and mortality risk across diverse subpopulations from NHANES IV: A cohort study. PLoS Med. 2018

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DunedinPACE (Pace of Ageing)



What it measures:

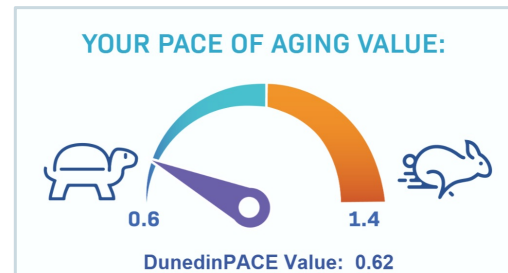
- Rate of biological ageing (how fast the body is ageing).
- Based on longitudinal, multi-system data.

Associated with:

- Chronic disease & mortality.
- Cognitive decline & brain ageing.
- Physical function (e.g. strength, balance).
- Visible (facial) ageing.

What it reflects:

- Cardiometabolic health, physical and cognitive performance.
- Inflammation, immune function & organ function (liver, kidney, lung).



Belsky DW, et al. DunedinPACE, a DNA methylation biomarker of the pace of aging. *Elife*. 2022; Whitman E, et al. A blood biomarker of the pace of aging is associated with brain structure: replication across three cohorts. *Neurobiol Aging*. 2024

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PhenoAge →
How old are you biologically?

DunedinPACE →
How fast are you ageing?



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EPIGENETIC (BIOLOGICAL) AGE RESEARCH

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BIOLOGICAL AGEING & COGNITION

- Faster biological ageing (epigenetic clocks) is associated with:
 - *worse cognitive performance*
 - *faster cognitive decline*
- Evidence observed across **multiple cohort studies**.
- **Strength and consistency vary by clock** (*stronger for pace-of-ageing measures*).

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Association of rate of cognitive decline with epigenetic clocks in a population-based study

Study Overview

- n = 2,296 adults (mean age ~62) / ~20 years longitudinal cognitive follow-up
- Epigenetic clocks: PhenoAge, GrimAge, DunedinPACE

Key Findings

- Faster biological ageing across all clocks associated with:
 - ↓ baseline cognition + ↑ rate of decline ~20 yrs
- Faster DunedinPACE is associated with preclinical cognitive ageing.
 - DunedinPACE = most consistent predictor of cognitive decline rate
- They adjusted for APOE genotype in their models
 - The association between biological ageing and cognitive decline remained significant

Abstract

Introduction: The geriatric syndrome of cognitive decline.

Methods: We analyzed longitudinal data from the Baltimore Longitudinal Study, a population-based study of aging. We analyzed data from 2,296 adults (mean age M = 62, SD = 9, range 45-90 years) who participated in a longitudinal cognitive follow-up study. We analyzed data from the DunedinPACE epigenetic clock, a measure of biological aging. Analysis tested if faster rate of cognitive decline was associated with faster rate of biological aging. Findings were similar for the PhenoAge and GrimAge epigenetic clocks.

Results: Participants with faster rate of cognitive decline experienced more rapid biological aging. Faster rate of biological aging was associated with faster rate of cognitive decline across population strata. Findings were similar for the PhenoAge and GrimAge epigenetic clocks.

Discussion: Faster pace of aging is a risk factor for preclinical cognitive decline. Metrics of biological aging may inform risk stratification in clinical trials and prognosis in patient care.

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dr.denise GENES, NUTRITION + LIFESTYLE

> Neurobiol Aging. 2024 Apr;136:23-33. doi: 10.1016/j.neurobiolaging.2024.01.008. Epub 2024 Jan 24.

A blood test predicts brain ageing

Ethan T Whitmer¹, Maxwell L Ellison², Tracy R Melzer³, Jiayi Zhou^{2,4}, Alzheimer's Disease Neuroimaging Initiative⁵

Affiliations +
PMID: 38301411

Study Overview: n~2,300 participants across 3 cohorts

- Dunedin Study (midlife), Framingham Offspring Cohort, ADNI (older adults / cognitive decline cohort)

Biological age clocks: DunedinPACE, GrimAge, first-generation clocks (Horvath, Hannum).

Brain MRI: Brain volume, hippocampal volume, cortical thickness, white matter lesions.

Key Findings

- Faster biological ageing ↑ DunedinPACE associated with brain ageing neurodegeneration:
 - ↓ Brain volume
 - ↓ Hippocampal volume
 - ↓ Cortical thickness
 - ↑ White matter lesions
- Models adjusted for APOE genotype - associations remained **significant**
- Biological ageing reflects brain changes **beyond genetic risk**.

Abstract
Biological ageing is a complex process involving many age-related changes. Some of these changes are associated with neurodegeneration, leading to associations between biological ageing and cognitive decline. To advanced age-related changes, we tested four biological age clocks (mean age=63.5 years) also tested four first-generation clocks (PhenoAge, an accelerated clock, and DunedinPACE, a faster clock). DunedinPACE was associated with a greater burden of white matter lesions, hippocampal volume, and cortical thickness. GrimAge had the strongest associations with brain changes. These findings suggest that single timepoint measures of multi-organ decline such as DunedinPACE could be useful for gauging nervous system health.

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> Alzheimers Dement. 2023;19(12):2023-2034

Epigenetic clocks and plasma biomarkers of biological ageing

Bowei Zhang ¹, Linda K. McEvoy ², Steve Horvath ³, Ake T. L. Lee ⁴, Susan M. Resnick ⁷, Michael J. McClellan ⁸, JoAnn E. Manson ^{12, 13}, L. R. McEvoy ¹⁴

Affiliations + expand
PMID: 41399190 PMCID: PMC10311111

Abstract

Introduction: Chronological age predicts the risk of developing dementias (ADRD). However, biological age with ADRD pathology is less clear.

Methods: In a cohort of cognitively normal older women, we examined associations of epigenetic clocks and plasma biomarkers of biological ageing with 15-year changes in brain biomarkers.

Results: At baseline, higher epigenetic age (AgeAccel) was associated with higher plasma Aβ₄₂ (Aβ₄₂:Aβ₄₀ ratio) and lower plasma Aβ₄₀ (Aβ₄₀:Aβ₄₂ ratio). These associations remained after adjusting for chronological age, education, and APOE genotype. Epigenetic age was also associated with plasma GFAP (neuroinflammation) and NfL (neurodegeneration). These associations remained after adjusting for chronological age, education, and APOE genotype.

Discussion: Accelerated biological ageing (epigenetic clocks) is associated with higher plasma Aβ₄₂:Aβ₄₀ ratio and lower plasma Aβ₄₀:Aβ₄₂ ratio, as well as higher plasma GFAP and NfL. These associations remained after adjusting for chronological age, education, and APOE genotype.

Highlights: We studied 2,366 cognitively normal older women. AgeAccelHorvath and AgeAccelGrim2, PCPhenoAge, and DunedinPACE were associated with plasma biomarkers of biological ageing.

