DEPARTMENT OF ELECTRONIC

AND

TELECOMMUNICATION ENGINEERING

UNIVERSITY OF MORATUWA

BM2102 - Modelling and Analysis of Physiological Systems

Assignment 4

Compartmental Modelling

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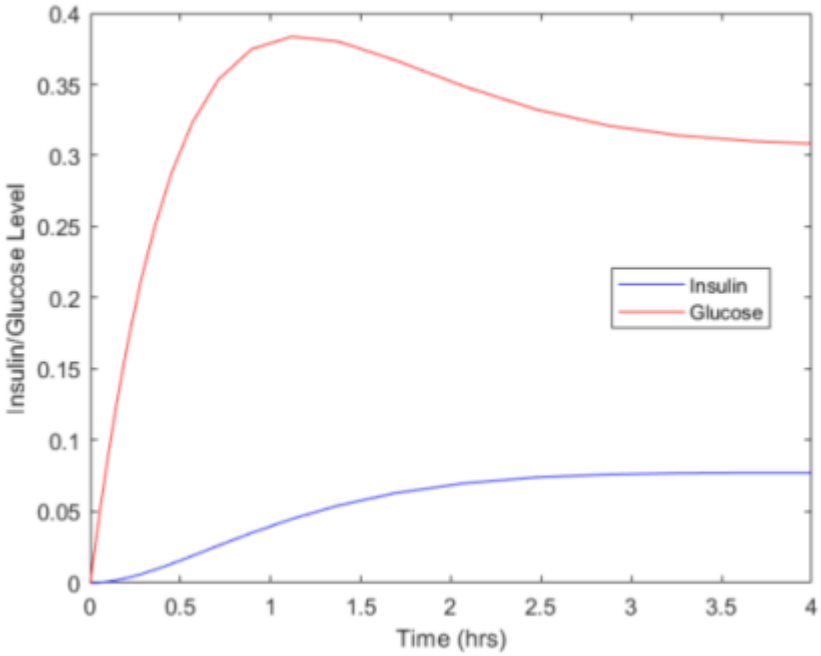
200003P

Part 1

Glucose step input



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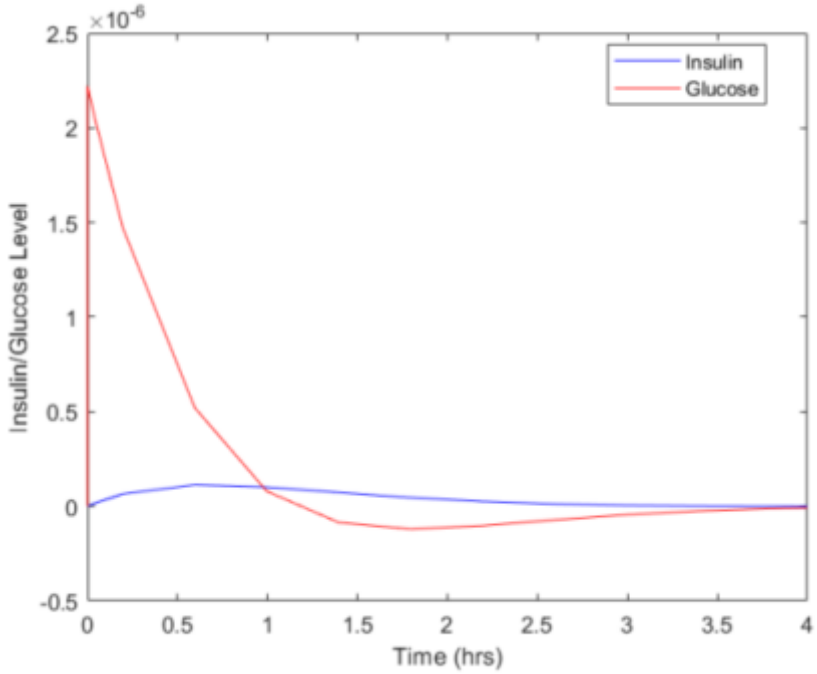
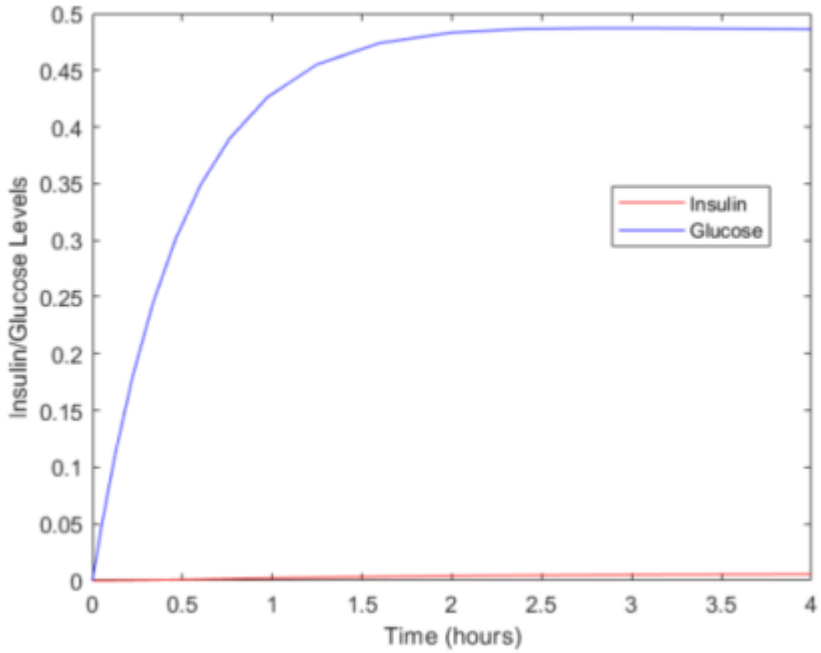
Glucose bolus input

