Models of Aging, Rapamycin Trials & Biological Clocks

A concise education deck: what aging models explain, what trials are testing, and how clocks should be interpreted.

Dr Rob Health - Longevity Education Series

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1. Models of Aging

Different models are useful for different questions - no single model explains everything.

Damage accumulation

DNA damage, telomere shortening, protein misfolding, mitochondrial stress and senescent-cell build-up gradually reduce resilience.

Hallmarks framework

The 2023 Hallmarks update organises aging into 12 connected biological processes, including inflammation and dysbiosis.

Systems model

Aging behaves like a network: metabolism, immunity, tissue repair, microbiome and environment push each other up or down.

Programmed/adaptive views

Some age-related changes may be regulated responses, but this does not prove aging is intentionally programmed.

AI/clock model

Machine learning estimates biological age from biomarkers, but prediction is not the same as proving causality.

2. What Interventions Try to Target

Most longevity ideas map to one or more aging pathways.

- Nutrient sensing: mTOR, AMPK, insulin/IGF-1; diet, exercise and rapalogs often sit here.
- Proteostasis/autophagy: Cell clean-up systems; heat shock proteins and autophagy are key repair pathways.
- Inflammation & senescence: Chronic low-grade inflammation and senescent cells can damage tissue signalling.
- Mitochondria & metabolism: Energy production, oxidative stress, muscle function and metabolic flexibility.
- Microbiome & dysbiosis: Gut ecology can influence immune tone, metabolites and inflammation.

3. Rapamycin Trials: Promise and Caution

Rapamycin inhibits mTOR; animal data are strong, human longevity proof is still incomplete.

- Why the interest: mTOR inhibition is linked to nutrient sensing, autophagy and lifespan extension in multiple animal models.
- Human reality: approved uses are medical, not general anti-aging; off-label longevity use remains uncertain.
- Trials are now more serious: 2026 National Institute on Aging-funded work at UT Health San Antonio is testing dosing, biology, safety and long-term outcomes in older adults.
- What to watch: immune effects, mouth ulcers, lipids/glucose, wound healing, infections, drug interactions and dose schedule.
- Best message: biologically plausible is not the same as clinically proven.

4. Biological Clocks

Useful for research and trend tracking - risky when marketed as a single “true age”.

Epigenetic clocks

DNA methylation patterns estimate biological age or pace of aging. Newer clocks often relate more strongly to disease risk.

Multi-omic clocks

Proteomic, metabolomic, immune, microbiome and imaging clocks can capture different biology.

Interpretation limits

A clock can move after an intervention, but that does not automatically prove longer life or better health.

- Good use: compare trends over time with the same method and context.
- Weak use: compare different commercial tests as if they measure the same thing.
- Clinical gap: clocks are promising research tools, but not yet a replacement for standard risk assessment.

5. Practical Takeaways

Practical summary and selected sources

- Use aging models as maps, not as proof.
- Rapamycin deserves serious study, but routine anti-aging use remains unproven.
- Biological clocks are useful signals, not final verdicts.
- Focus first on proven basics: exercise, sleep, nutrition, metabolic health and medical supervision.

Selected references

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5. Min M. et al. Critical review of aging clocks. Frontiers in Aging, 2024.