Study Overview

- Investigated whether **biological ageing predicts changes in brain biomarkers over time**
- 2,366 older women** cognitively normal older women
 - Longitudinal subset: **n = 873**, Follow-up: **~15 years**
- Epigenetic clocks: **PhenoAge, GrimAge, DunedinPACE**
- Plasma Biomarkers Measured
 - Amyloid:** Aβ₄₂:Aβ₄₀, **Tau:** p-tau181, p-tau217, **Neurodegeneration:** NfL and **Neuroinflammation:** GFAP

Key Findings

- DunedinPACE was strongest predictor** - Predicted longitudinal increases in:
 - p-tau181 & p-tau217 (tau pathology)
 - NfL (neurodegeneration)
 - GFAP (neuroinflammation)
- PhenoAge & GrimAge** - Associated with:
 - Amyloid changes (Aβ ratio)
 - Neurodegeneration (NfL)
- APOE genotype** → independently associated with brain biomarkers
 - Associations between epigenetic ageing and biomarkers **remained after adjusting for APOE**

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BIOLOGICAL AGEING & METABOLIC HEALTH

- Faster biological ageing (epigenetic clocks) are associated with:
 - *Impaired blood sugar regulation*
 - *Cardiovascular health outcomes*
 - *Liver and respiratory function*
 - *Broader age-related chronic conditions*
- Evidence observed across large longitudinal cohorts.
- Strength and consistency vary by biological age measure.

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> J Gerontol A Biol Sci Med Sci. 2025 Jun 10;80(7):glaf085. doi: 10.1093/gerona/glaf085.

Association of Prevalent and Incident Type 2 Diabetes with Epigenetic Age

Danmeng Lily Li¹, Allison F. Pierre-Antoine Dugue^{1, 2}

Affiliations + expand
PMID: 40256801 PMCID: PMC11511111

Abstract

Background: Type 2 diabetes (T2D) is a global health burden, and its pathogenesis involves complex interactions between genetic and environmental factors. Epigenetic age, a measure of biological aging, has been associated with T2D risk. However, the associations of epigenetic age with prevalent and incident T2D remain unclear.

Methods: We used data from the Dunedin Study, a population-based cohort study of 1032 New Zealanders born in 1978. We assessed the associations of epigenetic age (measured by five blood-based clocks: DNAmAge, GrimAge, PhenoAge, DunedinPACE, and DunedinPoAm) with prevalent and incident T2D. We used modified Poisson regression models to estimate the relative risk of T2D associated with epigenetic age.

Results: A standard deviation increase in epigenetic age was associated with a 1.22-fold increase in the risk of prevalent T2D (95% CI: 0.98-1.26) and a 1.33-fold increase in the risk of incident T2D (95% CI: 1.07-1.33). Prospectively, DunedinPACE showed the strongest association with incident T2D (hazard ratio = 1.22, 95% CI: 1.07-1.33), followed by PhenoAge (hazard ratio = 1.18, 95% CI: 1.03-1.33). DunedinPoAm and DNAmAge showed weaker associations. Analyses reclassifying participants into higher and lower risk groups based on fasting glucose levels (> 7 mmol/L for fasting glucose and > 10 mmol/L for nonfasting glucose) showed that higher epigenetic age was associated with higher risk of T2D, and this association was stronger in individuals with higher fasting glucose levels.

Conclusions: In middle-aged and older adults, higher epigenetic age, measured by DunedinPACE, was positively associated with the risk of prevalent and incident T2D, with stronger associations observed with DunedinPACE than with other epigenetic clocks. These findings have implications for understanding the pathogenesis of T2D and for identifying individuals at high risk of T2D.

Study Overview - Epigenetic Age and Type 2 Diabetes: Longitudinal Evidence

- n = 5,403 (middle-aged and older adults, mean age ~59)
- Prospective design with multiple follow-ups (up to ~10+ years)
- Investigated 5 Epigenetic clocks including: **PhenoAge, GrimAge, DunedinPACE**

Outcomes Assessed

- Prevalent T2D → *had at baseline*
- Incident T2D → *developed during follow-up*
- Fasting Glucose → *continuous measure across all participants (with and without T2D)*

Key Findings

Prevalent T2D (existing)

- Higher biological age associated with **T2D at baseline** observed across multiple clocks, including **PhenoAge** and **DunedinPACE**

Developed T2D (future risk)

- DunedinPACE showed the strongest association, PhenoAge showed an association initially, not significant after adjustment for risk factors.

Fasting Glucose

- Higher biological age associated with **higher glucose levels**, stronger associations observed with DunedinPACE, PhenoAge showed weaker associations.

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> Cardiovasc Diabetol. 2025 Jun 10;24(1):10. doi: 10.1007/s00125-025-06805-5.

Biological age and cardiovascular risk in individuals with type 2 diabetes: a prospective cohort study

Zhiwei Zeng^{1, 2, 3}, Chunyu Xueying Wang^{1, 2, 3}, Xi Li^{1, 2, 3}

Affiliations + expand
PMID: 40770719 PMCID: PMC11511111

Abstract

Background: Biological age (BA) is a measure of the rate of aging, and it has been associated with cardiovascular risk. However, the associations of BA with cardiovascular risk in individuals with type 2 diabetes (T2D) remain unclear.

Methods: We included 12,828 individuals with T2D from the UK Biobank cohort. We assessed the associations of BA (measured by PhenoAge and PhenoAgeAccel) with cardiovascular risk (including major cardiovascular outcomes, heart failure, atrial fibrillation, and venous thromboembolism). We used Cox proportional hazards models to estimate the hazard ratios of cardiovascular risk associated with BA.

Results: During a median follow-up of 10.5 years, higher BA was associated with an increased risk of major cardiovascular outcomes (hazard ratio = 1.18, 95% CI: 1.03-1.33). PhenoAgeAccel showed the strongest association with major cardiovascular outcomes (hazard ratio = 1.22, 95% CI: 1.07-1.33). Higher BA was also associated with an increased risk of heart failure (hazard ratio = 1.15, 95% CI: 1.01-1.30) and atrial fibrillation (hazard ratio = 1.12, 95% CI: 1.01-1.24). The associations of BA with cardiovascular risk were stronger in individuals with higher fasting glucose levels and higher levels of inflammatory markers (C-reactive protein and interleukin-6).

Conclusions: Biological age, measured by PhenoAge and PhenoAgeAccel, was positively associated with the risk of major cardiovascular outcomes, heart failure, atrial fibrillation, and venous thromboembolism in individuals with T2D. These findings have implications for understanding the pathogenesis of cardiovascular disease and for identifying individuals at high risk of cardiovascular disease.

Study Overview - Biological Age and Cardiovascular Risk

- n = 12,828 with T2M - UK Biobank cohort
- **PhenoAge** (derived from the 9 blood biomarkers + chronological age) assessed
- **PhenoAgeAccel** (difference between biological age and expected age) assessed
- Examined associations with **major cardiovascular outcomes**

Key Findings

PhenoAge and cardiovascular risk

- Higher PhenoAge associated with **increased risk across all major cardiovascular outcomes**

PhenoAge Acceleration

- Individuals with higher PhenoAge acceleration (faster biological ageing relative to chronological age) showed increased cardiovascular risk.

