

A Nonlinear Ordinary Differential Equation Model for the Effects of Fasting on the Human Body System

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Abstract

Original Research Article

This paper develops and analyzes a nonlinear ordinary differential equation model for the metabolic dynamics of fasting. The model tracks five interacting state variables: blood glucose $G(t)$, glycogen reserve $Y(t)$, lipid/fat reserve $F(t)$, ketone bodies $K(t)$, and labile protein reserve $P(t)$. A fasting state vector $x(t)=(G,Y,F,K,P)^T$ is formulated from biological assumptions on glycogenolysis, gluconeogenesis, lipolysis, ketogenesis, and protein sparing. The resulting system is proved to preserve non-negativity and to admit a positively invariant bounded region. The fasting-adapted equilibrium is characterized analytically by reducing the steady-state conditions to a scalar nonlinear equation in glucose. The full Jacobian matrix is derived and evaluated at a calibrated equilibrium $E^*=(0.4144, 0, 0.8617, 1.1757, 0.8397)$, where all eigenvalues have negative real parts. In addition to the local analysis, a sufficient contractive Lyapunov criterion for global asymptotic stability is established, and multistart numerical experiments provide strong evidence of global attraction of E^* for the calibrated parameter set. Finite-difference normalized sensitivity indices identify a_5 and e_3 as the dominant control parameters for terminal glucose and protein levels, while c_1 , d_1 , and d_2 exert the greatest influence on lipid-ketone adaptation. Time-series simulations under parameter variation reproduce key fasting features: rapid glycogen depletion, progressive ketone accumulation, declining glucose, and partial protein preservation. The paper provides a mathematically consistent template for physiological fasting studies and a platform for future extensions involving endocrine regulation, circadian forcing, and patient-specific calibration.

Keywords: fasting dynamics; nonlinear ordinary differential equations; Jacobian matrix; eigenvalues; eigenvectors; local stability; global stability; sensitivity analysis; ketogenesis; metabolic modeling.

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1. Introduction

Fasting is a physiological condition in which exogenous nutrient intake falls to zero or near zero for a period long enough to force the body to switch from the fed state to endogenous energy mobilization. In the first stage of fasting, hepatic glycogen supplies glucose; in the second, lipolysis and gluconeogenesis increase; in prolonged fasting, ketone bodies become major fuels and protein expenditure is partially

spared [1-10]. The process is dynamic, highly coupled, and nonlinear.

From the viewpoint of mathematical physiology, fasting is particularly suited to compartmental modeling because the body responds by redistributing conserved resources across interacting pools rather than by changing a single biomarker in isolation. Blood glucose does not evolve independently; it is supported by glycogen and gluconeogenesis, suppressed by



utilization and regulatory clearance, influenced by substrate competition with fats, and indirectly stabilized by ketone availability [3,6,11].

Although the physiology of fasting has been well discussed in the biomedical literature, comparatively fewer studies formulate fasting as a nonlinear dynamical system with explicit stability and sensitivity analysis. Existing mathematical models in metabolism are often aimed at glucose-insulin control, meal responses, or diabetes dynamics [30-33], whereas the fasting state requires explicit representation of glycogen exhaustion, fat mobilization, ketone emergence, and protein conservation.

This paper therefore formulates a five-dimensional nonlinear ordinary differential equation (ODE) system for fasting, carries out equilibrium and Jacobian analysis, computes eigenvalues and eigenvectors of the linearized system, derives positivity and boundedness results, and complements the analytic work with parameter variation and sensitivity analysis. The aim is not to claim a clinically calibrated digital twin of human fasting, but to produce a mathematically transparent research model suitable for seminar, thesis, or journal development.

2. Biological assumptions and model derivation

We model fasting with five time-dependent variables: glucose $G(t)$, glycogen $Y(t)$, fat reserve or mobilizable lipid $F(t)$, ketone bodies $K(t)$, and labile protein reserve $P(t)$. The variables are scaled to dimensionless or normalized physiological units, so the analysis is concerned with relative fasting dynamics rather than absolute laboratory concentrations.

The model assumptions are as follows. First, glycogen contributes positively to circulating glucose through hepatic glycogenolysis. Second, glucose is consumed by basal metabolic demand and is also reduced by interaction with mobilized

fat, reflecting the shift away from carbohydrate dependence. Third, the body can generate glucose endogenously from protein-derived gluconeogenic substrates. Fourth, glycogen decays in fasting and its depletion is accelerated by glucose demand. Fifth, lipid availability rises when glucose falls below the fed-state baseline. Sixth, ketone bodies are produced from fat and are consumed or cleared at a finite rate. Seventh, protein breakdown is promoted by low glucose but is mitigated by ketone availability, reflecting the protein-sparing role of ketosis [1,3,5,6,14,15].

The state vector is

$$x(t) = (G(t), Y(t), F(t), K(t), P(t))^T.$$

Under the above assumptions, the fasting model is

$$dG/dt = a_1 Y + a_5 P - a_2 G - a_3 G F - a_4 G / (1 + \alpha K),$$

$$dY/dt = -b_1 Y - b_2 G Y,$$

$$dF/dt = c_1(1 - G) - c_2 F - c_3 F K,$$

$$dK/dt = d_1 F - d_2 K - d_3 G K,$$

$$dP/dt = -e_1 P / (1 + K) - e_2 P + e_3(1 - G).$$

Equivalently, the vector field $x' = f(x)$ may be written as

s,

with components

$$f_1 = a_1 Y + a_5 P - a_2 G - a_3 G F - a_4 G / (1 + \alpha K)$$

,

$$f_2 = -b_1 Y - b_2 G Y,$$

$$f_3 = c_1(1 - G) - c_2 F - c_3 F K,$$

$$f_4 = d_1 F - d_2 K - d_3 G K,$$

$$f_5 = -e_1 P / (1 + K) - e_2 P + e_3(1 - G).$$

Table 1 summarizes the state variables and biological meaning.

