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Mathematical Modeling of Diabetes and Hypertension Co-Infection.

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Abstract - Lifestyle diseases have currently become rampant and take toll of the population due to lack of absolute treatment strategy, thus the biggest burden is on the control, which requires substantial financial resources in the care and treatment of infected patients. Diabetes and hypertension are among the common autoimmune diseases commonly occurring in patients, escalating the total effect on the infected and affected individuals. In this study, a mathematical model was formulated and analyzed to describe the disease dynamics, and determine the threshold values of significant parameters for optimal control strategy. The formulated model was tested for its positivity, stability and robustness and sensitivity of parameters evaluated. It was found that the model will guide the lifestyles desired to control the occurrence of the disease, thus improving the health living of the population and reduce the financial burden of treatment and care of patients.

Keywords - Diabetes, Hypertension, Stability, Robustness.

I. INTRODUCTION

Mathematical modeling involves the use of mathematical techniques and equations to represent and analyze real-world phenomena. In biology and chemistry, it serves as a powerful tool to understand complex systems and predict their behavior.

In biology, mathematical models can simulate population dynamics, disease spread, or even molecular interactions within cells. For instance, epidemiological models help predict the spread of infectious diseases and guide public health interventions. In chemistry, mathematical modeling is used to study reaction kinetics, optimize chemical processes, and explore molecular structures. Computational chemistry, for example, relies on models to predict how molecules interact, aiding in drug discovery and material design, while in biochemistry, reaction dynamics and immune pathways can be described using mathematical models using Michaelis Menten reaction kinetics.
By transforming intricate biological and chemical systems into manageable equations, usually differential equations, mathematical modeling enables scientists to uncover insights, make predictions, and solve real-world problems efficiently.

In this study, mathematical models are used to describe and analyze biochemical transduction pathways involved in the control of blood sugar and blood pressure, some common autoimmune problems commonly called Diabetes and Hypertension respectively.

Biochemical reaction is the process in which a substrate combines with an enzyme to produce a complex which disintegrate to a product and the enzyme. These biochemical reactions are evidenced in various biological processes which include digestion, gene regulation, plant and animal chemical control, selection and breeding, drug

interaction in disease treatment, immune response, genetic modification for tolerance and increased production, autoimmune disease control, among others. These biochemical reactions are inevitable in human system, and forms the basis for all living organisms, therefore important in this study.

Diabetes

Diabetes is a chronic autoimmune condition characterized by elevated blood glucose (blood sugar) levels due to the body's inability to use or produce insulin effectively (De Silva et al., 2012). Insulin is a hormone produced by the pancreas that plays a crucial role in regulating blood sugar levels and facilitating glucose uptake by cells for energy. When there's a disruption in insulin production or its action, blood sugar levels can rise or drop to unhealthy levels.

There are three main types of diabetes: Type 1 Diabetes is a condition where the body's immune system mistakenly attacks and destroys the insulin-producing cells in the pancreas, leading to low levels of insulin in the blood. People with type 1 diabetes require lifelong insulin therapy to boost their blood sugar levels. Type 1 diabetes usually develops into a metabolic disorders in which there are high blood sugar levels over a prolonged period.

Type 2 Diabetes: This is the most common type of diabetes and is often related to lifestyle factors such as obesity, physical inactivity, and a poor diet. In type 2 diabetes, the body becomes resistant to the effects of insulin, and the pancreas may not produce enough insulin to maintain normal blood sugar levels. It typically develops in adulthood, but due to increasing rates of obesity in younger populations, it's also becoming more common in children.

Type 3 Diabetes is also called Gestational Diabetes: This type occurs during pregnancy and is characterized by high blood sugar levels that develop and typically resolve after childbirth. However, women with gestational diabetes have an increased risk of developing type 2 diabetes later in life.

Hypertension

Hypertension, also called high blood pressure, is a condition present when blood flows through the blood vessels with a force greater than normal. It is known that blood is a means of transport of oxygen and food nutrients together with other vital cells and ions from one point to the other, and thus there is an optimum velocity that is expected to flow, to give time to various reactions and penetration of cells. Hypertension can strain the heart, damage blood vessels, and increase the risk of heart attack, stroke, kidney problems, and death.