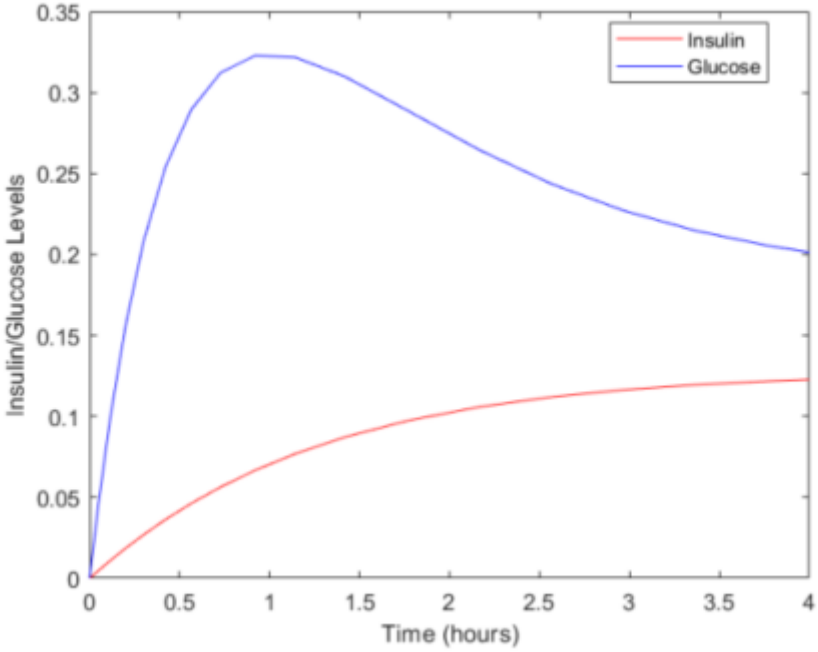


Diabetic Patient without external insulin supply



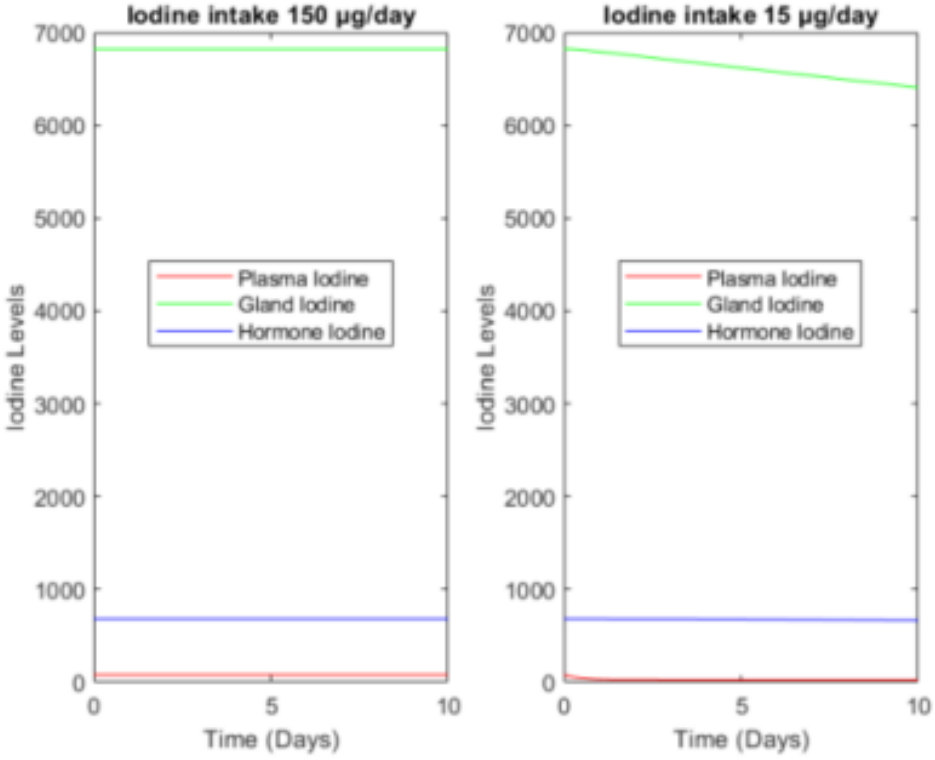
In diabetic patients, the beta cells in the pancreas may experience a reduced ability to secrete sufficient insulin to effectively convert blood glucose into glycogen. This impaired insulin secretion leads to a diminished dependence of insulin on glucose levels. This can be demonstrated by reducing the coefficient of the "g" term in the equation that describes the rate of change of insulin (di/dt). For example, if the coefficient is set to 0.01, it shows that the regulation of glucose levels is not adequately achieved. By reducing the coefficient of the "g" term, the equation implies that insulin production or its effectiveness in regulating glucose levels is compromised in diabetic individuals. As a result, the normal feedback mechanism that maintains stable glucose levels is disrupted, leading to difficulties in regulating blood sugar levels. This highlights the importance of proper insulin function in maintaining glucose homeostasis and the challenges faced by diabetic patients when insulin secretion is insufficient or ineffective.

