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Glucose level in the body of a fasting non-diabetic person

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Abstract

This study shows that the source of glucose to the cells of a fasting individual is not exclusively from the stored glucose in the Liver as this is shown to be exhausted within a very short period of time. For fasting to last for a relatively long time, it is shown that the rate of release of stored glucose in the Liver as well as the production of glucose by the Kidney and other parts of the body, must vary over the period of time in question.

Keywords
Glucose, Fasting, Liver, Kidney, Energy

1.0 Introduction

Human food substances are usually products of photosynthesis, Mbah and Ezeorah [5], by plants of which Carbohydrate is one of such products, Mbah [2]. We know that green plants in the presence of Sunlight use Water and Carbondioxide to produce Carbohydrates. It is in this form that the Glucose found in the human systems is derived when such Carbohydrates are taken as food. When Carbohydrates are taken as food, they are broken down as **Monosacharides, Polysacharides and Diasacharides.** Incidentally, glucose is a monosaccha-
ride. It is in this form, glucose, that the human cells absorb the food for the generation of energy, Mbah, et al [4], Mbah and Ezeorah [5].

When we take in food, this is broken down and then absorbed into the blood stream for circulation to the cells in the body. However, most of the time, we find that the glucose in the blood is in excess of what is required by the cells at that time. The result of this is that the excess glucose is converted to **Glycogen** and then stored in the Liver of the individual concerned. After some time, the glucose level in the blood returns to normal called the basal level. As far as no other food substances are taken, it remains at this level until the food is entirely absorbed to its maximal level and value.

If no food is further taken at this level, then the stored glucose in the form of glycogen will be reconverted back to glucose and sent to the blood for circulation to the cells of the body. If one observes ones normal feeding pattern, we discover that breakfast around 8.00 – 9.00 am, we usually feel heavy and not hungry until around 12.00 noon to 1.00 pm, depending on the type of breakfast food, when again we begin to feel hungry. We find that if nothing is taken within the next 30 minutes to one hour, then we seize to be hungry. The reason for this development is that the glycogen in the Liver is now being released into the blood in the form of glucose and this will beef up the blood-glucose level.

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Our problem then is that if this no-further food intake continues for some long period of time, we then say that the person is fasting. We wish to look for a mathematical model for such condition and possibly verify various states that many a time are observable in fasting individuals. We shall also look into the contributing factors that leads to the maintenance of the daily basal glucose level in such fasting individuals for such a long time until food is eventually taken.

2.0 The liver and the glucose release

The Liver is an organ of glucose production and consumption. It is exposed to insulin concentrations in the portal venous blood. The insulin concentration here is 3 – 10 folds greater than what obtains in the systemic circulation. The Liver is also the sole site of the blood gluco-regulating action of the glucagon. To consider the regulation of the blood glucose level by the Liver, we shall consider the period following an overnight fast and preceding the ingestion of the breakfast meal. Usually, after the overnight fast, the concentration of the hormones (Insulin and glucagon) and substrates (glucose, amino acids, fatty acids) return to the basal levels. The return of the insulin to the basal level ($10 - 20\mu U/m$) results in virtually total cessation of glucose uptake by the insulin-dependent tissues such as resting muscles, adipose tissues and the liver itself. However, the non-insulin-dependent tissues such as the brain, the formed elements of the body and the Renal medulla continues to use the glucose, which theoretically, is put at a combined rate of 2 – 3*mg/kg/minute*.

Under this fasting state therefore, the eventual build up or maintenance of the required basal level of the glucose is realized by the hepatic release of glucose at rates equal to tissue demands and utilization. However, this maintenance of the basal level is made possible through two pathways:- **glycogenolysis**: which is the synthesis of glucose from the glycogen and secondly, **gluconeogenosis:** which is the synthesis of glucose from the pyruvate, lactate, glycerol and amino acids. It is estimated that about 70 – 75 % of the hepatic glucose release is as a result of glycogenolysis while the remaining 30-25 % is due to gluconeogenosis, Vallence-Owen [6].

In reality, the relative contributions from these two pathways are influenced by:

(1) Total glycogen stored in the liver which is usually not less than 70g and about 450g in a 70kg man.