Blood pressure is controlled by a combination of organs and hormones, working together and independently. The liver and the kidney controls the blood volume, while the heart controls the pumping speed, meanwhile hormones like adrenaline, aldosterone and angiotensin regulate the heart rate, blood vessel constriction and fluid flow.

Hypertension and Diabetes Co-infection

In most cases, patients are diagnosed with both hypertension and diabetes simultaneously. This joint illness is called a co-infection. Hypertension (high blood pressure) and diabetes are closely linked, often coexisting as part of metabolic syndrome. Here are some key connections: Both conditions share common risk factors, such as obesity, sedentary lifestyle, and unhealthy diets. These factors contribute to the development of both hypertension and diabetes. Diabetes can damage blood vessels and the cardiovascular system, increasing the risk of hypertension. Elevated blood sugar levels can also lead to increased fluid retention, raising blood pressure. When both conditions are present, they can worsen each other. For example, hypertension can accelerate complications of diabetes, such as kidney damage, while diabetes can exacerbate cardiovascular strain caused by high blood pressure.

Because of this relationship, most people with diabetes will eventually have high blood pressure, along with other heart and circulation problems and vise versa.

Managing these conditions often involves lifestyle changes, medication, and regular monitoring to prevent complications. It's a delicate balance, but

understanding the connection helps in effective treatment and prevention. It is for this reason that this research studies hypertension and diabetes co-infection to understand the interaction dynamics and determine the significant parameters that when their threshold values are known, can help in the control and management of the two lifestyle diseases.

II. MATHEMATICAL MODELING

Various studies have been done in relation to diabetes and hypertension separately and also jointly along medical frontiers. Mathematical models have also been proposed to account for various disease dynamics of the two diseases, and in this section, a summary of what has been done is presented as a basis for this research and to show the knowledge gap under study.

Modeling Diabetes Infection

The existing literature surrounding diabetes models dealt with insulin and glucose dynamics Derouich and Boutayeb (2002), epidemiology of the disease, Staines et al. (1993) and cost and risk models (Bagust et al., 2002). The existing research have also presented numerous insulin-glucose models to represent the dynamics of diabetes. Magombedze et al. (2010) modeled immune regulation of Type 1 diabetes. They noted that Type 1 diabetes entails the progression of loss of pancreas cells leading to failed production of enough insulin for the conversion of glucose to energy. They formulated a mathematical model that incorporates the role of cytotoxic T-cell and regulatory T-cells in Type 1 diabetes. The study conclusively showed that diabetes is a combination of events and that no single event is fully responsible for its occurrence. The study also noted that control of diabetes could pay more when autolytic T-cells are regulated. The study further stated that diabetes-free state in diabetes individual is unpredictable and sensitive.

(Trudeau et al., 2003) studied diabetes of type 1 in NOD mice by quantifying autoreactive T-cells in the blood peripherals. In their study, compartmental model with five variables was used to formulate a system of five ordinary differential equations to account for activated T cells A(t), effector T-cells E(t), memory T-cells M(t), β-cells in the pancreas B(t) and the specific antigen peptides P(t) which feedback and affect the levels of activated T cells. The study showed that, the presence of cyclic T cell waves provide a predictor for humans becoming diabetic. This is a positive equilibrium, with elevated effector cells, which continually kill β-cells. This corresponds to autoimmune attack leading to the onset of diabetes.

Similar studies on detection of diabetes was done by Rosado (2009). This model monitored the concentration levels of glucose concentration, glucagon concentration and hormonal concentration together with externally supplied sources. The model formulated was 53% accurate in predicting diabetic patients using glucose tolerance test. This results are similar to the findings of Sandhya and Deepak (2011). In their study of glucose –insulin regulatory system, mathematical model was formulated using differential equations accounting for plasma glucose, plasma insulin concentration and insulin concentration in the peripheral compartments.

