

BENG 122A Fall 2025 HW #4
 Due *Friday, November 7 at 11:59pm*

This homework continues a tradition of reiterating on problems in previous homework, now finally getting at control design. As before, start from your previous work, and extend on the models and simulations that you already did using MATLAB or Simulink.

1. [50 pts] Here we reconsider the second-order biosystem of Problem 1 in HW #1 and #2, driven by input $f(t)$ and with output $u(t)$:

$$\begin{aligned}\frac{du}{dt} &= v(t) \\ \frac{dv}{dt} &= -a v(t) - b \sinh(c u(t)) + d f(t)\end{aligned}$$

and with the same parameters $a = 1 \text{ s}^{-1}$, $b = 1 \text{ m s}^{-2}$, $c = 2 \text{ m}^{-1}$, and $d = 2 \text{ kg}^{-1}$.

(a) [20 pts] Design a proportional-derivative (PD) controller that drives the output $u(t)$ of the biosystem towards a target $u_{\text{target}}(t)$ with critically damped response with time constant $\tau = 0.1 \text{ s}$ (*i.e.*, two coinciding poles at $s = -1/\tau$). You may assume small signals around zero baseline for the operating point ($\bar{u} = \bar{u}_{\text{target}} = 0$), and perfect measurement of $u(t)$ so that $f(t) = K_p e(t) + K_d \frac{d}{dt} e(t)$ where $e(t) = u_{\text{target}}(t) - u(t)$, and where K_p and K_d are the proportional and differential control parameters.

(b) [30 pts] Show the step response $u(t)$ of the closed-loop system, from zero initial conditions, for the following three values of the step Δu_{target} in the target $u_{\text{target}}(t)$ from zero baseline ($u_{\text{target}}(t) = 0$ for $t \leq 0$, and $u_{\text{target}}(t) = \Delta u_{\text{target}}$ for $t > 0$):

- $\Delta u_{\text{target}} = 1 \text{ cm}$;
- $\Delta u_{\text{target}} = 10 \text{ cm}$; and
- $\Delta u_{\text{target}} = 100 \text{ cm}$.

Is the response linear in the step size? What is the steady-state gain error? How would you fix it?

2. [50 pts] We again return to the model of regulation of glucose metabolism through insulin secretion, as studied in HW #1 through HW #3 (Problem 2). We now consider delay in the measurement of glucose concentration as given by a first-order lowpass response with time constant $\tau_{\text{meas}} = 2 \text{ min}$:

$$\frac{dG_{\text{meas}}}{dt} = \frac{1}{\tau_{\text{meas}}} (G(t) - G_{\text{meas}}(t)) \quad (1)$$

and we consider a combination of proportional, integral, and derivative (PID) control

$$I(t) = K_p e(t) + K_i \int_{-\infty}^t e(t) dt + K_d \frac{d}{dt} e(t) \quad (2)$$

in the control objective error

$$e(t) = G_{\text{meas}}(t) - T(t) \quad (3)$$

where $T(t)$ is the target glucose concentration. As before, the flow of insulin in the bloodstream and the kinetics in glycogenesis (conversion of glucose to glycogen) catalyzed by insulin are modeled as

$$\frac{dC}{dt} = \alpha I(t) - \frac{1}{\tau} C(t) \quad (4)$$

$$\frac{dG}{dt} = -k C(t) G(t) \quad (5)$$

with $\alpha = 0.2 \text{ L}^{-1}$, $\tau = 1 \text{ min}$, and $k = 1 \text{ L/s mmol}$.

- (a) [5 pts] Verify that the polarity of the difference between target and measured glucose concentrations in the error (3), contrary to the usual definition for setpoint control systems, is the correct one to use here for stable closed-loop dynamics.
- (b) [30 pts] As in HW #3, evaluate the response of the closed-loop system (1)-(5) to an initial 10 mmol of glucose in the 5 L blood volume, for a target concentration $T = 1 \text{ mmol/L}$, and with zero initial insulin in the bloodstream. Show the dynamics in the concentrations of insulin $C(t)$ and glucose $G(t)$, for three settings of the PID control parameters:
 - i. $K_p = 0.01 \text{ L/min}$, $K_i = 0$, and $K_d = 0$;
 - ii. $K_p = 0.01 \text{ L/min}$, $K_i = 0$, and $K_d = 5 \text{ L}$;
 - iii. $K_p = 0.01 \text{ L/min}$, $K_i = 0.01 \text{ L/min}^2$, and $K_d = 5 \text{ L}$.

Explain what you observe, in comparison with the setting in HW #3. How does the measurement delay affect the closed-loop dynamics, and how do the derivative and integral contributions to the control strategy impact the dynamics?

- (c) [15 pts] You may have noticed in your simulations that the control drive $I(t)$ goes negative. Even though insulin secretion can't go negative, there are other metabolic processes active in upregulating low glucose levels, in addition to insulin's role in downregulating high glucose levels.

Gluconeogenesis is the reversal of glycogenesis, converting glycogen back into glucose as catalyzed by the hormone glucagon. Hence insulin and glucagon are complementary regulators of blood glucose level, where insulin corrects for excess glucose, while glucagon corrects for shortage in glucose. Explain how to extend the insulin secretion control strategy (2) by coordinating the secretion of glucagon to allow both positive and negative corrective control action.