Diabetic patient with external insulin supply

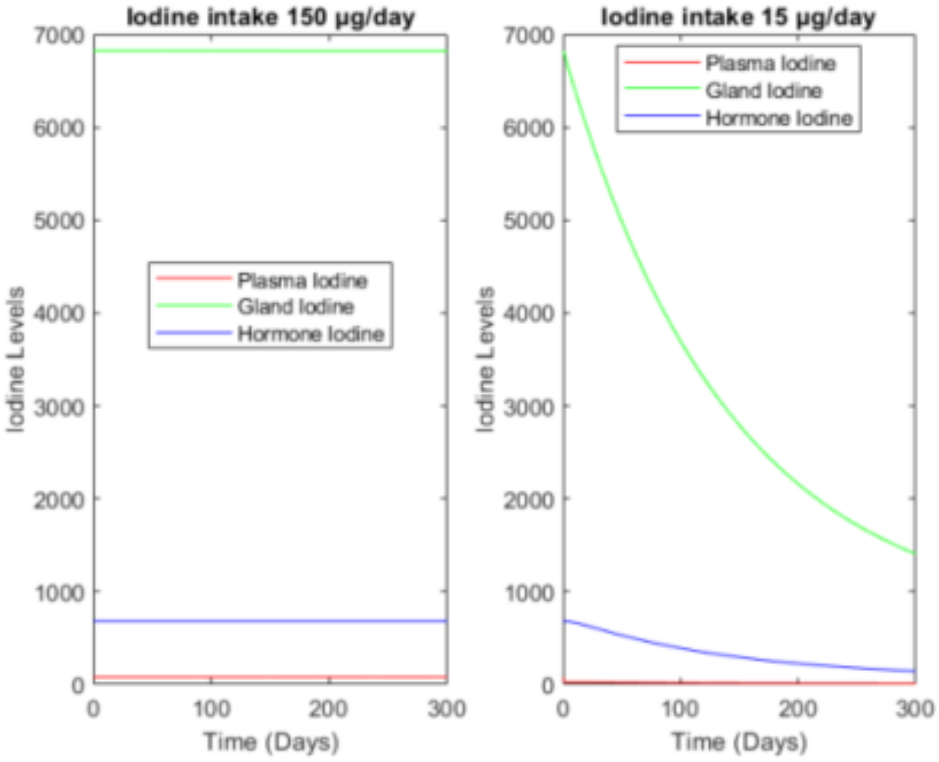


Sudden drop in Iodine input

0 to 10 days

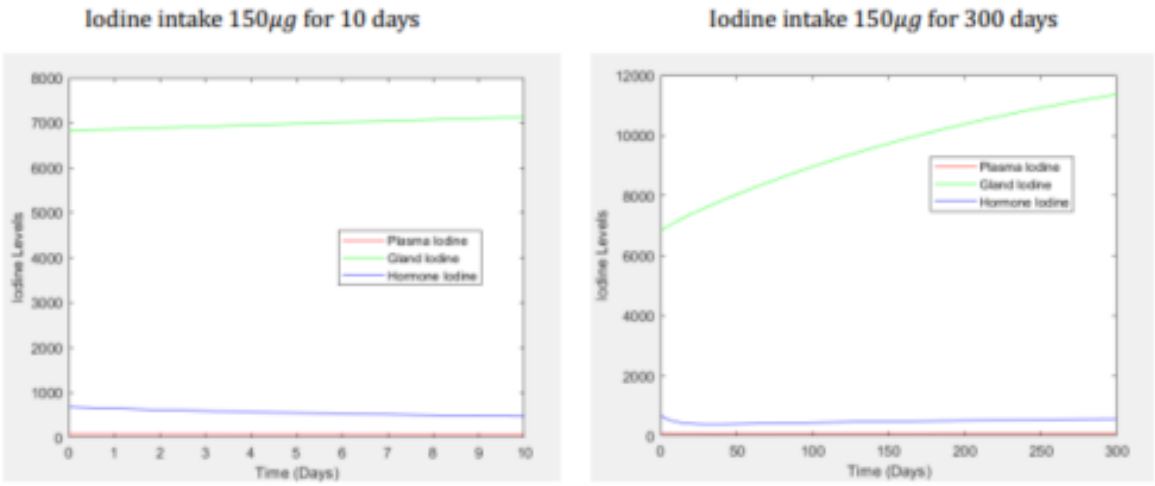


0 to 300 days



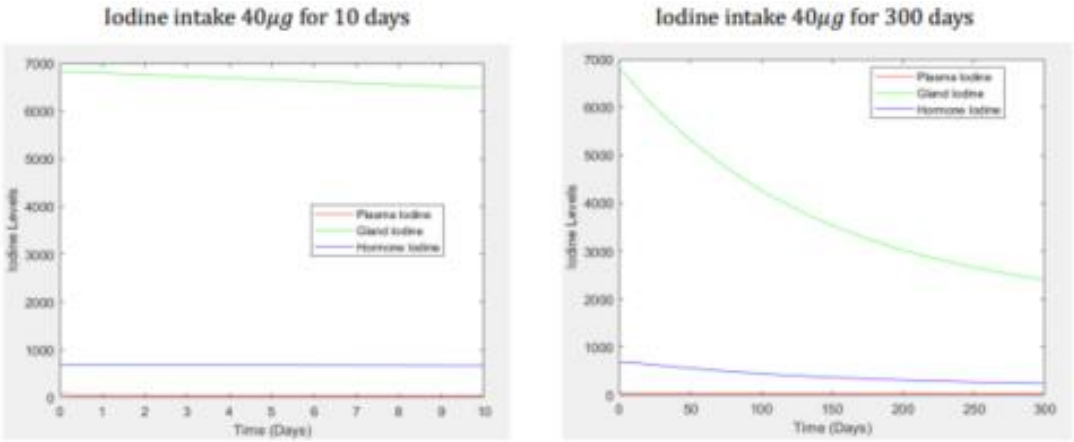
Hypothyroidism due to autoimmune thyroid disease

Autoimmune thyroid disease involves the immune system attacking the thyroid gland, leading to inflammation and damage. This impairs the thyroid's ability to produce thyroid hormones. In the Riggs model, simulating this condition involves decreasing the parameter k2 to a value of 0.005. This adjustment reflects the reduced capacity of the thyroid gland to generate hormones in the presence of the autoimmune attack. By observing the effects of this parameter change, researchers can better understand the disruption in thyroid hormone regulation caused by the disease.



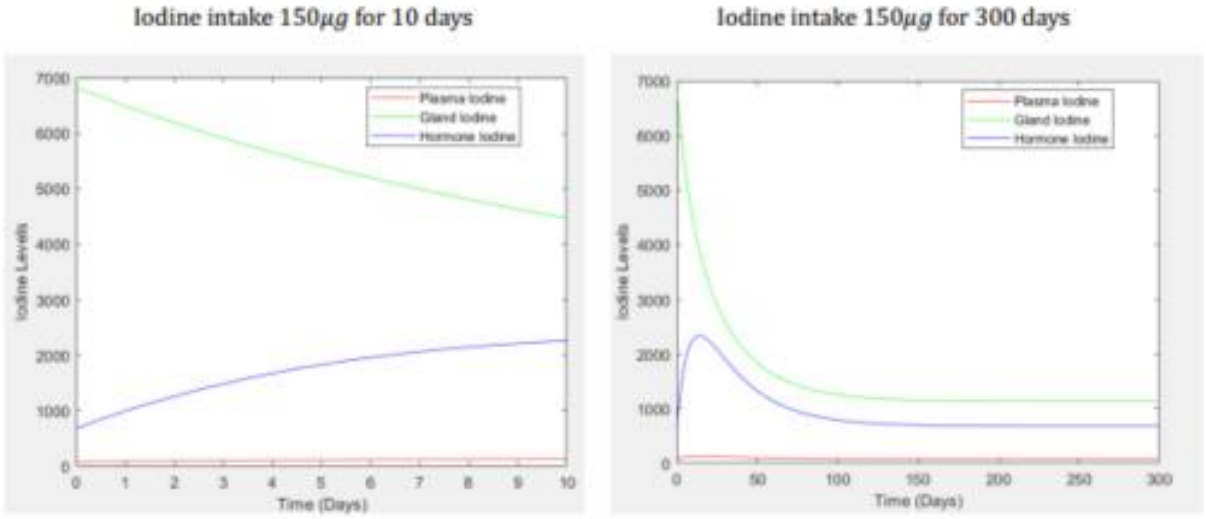
Hypothyroidism due to low Iodine intake

Autoimmune thyroid disease is not primarily caused by reduced intake of iodine. It is an autoimmune condition where the immune system mistakenly attacks the thyroid gland. However, iodine deficiency can contribute to certain thyroid disorders, such as goiter or hypothyroidism. In the Rigg's model, if one wishes to simulate the effect of reduced iodine intake on thyroid function, the input variable B1(t) can be decreased. By lowering the value of B1(t) from 150 to 40, it represents the reduced availability of iodine for thyroid hormone synthesis.



Hyperthyroidism due to Grave's disease

Grave's disease is a condition characterized by the excessive production of thyroid hormones. The thyroid gland produces more thyroid hormone than the body requires, resulting in symptoms such as weight loss, rapid and irregular heartbeat, and other hyperthyroid symptoms. To simulate the excessive thyroid hormone production seen in Grave's disease using the Rigg's model, the parameter k2 can be increased. By adjusting the value of k2 from 0.01 to 0.06 in the simulation, it represents the higher rate of thyroid hormone production associated with Grave's disease. Increasing the value of k2 in the simulation allows for the observation of the effects of accelerated thyroid hormone synthesis. It helps to capture the exaggerated hormone production seen in Grave's disease and provides insights into the altered regulation of thyroid hormones in affected individuals.



Goitre

A goitre is characterized by the enlargement of the thyroid gland, resulting in a visible lump in the front of the neck. This swelling is often caused by iodine deficiency, as the body requires iodine to produce thyroid hormones. Inadequate iodine intake in the diet leads to the thyroid gland enlarging in an attempt to capture as much available iodine as possible. This enlargement helps to compensate for the insufficient iodine and maintain the production of the necessary amount

of thyroid hormones required for the body's functioning. To model this situation using the Rigg's model, the input variable B1(t) can be reduced. By decreasing the value of B1(t), it represents the reduced availability of iodine for thyroid hormone synthesis. This adjustment in the model allows for the observation of the effects of inadequate iodine intake on thyroid gland functionality and the compensatory enlargement of the gland, mimicking the development of a goitre. It is important to note that while iodine deficiency is a common cause of goitre, there can be other factors involved, such as certain medications, genetic conditions, or autoimmune disorders.