The use of partial differential equations to model diabetes was done by (Anthena Makroglou, Jiaxu Li, & Kuang, 2006). They used partial differential equations in their study, to show that insulin and glucose regulatory system is the major determinant of diabetes infection. The study modeled the concentration of hexameric and dimeric insulin and glucose concentrations in the blood after injection. This was done to study the response of the regulatory system in controlling the blood sugar levels.

The effect of insulin receptor dynamics in the glucose regulatory system was also studied by (Ryan, Danielle, Daniel, Thomas, & Stephen, 2001). Using a system of ordinary differential equations, β-cell mass, insulin and glucose kinetics were analyzed. Findings indicated that basal levels of beta cell mass, insulin, glucose and insulin receptors approaches the physiological state of hyperglycemic glucose levels equilibrium or approach a saddle-node bifurcation that leaves the pathological equilibrium as a global attractor. They further showed that exercises can

increase insulin sensitivity by 36%, thus reducing the required insulin levels for a constant glucose concentration. Similarly, sedentary lifestyle, along with obesity can lower insulin sensitivity by 50-100%. In general, a person whose insulin sensitivity drops by 60% will be insulin resistant, and progress to be diabetic (Ryan et al., 2001).

Modeling Hypertension

Mathematical modeling of hypertension is a multidisciplinary approach that integrates mathematics, physiology, and computational techniques to understand the dynamics of blood pressure regulation and its pathological deviations. These models provide insights into the mechanisms of hypertension and aid in predicting the outcomes of medical interventions.

Blood pressure is measured using two values: systolic pressure and diastolic pressure. Systolic pressure is higher, indicating the pressure when the heart beats and pumps blood into the arteries. Diastolic pressure is the lower number, representing the pressure when the heart rests between beats. A normal blood pressure reading is typically around 120/80 mmHg (millimeters of mercury). Hypertension is often defined as having sustained blood pressure readings consistently higher than 130/80 mmHg.

Hypertension is common among patients with diabetes, and its prevalence is dependent on type and duration of diabetes, age, race, sex, presence of kidney disease, Body Mass Index (BMI) (De Boer et al., 2017). The blood pressure is dependent on the work rate of the heart and the resistance offered by the wall of the blood vessel. Hypertension has significantly been linked with chronic heart and kidney disease (Waezizadeh et al., 2018). Therefore, many researchers have come up with various models aiming to model hypertension. For instance, a study by Waezizadeh et al. (2018) used deterministic and stochastic approaches to model chronic heart disease and chronic kidney disease. Waezizadeh et al. (2018) relied on hypertension and stress to estimate the extent of damaging or removing nephrons, which are vital for kidney functions.

Mathematical models of hypertension often focus on the cardiovascular system, which includes the heart, arteries, veins, and blood. Blood flow and pressure dynamics are governed by complex interactions between these components. Models typically use equations derived from fluid dynamics, such as the Navier-Stokes equations, to describe blood flow. These equations account for the non-Newtonian properties of blood and the viscoelastic nature of blood vessel walls. In these models, there are always two equilibrium points; sick and healthy states as described by (Kutumova, Kiselev, Sharipov, Lifshits, & Kolpakov, 2022).

Diabetes and Hypertension Coinfection

Co-infection of diabetes and hypertension is a significant health concern that arises when an individual simultaneously experiences both conditions Krolewski et al. (1988). Diabetes, characterized by elevated blood glucose levels, and hypertension, characterized by high blood pressure, are two common chronic diseases that often coexist, leading to complex interactions and potential health complications. The co-occurrence of diabetes and hypertension is prevalent and has become a global health issue. These two conditions share risk factors such as obesity, a sedentary lifestyle, and a poor diet. Co-infection amplifies the risk of cardiovascular diseases, stroke, kidney problems, and other complications, making early detection and management crucial.