PhenoAge is derived from standard clinical biomarkers, it reflects underlying metabolic and inflammatory processes that are directly modifiable in practice.

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nature communications

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Biological Age Clocks: Large-Scale Evidence Across Chronic Health Outcomes

Christos Mavromatis
Vadim N. Gladyshev

Nature Communications
10k Accesses | 8 Citations

Abstract

Epigenetic Clocks (ECs) are mathematical models that predict chronological age from DNA methylation data. Such clocks are often used to study the relationship between age and health outcomes. However, the accuracy and utility of these clocks have been debated. Here, we present a large-scale analysis of 14 ECs across 18,859 individuals in a 10-year longitudinal study. We compare the performance of these clocks in predicting mortality and incident chronic health outcomes. Our results show that later-generation clocks significantly outperformed first-generation clocks (e.g. Horvath, Hannum). Strong associations were observed across multiple disease categories, including: Respiratory illness (including lung cancer), Liver disorder, and Metabolic – T2DM/ CVD. GrimAge, DunedinPACE, and PhenoAge showed the most consistent associations with chronic health outcomes.

- n = 18,859, 10-year longitudinal follow-up
- 14 epigenetic clocks compared
- Associations tested across 174 incident chronic health outcomes

Key Findings

- Later-generation clocks significantly outperformed first-generation clocks (e.g. Horvath, Hannum).
- Strong associations observed across multiple disease categories, including:
 - Respiratory illness (including lung cancer)
 - Liver disorder
 - Metabolic – T2DM/ CVD
- GrimAge, DunedinPACE, and PhenoAge showed the most consistent associations with chronic health outcomes.

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Biological age is not just theoretical, large-scale data shows it is measurable, reproducible, & associated with real-world health outcomes.

This type of large-scale analysis gives us confidence that these measures are not just research tools, they are increasingly relevant in clinical settings where we are trying to quantify & improve long-term health.

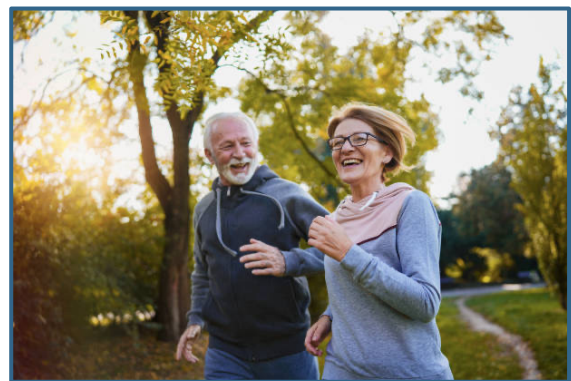
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A MULTI-MODEL APPROACH TO SUPPORT EPIGENETIC AGEING

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FROM LIFESTYLE TO TARGETED SUPPORT

- Lifestyle and nutrition form the foundation for healthy aging.
- There is growing interest in targeted nutritional and supplement strategies to support biological ageing.



The strongest evidence centres on supporting key biological systems and the molecular mechanisms involved in aging.

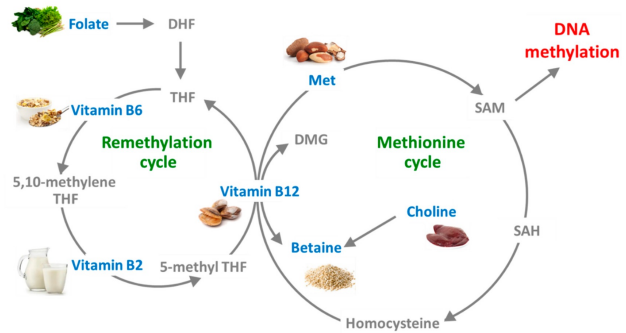
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METHYLATION, EPIGENETICS & BIOLOGICAL AGING

DNA methylation is a core mechanism underlying epigenetic clocks.

- B vitamins (folate, B12, B6, B2):
 - Support methylation capacity
 - Regulate homocysteine
 - Influence gene expression patterns



Disruptions → altered methylation → may influence biological ageing

Hussain SA, Sarker MI, Liu Y, Jin TZ. DNA Methylation and Its Role in Personalized Nutrition: Mechanisms, Clinical Insights, and Future Perspectives. *Int J Mol Sci.* 2026
Image: Cappuccilli, M. et al., Vitamin B Supplementation and Nutritional Intake of Methyl Donors: A Critical Review of the Impact on Epigenetic Machinery. *Nutrients* 2020

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Mitochondrial Function & Biological Aging

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GENES, NUTRITION & FERTILITY MEDICINE

NAD⁺ Precursors (NR / NMN) both increase NAD⁺ levels in humans.

- *NAD⁺ is a central cofactor in cellular energy metabolism and mitochondrial function.*

Nicotinamide Riboside (NR)

- Stronger clinical (human) evidence base.
- Modest biological age effects seen in DNA methylation clocks (↓ *PhenoAge*, ↓ *GrimAge*).
- Recent studies suggest possible benefits on brain-related biomarkers and brain perfusion.

Nicotinamide Mononucleotide (NMN)

- Strong preclinical evidence for ↑ mitochondrial function (ATP, oxidative phosphorylation), ↓ oxidative stress.
- Animal models show reduced amyloid pathology, improved memory and synaptic function.
- Limited human data for biological aging.

Orr M, et al., A randomized placebo-controlled trial of nicotinamide riboside in older adults with mild cognitive impairment. *Geroscience.* 2024; Yoshino J, et al., NAD⁺ Intermediates: The Biology and Therapeutic Potential of NMN and NR. *Cell Metab.* 2018; Wu C, et al., Cognitive biomarker effects of oral nicotinamide riboside (NR) supplementation in older adults with subjective cognitive decline and mild cognitive impairment. *A. Dement (N Y).* 2025.

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Review

Coenz

Madeleine
Jeffery M R

Affiliations
PMID: 4094

Abstract

Background
countries, c
metabolic a
adenosine t
flow, have t
Coenzyme
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CoQ10 on cognitive function in ageing and in neurodegenerative diseases, future studies are needed that target possible mechanisms and utilise a wider range of cognitive assessments.

CoQ10 / Ubiquinol: Mitochondrial & Translational Evidence

Preclinical Evidence (Animal Models)

- ↑ Mitochondrial function (ATP production, ETC activity)
- ↓ Oxidative stress and neuronal damage
- ↓ Amyloid plaque accumulation
- ↑ Memory and cognitive performance

Human Clinical Trials, 8 trials identified:

- 4 showed improvements in cognition
- 2 showed increased cerebral blood flow
- Remaining → mixed or no significant effect

Less consistent than preclinical data

Ubiquinone vs Ubiquinol

- **Ubiquinol (reduced form):** Greater bioavailability, antioxidant activity, may be more relevant in ageing populations, and higher oxidative stress states.