Tumours

In the initial stages, thyroid tumours often do not cause noticeable symptoms. However, as they grow over time, a small lump becomes visible in the neck. Symptoms such as changes in voice and difficulty swallowing can occur in association with thyroid tumours. These lumps, known as thyroid nodules, can sometimes lead to overproduction of thyroid hormone, resulting in a condition called hyperthyroidism.

To represent this scenario in the Riggs' model, the parameter k2 can be increased. By adjusting the value of k2 from 0.01 to 0.3 in the simulation, it reflects the higher rate of thyroid hormone production associated with thyroid nodules and hyperthyroidism.

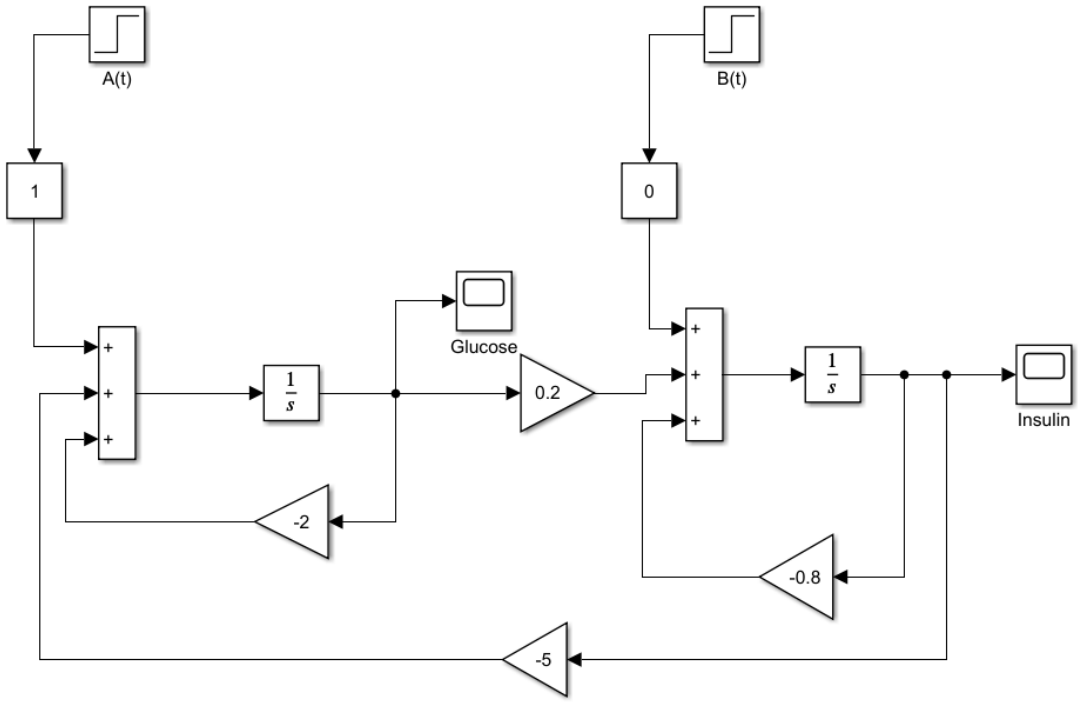
Increasing the value of k2 in the simulation allows for the observation of the effects of accelerated thyroid hormone synthesis, mimicking the overproduction seen in thyroid nodules and hyperthyroidism. This adjustment helps in understanding the impact of thyroid tumour growth on hormone regulation and the resulting hyperthyroid symptoms.