The mechanisms underlying the co-infection are intricate and multifaceted. Insulin resistance, oxidative stress, inflammation, and endothelial dysfunction are common pathways that link diabetes and hypertension. Understanding these mechanisms is vital for designing effective treatment strategies. Through research, mathematical modeling, various variables and parameters can be brought together and simulated to give a clear prognostic picture crucial for improving the well-being of individuals affected by these conditions. This paper handles this problem using reaction kinetics of enzymes and substrates to understand the genetic transduction pathways of controlling the co-infection.

III. MATERIALS AND METHODS

In this article, the methods to be used in the study of the model are presented. We formulated a mathematical model using differential equations based on the schematic diagram below. Mathematical models are very fundamental techniques used to understand the biochemical reactions and therefore single out specific variables that may need to be studied further using experiments. Models help in the understanding of metabolism, genes and diseases as discussed by (Nijhout, Best, & Reed, 2015). In this section, two models are formulated: one for hyperglycemia (high blood sugar) and the other for hypertension, then thee two are merged to describe a co-infection situation.

Diabetes Model description

Regulation of blood sugar involves various enzymes, hormones and organs involved in the production of the enzymes and hormones. The normal regulation of blood sugar is described as follows: Through uptake of sugar or glucose, the digestive system will process the food leading to high blood glucose levels. If these levels go beyond the normal range of 70 – 140mg/dL, insulin is secreted by beta cells in the Pancreas in response to this. Insulin is a hormone which activates uptake of glucose from the blood stream and glycogenesis, a process where glucose is converted to glycogen and stored in the liver or muscles. At the same time, insulin activates lipogenesis, a process of converting excess glucose to fatty acids and storage in the adipose tissue. A feedback mechanism determined by the level of glucose in the blood sugar is relayed to the beta cells in the Pancreas. This process continues until equilibrium is achieved. On the other hand, If the blood glucose is low, and needed for metabolism, the alpha cells in the Pancreas is activated to produce Glycagon hormone, which activates Glyconeogeneses and Glycogenolysis processes, which involves synthesis of glycose from amino-acids in the adipose tissue and breakdown of glycogen into glucose in the liver respectively. This will increase the blood glucose levels. Diabetes occurs when beta cells are destroyed, and thus producing insufficient insulin or alternatively, the uptake is reduced die to insulin resistance by cells, despite sufficient insulin secretion.

Diabetes Model Flow chart

The description of blood glucose levels regulation and the occurrence of diabetes can be represented in flow chart below.

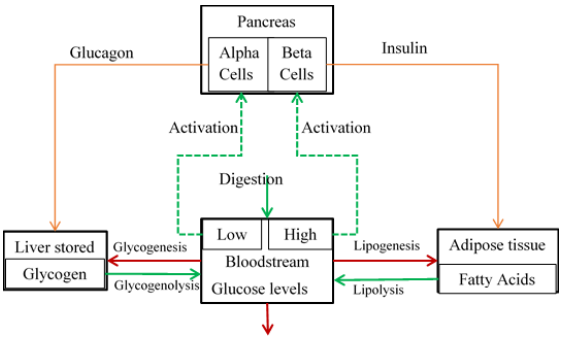

Figure 1 Flow chart showing Blood Glucose Level Regulation

From the flow chart in Figure 1 above, the processes indicated are carried out by reaction of enzymes and substrates, which are activated and controlled by hormones; Glucagon and Insulin. The following is a description of the enzyme – substrate reaction kinetics represented in the chart in Figure 1 above.

Substrate–Enzyme Reaction Kinetics

Two main processes are involved in the blood glucose regulation dynamics; one involving raising blood glucose level; and the decreasing blood glucose levels, explained below.

Increasing Blood Glucose Levels

The main hormone responsible for this is Glucagon, secreted by alpha cells in the Pancreas. When low level of blood glucose is detected, alpha cells in the Pancreas are triggered, and they secrete Glucagon hormone, which activates Glycogen Phosphorylase enzyme. This enzyme reacts with glycogen substrate stored in the liver and muscles, producing an Enzyme Substrate complex (Glucose-1-Phosphate and Glucose-6-Phosphate), which disintegrates to Glucose as a product and the enzyme remains unconsumed. The same process can be triggered by Epinephrine hormone (in the adrenal glands) during stress or physical activity. In this research, we limit ourselves to the normal circumstances. This process

of breaking down glycogen to glucose is called Glycogenolysis.