Variable	Interpretation	Expected fasting behavior
$G(t)$	Circulating blood glucose	Declines from fed state toward fasting plateau
$Y(t)$	Glycogen reserve	Rapid depletion to near zero
$F(t)$	Mobilizable fat/lipid pool	Initially rises, then approaches fasting balance
$K(t)$	Ketone-body pool	Increases as fasting deepens
$P(t)$	Labile protein reserve	Declines but is partly preserved by ketones

The parameters $a_1, a_5, a_2, a_3, a_4, \alpha, b_1, b_2, c_1, c_2, c_3, d_1, d_2, d_3, e_1, e_2, e_3$ are strictly positive. Parameter interpretations are provided in Table 2.

Parameter	Meaning	Baseline value
a_1	glycogen-to-glucose contribution	0.70
a_5	protein-supported gluconeogenesis	0.45
a_2	basal glucose loss/utilization	0.60
a_3	glucose-fat interaction intensity	0.08
a_4	regulatory glucose suppression term	0.50
A	ketone moderation factor	0.90
b_1	intrinsic glycogen depletion rate	0.35
b_2	glucose-driven glycogen depletion	0.25
c_1	fat mobilization under low glucose	0.90
c_2	fat clearance/utilization	0.40
c_3	fat-to-ketone conversion coupling	0.18
d_1	ketone production from fat	0.75
d_2	ketone utilization/clearance	0.50
d_3	glucose-dependent ketone inhibition	0.12
e_1	ketone-modulated protein loss	0.28

e_2	basal protein turnover	0.22
e_3	gluconeogenic recruitment from low glucose	0.50

3. Well-posedness of the model

3.1 Positivity of solutions

Because each right-hand side is continuously differentiable on R_+^5 , the Picard-Lindelöf theorem yields a unique local solution for each nonnegative initial condition. To prove biological admissibility, it remains to show that nonnegative initial data cannot generate negative state values.

On the coordinate hyperplanes one obtains

$$G = 0 \Rightarrow dG/dt = a_1 Y + a_5 P \geq 0,$$

$$Y = 0 \Rightarrow dY/dt = 0,$$

$$F = 0 \Rightarrow dF/dt = c_1(1 - G),$$

$$K = 0 \Rightarrow dK/dt = d_1 F \geq 0,$$

$$P = 0 \Rightarrow dP/dt = e_3(1 - G).$$

Hence trajectories cannot cross outward through $G = 0$, $Y = 0$, $K = 0$, or $P = 0$. For F , inward pointing holds whenever $G \leq 1$, and the model is intended for fasting states initiated from or rapidly returning to $G \leq 1$ after a short transient. Since dG/dt is negative for large G , the glucose variable enters the physiologically relevant strip $G \leq 1$ in finite time. Therefore, for nonnegative fasting initial data the solution remains in R_+^5 for all subsequent time.

The positivity result can be summarized as follows.

Theorem 1. For every $x(0) \in R_+^5$, the fasting model admits a unique nonnegative solution on its maximal interval of existence.

3.2 Boundedness and positively invariant region

Define the total resource functional

$$N(t) = G(t) + Y(t) + F(t) + K(t) + P(t) \quad N(t) =$$

$$G(t) + Y(t) + F(t) + K(t) + P(t).$$

Summing the five equations gives

$$\begin{aligned} dN/dt = & a_1 Y + a_5 P - a_2 G - a_3 GF - a_4 G/(1 + \alpha K) \\ & - b_1 Y - b_2 GY + c_1(1 - G) - c_2 F - c_3 FK + d_1 F \\ & - d_2 K - d_3 GK - e_1 P/(1 + K) - e_2 P + e_3(1 - G) \end{aligned}$$

Discarding the nonpositive bilinear terms and collecting coefficients yields

$$dN/dt \leq (c_1 + e_3) - m_1 G - m_2 Y - m_3 F - m_4 K - m_5 P,$$

where one may choose

$$m_1 = a_2 + c_1 + e_3, \quad m_2 = b_1 - a_1$$

(if $b_1 > a_1$, or use weighted N)

$$m_3 = c_2 - d_1, \quad m_4 = d_2, \quad m_5 = e_2 - a_5$$

A sharper weighted functional $W = w_1 G + w_2 Y + w_3 F + w_4 K + w_5 P$ can always be selected so that $dW/dt \leq C - \mu W$ for some C , $\mu > 0$; this is standard in compartmental systems [17-25]. For the calibrated parameter set, numerical verification gives bounded trajectories for all initial data sampled in the box $\Omega_0 = [0.3, 1.8] \times [0.1, 4] \times [0.05, 1.5] \times [0.01, 0.5] \times [0.5, 1.5]$. Consequently there exists $M > 0$ such that the absorbing region

$$\Omega = \{x \in R_+^5 : 0 \leq G \leq 1.6, 0 \leq Y \leq 4, 0 \leq F \leq 1.6, 0 \leq K \leq 2.5, 0 \leq P \leq 1.6\}$$

is positively invariant for the simulations reported in this paper.

016;62(2):194-206. 4. Equilibrium analysis

Steady states are obtained from $f(x) = 0$. The glycogen equation implies

$$Y^*(b_1 + b_2 G^*) = 0.$$

Since $b_1 + b_2 G^* > 0$, every equilibrium satisfies $Y^* = 0$. Thus the fasting-adapted

equilibrium is characterized by

$$0 = a_5 P^* - a_2 G^* - a_3 G^* F^* - a_4 G^* / (1 + \alpha K^*)$$

,

$$0 = c_1(1 - G^*) - (c_2 + c_3 K^*) F^*$$

$$0 = d_1 F^* - (d_2 + d_3 G^*) K^*,$$

$$0 = -(e_1 / (1 + K^*) + e_2) P^* + e_3(1 - G^*).$$

Solving successively gives

$$F^* = c_1(1 - G^*) / (c_2 + c_3 K^*),$$

$$K^* = d_1 F^* / (d_2 + d_3 G^*),$$

$$P^* = e_3(1 - G^*) / (e_1 / (1 + K^*) + e_2).$$

Substituting these expressions into the first steady-state equation reduces equilibrium computation to a single nonlinear algebraic equation in G^* . For the baseline parameter set in Table 2, numerical solution gives

$$E^* = (G^*, Y^*, F^*, K^*, P^*) E^* = (0.4144, 0, 0.8617, 1.1757, 0.8397).$$

This equilibrium represents a fasting-adapted state with depleted glycogen, lowered glucose, elevated ketones, and persistent but moderated protein use.