(2) Stored protein-derived amino acids.

We have to note that when the stored glycogen is depleted due to the fact that only the brain requires about 150g daily, then the Splanchnic uptake of alanine gets stimulated. As a result of this, the Kidney becomes an important source of glucose synthesis and this accounts for approximately 50% of the total glucose release into the blood stream. For very long period of starvation, glucose utilization reduces to as much as 50% and at such periods, the brain uses ketonic acid as its main oxidative fuel in place of glucose. More details about glucose level in the blood for fasting individual can be found in Mbah [2] and Felig et al [7].

3.0 The Mathematical models

Davis [1] and later generalized by Mbah [3] had given a mathematical model for an insulin-dependent diabetic patient as:

$$\frac{dx}{dt} = a_1 z - a_2 xy - a_3 x + a_4(\bar{x} - x) \tag{3.1}$$

$$\frac{dy}{dt} = b_1 x - b_2 + b_3(\gamma t + \delta) \tag{3.2}$$

where x = glucose level, y = insulin level, z = food intake in the form of glucose, $\bar{x}$ = the basal level of the glucose, a_1, a_2, a_3, a_4, b_1, b_2, b_3, γ and δ are all constants, Mbah [3].

If we now consider an individual who is non-diabetic, then equation (3.2) now becomes:

$$\frac{dy}{dt} = b_1 x - b_2 y \tag{3.3}$$

Specifically, b_1 measures the level of the glucose stimulation of the pancreas for the release of of insulin. a_1 measures the absorption level of the eaten food into the blood stream in the form of glucose, a_2 measures the rate of conversion of the excess glucose in the blood to glycogen which is the stored in the liver, a_3 measures the general body usage of the glucose as a source of energy for proper cell function in the body, a_4 measures the level of glucose release by the liver as a result of low glucose level in the blood and finally, b_2 measures the insulin usage or consumption as a hormone in the body.

In this present study, the individual of interest is fasting and non-diabetic so that $b_3 = 0$, $a_1 = 0$, and $a_2 = 0$. Of particular note is the fact that $a_2 = 0$ because there is now no excess glucose in the blood stream so that no glucose will be required to be converted to glycogen. If this is the case, then we shall re-model our equations to reflect the theoretical findings as:

$$\frac{dx}{dt} = -a_3 x + a_4 x e^{-\alpha t} + a_5 x \tag{3.4}$$

$$\frac{dy}{dt} = b_1 x - b_2 y \tag{3.5}$$

In equation (3.4), we defined the term $a_4(\bar{x}$ -x) as in equation found in equation (3.1) as shown here because the stored glucose gets depleted exponentially. The term $a_5 x$ reflects other glucose productions from the kidney and other sources other than the Liver.

We shall solve these equations by considering two special cases in relation to the nature of the glucose consumption mechanism by the cells of the body while fasting.

Case 1: When a_3 is constant at all times.

If this is the case, we solve equations (3.4) and (3.5) to obtain the solutions:

$$x(t) = x_o \left\{ \exp\left[\frac{a_4}{\alpha}\left(1 - e^{-\alpha t}\right) + (a_5 - a_3)t \right] \right\} \tag{3.6}$$

$$y = b_1 e^{-b_2 t} x_o \int \exp\left[\frac{a_4}{\alpha}\left(1 - e^{-\alpha t}\right) + (a_5 - a_3)t \right] e^{b_2 t}\, dt + c e^{-b_2 t} \tag{3.7}$$

where x_0 is the glucose level stored in the liver just before the fasting began.

Equation (3.7) measures the producible glucose level in the blood or body for such fasting individual. We shall note that for such a person, the released insulin is principally for utilization by the individual cells in the body in the process of glucose entry into the cells, Mbah [2]. Hence, the quantity released is just enough to perform this function since there is no excess glucose in the blood. However, if for any reason we have excess glucose, as will be shown in the analysis, then much of the insulin will be released which again will return the glucose level to the basal level. In general, the stimulating effect of the glucose for the release of insulin is very minimal in a fasting individual so that we can assume b_1 to be very small.