Decreasing Blood Glucose Levels

When the blood glucose level is high, the beta cells in the Pancreas are triggered, and they secrete insulin, and hormone which activates Glycogen Synthase enzyme, which reacts with Glucose-6-Phosphate substrate to produce Glycogen substrate. This process is called Glycogenesis. Other processes of reducing blood sugar include glycolysis (breakdown of glucose to produce energy) and Lypogenesis (conversion of excess glucose into fats). Thus research looks into the glycogenesis process only.

Hypertension Modeling

Blood pressure is maintained by a remarkably intricate balance of enzyme-mediated pathways, hormonal signals, and substrate conversions. These processes work together to ensure the cardiovascular system responds appropriately to changes in blood volume, flow, and vascular tone. There are four pathways of blood pressure regulation, these include: The Renin-Angiotensin-Aldosterone System (RAAS): The Nitric Oxide (NO) Pathway: The Endothelin System: and The Prostacyclin Pathway. The first two are the major players in balancing blood pressure and will be dealt with in detail.

As described earlier, this is a condition where the blood pressure exceeds the normal range. This normal range is achieved when enzymes and substrate reaction is at an equilibrium (Dillon, 2014). This equilibrium is created by joint operation by RAAS and NO pathways.

Renin Angiotensin Aldosterone System

This system is vital in the process that leads to increase in the blood volume and subsequently increase blood pressure. Renin, and enzyme produced from the kidney converts Angiotensinogen, a substrate stored in the liver into Angiotensin – I, which is further converted to Angiotensin – II by ACE enzymes secreted by the endothelial cells. The Angiotensin – II is a vasoconstrictor complex, which will stimulate the adrenal cortex in the Kidney to release Aldosterone hormone, which will increase sodium and water reabsorption in the kidney, increasing the water volume, thus increasing blood pressure (Genest, 1961; Hall, 1991).

High blood pressure is experienced when there is a defective RAAS mechanism, where excessive production of Angiotensin II leads to chronic vasoconstriction, and excess production of Aldosterone hormone. This will lead to excess retention of water and sodium, making the heart to overwork trying to pump the excess blood volumes against increased vascular resistance, leading to sustained elevated blood pressure.

Nitric Oxide (NO) Pathway

Here, the mechanism is meant to downregulate or counter balance vasoconstriction by producing a vasodilator to ensure that blood pressure is not excessive. This counterbalance is achieved by a mechanism where Nitric Oxide Synthase enzyme from the endothelial cells react with L-Arginine substrate in the liver to produce Nitric Oxide complex, which activates the production of soluble Guanylate Cyclase (sGC), which diffuse into the smooth muscles causing relaxation and vasodilation of blood vessels, subsequently reducing blood volume.

In the case of hypertension, the NO pathway is impaired, lowering sensitivity of NO by muscles or inadequate production of NO is experienced, thus leading to inadequate vasodilation and consequently leading to persistent elevated blood pressure (Lind, 2000).

Blood Pressure Regulation Schematic Diagram

The following diagram illustrates the action and reaction of involved enzymes, substrates and hormones in the regulation of blood pressure.