POLYPHENOLS & CELLULAR DEFENCE

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Plant-derived bioactive compounds found in foods and botanicals that influence:

- cellular signalling pathways, including Nrf2
- oxidative stress
- inflammation
- metabolic function

Resveratrol has over two decades of research demonstrating effects on metabolic function (glucose, insulin, lipids) and cellular signalling pathways involved in oxidative stress and inflammation.

Brown K, et al., Resveratrol for the Management of Human Health: How Far Have We Come? A Systematic Review of Resveratrol Clinical Trials to Highlight Gaps and Opportunities. Int J Mol Sci. 2024



PILOT PROGRAM OUTLINE

Lead Investigator: Denise Furness (PhD), The Academy for Precision Health
Co-Investigator: Paul Taylor (PhD), University Newcastle

Aim: To determine if a 3-month *and* 6-month nutrition, supplement and lifestyle intervention program can alter DNA methylation and biological age.

Methods:

- This was a real-world, practitioner-led intervention designed to reflect clinical practice rather than a controlled trial setting.
- Biological age is measured using the TruAge (TruDiagnostic Lexington, KY) test, via an at home blood spot collection kit.
- Baseline DNA methylation biological age markers were measured at the start of the study before intervention and again at the end of the study after the 3-month and 6-month intervention to assess any changes.

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-Drink filtered water throughout the day - 250-300ml per 10 kg of body weight – more if training or using the sauna.	-Start the day with a hydrating drink before coffee as many days as possible.	-Aim for a minimum 12 hr fast as many days as possible (fasting range is 12-16 hrs).	-Eat organic (or spray-free), wild or free-range foods as many times as possible.
-Aim to get most of your carbohydrates from vegetables, legumes, nuts, seeds and fruits.	-Have your last meal approx. 3 hrs before bed (and avoid snacks before bed) as many days as possible.	-Avoid added sugars, highly processed foods and refined grains (twice-cooked starch is accepted).	-Aim for a maximum of 2 coffees (not after 2 pm) as many days as possible. Drink herbal tea as desired.
-Limit alcohol to 2 standard drinks per week (preferably red wine).	-Minimise plastic food and drink containers (never heat food in these).	-Minimise chemical-laden household cleaners, fragrances, skincare and beauty products.	

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GENES, NUTRITION, LIFESTYLE MEDICINE

INTERVENTION

Dietary Advice:

- Wholefoods, high vegetable and protein intake, high fiber carbohydrates, nuts, seeds, fermented foods, herbs/spices.

Lifestyle Advice:

- Exercise 30 mins day, including 3 x week strength training. Sleep 7-9 hrs most nights. Reduce exposures. Mindfulness daily 5-10 mins.

Supplements:

- To support methylation, mitochondrial function and cellular health.

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GENES, NUTRITION, LIFESTYLE MEDICINE

NUTRACEUTICALS

Five core products recommended daily in the protocol.

****If required:**

- Omega 3's
- Vitamin D
- Iron

B complex - 400 mcg 5-MTHF, 400 mcg MethylB12, B1, B2, B3, B5, B6

Resveratrol - 100 mg trans-resveratrol

Ubiquinol - 200 mg

Nicotinamide Riboside – 100 mg plus magnesium

Liver support - NAC, sulforaphane, curcumin, milk thistle, ALA, AA's

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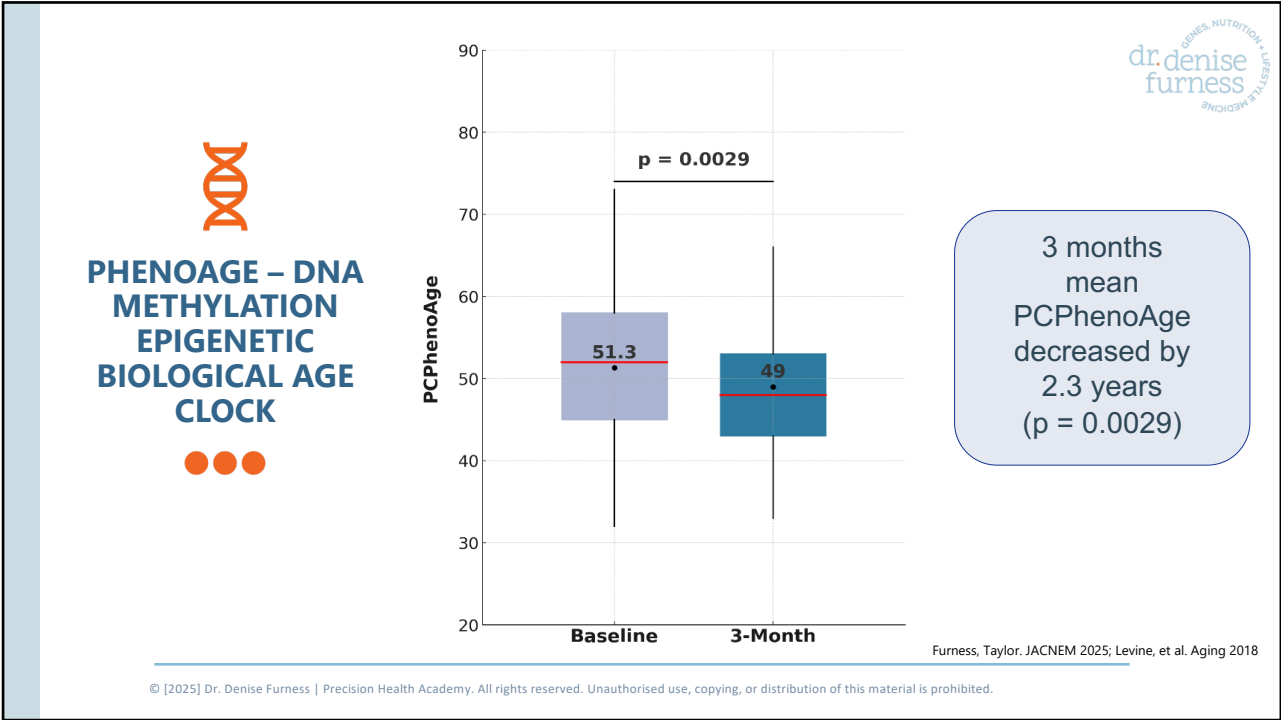
PARTICIPANTS WHO COMPLETED THE 3 MONTH-STUDY

CATEGORY	VALUE
Total Participants	41
Female	30
Male	11
Age Range	30 - 81 years
Mean Age	55.4 years
Median Age	56 years