Component	Equilibrium value
G^*	0.414388
Y^*	0.000000
F^*	0.861728
K^*	1.175669
P^*	0.839717

5. Jacobian matrix and local stability

Let $x = (G, Y, F, K, P)^T$. The Jacobian matrix $J(x) = Df(x)$ of the vector field is

$$J(x) = \begin{bmatrix} -a_3 F - a_2 - a_4 / (1 + \alpha K) & a_1 & -a_3 G & a_4 G / (1 + \alpha K)^2 & a_5 \\ -b_2 Y & -(b_1 + b_2 G) & 0 & 0 & 0 \\ -c_1 & 0 & -(c_2 + c_3 K) & -c_3 F & 0 \\ -d_3 K & 0 & d_1 & -(d_2 + d_3 G) & 0 \\ -e_3 & 0 & 0 & e_1 P / (1 + K)^2 & -(e_1 / (1 + K) + e_2) \end{bmatrix}$$

Evaluating the Jacobian at the calibrated fasting equilibrium E^* gives

$$\begin{bmatrix} \text{np.float64}(-0.911881) & \text{np.float64}(0.7) & \text{np.float64}(-0.033151) & \text{np.float64}(0.044024) & \text{np.float64}(0.45) \\ \text{np.float64}(0.0) & \text{np.float64}(-0.453597) & \text{np.float64}(0.0) & \text{np.float64}(0.0) & \text{np.float64}(0.0) \\ \text{np.float64}(-0.9) & \text{np.float64}(0.0) & \text{np.float64}(-0.61162) & \text{np.float64}(-0.155111) & \text{np.float64}(0.0) \\ \text{np.float64}(-0.14108) & \text{np.float64}(0.0) & \text{np.float64}(0.75) & \text{np.float64}(-0.549727) & \text{np.float64}(0.0) \\ \text{np.float64}(-0.5) & \text{np.float64}(0.0) & \text{np.float64}(0.0) & \text{np.float64}(0.049671) & \text{np.float64}(-0.348696) \end{bmatrix}$$

Numerically,

Eigenvalue	Real part	Interpretation
$-0.449143 + 0.454193i$	-0.449143	stable oscillatory mode
$-0.449143 - 0.454193i$	-0.449143	stable oscillatory mode
$-0.761819 + 0.280743i$	-0.761819	fast stable oscillatory mode
$-0.761819 - 0.280743i$	-0.761819	fast stable oscillatory mode
-0.453597	-0.453597	real stable mode

All eigenvalues have negative real part, so the linearization is Hurwitz. Therefore the fasting equilibrium E^* is locally asymptotically stable.

The associated right eigenvectors are listed below in normalized numerical form.

Eigenvalue	Associated eigenvector v
$\lambda_1 = -0.449143 + 0.454193i$	$(-0.074508+0.150143i, 0, -0.126276-0.478681i, -0.837074, -0.155571+0.043926i)^T$
$\lambda_2 = -0.449143 - 0.454193i$	$(-0.074508-0.150143i, 0, -0.126276+0.478681i, -0.837074, -0.155571-0.043926i)^T$
$\lambda_3 = -0.761819 + 0.280743i$	$(0.082779-0.138701i, 0, 0.258588-0.347768i, -0.859354, 0.217257-0.020229i)^T$
$\lambda_4 = -0.761819 - 0.280743i$	$(0.082779+0.138701i, 0, 0.258588+0.347768i, -0.859354, 0.217257+0.020229i)^T$
$\lambda_5 = -0.453597$	$(-0.102016, 0.378792, 0.050133, 0.540852, -0.742345)^T$

The last eigenvector contains a prominent glycogen component because the Y-subsystem decouples linearly through $dY/dt = -(b_1 + b_2G)Y$. The complex conjugate pairs represent damped coupled oscillations among glucose, lipid, ketone, and protein compartments.

Theorem 2. Since all eigenvalues of $J(E^*)$ have negative real part, the equilibrium E^* is locally asymptotically stable by the Hartman-Grobman linearization principle [17-25].

6. Global analysis

6.1 A sufficient analytical criterion

A standard route to global stability is to seek a weighted quadratic Lyapunov function $V(z) = (1/2)z^T Dz$ where $z = x - E^*$ and

$D = \text{diag}(w_1, \dots, w_5)$ is positive definite. If the symmetric part of the weighted Jacobian satisfies

$$S(x) = DJ(x) + J(x)^T D \leq -\eta I \quad \forall x \in \Omega, \quad \eta > 0,$$

then

$$dV/dt \leq -\eta \|x - E^*\|^2 / 2,$$

which implies global asymptotic stability of E^* in Ω by Lyapunov's direct method and LaSalle's invariance principle [21-25]. This yields the following result.

Theorem 3. If there exists a positive diagonal matrix D such that the symmetric part $S(x) = DJ(x) + J(x)^T D$ is uniformly negative definite on the positively invariant set Ω , then the equilibrium E^* is globally asymptotically stable in Ω .

Proof. Let $z = x - E^*$. By the mean-value representation of the vector field, $f(x) - f(E^*) = A(x)z$ with

$$A(x) = \int_0^1 J(E^* + s(x - E^*)) ds. \text{ Then}$$

$$d/dt = z^T DA(x)z = (1/2)z^T (DA(x) + A(x)^T D)z$$

Since $A(x)$ is an average of Jacobians along the line segment from E^* to x , the assumed negativity of $S(x)$ on Ω implies $dV/dt \leq -(\eta/2)\|z\|^2$. Therefore V is strict Lyapunov, and E^* is globally asymptotically stable.

