CONNECT
HEALTH

LONGEVITY 100

A Science Driven Approach to Healthy Aging

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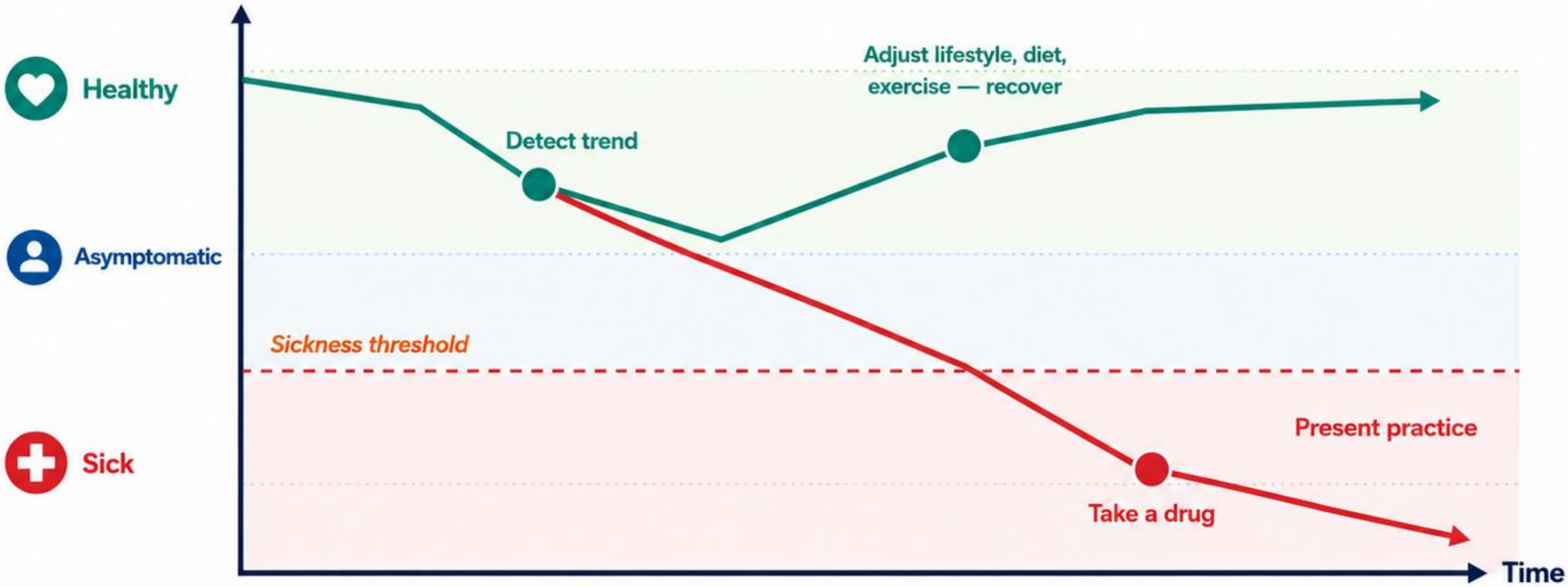
26th Annual Healthcare Summit

EARLY DETECTION

Catching trends — a major opportunity

Detecting the early transition into illness is an opportunity to potentially reverse the trend back toward health.



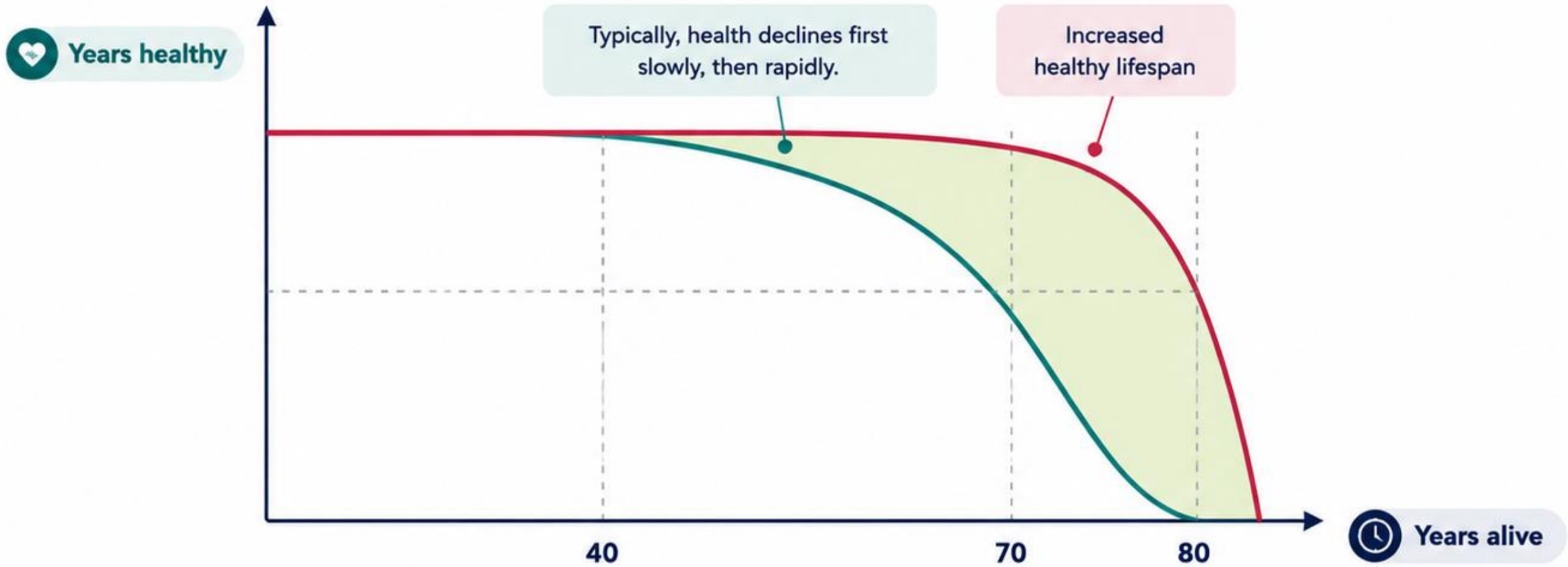
Proactive — detect & adjust | Present practice — react after threshold

The opportunity: *personalized diet, exercise, and supplement programs based on the patient's own data.*

THE FRAME

Healthspan vs. lifespan

Extending years alive is the 20th-century achievement. ***Extending years lived well is the goal now.***



The goal: increase years alive (*lifespan*) while extending the years lived with health and vitality (*healthspan*).

THE SHIFT

From treating disease to extending healthspan

TRADITIONAL CARE
Reactive

- Treats disease after symptoms emerge
- Episodic, problem-list visits
- Organ-system silos
- Limited longitudinal data between visits

PRECISION LONGEVITY
Proactive

- Detects dysfunction before disease
- Continuous physiologic monitoring
- Systems-biology and root-cause framing
- Targets healthspan, not only lifespan

THE PREMISE: *Aging itself is the dominant risk factor for cardiovascular disease, cancer, neurodegeneration, diabetes, and frailty.*

DEFINITION

What is longevity medicine?

A clinical discipline that applies precision diagnostics, lifestyle science, and evidence-based therapeutics to **detect dysfunction before disease** and extend the period of life lived in good function.

PROACTIVE

Acts before symptoms

PRECISION

Tailored to the individual

LONGITUDINAL

Tracks change over time

EVIDENCE-BASED

Anchored in peer-reviewed data

MECHANISM

The hallmarks of aging

A unifying framework — twelve interconnected mechanisms drive biological aging across tissues. They are why the Big Six cluster.

PRIMARY

Sources of cellular damage

Genomic instability • telomere attrition • epigenetic alterations • loss of proteostasis • disabled macroautophagy