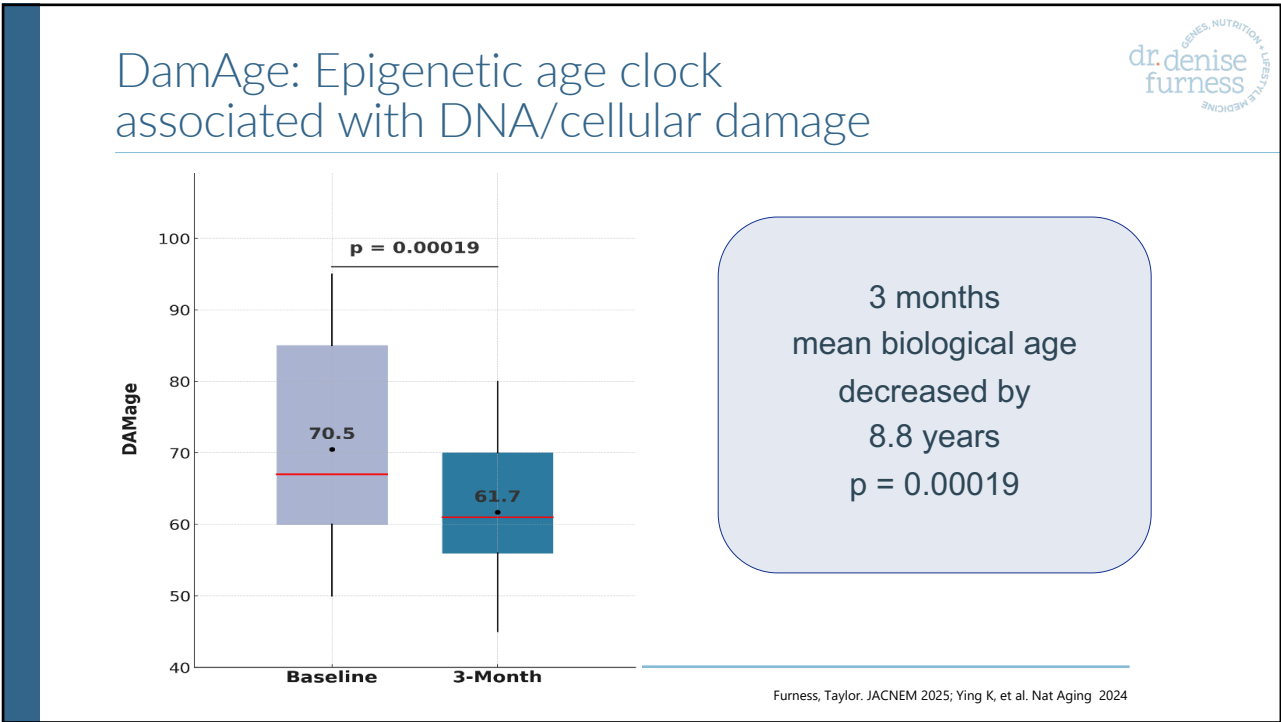
- 60 adults were enrolled through self-selection and received no financial compensation.
- 41 participants (30 females, 11 males) completed both baseline and follow-up DNA methylation testing with samples that passed laboratory quality control.
- Non-completion was due to one or more of the following: (1) medical contraindications, (2) voluntary withdrawal, (3) failure to complete follow-up testing within the designated timeframe, (4) sample errors (e.g., insufficient blood spot card), or (5) sample loss or damage during shipping.

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3-MONTH POST PROTOCOL OUTCOMES



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Changes in Epigenetic Clock Measures Following 3-Month Intervention



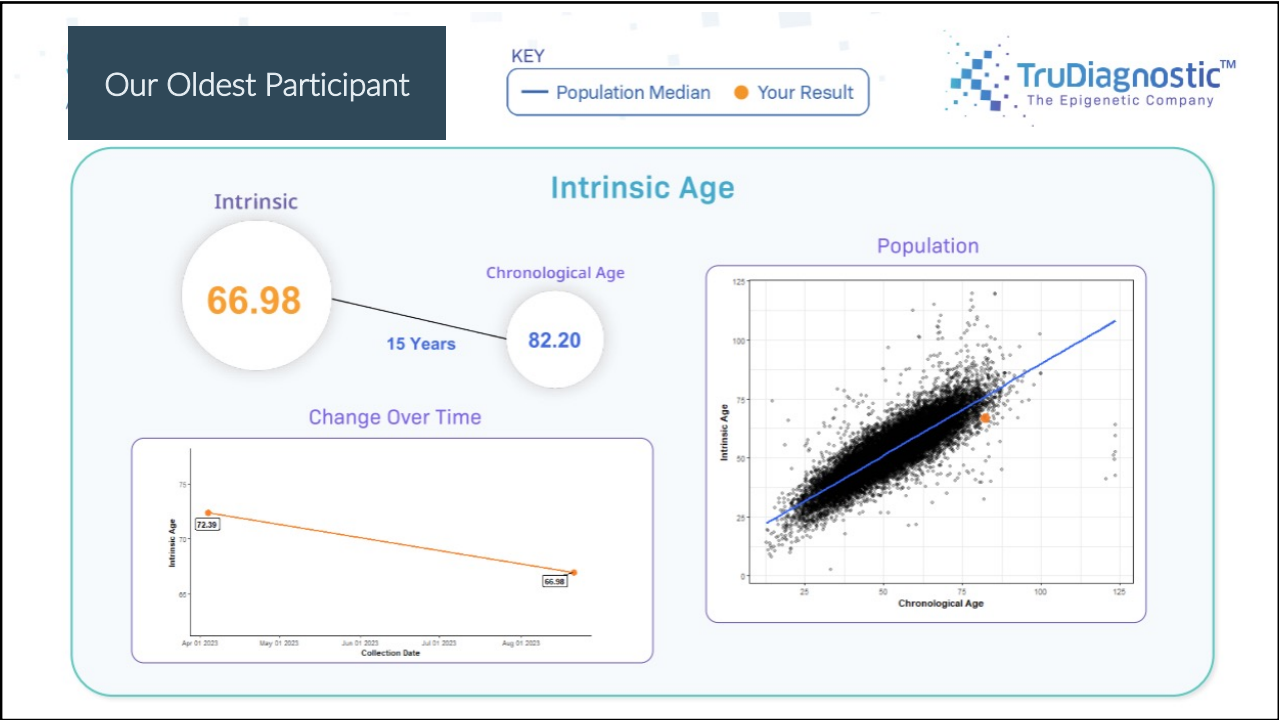
Epigenetic Clock	Baseline Mean	3-Month Mean	Change Mean	p-value
PC_PhenoAge	51.3	49	-2.3	0.0029
DAMAge	70.5	61.7	-8.8	0.00019
Intrinsic Epigenetic Age	54.2	53.8	-0.4	0.71
DunedinPACE	0.88	0.87	-0.01	0.59