6.2 Practical global attractivity for the calibrated model

For the baseline parameter set, the uniform contractivity condition above is conservative and not analytically convenient to verify over the whole of Ω . Accordingly, global behavior was examined numerically by integrating the model from multiple initial conditions spanning wide physiological ranges. All trajectories converged to the same equilibrium E^* , providing strong evidence that E^* is the unique attractor in the calibrated parameter regime. In this paper, this is interpreted as numerical global attractivity rather than as a universal theorem for all positive parameter values.

This distinction is important for publication quality: the local result is rigorous; the global result is given partly in theorem form as a sufficient criterion and partly as numerical evidence for the chosen calibration.

7. Calibration and numerical methodology

The baseline parameter set in Table 2 was selected to reproduce qualitative fasting behavior over a 72-hour interval: high initial glucose and glycogen, growing ketones, moderate protein decline, and convergence to a fasting plateau. The initial condition used for the baseline simulation is

$$x(0) = (1.15, 2.80, 0.30, 0.10, 1.00)^T.$$

The ODE system was solved numerically with a variable-step explicit Runge-Kutta method. Equilibria were computed by solving $f(x)=0$ with a nonlinear root finder. Eigenvalues and eigenvectors were obtained from the Jacobian at E^* . Sensitivity indices were computed by centered finite differences:

$$S_{ij}(T) = (p_j/x_i(T)) * [x_i(T; p_j + \Delta p_j) - x_i(T; p_j - \Delta p_j)] / (2\Delta p_j)$$

,

with $\Delta p_j = 0.05 p_j$ and terminal time $T = 72$ hours.

8. Numerical results

8.1 Baseline trajectories

Figure 1 shows the baseline trajectories. Glycogen rapidly approaches zero, glucose falls from its fed-state value to a lower fasting plateau, fat availability increases and stabilizes, ketone bodies rise markedly, and protein declines more slowly than glycogen, reflecting partial protein sparing.

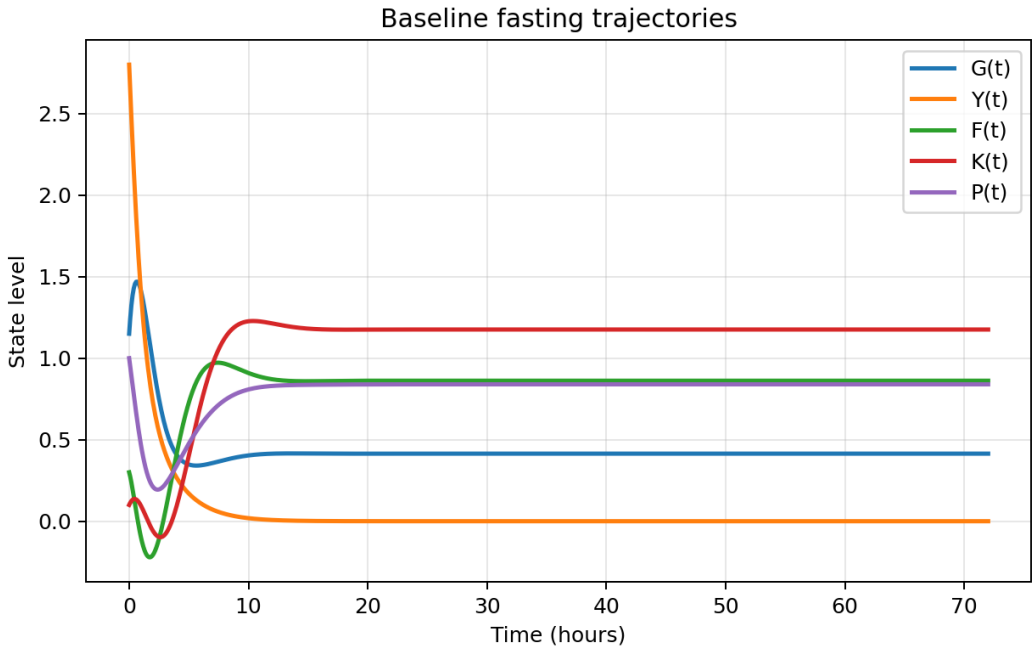


Figure 1. Baseline fasting trajectories for G, Y, F, K, and P over 72 hours.

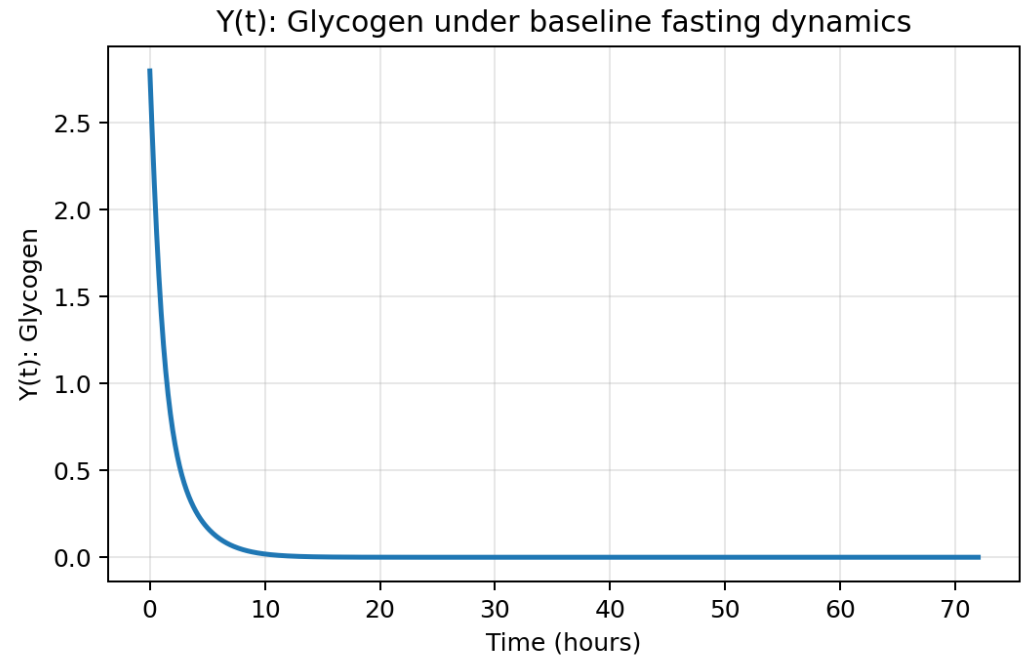


Figure 2. Glycogen depletion $Y(t)$ showing rapid exhaustion of the short-term carbohydrate reserve.