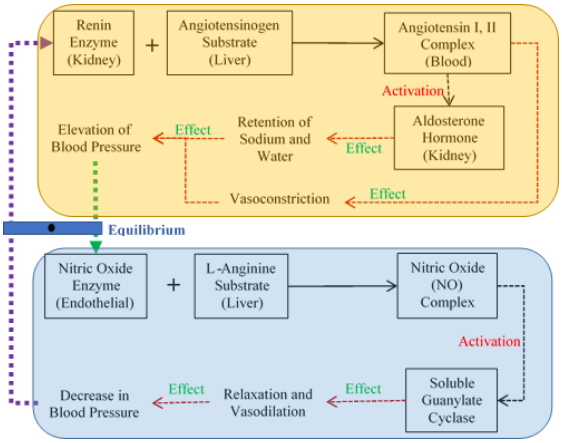

Figure 2 Blood Pressure Regulation Schematic Diagram

Hypertension Model Equations

From the diagram in Figure 1 and Figure 2 above, the following schematic representation of reactions is generated. Define the following variables to represent enzymes and substrates involved in the reaction as follows: Let S_Hr,E_Hr,C_Hr P_Hr,S_Hl,E_Hl,C_Hl and P_Hl denote the Substrate, Enzyme, Complex and Product respectively with the subscripts Hr and Hl denoting Hypertension raising and Hypertension lowering respectively. Then we have the following schematic representation of reaction.

$$S\_Hr+E\_Hr \rightarrow\_(k\_1\ )\ C\_Hr \rightarrow\_(k\_2\ )\ P\_Hr+E\_Hr \quad (3.1)$$

$$S\_Hl+E\_Hl \rightarrow\_(k\_3\ )\ C\_Hl \rightarrow\_(k\_4\ )\ P\_Hl+E\_Hl \quad (3.2)$$

Equations (3.1) and (3.2) relate to regulation of Hypertension, with the constants k_i,i=1,2,…,4 representing the reaction rates.

From the system of equations in (3.1 – 3.2), we arrive at the following differential equations. Let the concentrations of the enzymes, substrates, products and the complexes involved be denoted by s=[S], then we have:

$$(ds\_hr)/dt=a\_1\ h\_1-k\_1\ \ ⟦s\_hr\ e⟧\ \_hr \quad (3.3)$$

$$(de\_hr)/dt=-k\_1\ \ ⟦s\_hr\ e⟧\ \_hr+k\_2\ c\_hr \quad (3.4)$$

$$(dc\_hr)/dt=k\_1\ \ ⟦s\_hr\ e⟧\ \_hr-k\_2\ c\_hr \quad (3.5)$$

$$(dp\_hr)/dt=k\_2\ c\_hr \quad (3.6)$$

$$(ds\_hl)/dt=a\_2\ h\_2-k\_3\ \ ⟦s\_hl\ e⟧\ \_hl \quad (3.7)$$

$$(de\_hl)/dt=-k\_3\ \ ⟦s\_hl\ e⟧\ \_hl+k\_4\ c\_hl \quad (3.8)$$

$$(dc\_hl)/dt=k\_3\ \ ⟦s\_hl\ e⟧\ \_hl-k\_4\ c\_hl \quad (3.9)$$

$$(dp\_dr)/dt=k\_4\ c\_hl \quad (3.10)$$

Where h_1and h_2 are hormone 1 and hormone 2 activating increase in blood pressure and activating decrease in blood pressure at a rate of a_1 and a_2 respectively. Apart from the eight equations (3.3 – 3.10), another equation linking the equilibrium of the increasing and decreasing factors of blood pressure need to be formulated representing the blood pressure levels (b_p) in terms of the hormones activating the increase and decrease, together with the products of the enzyme-substrate reactions.

Let b_p denote the blood pressure levels, let ξ be the proportional increase of pressure due to Aldosterone Hormone product, and let η be the overall decreasing effect of sGC product, then

$$(db\_p)/dt=ξk\_2\ c\_hr\ b\_p-ηk\_4\ c\_hl\ b\_p \quad (3.11)$$

Equation (3.11) describes the blood pressure levels, and it is at equilibrium if $ξk\_2=ηk\_4$.

Diabetes Model Equations

From the diagram in Figure 1, the model schematic flow of enzyme-substrate reaction in blood sugar regulation is presented below. The molecules involved are: Glucogen enzyme reacting with glucagon Phosphatase substrate to yield Gluco-1-Phosphate and gluco-6-phosphate, which disintegrate to yield glucose product and free enzyme. This is activated by glucagon hormone when the blood sugar is low.