Part 2

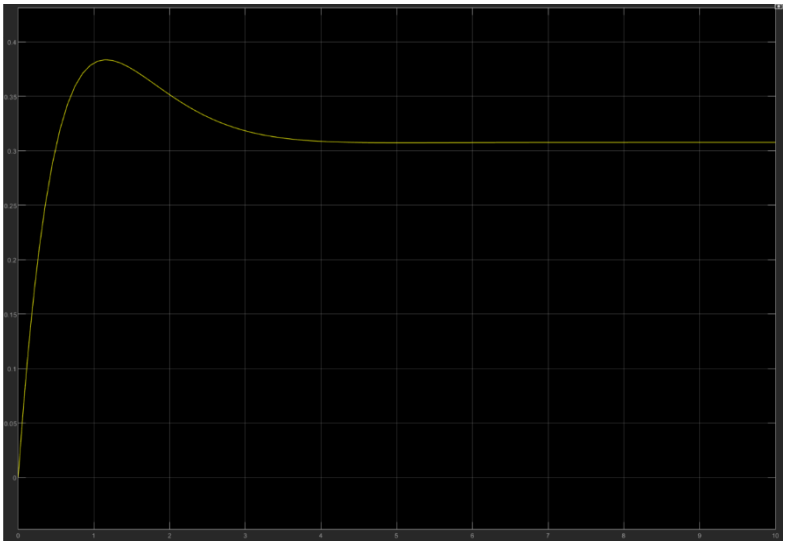
Model 1

$$\frac{di}{dt} = -0.81i + 0.2g + B(t)$$

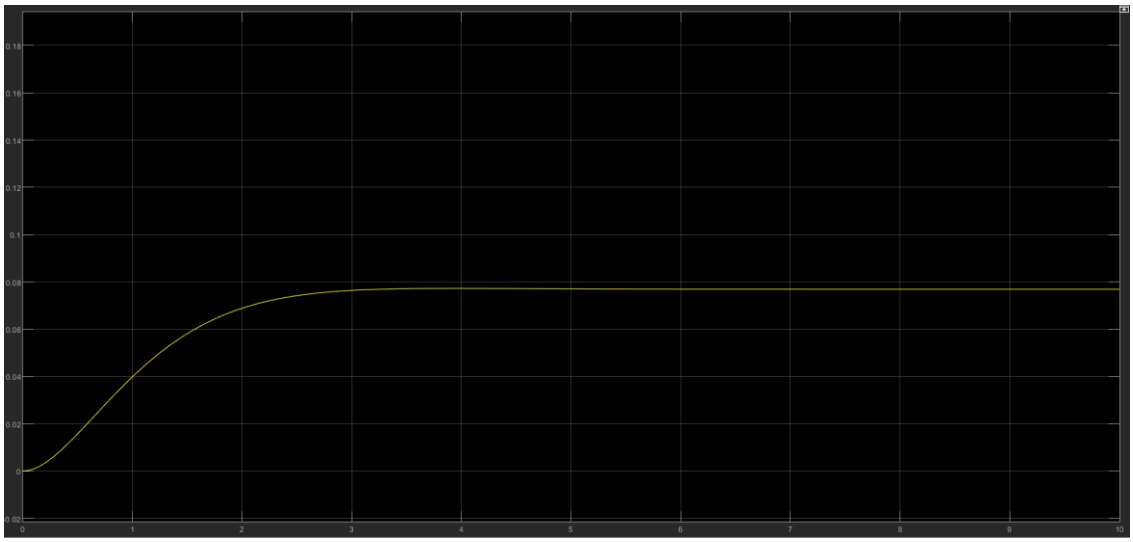
$$\frac{dg}{dt} = -5i - 2g + A(g)$$



Glucose



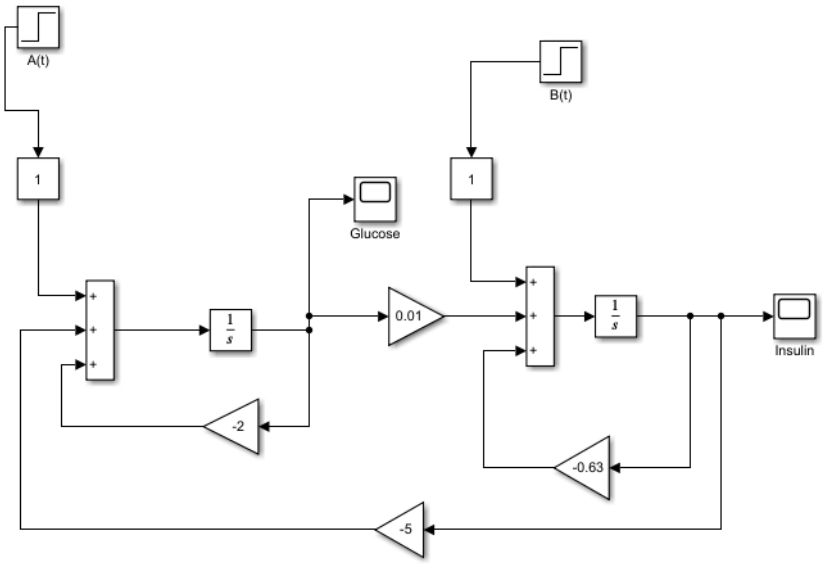
Insulin



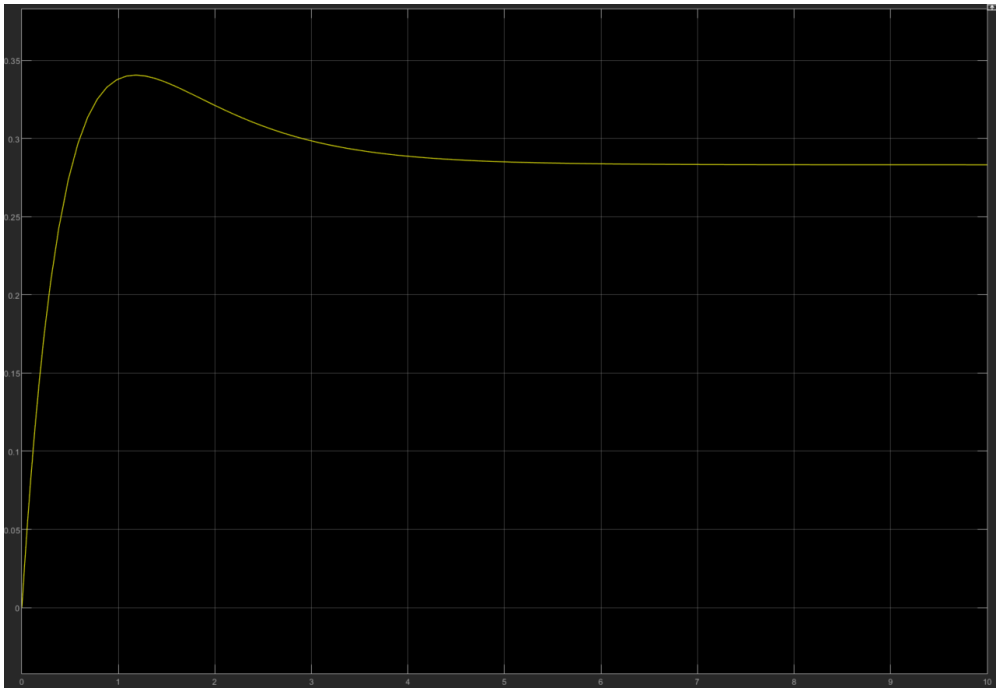
Model 2

$$\frac{di}{dt} = -0.63i + 0.13g$$

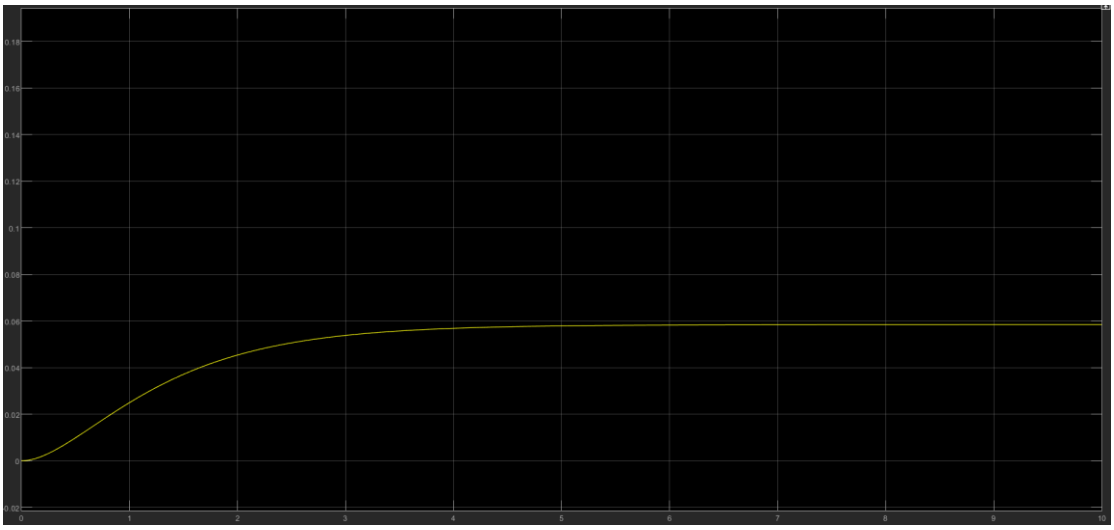
$$\frac{dg}{dt} = -5i - 2.5g + A(t)$$



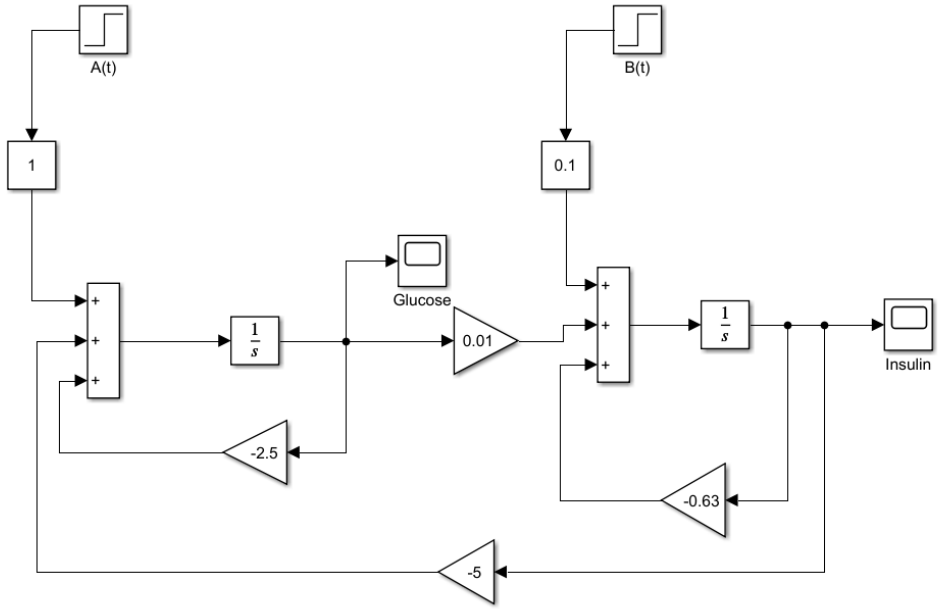
Glucose



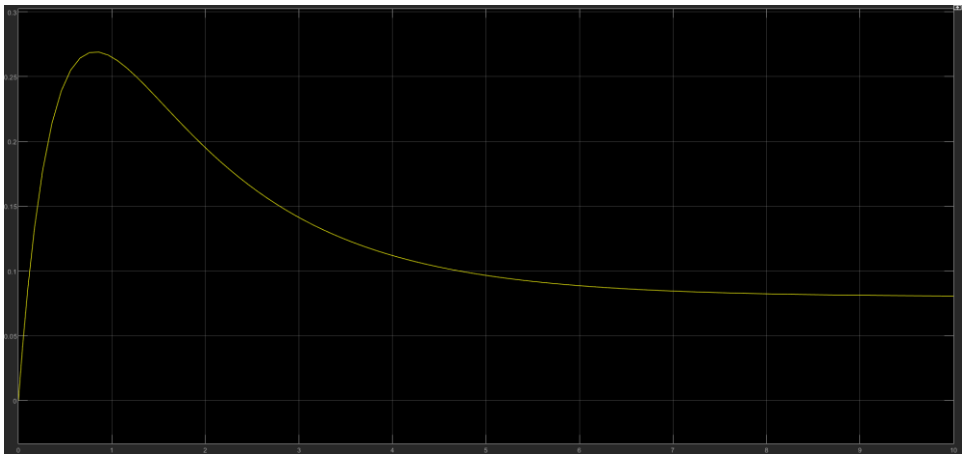
Insulin



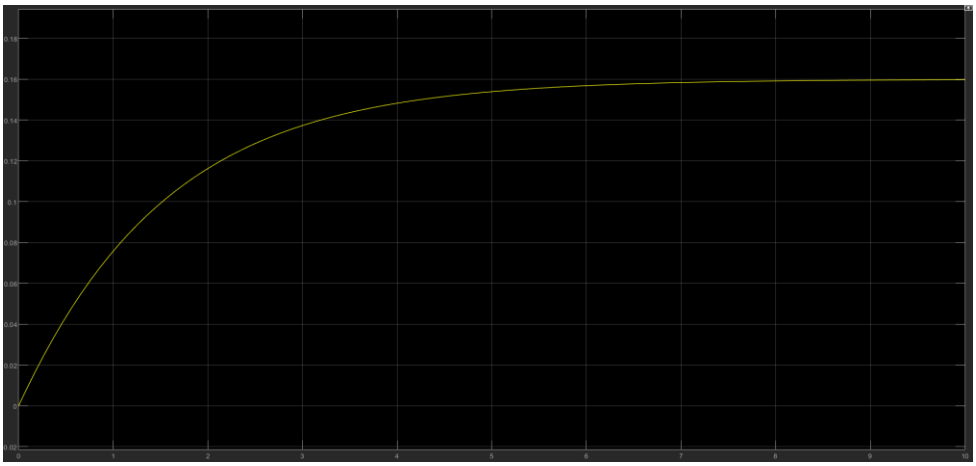
B(t) = 0.1 U/kg/h in a diabetic subject



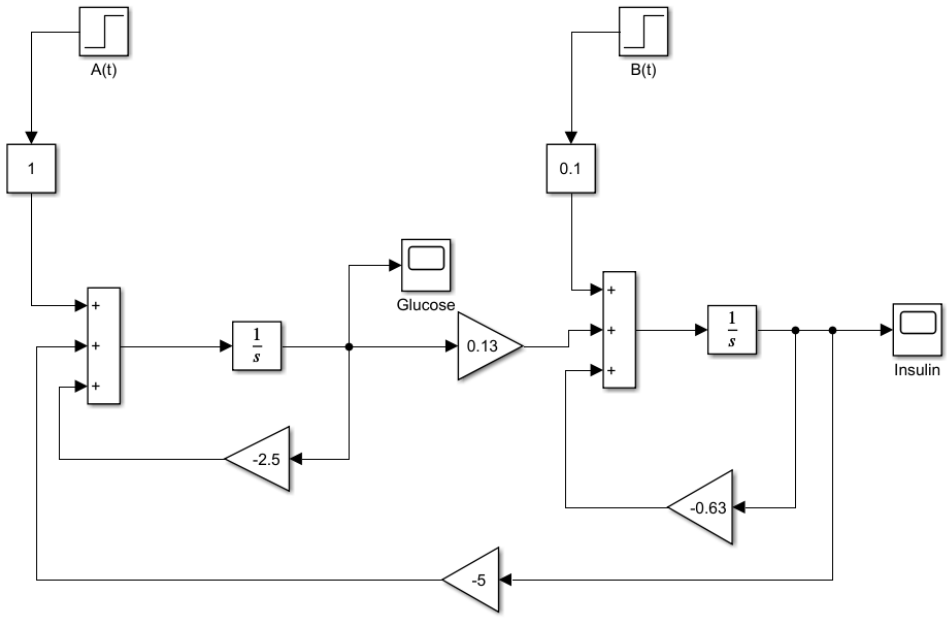
Glucose



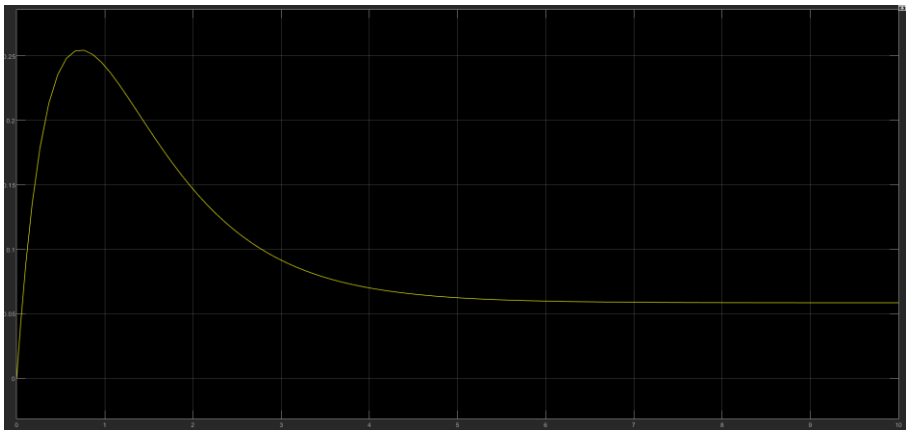
Insulin