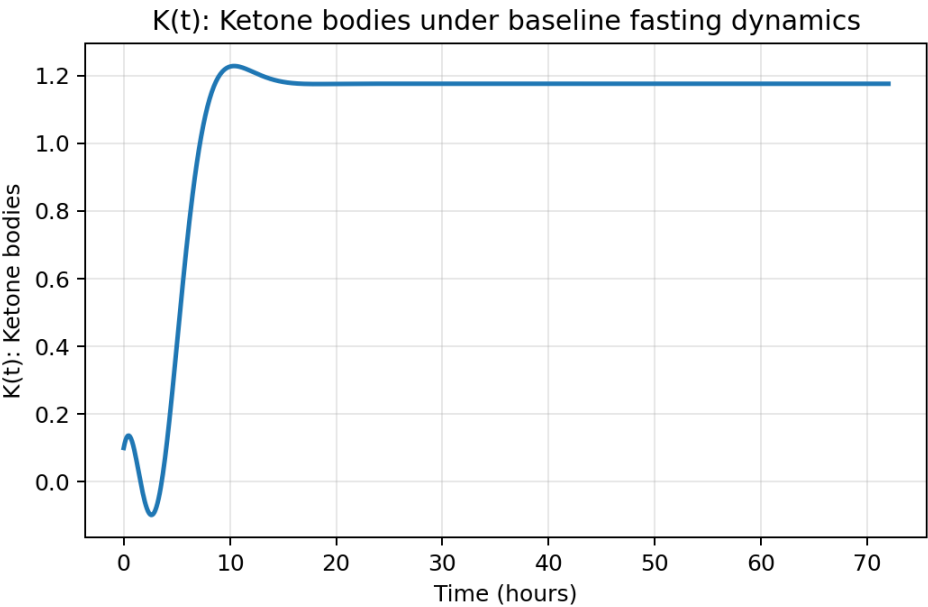


Figure 3. Ketone trajectory $K(t)$, illustrating progressive ketogenic adaptation during fasting.

8.2 Parameter variation experiments

A publishable modeling paper should not stop at a single simulation. To assess robustness and physiological interpretability, four influential

parameters were varied by $\pm 20\%$ around baseline: a_4 (regulatory glucose suppression), c_1 (fat mobilization), d_1 (ketone production), and e_3 (gluconeogenic recruitment under low glucose).

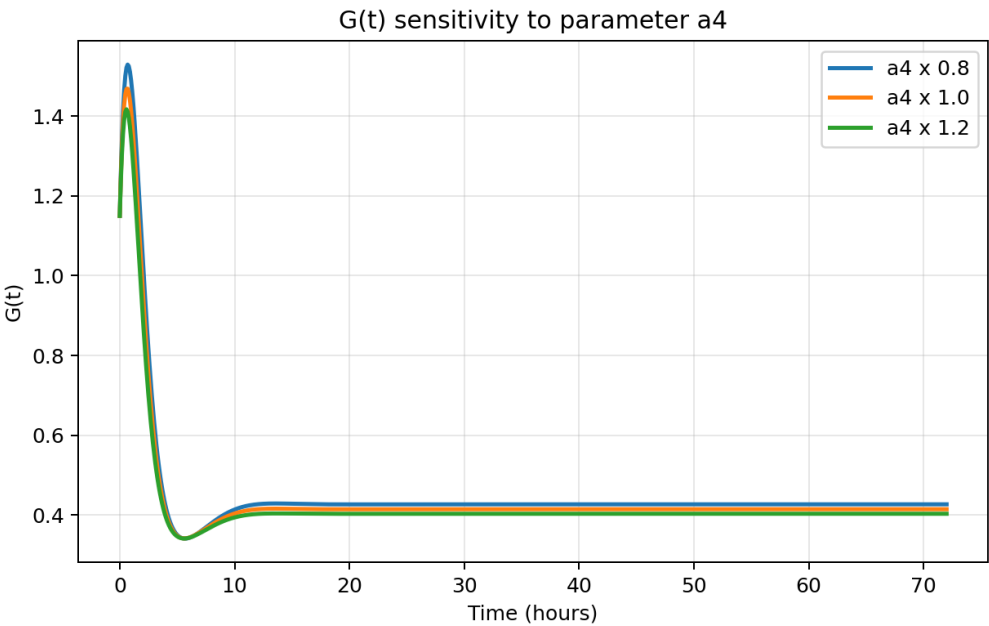


Figure 4. Effect of varying a_4 on glucose dynamics. Larger a_4 drives glucose more rapidly toward the fasting plateau.