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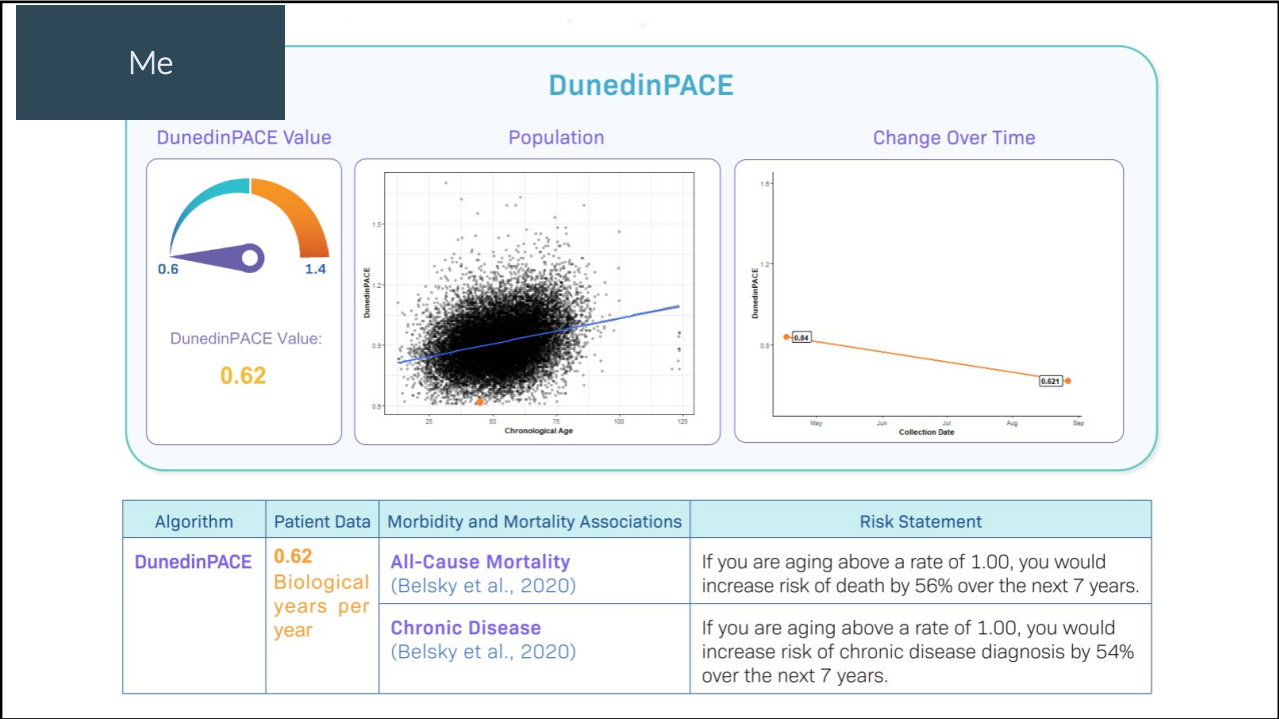
FROM GROUP RESULTS TO INDIVIDUAL RESPONSES