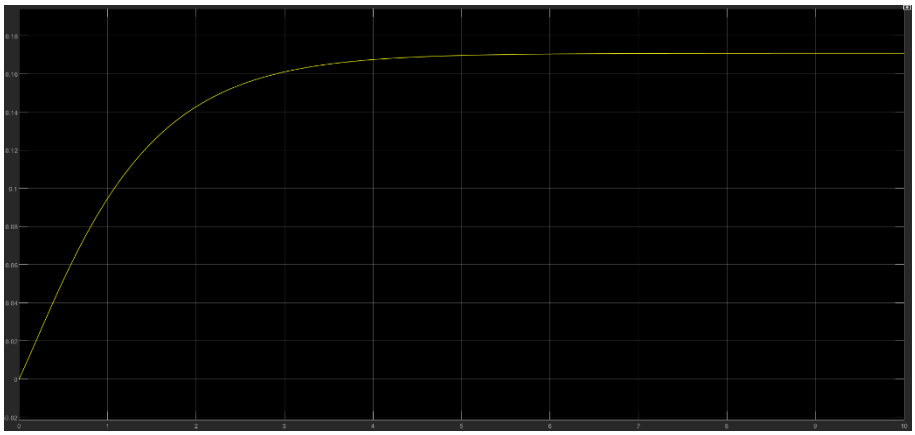
B(t) = 0.1 U/kg/h in a normal subject



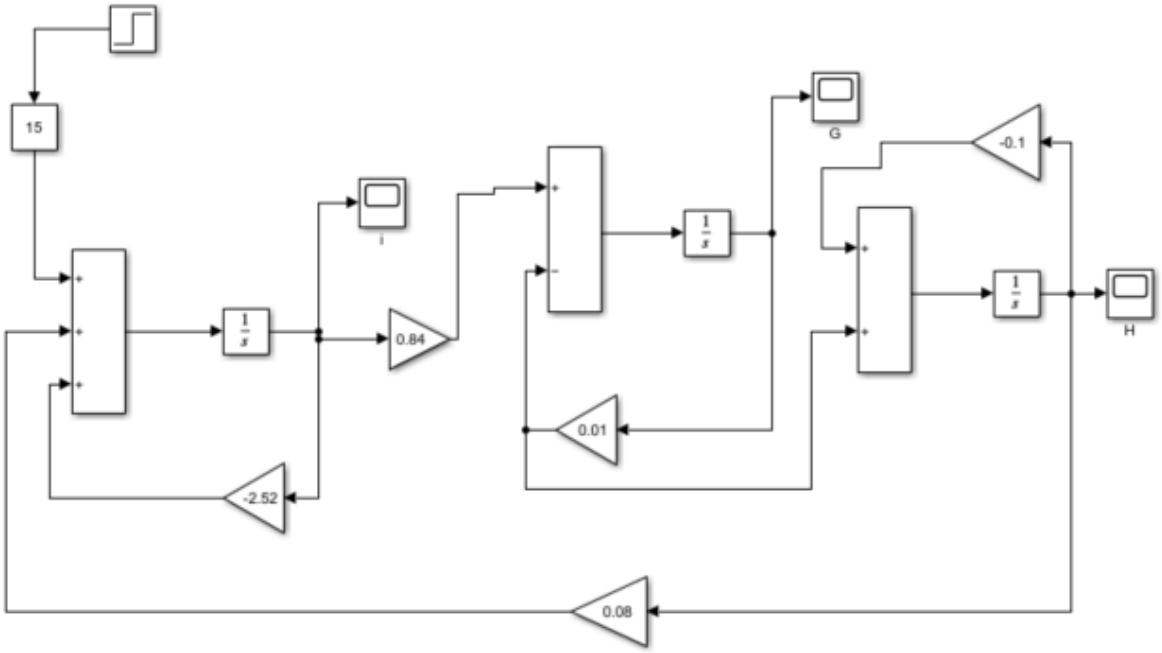
Glucose



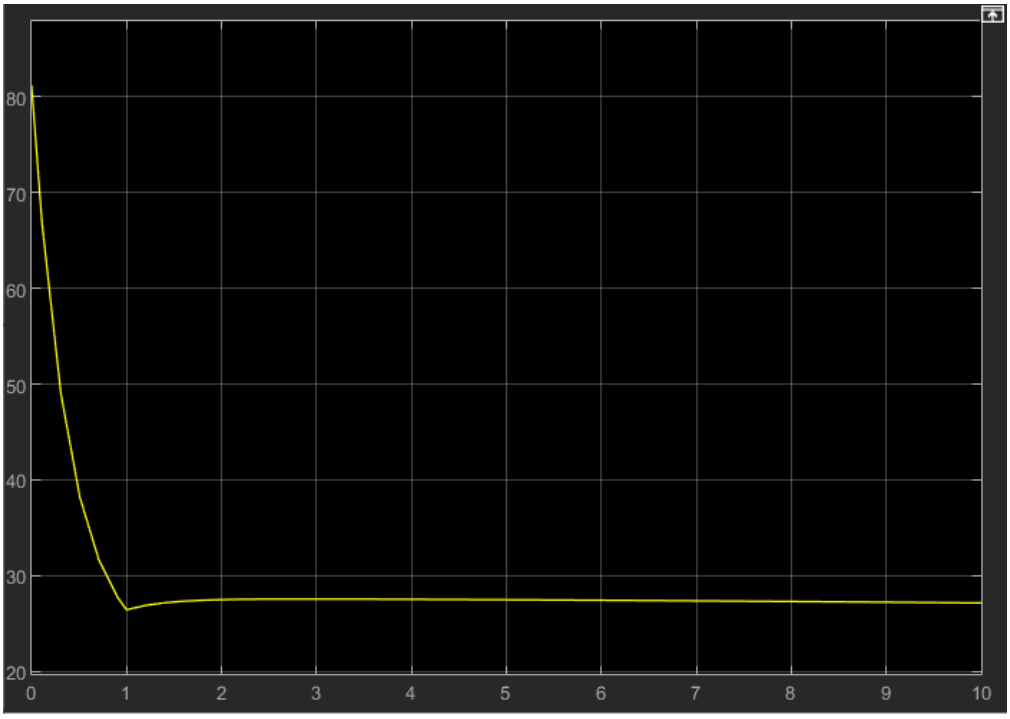
Insulin



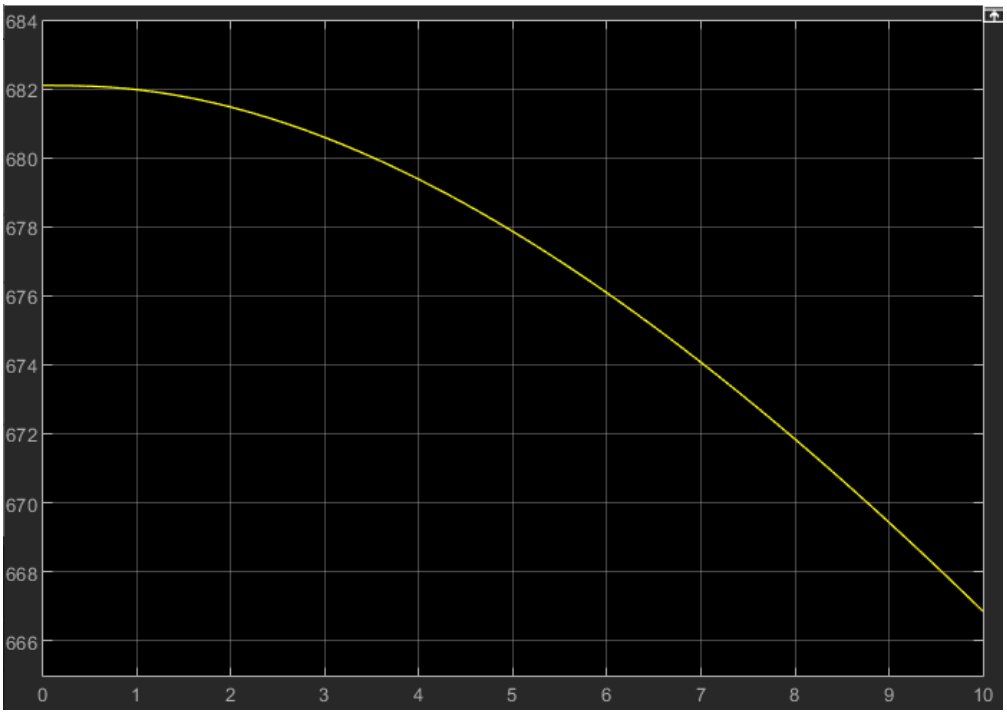
Rigg's Iodine model



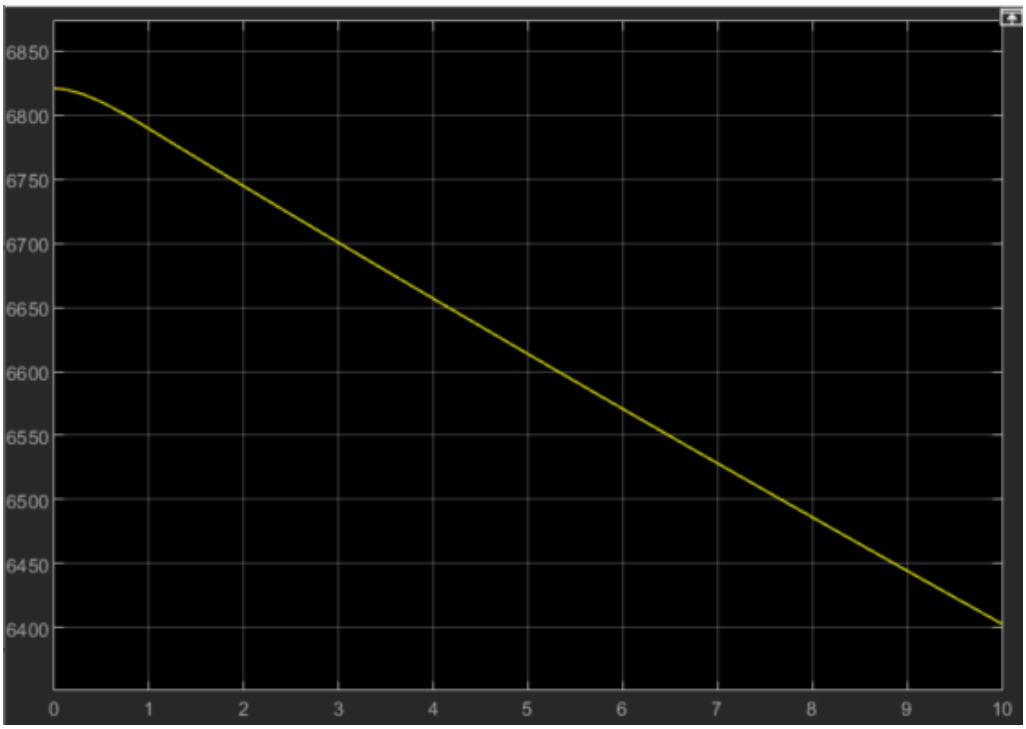
Plasma Iodine



Hormone iodine



Hormone iodine



Part 3

$$\frac{di}{dt} = -k_1 i + k_3 g + B(t)$$

$$\frac{dg}{dt} = -k_4 g - k_6 i + A(t)$$

$$\mathrm{B(t)} = 0$$

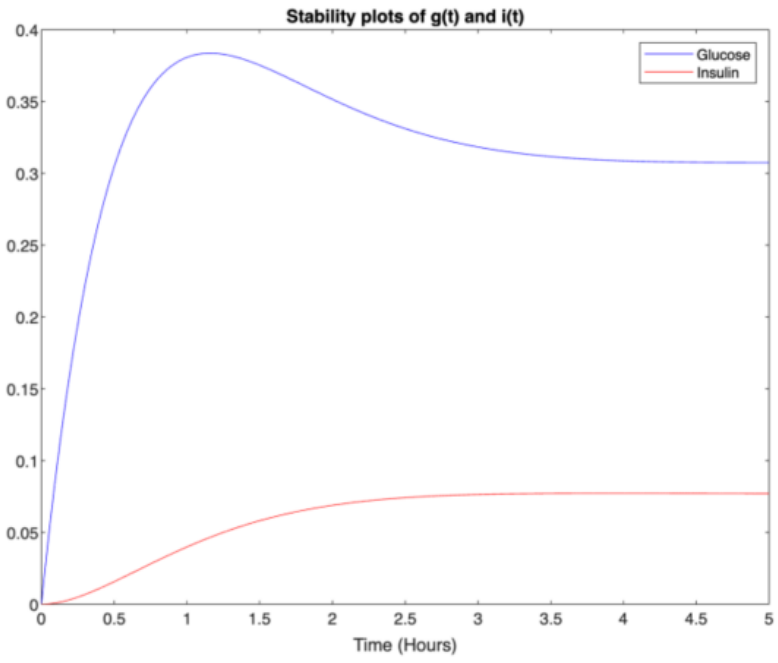
$$\mathrm{A(t)} = \mathrm{a.u(t)}$$

$$k_1 = 0.8h^{-1}, k_3 = 1Uh^{-1}g^{-1}, k_4 = 2h^{-1}, k_6 = 5gh^{-1}lU^{-1}, a = 1$$

By solving 2 differential equations

$$g(t) = e^{-1.4t}\left(-\frac{5}{13}cos0.8t - \frac{35}{52}sin0.8t\right) + \frac{4}{13}$$

$$i(t) = e^{-1.4t}\left(-\frac{1}{13}cos0.8t - \frac{7}{52}sin0.8t\right) + \frac{1}{13}$$



The pancreatic beta cells play a crucial role in producing insulin, which converts glucose into glycogen and stores it in the liver. On the other hand, pancreatic alpha cells produce glucagon, which performs the opposite function by converting stored glycogen in the liver back into glucose.

In the graph, a sudden rise in glucose level occurs due to the initial injection of glucose at a rate of 1 unit per hour. However, there is a time delay for the pancreatic beta cells to detect the increase in plasma glucose level. As a result, the insulin level peaks slightly after the peak of the glucose level.

Insulin works to reduce the amount of glucose in the bloodstream, and this decrease is recognized by the pancreatic alpha cells. In response, the alpha cells produce glucagon, which increases the plasma glucose level by breaking down stored glycogen into glucose. Consequently, the steady-state glucose level is slightly higher compared to a scenario where the effect of glucagon was not considered (as in the first case).

In this scenario, both glucagon and insulin levels gradually increase over time compared to the first case. This represents the dynamic interaction between insulin and glucagon in regulating blood glucose levels, with their levels adjusting and responding to changes in glucose concentration.