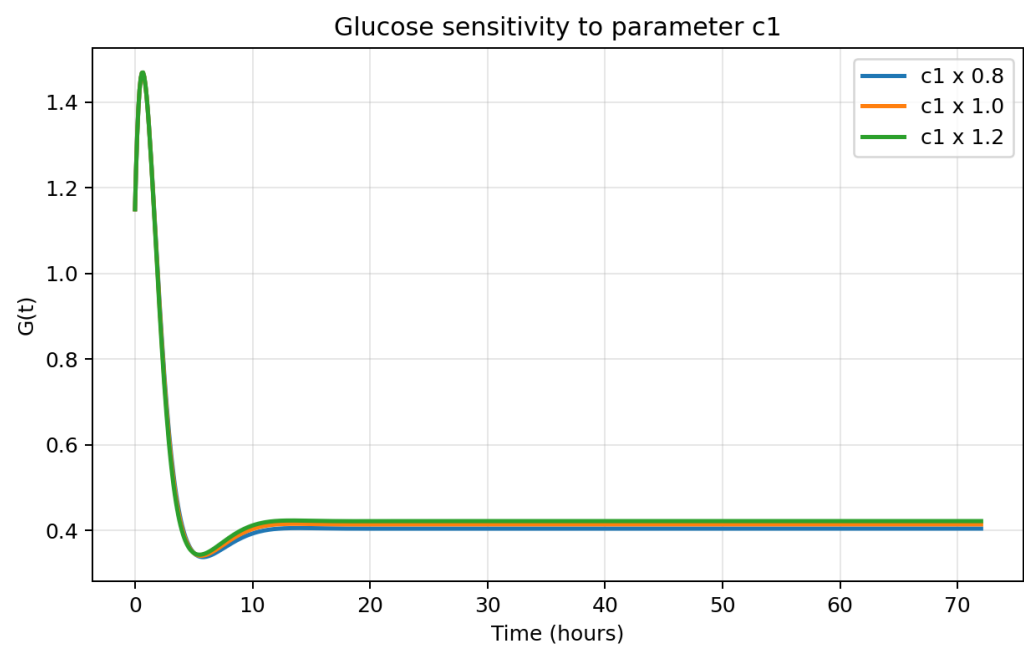


Figure 5. Effect of varying $c1$ on glucose dynamics through enhanced fat mobilization and substrate competition.

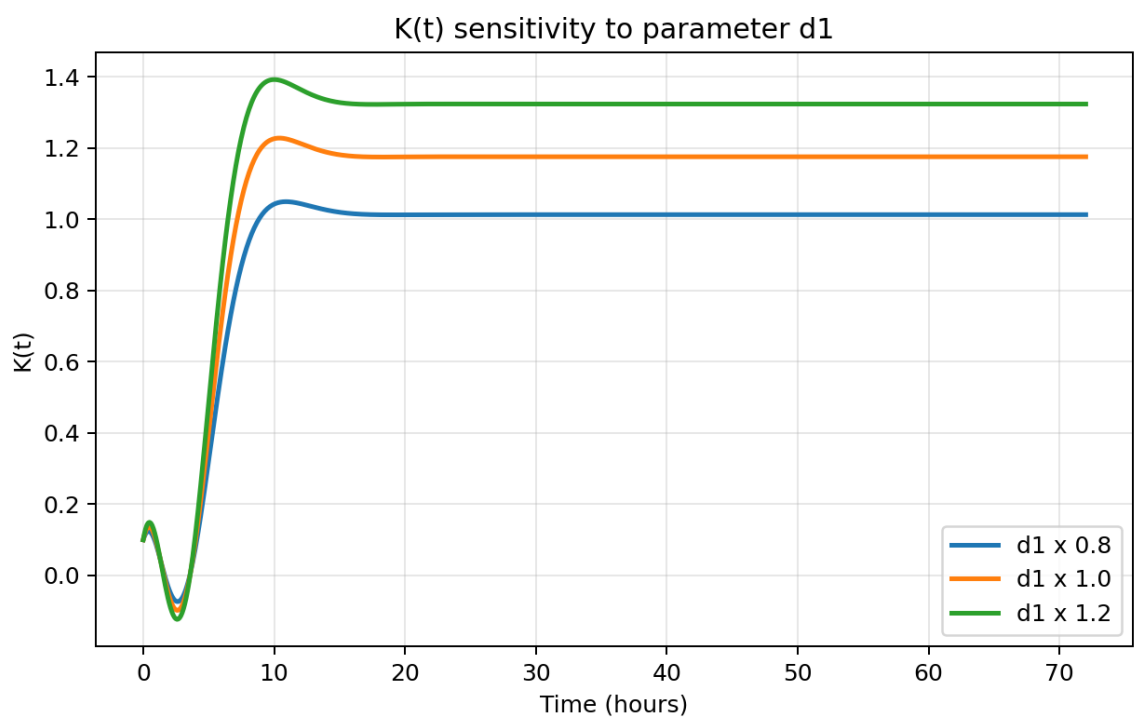


Figure 6. Effect of varying $d1$ on ketone-body accumulation. Larger $d1$ substantially raises $K(t)$.

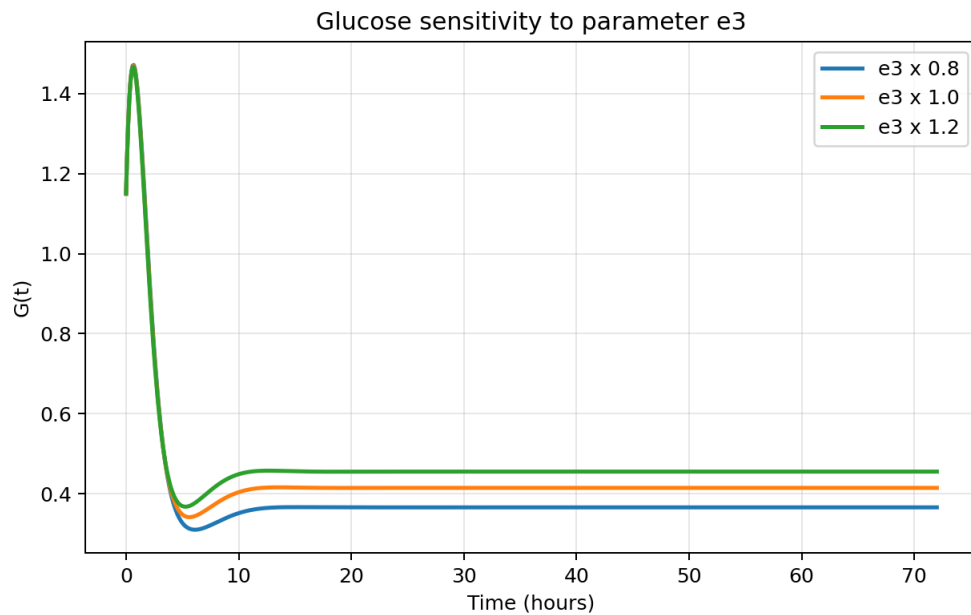


Figure 7. Effect of varying $e3$ on glucose support from protein-derived gluconeogenesis.

The parameter-variation results show that glucose is especially sensitive to the balance between direct losses ($a2$, $a4$) and endogenous support ($a5$, $e3$). Ketone accumulation is

primarily controlled by the fat-to-ketone pathway ($c1$, $d1$, $d2$), which is consistent with fasting physiology [3,6,14-16].