ANTAGONISTIC

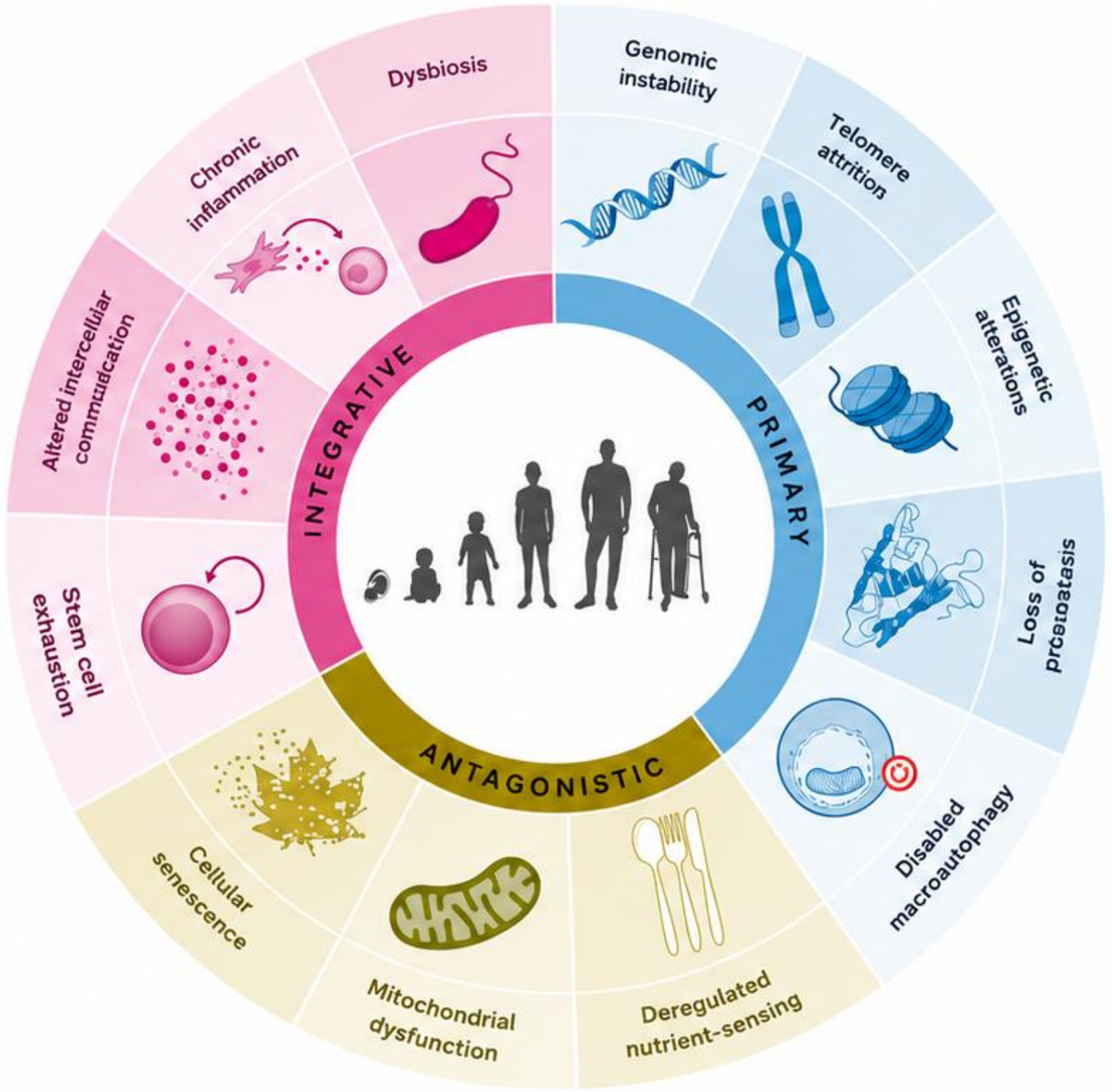
Compensatory responses gone wrong

Deregulated nutrient sensing • mitochondrial dysfunction • cellular senescence

INTEGRATIVE

Tissue- and system-level effects

Stem cell exhaustion • altered intercellular communication • chronic inflammation • dysbiosis



López-Otín et al., Cell 2023;186(2):243–278

Longevity medicine seeks interventions that address the root drivers of aging biology.

Precision Longevity Medicine

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The Big Six — chronic diseases of aging

Six disease categories drive most morbidity and mortality after age 50 in Canada. They share underlying biology — which is why precision longevity medicine attacks them together, not separately.

CANCER

84,629

Canadian deaths/yr — #1 cause of death (25.9%)

CARDIOVASCULAR

57,890

Canadian deaths/yr — #2 cause of death

NEURODEGENERATIVE

~750K

Canadians living with dementia — set to double by 2050

METABOLIC

11.9M

Canadians with diabetes or prediabetes (30%)

IMMUNE AGING

~24K

Canadian flu, pneumonia & COVID-19 deaths in ≥65 (2022)

SARCOPENIA & FALLS

7,189

Canadian fall deaths in adults ≥65 — up 51% since 2017

Shared biology = shared targets.
Addressing common drivers can help prevent, delay, and treat all six together.

EARLY DETECTION

Detect earlier. Act sooner.
Change the trajectory.

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RESULTS OF EARLY SCREENING

Early screening identified unrecognized health risks

Pilot cohort: 30 healthy adults, ages 45-65

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Study context

n = 30

healthy adults

Ages 45-65

Results shown are clinically significant findings from an early screening program.

New diagnoses identified

2
Diabetes

1
Celiac disease

New clinically relevant screening findings

	% of cohort
Low vitamin D	64%
Low omega-3	60%
Suboptimal lipids	60%
Elevated visceral fat / fatty liver	55%
Osteopenia (bone thinning)	50%
Insulin resistance	40%
Hypertension	35%
Significant finding on MRI	>30%
Cancer screening not guideline-concordant	25%
ApoE4 risk allele	11%

Percentages sorted to highlight relative frequency; new diagnoses shown separately.

TOOLS

Expanded Diagnostics — System by System

What we order beyond the standard panel.

CARDIAC

ApoB · Lp(a) · vascular specific inflammation markers, absorption vs production markers, CAC/CT Angio

METABOLIC

Visceral fat (MRI/DEXA) · fasting insulin · HOMA-IR · CGM

MUSCULOSKELETAL

DEXA body comp · bone density · lean muscle mass · myomarkers · c-telopeptide

CANCER

Earlier screening using routine modalities + Whole Body MRI + blood based testing +/- hereditary cancer panels

BRAIN

APOE genotype · brain MRI · cognitive batteries · sleep architecture tracking

GUT

Celiac serology · microbiome · zonulin · stool inflammatory markers

The Pattern:
Each system has 1–2 high-yield advanced tests that change clinical decisions for a meaningful subset of patients.

Our Approach:
Targeted. Evidence-based. Personalized.

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Deeper insights. Precision decisions. Better outcomes.

Atherosclerosis: Opportunities for Early Detection

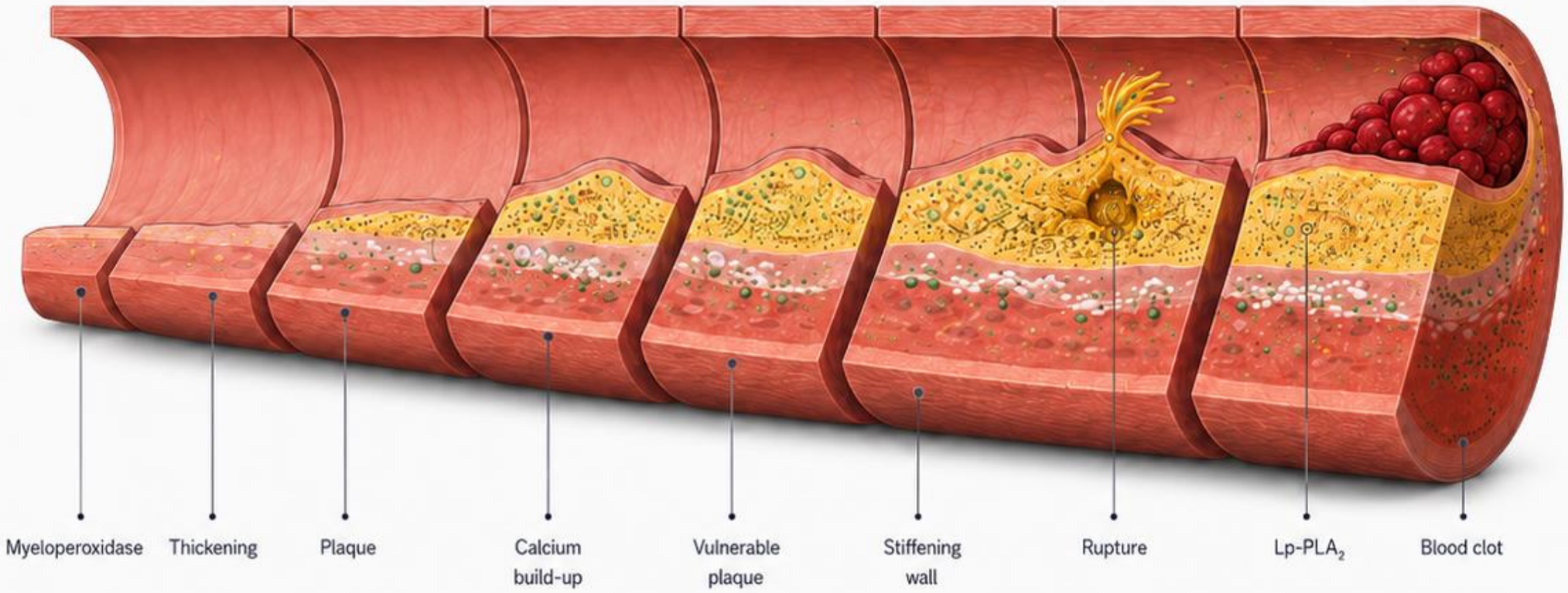
Subclinical disease progression precedes cardiovascular events by decades

	Oxidation		Disease initiation / Plaque growth	Plaque maturation / Vulnerable plaque	Acute coronary syndrome
Representative biomarkers	F_2-IsoPs	OxLDL	hsCRP Microalbumin	MPO Lp-PLA_2	Troponin T CK-MB

Leading cause of mortality worldwide.

Disease develops over decades.

Early detection may enable earlier intervention.



Conceptual representation of atherosclerotic disease progression and associated biomarkers. Not to scale.

References

1. Libby P. Inflammation in atherosclerosis. *Nature*. 2002;420:868–874.
2. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med*. 2005;352:1685–1695.
3. Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. *Circulation*. 2004;109:363–369.