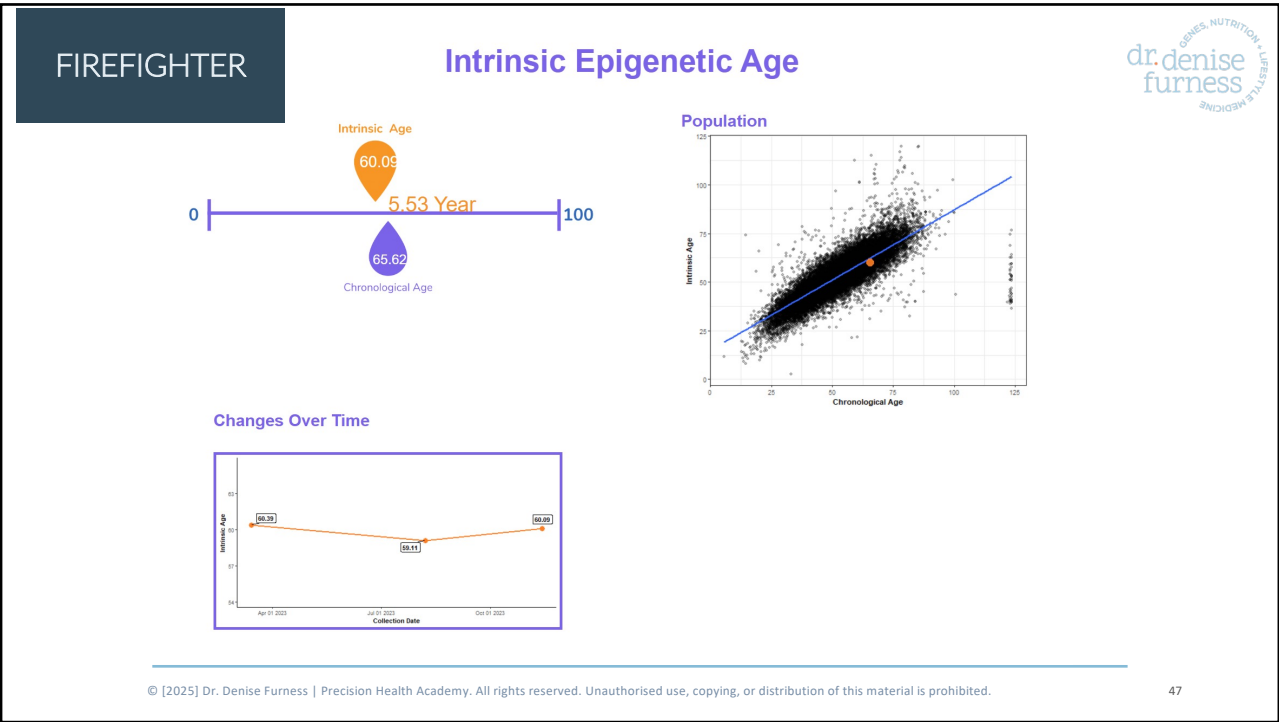


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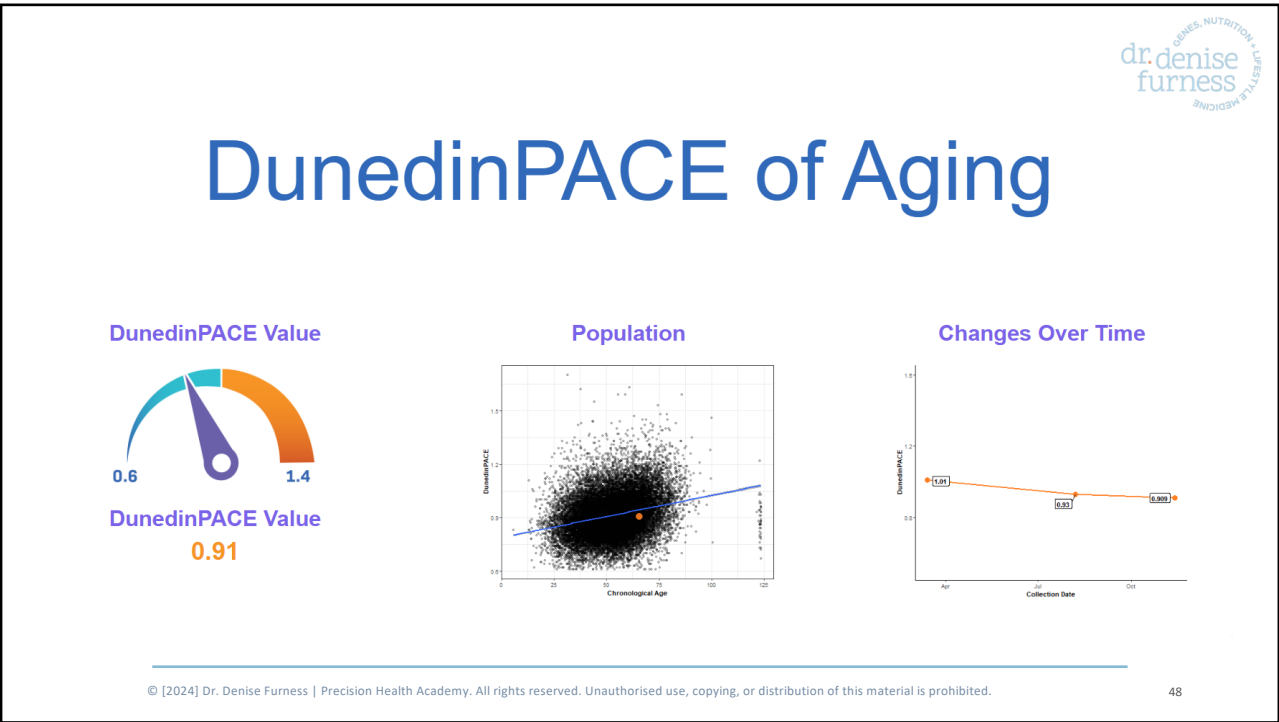


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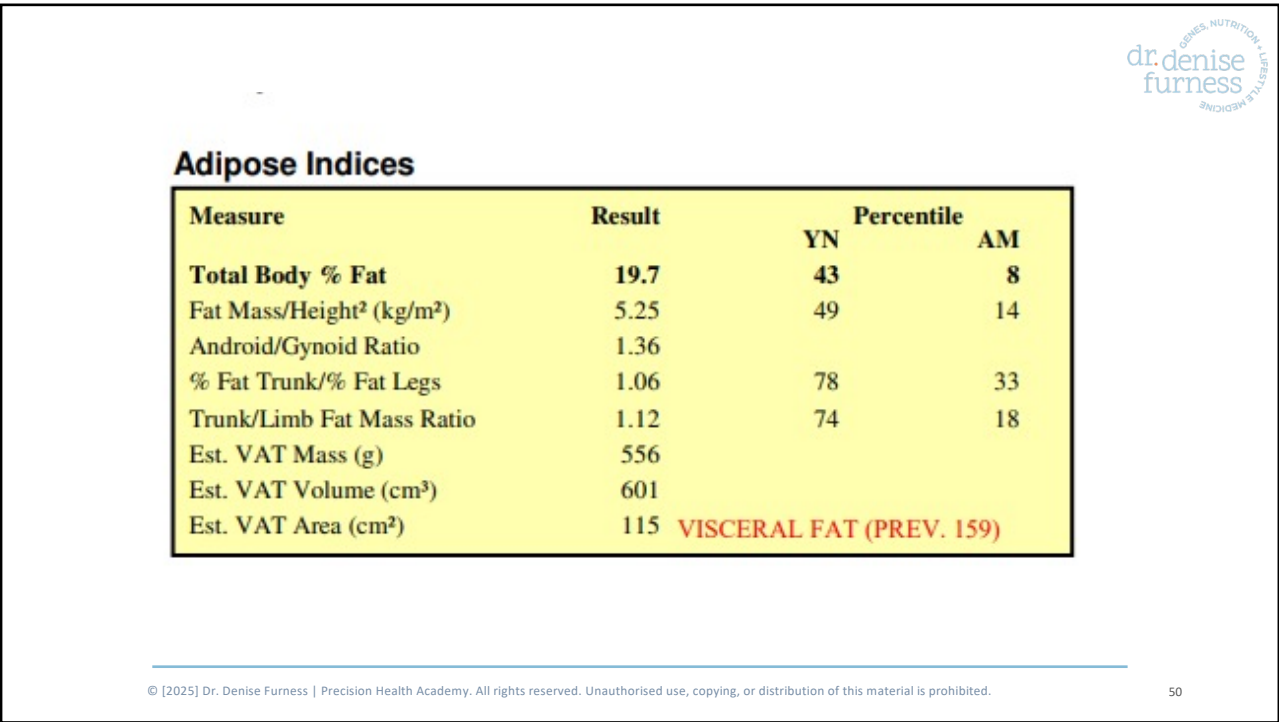
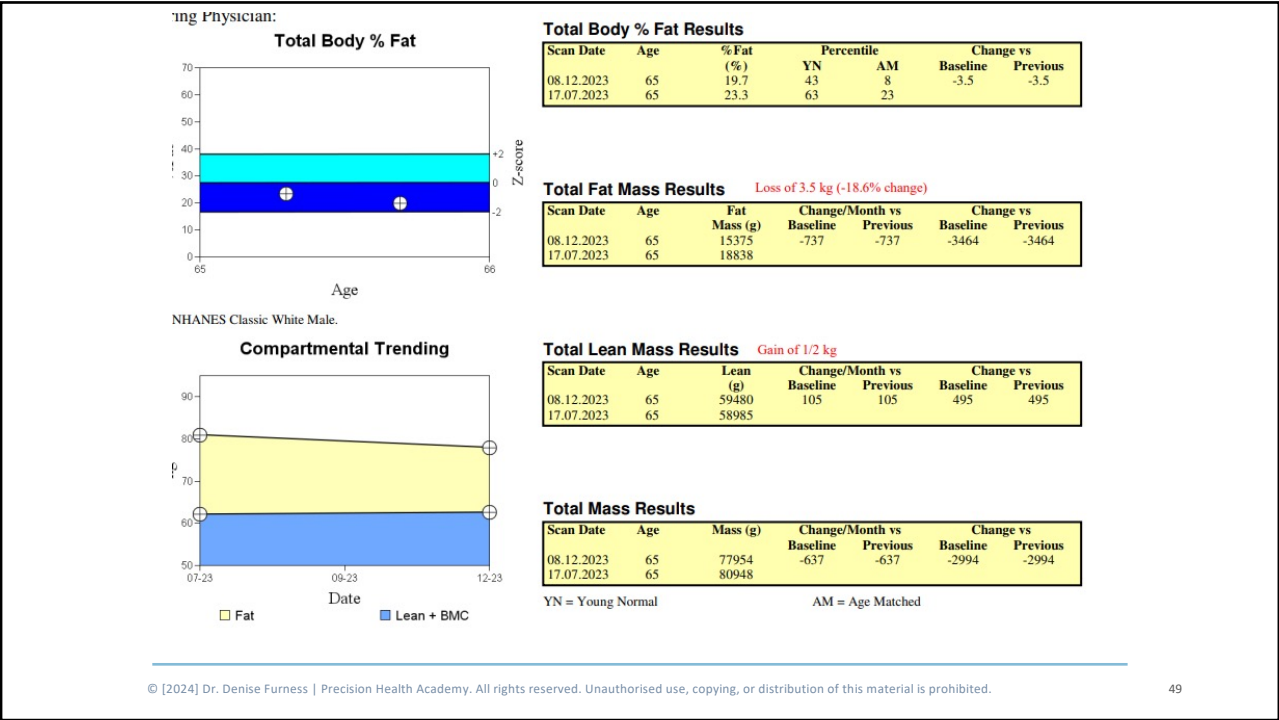