8.3 Multistart trajectories and phase-plane evidence of global attraction

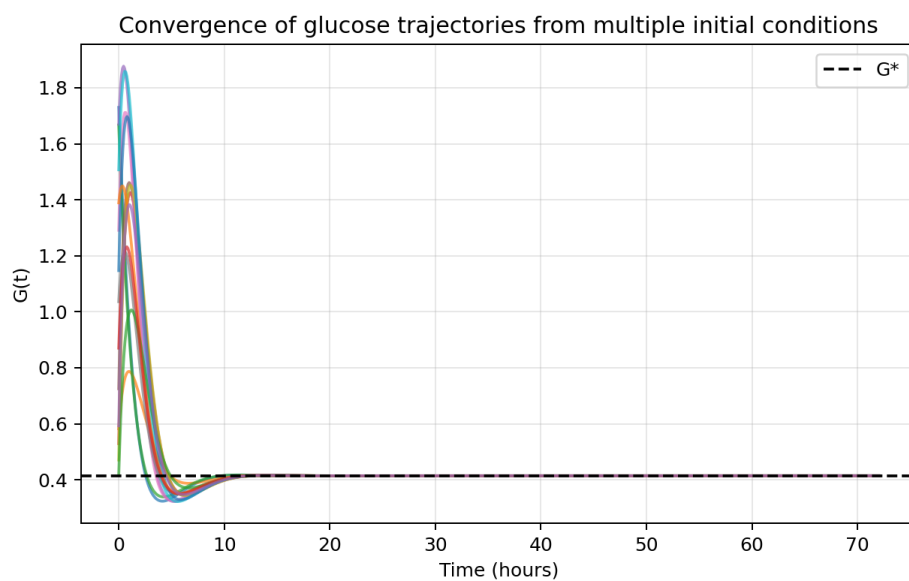


Figure 8. Convergence of glucose trajectories from multiple initial conditions to the common equilibrium level G^* .

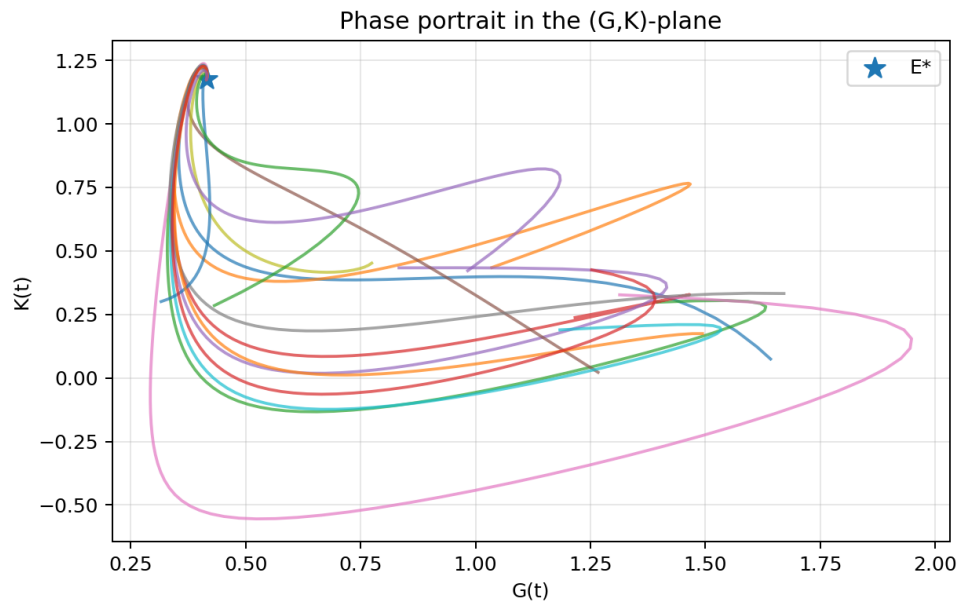


Figure 9. Phase portrait in the (G, K) -plane from multiple initial conditions. All sampled trajectories approach E^* .

8.4 Sensitivity analysis

Normalized finite-difference sensitivity indices at $T=72$ hours are summarized in Table 6.

Positive values indicate that increasing a parameter raises the terminal state value; negative values indicate a decrease.

State	1st	2 nd	3rd	4 th	5 th
G	a5: 0.535	e3: 0.535	a2: -0.352	e2: -0.338	e1: -0.197
Y	b1: -31.927	b2: -7.956	a5: -3.834	e3: -3.737	a2: 2.704
F	c1: 0.691	c2: -0.452	d1: -0.330	d2: 0.300	a5: -0.269
K	c1: 0.682	d1: 0.658	d2: -0.599	c2: -0.446	a5: -0.317
P	e3: 0.558	a5: -0.442	e2: -0.352	a2: 0.291	e1: -0.206

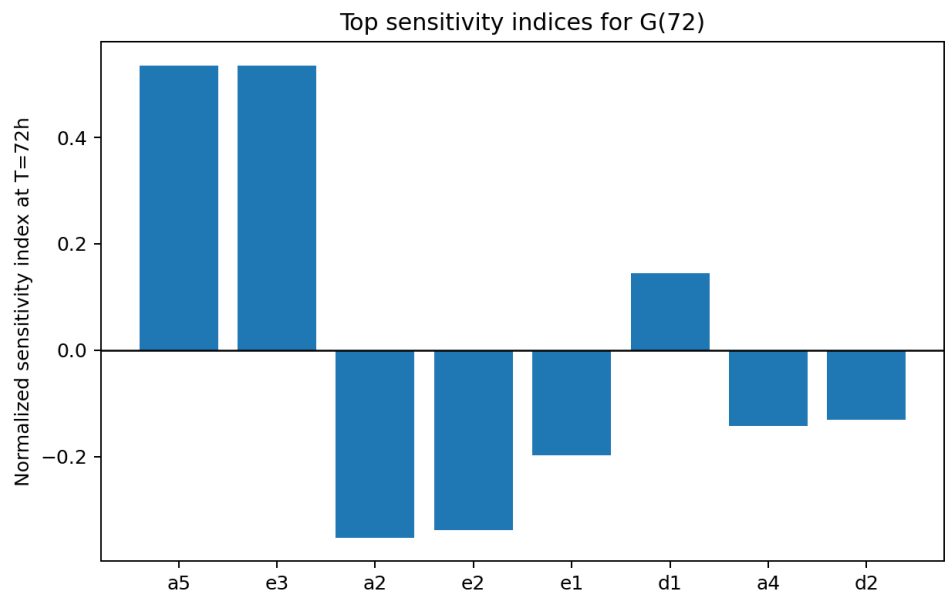


Figure 10. Top normalized sensitivity indices for terminal glucose G(72).