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Cardiac Screening Strategy

LIPID ASSESSMENT
Standard & advanced lipid profiling
ApoB, Lp(a), absorption vs production markers

INFLAMMATORY & METABOLIC RISK
hs-CRP, Lp-PLA2, homocysteine, uric acid
Metabolic health evaluation

GENETIC & PRECISION MEDICINE
ApoE, Lp(a) genetics
Pharmacogenomics

FUNCTIONAL TESTING
Blood pressure assessment
Stress testing

CARDIOVASCULAR IMAGING
CT calcium score / CT coronary angiogram
Echocardiography | ECG

FOUNDATIONAL NUTRIENTS
Omega-3 | Magnesium

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Cancer Screening

- **Wholebody** MRI
- **Skin:** full skin checks every 6 months
- **Colon:**
  - stool for blood testing starting at age 40 or earlier
  - Colonoscopy
- **Oral:** dental exam for oral cancer every 6-9 months
- **Prostate:** yearly MRI, PSA
- **Breast:** mammogram + US or MRI
- **Other:** wholebody MRI
- **Molecular testing:**
  - Circulating tumour DNA (ie. GRAIL)
  - Metabolic signatures
  - Cancer genetics

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WHOLE-BODY MRI FOR CANCER SCREENING

BENEFITS AND DRAWBACKS

Whole-body MRI (WB-MRI) is a radiation-free imaging tool that can detect cancers throughout the body in a single scan.

BENEFITS

RADIATION-FREE
No ionizing radiation—safer for repeated screening and younger individuals.

WHOLE-BODY, COMPREHENSIVE
Assesses all major organs and tissues in one session.

DETECTS EARLY, OCCULT CANCERS
Can identify cancers at an earlier stage, when treatment is more effective.

FINDS CANCERS WITHOUT A USUAL SCREENING PATHWAY
WB-MRI can detect cancers that are not typically screened for (e.g., pancreas, adrenal, sarcoma, ovarian, bile duct, rare organ cancers).
Opportunistic detection beyond standard care.

NO KNOWN BIOLOGICAL HARM
MRI has no known carcinogenic effects.

DRAWBACKS

INCIDENTAL FINDINGS ARE COMMON
Abnormalities are reported in up to ~94% of scans; most are benign and may prompt additional testing.

LOW CANCER DETECTION RATE IN AVERAGE-RISK POPULATIONS
Cancers are detected in ~1–2% of asymptomatic, average-risk individuals.

DOWNSTREAM TESTING AND PROCEDURES
Up to ~30% may undergo further imaging, biopsies, or referrals—leading to cost, anxiety, and potential complications.

COST AND ACCESS
Higher upfront cost and limited insurance coverage for screening in average-risk individuals.

UNCERTAIN IMPACT ON OUTCOMES
No randomized trials showing reduced cancer mortality in average-risk populations; risk of overdiagnosis.

KEY TAKEAWAY

WB-MRI is a powerful, radiation-free tool that can detect a broad range of cancers, **INCLUDING MANY WITHOUT A USUAL SCREENING PATHWAY.**

Greatest evidence of benefit is in high-risk populations (e.g., Li-Fraumeni syndrome).
In average-risk individuals, it detects cancers but has unproven impact on long-term outcomes.

Evidence sources: Systematic reviews and meta-analyses, 2016–2025; incl. JAMA Oncology 2019 (TP53/Li-Fraumeni), European Radiology 2024, Radiology Advances 2026, and Tools for Practice 2026.

Precision Cancer Screening & Emerging Testing

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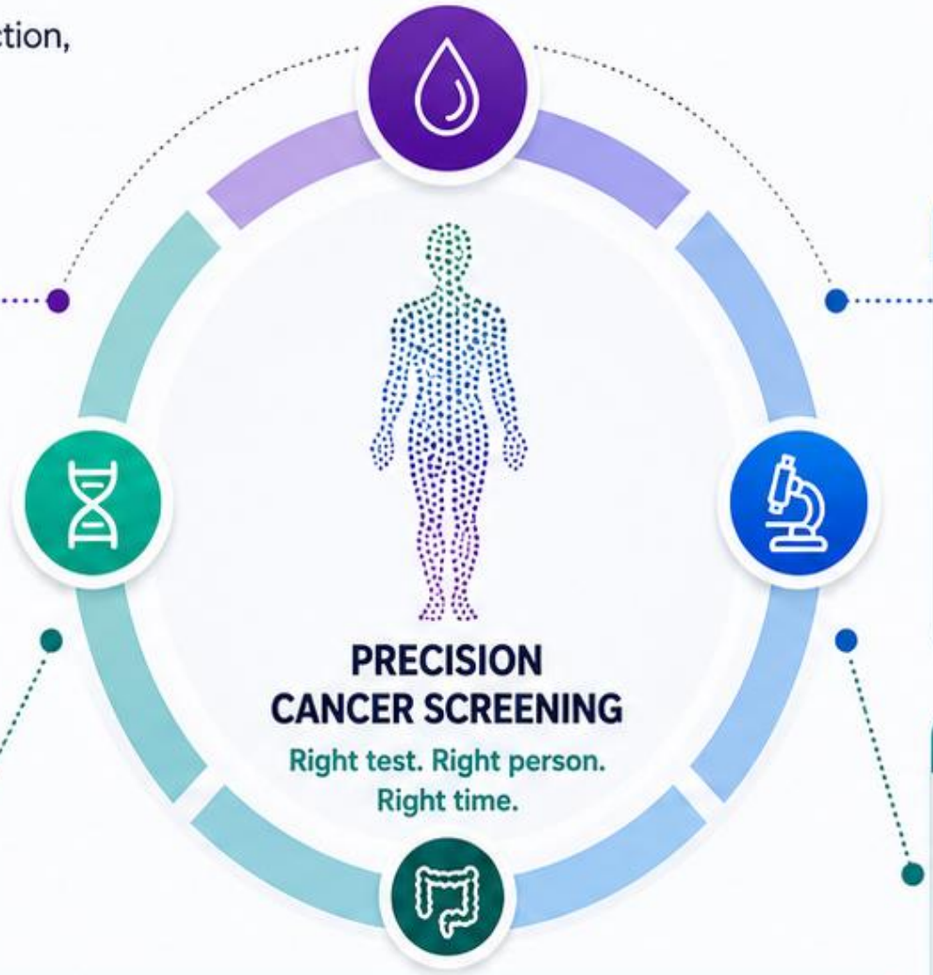
The next generation of tests expanding early detection, risk assessment, and personalized care.



MULTI-CANCER EARLY DETECTION (MCED) BLOOD TESTS

GRAIL — Galleri® test – designed to detect a signal shared by many cancers

cancer seek® — AI-powered assay to detect multiple cancers and predict tissue of origin

one test™ — OneTest® – multi-omics blood test using machine learning

GENETIC RISK & HEREDITARY TESTING

INVITAE® — Comprehensive hereditary cancer gene testing (200+ genes)

SIGNATARA™ — Tumor-informed MRD testing and recurrence monitoring

TISSUE-BASED & PROTEOMIC TESTING

CANCER CHECK LABS® — 8-protein blood test providing risk score for 10 common cancers

SIGNATARA™ — Post-treatment MRD testing to detect minimal residual disease and monitor recurrence

COLORECTAL CANCER SCREENING

GUARANT 360® — AI-powered platform integrating personal, family, and clinical data to estimate risk and recommend screening pathways

cologuard® PLUS® — Non-invasive stool DNA test detecting DNA markers and blood for colorectial cancer

ADDITIONAL EMERGING & INNOVATIVE TESTS

Thrive	Natera® Empower™	Lucence INSIGHT®	Freenome®	Delfi Diagnostics
MCED blood test	MCED blood test	MCED blood test	MCED blood test	Blood-based protein signature

Why It Matters

Detect earlier
Identify cancer sooner, when intervention matters most

Personalize risk
Tailor screening based on genetics, biology, lifestyle, and family history

Improve outcomes
Enable proactive care, reduce late-stage diagnosis

Integrate & empower
Combine data and testing to support shared decision-making and ongoing monitoring

Colon Cancer Screening: Where We Are and the Opportunity Ahead

Earlier screening. Higher participation. Better outcomes.

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WHEN TO START SCREENING

BRITISH COLUMBIA

Screening starts at

50 years old

UNITED STATES

Screening starts at

45 years old

SCREENING PARTICIPATION IN CANADA

Only about 50% of eligible Canadians are up to date with recommended colorectal cancer screening.

THE OPPORTUNITY

1 in 2 eligible Canadians are not getting screened on schedule.

Improving participation and starting screening at the right time **saves lives**.

A system-level opportunity to **prevent more cancers** and reduce long-term health costs.

Our Goal: Close the Gap. Save More Lives.

Through earlier screening, higher uptake, and personalized prevention.

Sources: Statistics Canada 2024 (FIT/FOBT within 2 yrs or colonoscopy within 10 yrs); Canadian Partnership Against Cancer 2023; Springer 2021.

Alzheimer's Disease Risk Assessment

APOE Genotyping & Biomarkers

APOE ε4/ε4 Genotype

Individuals with two APOE ε4 alleles (ε4/ε4) have an approximately

8–12×

INCREASED RELATIVE RISK

of late-onset Alzheimer's disease compared with APOE3/3.

Risk is influenced by age, sex, ethnicity, and lifestyle and health factors.

KEY POINTS

- APOE ε4 is the strongest common genetic risk factor for late-onset Alzheimer's disease.
- APOE status is a susceptibility marker, not deterministic.
- Many individuals with APOE ε4 never develop Alzheimer's.
- Clinical risk is modified by multiple factors, including vascular, metabolic, inflammatory, and lifestyle factors.

