Core Assumptions

Three interacting subsystems:

$$i \in \{\text{Cardio, Musc, Neuro}\}.$$

Two variables per subsystem:

- **Functional health** $X_i(t) \in [0, 1]$: fast variable, real-time performance.
- **Structural damage** $D_i(t) \geq 0$: slow variable, accumulated aging.

External shocks $S_i(t)$**:** random events occurring at a constant rate, independent of age; their impact grows because damaged systems are more fragile.

Aging is endogenous: damage accelerates decay, weakens recovery, and increases shock propagation.

Three core mechanisms:

1. Damage-dependent drift of X_i (decay vs. recovery).
2. Random shocks that reduce function and raise damage.
3. Frailty coupling: shocks spread more as systems deteriorate.

Core Equations

Functional health dynamics

$$\underbrace{X_i(t+\Delta t)}_{\text{new function}} = \underbrace{X_i(t)}_{\text{current state}} \underbrace{- \delta_i(D_i)\, X_i(t)\, \Delta t}_{\text{damage-amplified decay}} \underbrace{- S_i(t)}_{\text{shock impact}} \underbrace{+ r_i(D_i)\big(X_{\max,i}(D_i) - X_i(t)\big)\Delta t}_{\text{recovery toward ceiling}} \underbrace{+ \varepsilon_i(t}_{\text{noise}}$$

Decay, recovery, and ceiling shrink with structural damage:

$$\delta_i(D_i) = \delta_{0,i}\big(1 + \beta_{\text{decay},i} D_i\big), \qquad r_i(D_i) = r_{0,i}\big(1 - \gamma_{\text{rec},i} D_i\big),$$
$$X_{\max,i}(D_i) = 1 - k_{\text{ceil},i} D_i.$$

Shock propagation (frailty amplification)

$$\boldsymbol{S}(t) = \boldsymbol{S}^{\text{local}}(t) + C(D)\, \boldsymbol{S}^{\text{local}}(t), \qquad C_{ij}(D) = C_{ij}^{\text{base}} \left(1 + \gamma_{\text{coup}} \frac{D_i + D_j}{2}\right).$$

Structural damage accumulation

$$\underbrace{D_i(t+\Delta t)}_{\text{new damage}} = \underbrace{D_i(t)}_{\text{current damage}} + \underbrace{\alpha_X(1 - X_i(t))\Delta t}_{\text{wear from low function}} + \underbrace{\beta_S\, S_i(t)\Delta t}_{\text{damage from shocks}} .$$

Healthspan / Lifespan

$$\text{Healthspan: } \frac{1}{3}\sum_i X_i(t) > X_{\text{functional}}, \qquad \text{Death when any } X_i(t) < X_{\text{death}}.$$

Exercise Intervention

Biological idea: lifestyle interventions (e.g. exercise) improve resilience but do not literally make shocks rarer. They act by:

- improving recovery of function after perturbation,
- slowing the rate at which low function accumulates structural damage.

Implementation in the model
For ages $t \geq t_{\text{exercise}}$:

$$r_i(D_i) \;\mapsto\; r_i^{(\text{ex})}(D_i) = r_i(D_i)\big(1 + \Delta r_{\text{ex}}\big),$$

$$\alpha_X \;\mapsto\; \alpha_X^{(\text{ex})} = \alpha_X\big(1 - \Delta\alpha_{\text{ex}}\big), \quad 0 < \Delta\alpha_{\text{ex}} < 1.$$

All other terms (shock statistics, coupling $C(D)$, baseline decay law) are unchanged.

Consequences:

- $X_i(t)$ stays closer to its damage-limited ceiling for longer.
- Structural damage $D_i(t)$ accumulates more slowly.
- In simulations: clear increase in **healthspan**, moderate effect on lifespan.

This captures the empirical intuition: exercise compresses morbidity but does not make the organism shock-proof.

Drug Intervention (Shock-Modulating)

Biological idea: pharmacological interventions can reduce the probability or severity of acute events (e.g. statins, anticoagulants, anti-arrhythmic drugs), without fundamentally changing slow aging drift.

Implementation in the model
For ages $t \geq t_{\text{drug}}$:

- *Shock probability and magnitude* are scaled:

$$S_i^{\text{local}}(t) \;\mapsto\; S_i^{\text{local,drug}}(t) = f_{\text{prob}}\, S_i^{\text{local}}(t), \qquad 0 < f_{\text{prob}} < 1,$$

  or equivalently by reducing both occurrence probability and mean size.

- *Shock-induced damage* is attenuated:

$$\beta_S \;\mapsto\; \beta_S^{(\text{drug})} = \beta_S \big(1 - \Delta\beta_{\text{drug}}\big).$$

The drift terms $\delta_i(D_i)$ and $r_i(D_i)$ are left unchanged.

Consequences:

- Fewer catastrophic drops of X_i and slower jump-like increases in D_i.
- Subsystems can survive longer at relatively low function.
- In simulations: more gain in **lifespan** than in healthspan (people stay alive longer, but not necessarily vigorous).

Replacement Intervention

Biological idea: replacement (e.g. organ transplant, engineered tissue, future rejuvenation surgery) directly resets the state of a subsystem instead of merely changing the rules of evolution.

Implementation as a discrete reset
At a chosen intervention time t_{repl}, for a set of subsystems $i \in \mathcal{R}$:

$$X_i(t_{\text{repl}}^+) = X_i^{(\text{repl})}, \qquad D_i(t_{\text{repl}}^+) = D_i^{(\text{repl})},$$

with $X_i^{(\text{repl})}$ close to 1 and $D_i^{(\text{repl})}$ close to a young-adult value.
Immediately this:

- raises the damage-limited ceiling $X_{\max,i}(D_i)$;
- lowers $\delta_i(D_i)$ and increases $r_i(D_i)$;
- weakens shock propagation from / to the rejuvenated subsystem (via lower D_i).

Interpretation:

- Exercise and drugs act on *process parameters* (slopes of trajectories).
- Replacement acts on the *state* (X_i, D_i) itself, effectively resetting local biological age.
- In the model this can improve both healthspan and lifespan in a qualitatively different way.