When blood sugar is high, Insulin is released which activates glucogen synthase enzyme, reacting with glucose-6-phosphatase substrate to yield a complex which disintegrates into glycogen substrate whichreduces blood sugar levels. This is represented by the schematic equations;

S_Dr+E_Dr →_(k_5) C_Dr →_(k_6) P_Dr+E_Dr (3.12)
S_Dl+E_Dl →_(k_7) C_Dl →_(k_8) P_Dl+E_Dl
(3.13)

From the system of equations in (3.12 – 3.13), we arrive at the following differential equations. Let the concentrations of the enzymes, substrates, products and the complexes involved be denoted by s=[S], then we have:

(ds_dr)/dt=a_3 h_3-k_5 〖s_dr e〗_dr
(3.14)

(de_dr)/dt=-k_5 〖s_dr e〗_dr+k_6 c_dr
(3.15)

(dc_dr)/dt=k_5 〖s_dr e〗_dr-k_6 c_dr
(3.16)

(dp_dr)/dt=k_6 c_dr
(3.17)

(ds_dl)/dt=a_4 h_4-k_7 〖s_dl e〗_dl
(3.18)

(de_dl)/dt=-k_7 〖s_dl e〗_dl+k_8 c_dl
(3.19)

(dc_dl)/dt=k_7 〖s_dl e〗_dl-k_8 c_dl
(3.20)

(dp_dr)/dt=k_8 c_dl
(3.21)

Apart from the eight equations (3.14 – 3.21), another equation linking the equilibrium of the increasing and decreasing factors of blood sugar need to be formulated representing the blood sugar levels (b_s) in terms of the hormones activating the increase and decrease, together with the products of the enzyme-substrate reactions.

Let b_s denote the blood pressure levels, let δ be the proportional increase of pressure due to Glucagon Hormone product, and let σ be the overall decreasing effect of Insulin hormone, then

(db_s)/dt=δk_6 c_dr b_s-σk_8 c_dl b_s
(3.22)

Equation (3.22) describes the blood sugar levels, and it is at equilibrium if δk_6=σk_8.

3.5 Hypertension – Diabetes Coinfection Model

In an event that an individual has defective blood pressure regulation and defective blood sugar regulation, a new equilibrium of coinfection is created. The stability and parameter values for the existence of this equilibrium is studied using the equations (3.11) and (3.22) with a competitive feedback mechanism connecting the two. This is given by;

(db_p)/dt=ξk_2 c_hr b_p-ηk_4 c_hl b_p-r_1 β_12 (b_p b_s)/K_1 (3.23)

(db_s)/dt=δk_6 c_dr b_s-σk_8 c_dl b_s-r_2 β_21 (b_p b_s)/K_2 (3.24)

Where the parameters r_1,r_2 are the production rates of the respective molecules with carrying capacity of K_1,K_2 respectively while β_12,β_21 are the competitive rates for the same molecules, which can be enzymes, substrates or hormones.

IV. CONCLUSION

In this study, we developed a mathematical model to analyze the dynamics of co-infection with diabetes and hypertension within a population. The model incorporates key biological and epidemiological factors, including transmission rates, progression between disease stages, treatment interventions, and interactions between the two conditions. Our analysis reveals that the coexistence of diabetes and hypertension significantly increases the disease burden in affected individuals and the overall population. The model shows that the presence of one condition can exacerbate the progression and complications of the other, highlighting the importance of integrated management strategies. Sensitivity analysis identifies critical parameters—such as the rates of diagnosis, treatment adherence, and lifestyle modification—as effective points for intervention.

Furthermore, the model demonstrates that without coordinated public health efforts, including early screening and consistent treatment, the co-infection prevalence can rise steadily, posing a serious threat to public health systems. We conclude that mathematical modeling serves as a valuable tool in understanding the complex interplay between chronic diseases like diabetes and hypertension. It provides a framework for predicting future trends and evaluating the impact of different control strategies. Policymakers and healthcare providers should prioritize joint interventions, awareness campaigns, and policies that address these diseases concurrently to reduce long-term morbidity and healthcare costs.

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