COMPLEMENTARY BIOMARKERS

- Amyloid PET
- Plasma p-tau
- Aβ42/40 ratio
- CSF biomarkers (Aβ42, p-tau, t-tau)
- Neurofilament light (NfL)

APOE GENOTYPE FREQUENCY IN THE GENERAL POPULATION

Genotype	E2/E2	E2/E3	E3/E3	E2/E4	E3/E4	E4/E4
Approximate Population Frequency	1%	10–12%	55–65%	1–3%	20–25%	2–5%

APOE4 increases risk — it does NOT determine destiny.
Integrate genetics with biomarkers and clinical context for personalized risk assessment.

References:
Farrer LA, et al. Neurology. 1997;48:912-913.
Corder EH, et al. Nat Genet. 1993;3:160-165.

The Potential of Next-Generation Testing

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Deeper insights. Earlier detection. Personalized action.

Multi-omics testing evaluates biology at a system level—integrating genomics, proteomics, metabolomics, and microbiomics to uncover root causes, identify risk earlier, and guide precision prevention.

Multi-omics. One comprehensive view.

GENOMICS

Clinic-Ready

Inherited risk, mutation detection, and pathway insights for proactive prevention.

Examples: APOE, MTHFR, CYP variants, cancer risk panels

PROTEOMICS

Emerging

Protein signatures reveal early changes in aging, organ function, and disease risk.

Examples: Plasma protein panels, aging clocks, organ-specific decline signatures

METABOLOMICS

Emerging

Metabolic phenotype reflects real-time physiology and response to lifestyle.

Examples: Energy metabolism, oxidative stress-specific decliral function

MICROBIOMICS

Exploratory

Microbiome composition impacts immunity, metabolism, and inflammation.

Examples: 16S rRNA, shotgun sequencing, microbial diversity

Why it matters

Earlier detection
Identify biological changes before symptoms appear.

Root cause insight
Move beyond symptoms to understand why.

Personalized prevention
Tailor strategies to your unique biology for better outcomes.

Long-term impact
Proactive care today reduces disease risk and future healthcare costs.

The Opportunity

Harnessing multi-omics data empowers truly personalized, preemptive, and precision healthcare.

~50% of eligible Canadians are not up to date with recommended colorectal cancer screening.*

Advanced testing + better participation = more lives saved.

Example: Molecular You

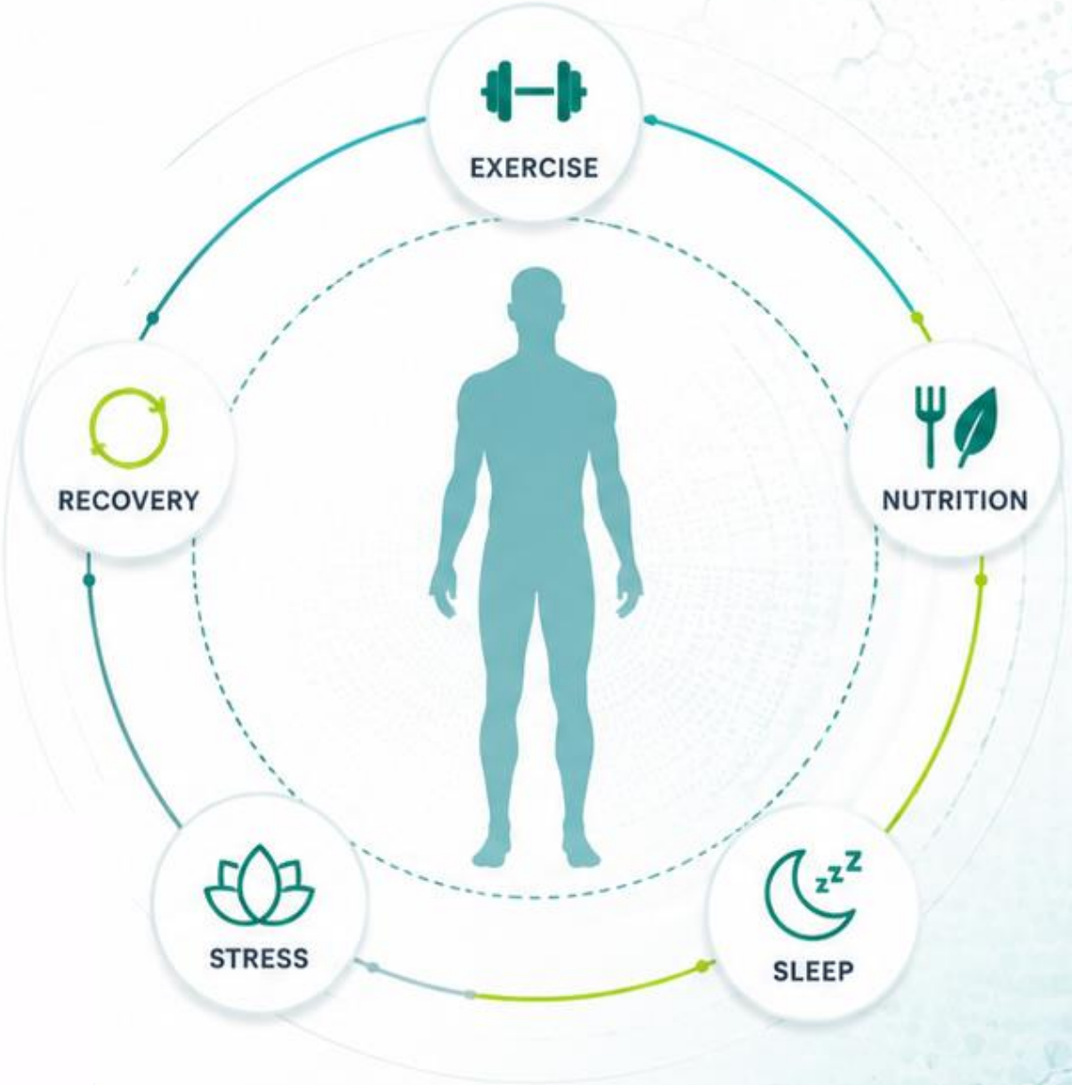
A comprehensive blood-based test that measures hundreds of biomarkers across multiple biological pathways—one test, one tube, powerful insights.

- Simple blood collection
- Comprehensive biomarker panel
- Actionable results
- Personalized care

*Sources: Statistics Canada 2024; Canadian Partnership Against Cancer 2023; Springer 2021.

LIFESTYLE OPTIMIZATION

Quantify. Personalize. Optimize.
Small changes. Big impact.



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Lifestyle Optimization — quantified, benchmarked and tracked

The highest-evidence interventions for disease prevention and longevity are lifestyle.
How can we be more personalized and enable behavior change.

Exercise

Measure it.

Nutrition

Measure it.

Sleep

Measure it.

Stress

Measure it.

Recovery

Measure it.

• THE MEASUREMENT STACK •

WEARABLES

HRV · sleep · recovery · activity

CGM

Glucose response to food, sleep, stress

TRACKERS

Food logs · training load · symptoms

AI COACHING

Pattern recognition, contextual nudges

Real time feedback enables behavior change. *Wearables, CGM, and HRV move the foundational levers into N=1 experiments.*

EVIDENCE

The Biggest Modifiable Drivers of Longevity

Lifestyle factors have the largest impact on survival

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WHAT ACTUALLY MOVES THE CURVE

Effect sizes from large peer-reviewed cohort studies on all-cause mortality reduction.

These are not marketing claims.

LANDMARK FINDING

5x

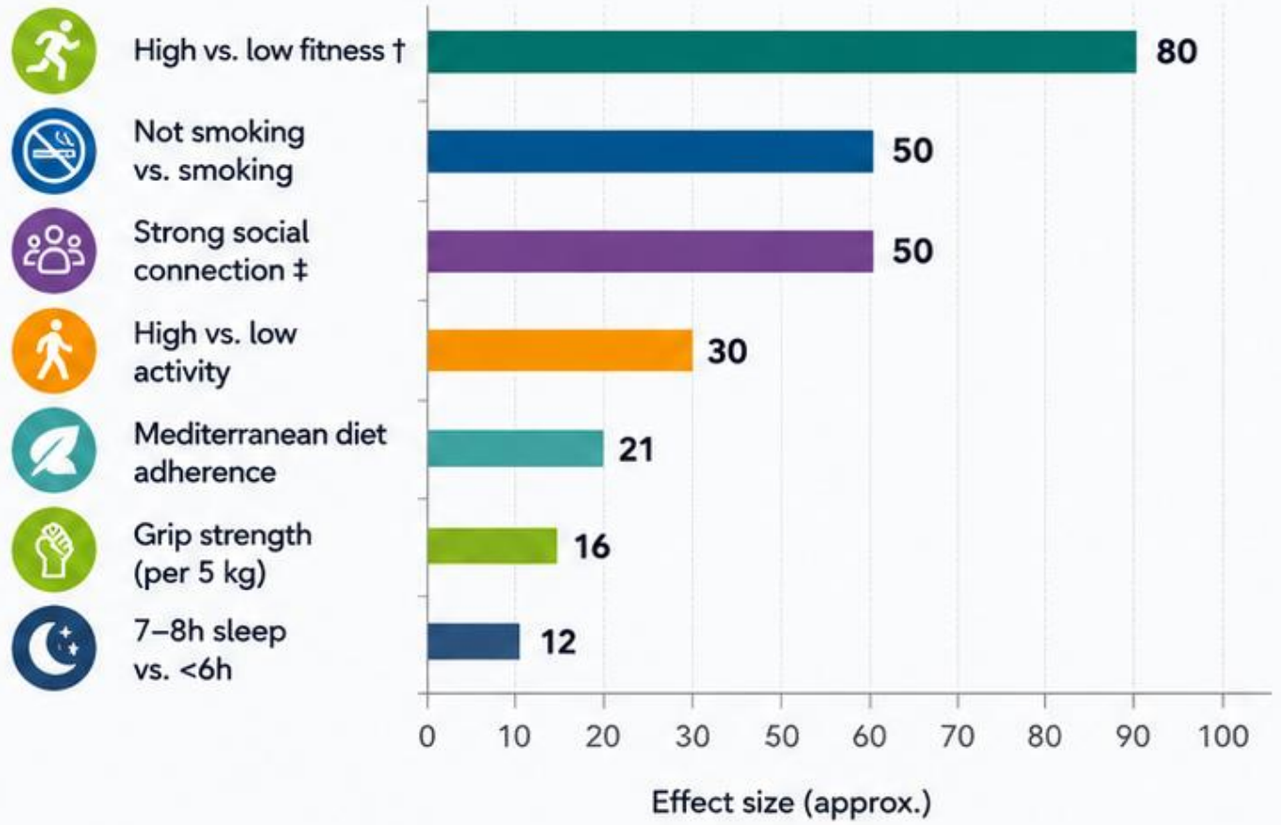
Low cardiorespiratory fitness carries ~5x the all-cause mortality risk of high fitness — comparable to or greater than smoking, diabetes, and CAD.