Case 2: When a_3 is a function of time.

Suppose that a_3 is a function of time since it can be reduced to as low as 50% of the former value as the days of fasting increases. Suppose further that this variation is linear in time so that we can define a_3 as:

$$a_3 = a_3(1 - \beta t) \tag{3.8}$$

If this is the case, then equation (3.4) becomes

$$\frac{dx}{dt} = -a_3(1 - \beta t)x + a_4 x e^{-\alpha t} + a_5 x \tag{3.9}$$

$$\Rightarrow \frac{dx}{dt} = \left[-a_3(1 - \beta t) + a_4 e^{-\alpha t} + a_5 \right] x\,.$$

Solving this equation, we get:

$$x(t) = x_o \exp\left\{\left[a_5 - a_3\left(1 - \frac{\beta t}{2}\right)\right]t + \frac{a_4}{\alpha}\left(1 - e^{-\alpha t}\right)\right\} \qquad (3.10)$$

Equation (3.10) gives the producible glucose level in the entire body of the individual at all times given that the cell absorption level decreases with time and defined as stated above.

4.0 Analysis

In this analysis, we wish to see the effect of variations in the constants as well as variations in the glucose level producible over the fasting period of time. To do this, we assume the values $\alpha = 0.05$, $a_5 = 0.01$, $a_4 = 0.01$, $x_0 = 250$ and $a_3 = 0.095$. In figures (4.1) and (4.2), we can see that after 14dys of fasting, the much that can be released by the liver, kidney and other sources will be such that the storage in the liver will be exhausted even before then. Beyond this 14 days period, we can see from the figures that the glucose level will be below the required basal level and the individual will loose weight very highly.

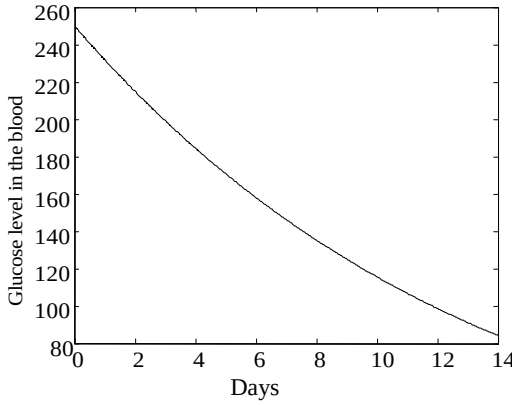

Figure 4.1: Variation in the glucose level over 14 days

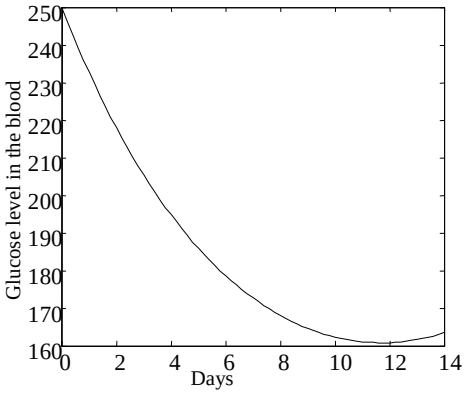

Figure 4.2: Variation in the glucose level over 14 days due to change in a_3

Figure 4.2 is obtained by assuming that a_3 varies and can reduce to as low as 50% of the original value at the start of the fasting. In getting the graph in figure 4.2, we assumed that the 50% reduced value of a_3 is usually attained after 7 days. However, from the figure 4.2, we can see that after 12 days, the curve start increasing. From further analysis of our result, we discover that a lot of things or factors may be responsible for this among which may be our wrong assumption about the time it takes a_3 to reduce to about 50% of its original value. Another possible reason may be that a_5 may have smaller value than assigned to it. This means that if actually glucose usage by the body decreases over the period of fasting, it might equally mean that a_5 may equally be reducing or increasing over time. In our graph above we assumed constant value for a_5. We therefore in figures (4.3) and (4.4) show the variation in the value of a_5 for the two cases as treated. We retained the same values for the constants as in figures (4.1) and (4.2). These are shown as:

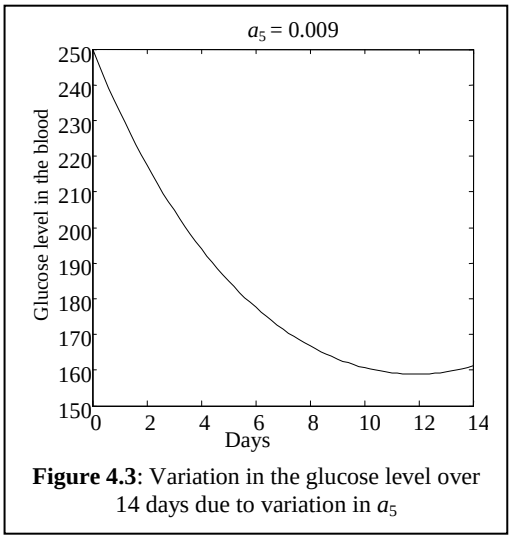

Figure 4.3: Variation in the glucose level over 14 days due to variation in a_5

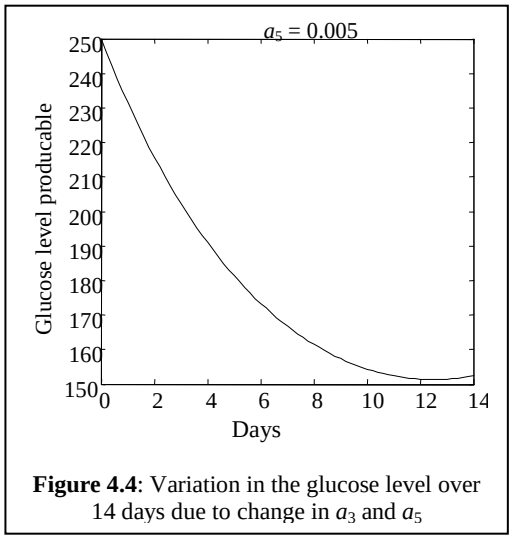

Figure 4.4: Variation in the glucose level over 14 days due to change in a_3 and a_5

When a_5 = 0.009, the curve starts to increase after about 12.5 days and the producible glucose level is 159.139 which is less than as in figure (4.2) where it was 161.157. In figure (4), a_5=0.005, the glucose level did not start to increase even up to the 14th day. Comparing this with figure (4.2), we see that the producible glucose level is still high (152.4563) as against (84.135) in figure\(4.2).

For figures (4.5) and (4.6), we still used the same values but for a_4 which changes. As can be seen in the figures, the glucose levels are still very high (93.0211 and 180.83). a_4 varying means that the glucose release rate from the Liver is not constant over time. This assumption may be true and if so, may be partly responsible for admissible long period of fasting without the individual dying. We noticed on further investigation that if a_5=0 and a_3 = 0.095, the contribution in the blood glucose level from the Liver is about 80.8687 units. This goes to confirm that if actually somebody can fast beyond two weeks, then there must be other sources of glucose release to account for the needed glucose for such period. It is generally noticed that for a long period of fasting, the individual starts loosing consciousness once the releasable glucose from the liver is nearly exhausted.

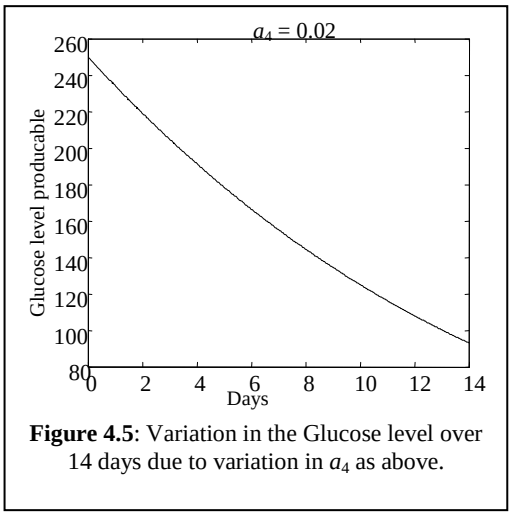

Figure 4.5: Variation in the Glucose level over 14 days due to variation in a_4 as above.