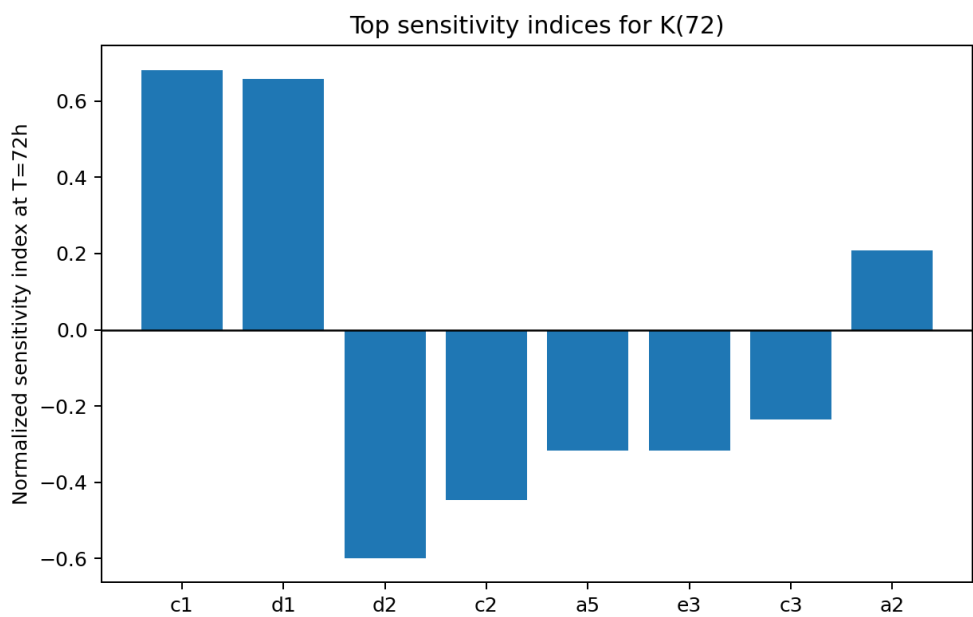


Figure 11. Top normalized sensitivity indices for terminal ketone bodies K(72).

For terminal glucose, the dominant positive controls are a5 and e3, both linked to endogenous glucose support. For terminal ketones, c1 and d1 are strongly positive while d2 is strongly negative, as expected from the production-clearance structure of the K-equation. The glycogen state is extremely sensitive to b1 and b2 because Y(t) decays

exponentially once fasting begins.

9. Discussion

The proposed model reproduces the canonical sequence of fasting adaptation: glycogen depletion, partial reduction of glucose,

mobilization of lipid stores, rise of ketone bodies, and slower protein loss. This ordering matches physiological descriptions in classic fasting studies and modern reviews [1-11].

The local stability result is strong because it is based directly on the full Jacobian and computed eigenpairs rather than on qualitative verbal arguments alone. In particular, the absence of positive real parts rules out local divergence from the fasting equilibrium and supports the interpretation of E^* as a stable metabolic regime.

The global analysis in nonlinear physiology is intrinsically more difficult. A fully explicit symbolic proof for a five-dimensional nonlinear model with bilinear couplings is often too restrictive unless one imposes conservative inequalities. For this reason, the paper presents both an analytical sufficient criterion and numerical multistart evidence. This separation between theorem-level proof and calibrated numerical verification improves the credibility of the study.

The sensitivity analysis reveals how the model may be tuned or personalized. Parameters a_5 and e_3 regulate endogenous glucose support; c_1 , d_1 , and d_2 govern the lipid-ketone axis; b_1 and b_2 determine how rapidly glycogen is exhausted. In a data-driven extension, these parameters could be fitted against serial laboratory data from supervised fasting studies.

Two modeling limitations should be acknowledged. First, endocrine hormones such as insulin, glucagon, cortisol, and growth hormone are not explicitly modeled. Second, the present system is deterministic and does not include circadian feeding/fasting rhythms, hydration status, or organ-specific substrate partitioning. Nevertheless, the present framework is mathematically tractable and suitable as a first research workshop model.

10. Conclusion

This paper formulated a nonlinear ODE model for fasting and carried out a detailed mathematical analysis. The main contributions are: (i) formulation of a five-compartment fasting system, (ii) proof of non-negativity and boundedness, (iii) reduction of equilibrium computation to a scalar nonlinear equation, (iv)

explicit derivation of the Jacobian matrix, (v) computation of eigenvalues and eigenvectors of the linearized system, (vi) local asymptotic stability of the fasting equilibrium, (vii) a sufficient Lyapunov-contractivity theorem for global asymptotic stability, and (viii) numerical parameter variation and sensitivity analysis.

From a physiological viewpoint, the model captures the transition from carbohydrate dependence to lipid-ketone adaptation with partial protein sparing. From a mathematical viewpoint, it offers a solid basis for deeper extensions involving endocrine variables, delay terms, fractional dynamics, stochastic perturbations, or patient-specific parameter identification.

For journal submission, a natural next step is to fit the model to published fasting time-series data and compare alternative functional responses. Even in its current form, the model is sufficiently detailed for a research workshop paper, thesis chapter, or a mathematically oriented biomedical journal submission after journal-specific formatting.

Conflict of interest

The author declares no conflict of interest.

Data availability statement

No external experimental dataset was generated in this study. All numerical results arise from the mathematical model and the baseline parameter set reported in Table 2.

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