Mandsager et al., JAMA Network Open 2018 (n = 122,007)

Reported figures are typical high-vs-low contrasts from major cohort and meta-analytic studies; effect sizes vary by population and design.

EFFECT ON MORTALITY / SURVIVAL

Approximate effect sizes from peer-reviewed cohort and meta-analytic studies (observational)



Fitness is foundational
Low fitness is the strongest modifiable predictor of mortality.

Connections matter
Strong social connection is as impactful as not smoking.

Daily habits add up
Movement, nutrition, strength, and sleep all contribute meaningfully.

Small changes, big outcomes
Sustainable lifestyle changes yield the greatest long-term benefit.

† *High vs. low fitness = **the same Mandsager finding as the 5× callout** (elite vs. low; aHR 0.20). Bars mix hazard and odds ratios — read as magnitude, not exact rank.*

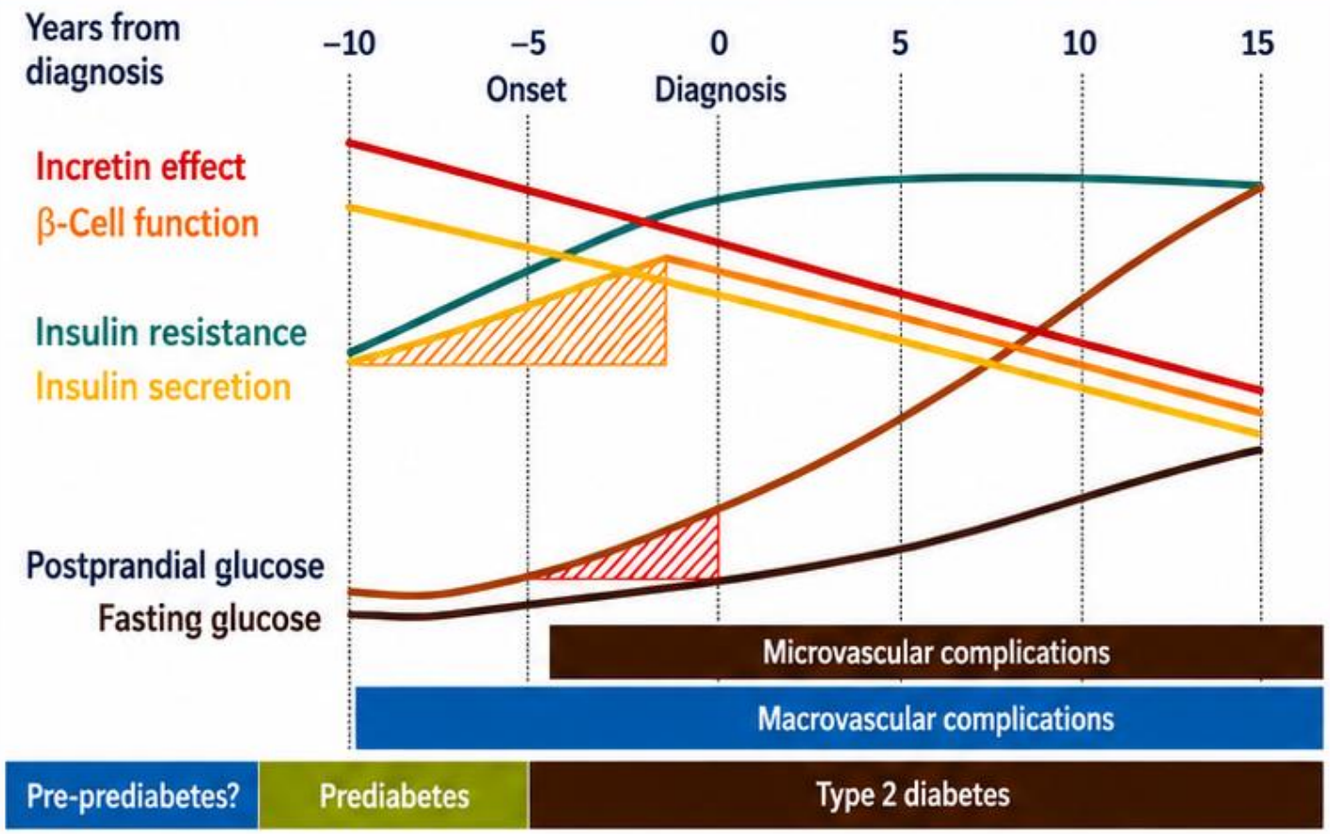
‡ *Social connection = **OR 1.50 for survival** (Holt-Lunstad 2010), an odds ratio.*

LIFESTYLE IS MEDICINE. *The choices we make every day shape how long — and how well — we live.*

Metabolic Dysregulation Before Hyperglycemia

Insulin resistance and compensatory hyperinsulinemia may precede abnormal glucose, A1C, and OGTT findings

THE NATURAL HISTORY OF TYPE 2 DIABETES



EVIDENCE: EARLY INSULIN RESISTANCE MATTERS

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openheart

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EDITORIAL ▸ Open Heart. 2017 Nov 27;4(2):e000656. doi: 10.1136/openhrt-2017-000656

Postprandial insulin assay as the earliest biomarker for diagnosing pre-diabetes, type 2 diabetes and increased cardiovascular risk

James J DiNicolantonio [1], Jaikrit Bhutani [2], James H O'Keefe [1], Catherine Crofts [3]

▸ Author information ▸ Article notes ▸ Copyright and License information

PMCID: PMC5708305 PMID: 29225902

WHY EARLY DETECTION IS CRITICAL

1. Insulin resistance precedes hyperglycemia

Metabolic dysfunction may emerge years before glucose-based markers become abnormal.

2. Compensatory insulin secretion rises early

Hyperinsulinemia can maintain normal glucose while metabolic risk progresses.

3. Earlier identification enables prevention

Detecting risk earlier creates a wider window for lifestyle and therapeutic intervention.

4. Glucose alone may miss early risk

Normal fasting glucose, A1C, or OGTT may not exclude underlying insulin resistance.

ASSESS METABOLIC RISK BEFORE DYSGLYCEMIA IS APPARENT.

Detect insulin resistance before glucose-based markers become abnormal.

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DiNicolantonio JJ, Bhutani J, O'Keefe JH, Crofts C. *Open Heart*. 2017;4(2):e000656. doi: 10.1136/openhrt-2017-000656.