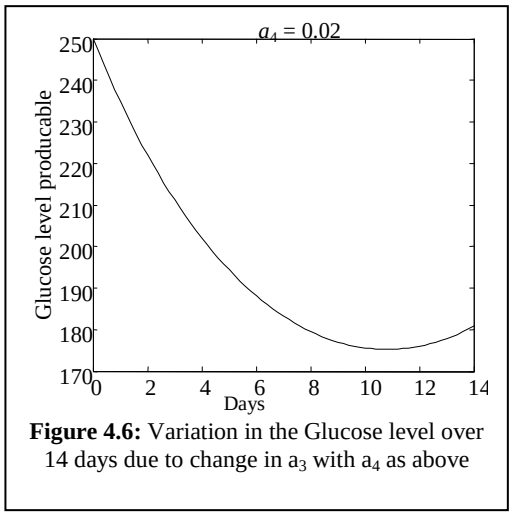

Figure 4.6: Variation in the Glucose level over 14 days due to change in a_3 with a_4 as above

In figures (4.7) and (4.8), we considered a case where all the variables are the same as before while only x_0 changes. For the two cases, we considered a case where x_0 = 300 instead of 250. We saw that the producible glucose level is higher here as compared to what obtained in figures (4.1) and (4.2). This is expected and goes to confirm that eating well before embarking on fasting gives room for greater glucose storage and thus longer period of successful fasting.

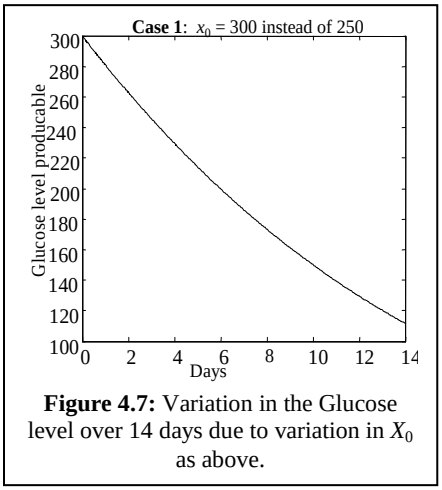

Figure 4.7: Variation in the Glucose level over 14 days due to variation in X_0 as above.

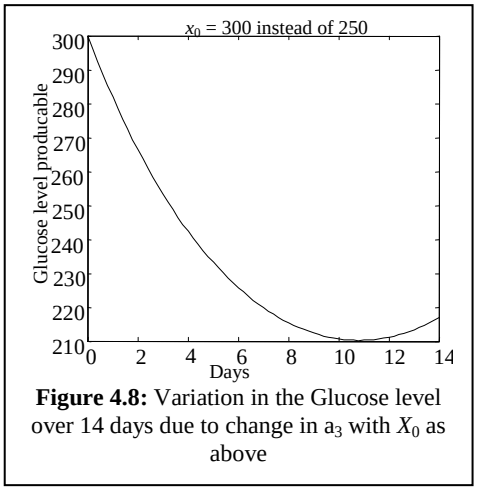

Figure 4.8: Variation in the Glucose level over 14 days due to change in a_3 with X_0 as above

Summarizing therefore, I would wish to say that fasting by individuals has to be properly checked to ensure that people who has the ability can only engage in it. We have seen that the quantity of glucose storable in the Liver and other convertible materials by the Kidney and other sources determine to a large extent the length of time one can fast. Also, the rate of body usage of the stored glucose is very vital in the conservation of glucose for longer fasting. Engagement in extensive energy sapping activities while fasting can be very dangerous and should therefore be discouraged to avert death due to hypoglycemia or coma due to loss of memory.

5.0 Conclusion

We have been able to show that the release of glucose from the liver of a fasting non-diabetic person varies over time. We similarly showed that the subsequent generation of glucose through the creb cycle using the fats and other deposits in the body similarly varies over the time. We equally showed that the level of Glucose stored in the body or storable in the body has some good influence on the length of time one can fast. Altogether, we have shown that many factors contribute to the length of time one can fast.

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