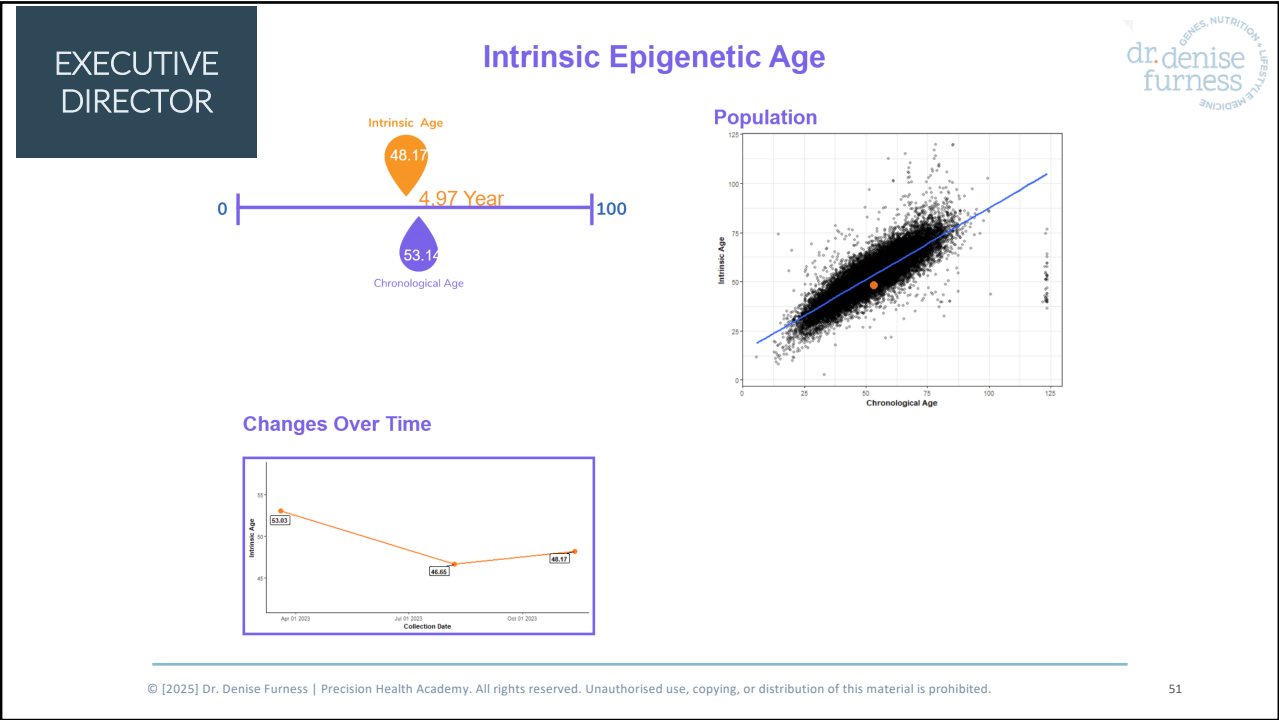


47

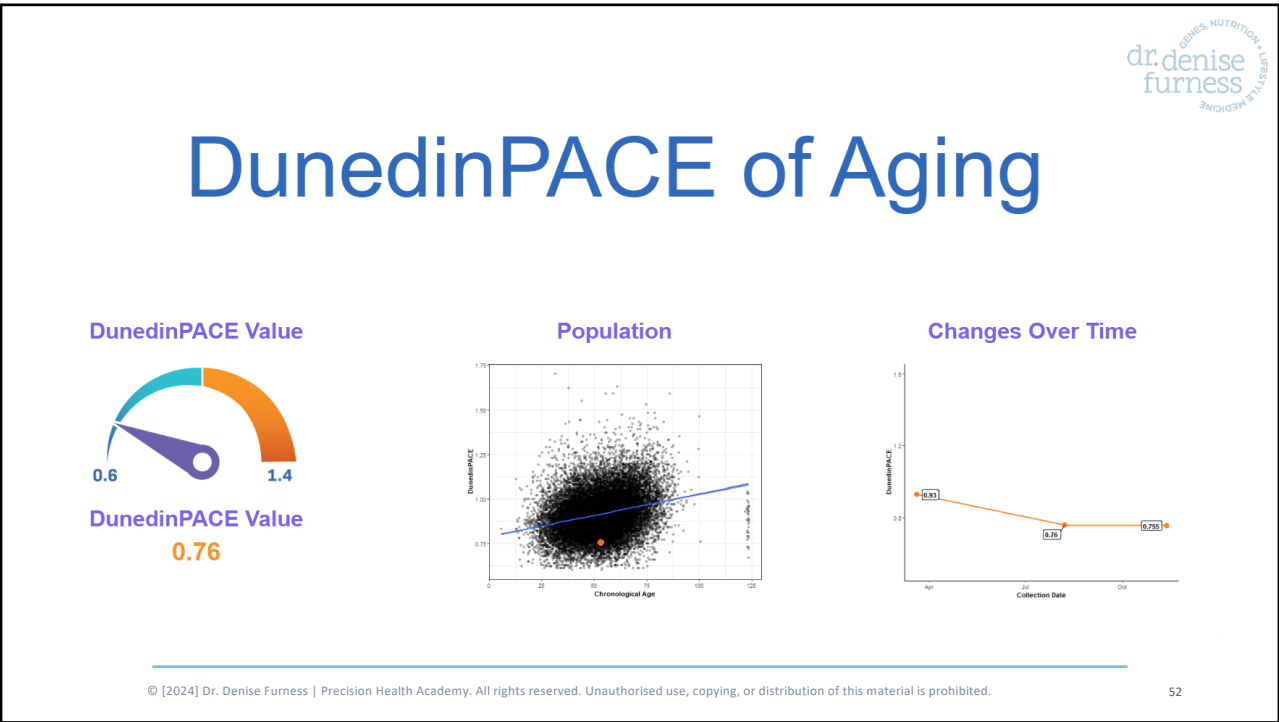


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SUMMARY



- **Ageing Is Modifiable** – Lifestyle, nutrition, and targeted interventions can improve biological age measures and slow aspects of the ageing process.
- **Epigenetic Clocks as Tools** – Multiple clocks reveal different aspects of ageing, providing valuable insights for guiding longevity medicine.
- **Real-World Evidence** – Short-term interventions produced significant reductions in biological age within just 3 months.
- **From Research to Practice** – Translating these findings empowers clinicians to personalise strategies that extend healthspan and improve patient outcomes.

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