EARLY DETECTION OF METABOLIC DYSREGULATION
KEY TO PREVENTING PROGRESSION TO DIABETES

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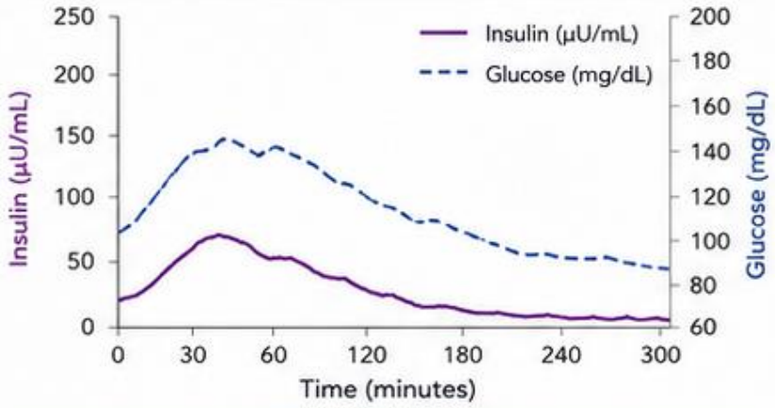
Insulin resistance and β-cell dysfunction develop gradually. **Early detection enables early intervention.**

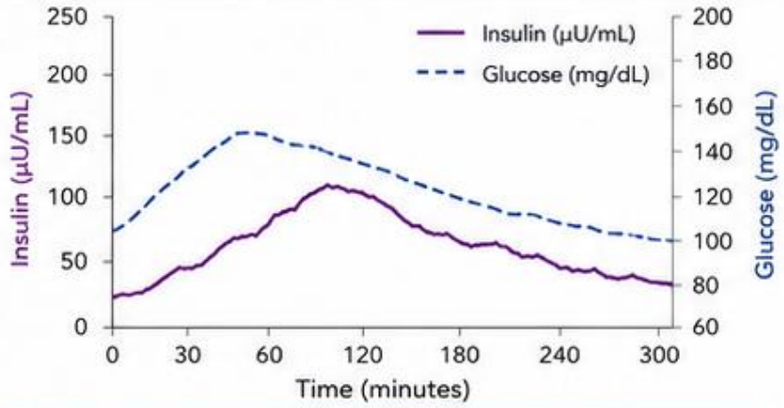
INSULIN RESPONSE PATTERNS DURING 75 g ORAL GLUCOSE TOLERANCE TEST (OGTT)

PATTERN I – NORMAL



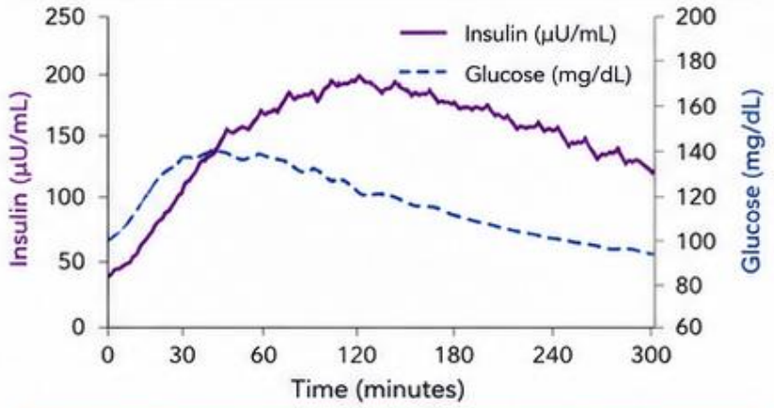
Early insulin peak (30–60 min) with return to baseline by 2–3 hours. Normal insulin sensitivity.

PATTERN II – DELAYED HYPERINSULINEMIA



Delayed insulin peak (90–120 min) with prolonged elevation at 2–3 hours. Early insulin resistance.

PATTERN III – SEVERE HYPERINSULINEMIA



Markedly elevated and delayed insulin response (peaks 120–180 min) with prolonged elevation through 5 hours. Glucose may be normal.

These patterns reflect the body's ability to secrete insulin and maintain glucose homeostasis after a glucose load.
Progression from **Pattern I → II → III** increases risk for impaired glucose tolerance and type 2 diabetes.

CONTINUOUS GLUCOSE MONITORING (CGM): EARLY INSIGHT, BETTER OUTCOMES

DETECT EARLY DYSREGULATION
Identify glucose patterns and variability before lab abnormalities appear.

INTERVENE EARLY
Address insulin resistance and lifestyle factors to restore metabolic health.

PREVENT PROGRESSION
Reduce risk of impaired glucose tolerance, type 2 diabetes, and cardiovascular disease.

PERSONALIZE CARE
CGM data empowers tailored nutrition, exercise, and behavioral strategies.

EARLY DETECTION TODAY. BETTER HEALTH TOMORROW.

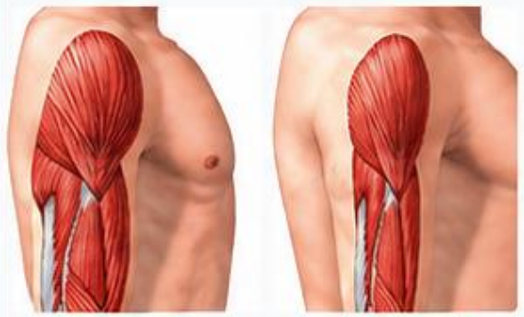
Integrating OGTT insights with CGM technology enables precision prevention and optimal metabolic health.

Kraft JR. Insulin response patterns. JAMA. 1970;212(5):931–933. Conceptual representation for educational purposes.

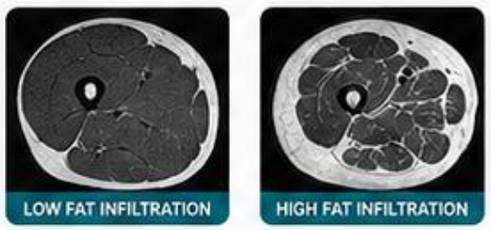
Body Composition & Musculoskeletal Health in Aging

Muscle mass, muscle quality, visceral fat, and bone density are modifiable determinants of healthspan.

1 Muscle Mass & Strength

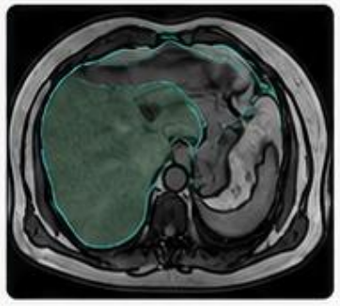


- Sarcopenia: age-related loss of muscle mass and strength
- 1–2% loss of muscle mass per year after age 50
- Infiltration of fat within muscle reduces quality and function



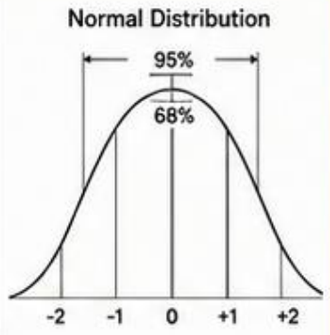
2 Visceral Fat & Metabolic Risk

- High visceral adipose tissue is an independent risk marker for cardiometabolic morbidity and mortality
- Assess with DXA, BIA, and MRI for deeper insight



Visceral adipose tissue (shaded) surrounds internal organs and predicts risk.

3 Bone Density & Fracture Risk



- Low bone density increases risk of falls and fractures, especially in older age
- Bone loss accelerates with advancing age and is a major driver of morbidity
- Early detection enables intervention and fracture-risk reduction

Fractures are a leading cause of loss of independence, disability, and mortality in older adults.

4 Muscle Health Testing

- Comprehensive, non-invasive testing across key domains
- Track changes over time and guide personalized interventions
- Actionable insights drive better outcomes

Functional reserve → healthspan

Mobility
Maintain strength and physical function

Metabolism
Improve metabolic health and disease resistance

Falls/Fractures
Reduce risk of falls and fractures

Independence
Preserve autonomy and quality of life

Better metrics. Earlier insight. Targeted action. Optimize body composition and musculoskeletal health to extend healthspan.

Wearables:
Your Health. Connected.

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Leverage advanced wearable technology and intelligent insights to optimize your health, performance, and longevity.

Track What Matters

- Heart Rate & HRV
- Sleep Stages & Quality
- Activity & Movement
- Recovery & Stress
- Nutrition & Metabolism
- Blood Glucose (CGM)
- VO_2 Max & Performance
- Functional Aging Metrics

Sleep Tracking for Real-time Feedback

Smart Plans. Smarter You.

Personalized tracking and AI-powered coaching

AI Coaching
Get personalized insights and actionable guidance.

Nutrition Tracking
Log meals, track macros, and optimize your fueling.

Performance Metrics
Monitor VO_2 max, grip strength, movement, and more.

Top Wearable Partners

OURA | WHOOP | APPLE WATCH

Integrated. Intelligent. Impactful.

Built for Your Life.

- Seamless data integration
- Real-time feedback
- Long-term trend tracking
- Empowers daily decisions
- Supports longevity & functional aging

Clinical Evidence for Wearable Health Technologies

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High-quality research consistently demonstrates that wearable technology can improve physical activity, health behaviors, and clinical outcomes across diverse populations and settings.

1 The Lancet Digital Health (2022)

Effectiveness of wearable activity trackers to increase physical activity and improve health: a systematic review of systematic reviews and meta-analyses

Umbrella review of reviews across healthy and clinical populations

Key Findings

- Increased daily steps
- Increased moderate-to-vigorous physical activity
- Reduced sedentary behavior

thelancet.com/journals/landig/article/PIIS2589-75002200111-X/fulltext

2 JMIR Publications (2025)

Wearable Activity Tracker–Based Interventions for Physical Activity, Body Composition, and Physical Function Among Community-Dwelling Older Adults: Systematic Review and Meta-Analysis of Randomized Controlled Trials

23 randomized controlled trials in older adults

Key Findings

- Significantly increased physical activity time
- Significantly increased daily step count
- Effective for improving activity behavior

jmir.org/2025/1/e59507

3 JAMA Network Open (2023)

Interventions Using Wearable Activity Trackers to Improve Patient Physical Activity and Other Outcomes in Adults Who Are Hospitalized: A Systematic Review and Meta-analysis

Hospitalized adults in systematic review/meta-analysis

Key Findings

- Higher physical activity
- Less sedentary time
- Better physical function during recovery
- Compared with usual care

jamanetwork.com/journals/jamanetworkopen/fullarticle/2806101

4 Journal of Medical Internet Research (2022)

The Influence of Wearables on Health Care Outcomes in Chronic Disease: Systematic Review

Chronic disease populations (e.g., diabetes, CVD)

Key Findings

- Improved self-management
- Improved adherence
- Increased engagement
- Improved clinical outcomes (e.g., glucose monitoring adherence, exercise compliance, remote monitoring)

pmc.ncbi.nlm.nih.gov/articles/PMC9288104/

THE TAKEAWAY

High-quality evidence across diverse populations consistently shows that wearable technology drives meaningful behavior change and improves health outcomes.

Precision Nutrition in Clinical Practice

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A data-informed workflow for individualized nutrition prescriptions in clinic

1 ASSESS

Patient-specific inputs

Labs and risk
metabolic health; lipids; cardiac risk; ApoE

Body composition
muscle mass; visceral fat; overall body composition

Dietary adequacy
protein; fiber; key micronutrients; amino acids

Behavioral context
intake tracking; timing patterns; nervous system state

2 STRATIFY

Nutrition priority

Nourishment x Muscle Mass

NOURISHMENT \ MUSCLE MASS	Under-muscled	Adequately muscled
Overnourished	Overnourished Under-muscled	Overnourished Adequately muscled
Adequately nourished	Adequately nourished Under-muscled	Adequately nourished Adequately muscled

Translate findings into priorities
Replete • Reduce • Build • Maintain

3 PRESCRIBE

Select nutrition levers

CR
Energy dose
caloric restriction when indicated

DR
Diet composition
food quality and restrictions

TR
Meal timing
time restriction and window

HOW
Implementation
nervous system, adherence

Output: individualized plan
Targets, sequence, metrics, follow-up.

4 TRACK AND ADJUST OVER TIME

Close the loop by comparing the prescription with follow-up biomarkers, body composition, intake data, symptoms, and adherence.

Follow-up labs

Lipids + cardiac risk

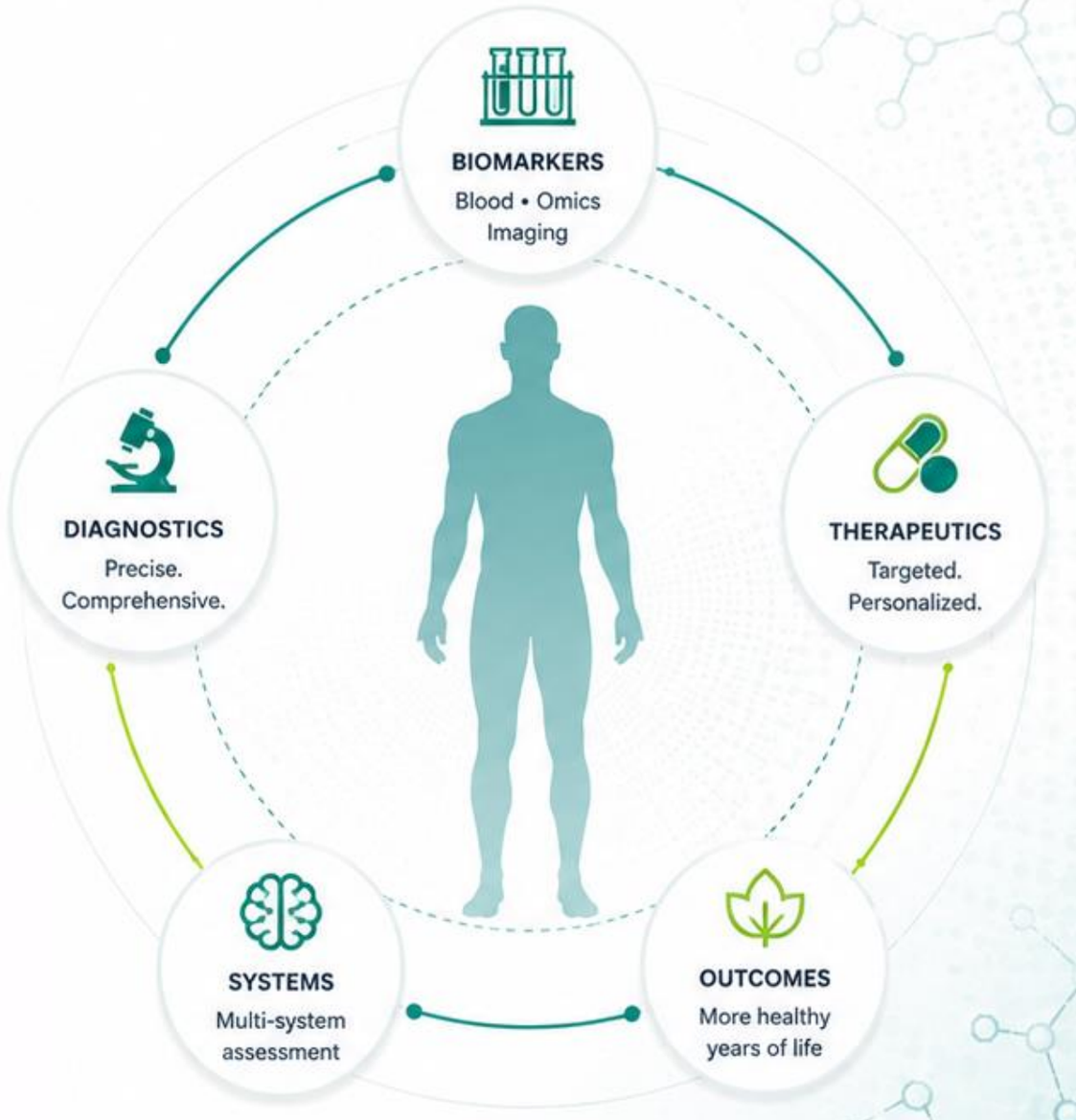
Body composition

Cronometer intake

Symptoms + adherence

LONGEVITY DIAGNOSTICS AND THERAPEUTICS

Advanced diagnostics. Targeted therapeutics.
Extend healthspan.



CONNECT
HEALTH

What is biological age?

Two people can share a birth year and be biologically decades apart.
Chronological age counts the calendar; biological age measures how well the body is actually functioning.

AGING SLOWER

Chronological age **60**

Biological age

50 *years*

Functions like a typical 50-year-old across cardiovascular, immune, muscular, and cognitive systems.

SAME BIRTH YEAR

AGING FASTER

Chronological age **60**

Biological age

70 *years*

Resembles the cellular and physiological decline of a typical 70-year-old despite the same birth year.

The verdict: *biological age is not years on the calendar—it is a measure of how well the body is actually functioning.*

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How we measure biological age

No single clock is definitive. We estimate biological age from several validated clocks, then reconcile them into one interpretable result.

THE INPUTS — AGING CLOCKS

Epigenetic clocks

DNA methylation marks that change predictably with age.

GrimAge2 · DNAm PhenoAge · OMICmAge · SYMPHONYAge

Blood-based clocks

Patterns in plasma proteins and routine clinical biomarkers.

PhenoAge (clinical) · proteomic & metabolomic aging scores

Each clock gives an age estimate

OUR APPROACH

Cross-check for consensus

Look for agreement across multiple clocks before trusting a signal.

Reconcile into one score

Combine the reliable estimates into a single, interpretable biological age.

The result: *a more reliable biological-age estimate than any single test — and the caveat that these are* ***trend tools, not one-time verdicts****.*

Functional aging measures

Biological age clocks get the attention. Functional measures get the predictions.

VO_2 MAX

Gold standard

Cardiorespiratory fitness · single best mortality predictor

GRIP STRENGTH

PURE study

Each 5 kg decrement = 16% ↑ all-cause mortality risk

GAIT SPEED

>1.0 m/s

Below 0.8 m/s in older adults predicts mortality and falls

SIT-TO-STAND

Function

Lower-body strength, balance, and coordination in one test

DEXA LEAN MASS

Sarcopenia

Muscle quantity vs. fat — body composition over BMI

HRV (rMSSD)

Autonomic

Parasympathetic tone, recovery capacity, stress resilience

FRONTIER

Potential gerotherapeutic targets

A gerotherapeutic targets a **biological mechanism of aging itself** — rather than any single disease — to slow aging and extend *healthspan*. Acting upstream on shared aging biology, one agent may delay multiple age-related conditions at once.

Evidence ranges from animal lifespan data to early human trials — none is established for healthy-lifespan extension in people.

01 **Rapamycin / rapalogs**
mTOR inhibition; robust animal lifespan data

02 **Metformin**
AMPK; human TAME trial ongoing

03 **Menopausal HRT**
Estrogen, appropriately timed near menopause

04 **GLP-1 receptor agonists**
Cardiometabolic outcomes; aging data emerging

05 **SGLT2 inhibitors**
Mouse lifespan + senolytic activity

06 **Acarbose**
Glucose control; mouse lifespan (ITP)

07 **Senolytics**
D+Q, fisetin; early human trials

08 **Testosterone replacement**
Hypogonadal men only; not anti-aging

09 **NAD$^+$ precursors**
NMN, NR; largely preclinical

10 **Spermidine**
Autophagy; largely preclinical

The digital platform — PALM – Collaboration with Polymorphic Biosciences.

None of this works at scale without an aggregation layer.

INPUTS

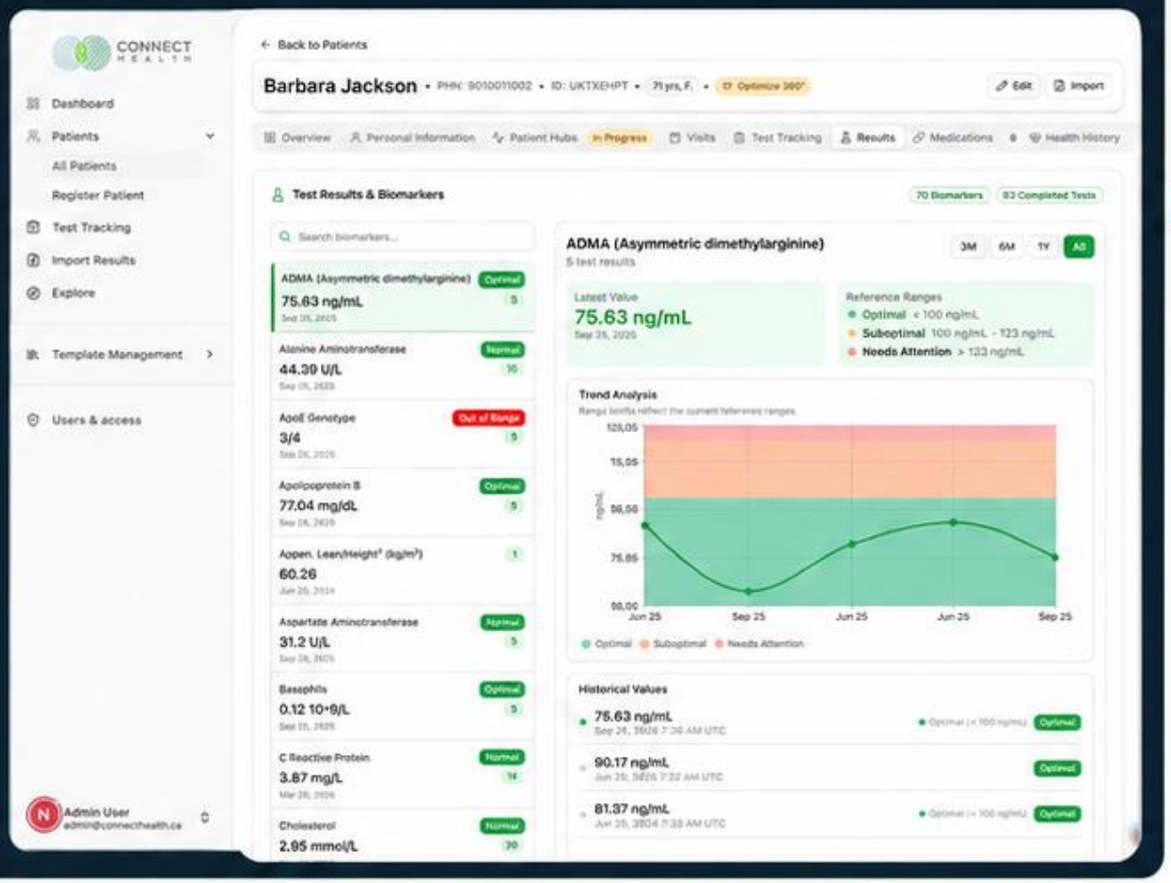
Data sources

- Labs · omics · imaging
- Wearables · CGM · HRV
- Functional measures
- Patient-reported outcomes

ENGINE

Aggregation · AI · longitudinal modeling

- Unified longitudinal record
- AI pattern recognition
- Cross-modality trends
- Clinical decision support



OUTPUTS

Clinical workflow

- Personalized dashboards
- Risk stratification
- Intervention guidance
- Outcome tracking

The shift: from a folder of disconnected reports to a continuously updated, longitudinal, clinically actionable record.

CONNECT HEALTH | Polymorphic Biosciences

APPROACH

Precision medicine — N of 1, not N of average

Precision medicine—using the individual's biology, genetics, environment and longitudinal data to choose what works specifically for them.

POPULATION MEDICINE

- Treats the average patient
- Guidelines from RCT means
- Reactive: starts at diagnosis
- Same protocol for similar phenotypes

PRECISION MEDICINE

- Treats this patient
- Uses genomics, biomarkers, omics
- Proactive: starts pre-symptomatic
- Protocols adjusted to N=1 response

WHAT IT REQUIRES

- Advanced diagnostics
- Longitudinal data infrastructure
- Deep phenotyping
- Time, context, judgment

THE GOAL: increase years alive (*lifespan*) while extending the years lived with health and vitality (*healthspan*).

SAFER PRESCRIBING

Pharmacogenomics — the case for testing once

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THE PROBLEM IN CANADA

4th

leading cause of death in Canada — adverse drug reactions

10,000–22,000 deaths per year

~200,000 severe ADRs requiring hospitalization

~$13.7B annual healthcare system cost

PREPARE trial (Lancet 2023): pre-emptive PGx panel reduced clinically relevant ADRs by 30% — potentially 3,000–6,600 Canadian deaths/year preventable.

POTENTIAL CLINICAL UTILITY

A one-time pre-emptive PGx panel can guide prescribing across a lifetime of medications.

COMMON MEDICATIONS WITH PGx-GUIDED RECOMMENDATIONS

- **Clopidogrel** — CYP2C19: efficacy after stenting
- **Warfarin** — CYP2C9/VKORC1: bleeding risk
- **Simvastatin** — SLCO1B1: myopathy risk
- **SSRIs/TCAs** — CYP2D6/2C19: efficacy & side effects
- **Codeine/tramadol** — CYP2D6: efficacy & toxicity
- **Abacavir** — HLA-B*57:01: hypersensitivity

CPIC guidelines cover >100 drug–gene pairs

PHARMACOGENOMICS CAN SUPPORT SAFER, MORE PRECISE PRESCRIBING.

One-time pre-emptive testing may inform medication choice and dosing across future prescribing decisions.

CONNECT HEALTH

LONGEVITY 100 PROGRAM

A structured clinical framework for optimizing healthspan and evaluating emerging longevity therapies in a medical setting—delivering evidence-informed, science-driven care.

WHAT WE MEASURE

Multi-omic phenotyping

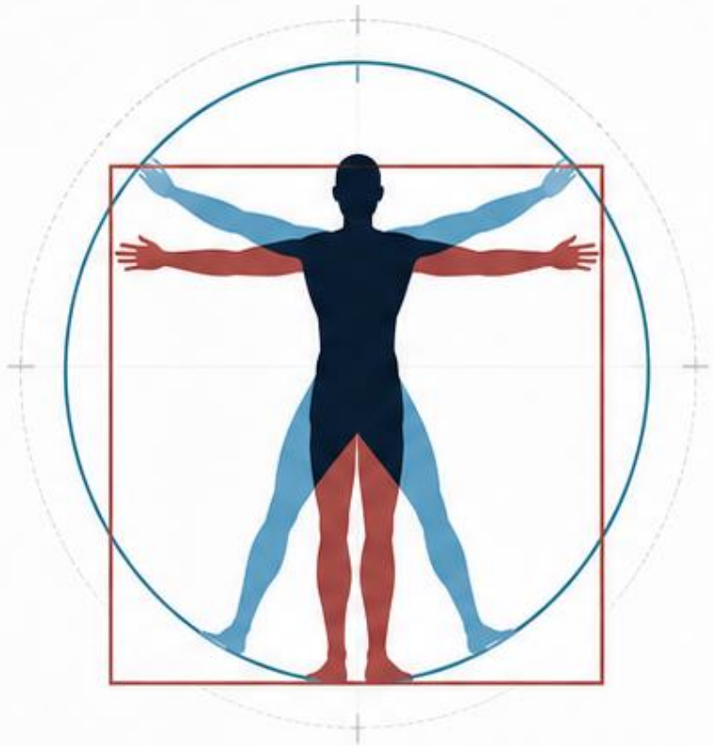
Aging clocks · genome · proteome · metabolome · microbiome

Deep clinical phenotyping

Organ function · biochemistry · body composition · VO_2 max · whole-body MRI

Digital health tracking

Continuous glucose · sleep analysis · fitness & recovery tracking



THE PATIENT

WHAT WE DO

Lifestyle optimization

Personalized nutrition, sleep, exercise & stress-resilience programs

Gerotherapeutics

Supplements · repurposed medications · hormones

Psychological & social

Stress management · social connection · purpose

N = 1 Personalization:

1. **Early Detection**
2. **Lifestyle Optimization**
3. **Potential healthspan strategies**

CONNECT HEALTH

Thank You

PARTNERING FOR A LONGER, HEALTHIER FUTURE.

At Connect Health, we combine cutting-edge science with the principles of functional and integrative medicine to deliver **optimized health, vitality, and longevity.**

We focus on **impact** and **efficiency**—helping you live a long, healthy life that is **rewarding** and feels **effortless.**

Our mission is to transform healthcare into a proactive, preventative, and systems-oriented model that prioritizes **root-cause resolution** and **whole-person health.**

> **"Always imagine *your future to be bigger than your past"***
>
> *– Dan Sullivan*

Presentation Materials
Scan the QR code to access today's slides and resources.

Dr. Lawrence Cheng
MD, CCFP (EM), MPH
Medical Director
& Co-Founder

*A unique blend of conventional expertise and integrative insight to set a new standard in **personalized medicine for healthy aging.***

Dr. Ashley Riskin
MD, BSc, CCFP
Clinical Director
& Co-Founder

Better data. Deeper insight. Personalized care. Lasting impact.

Presentation materials