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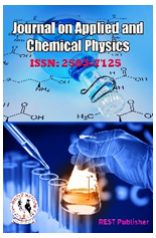

The Role of the Renin-Angiotensin-Aldosterone System (RAAS) in Blood Pressure Regulation and its Implications in Hypertension

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ABSTRACT

The renin-angiotensin-aldosterone system (RAAS) controls both blood pressure and cardiac function. When dysregulated, it can contribute to conditions like hypertension, cardiovascular, and renal diseases. Targeting the RAAS with medications can help improve these conditions. The RAAS maintains fluid balance and electrolyte levels, crucial for regulating blood pressure through a complex network involving the endocrine system and vascular resistance. Under normal conditions, the RAAS responds to decreased blood volume by activating juxtaglomerular cells in the kidney, leading to the secretion of renin The primary pathology in cases of chronic hypertension, kidney, and heart failure is attributed to a dysfunctional to sustained high blood pressure, necessitating its regulation. Typically prescribed for managing high blood pressure and heart failure, ACE inhibitors serve as key medications. angiotensin II, a hormone implicated in various adverse cardiovascular effects such as hypertrophy, arrhythmias, and impaired ventricular function. Furthermore, excess angiotensin II may provoke organ damage through increased membrane permeability, epithelial cell apoptosis, and cytokine-induced inflammation. The vasodilator effects of ACE inhibitors, facilitated by the Preventing the action of angiotensin II aids in additional reduction of blood pressure regulation. Similarly, Angiotensin Receptor Blockers (ARBs) are employed to manage hypertension and heart failure. By antagonizing the angiotensin II receptors, ARBs prevent the detrimental effects associated with angiotensin II activation. Additionally, aldosterone, another hormone stimulated by angiotensin II, promotes sodium retention in the kidneys and increases water retention, further elevating blood pressure. Furthermore, other factors such as hormones (e.g., adrenaline, vasopressin), endothelial function, and blood viscosity also contribute to blood pressure regulation. Plasma Renin Activity, Angiotensin II Level, Aldosterone Level, Blood Pressure, Hypertension Status: Diagnosed and Medication: ACE Inhibitor (Enalapril). The DEMATEL analysis indicates that Plasma Renin Activity ranks highest, while Aldosterone Level ranks lowest in the RAAS.

1. INTRODUCTION

The Renin-Angiotensin-Aldosterone System (RAAS) plays a pivotal role in regulating blood pressure, electrolyte equilibrium, and fluid levels within the body. Initiated by decreased blood flow to the kidneys or low sodium levels, the enzyme renin is released, Angiotensinogen conversion into angiotensin from promoting entering Angiotensin-converting enzyme The enzyme, or ACE, then This changes the precedent powerful vasoconstrictor called angiotensin II. In addition to narrowing blood arteries and increasing blood pressure, angiotensin II also causes the adrenal cortex to generate more aldosterone [1].

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Because of the way that aldosterone affects the kidneys, blood circulation and blood pressure rise as potassium elimination is enhanced and salt and water reabsorption is promoted. Moreover, angiotensin II directly influences cardiovascular function by stimulating cardiac contractility and promoting the release of anti-diuretic hormone (ADH), further contributing to fluid retention [2]. Dysregulation of the RAAS, such as in cases of hypertension or heart failure, underscores its critical role in cardiovascular health Blood pressure regulation is a vital physiological process essential for ensuring adequate blood flow to tissues and organs while maintaining overall cardiovascular health. It involves a delicate balance in systemic blood flow and the output of the heart, which are impacted by a number of internal processes and feedback cycles. The parasympathetic and parasympathetic of the autonomous nervous system, in particular, are essential for modulating heart rate, myocardial contractility, and vascular tone to adjust blood pressure in response to changing physiological demands [3]. Additionally, hormonal systems secretion of vasopressin (anti-diuretic hormone) by the hypothalamus contribute to blood pressure regulation by modulating vascular tone and fluid balance. Baroreceptors found in the inner layers of the main arteries, monitor blood pressure variations and send messages to the brain, triggering compensatory adjustments to maintain homeostasis. Endothelial cells lining blood vessels also release vasoactive substances such as nitric oxide and prostaglandins, which regulate vascular tone and endothelial function [4]. Additionally, kidney filtration and regenerative processes are crucial in renal mechanisms, which play fluid and Electrolyte balance an important role in regulation, particularly in the context of long-term hypertension. These regulatory mechanisms are intricate and can be implicated in complications such as hypertension, heart attacks, strokes, and cardiovascular diseases, including kidney damage. Hypertension, marked by consistently high blood pressure levels, significantly contributes to adverse health outcomes, including cardiovascular diseases like heart failure, thereby amplifying the risk of various cardiovascular events [5]. Renin-angiotensin-aldosterone system (RAAS) in hypertension in Pathophysiology plays an important role, with dysregulation leading to sustained vasoconstriction, Sodium retention along with water leads to an increase in blood volume results in elevated Normal blood pressure and vascular resistance and implications of RAAS dysregulation in hypertension extend beyond cardiovascular health, impacting renal function, vascular integrity, and metabolic homeostasis [6]. Chronic activation of the RAAS can contribute to renal damage, endothelial dysfunction, and arterial stiffness, further exacerbating hypertension and increasing the risk of organ damage. Additionally, Inflammation and Pro-fibrotic consequences RAAS activation contribute to the progression of vascular remodelling and atherosclerosis, exacerbating cardiovascular complications associated with hypertension [7]. Pharmacological interventions targeting the RAAS, have become cornerstone treatments for hypertension management, highlighting the clinical significance of understanding RAAS-mediated pathways in Pathogenesis of high blood pressure formulation and treatment emerged as a potent strategy in managing kidney disease progression. Over the last decade, research has illuminated Angiotensin II (AngII) as not just a vasoconstrictor but also as a cytokine with growth factor and profibrogenic properties, resembling hemodynamic cytokines [8]. These non-hemodynamic properties contribute significantly to the harm inflicted by RAAS dysregulation, exacerbating kidney dysfunction even in patients with modest impairment, predisposing them to cardiovascular ailments long before aimed at impeding disease progression or even inducing regression to restore renal function. Irrespective of the initiating factors, kidney disease progression is characterized by a sequence of pathological alterations starting from early nephritis and culminating in tubulointerstitial fibrosis, tubular atrophy, and glomerulosclerosis [9]. The RAAS is intricately involved in many of these pathophysiological changes, underscoring its significance affecting its function. The severity of Covid-19 and patient behaviour can influence the absorption of RAAS inhibitors or lead to discontinuation despite medical advice. Despite recommendations from many professional scientific societies against discontinuing RAAS inhibitors, reports indicate a withdrawal of these drugs in patients with conditions where they are commonly used, such as hypertension and heart failure [10]. This withdrawal can result in a notable increase in the risk of complications and death. Specific drugs within the RAAS inhibitor class, such as renin inhibitors and sacubitril, are mentioned. Sacubitril is exclusively used for heart failure treatment. Additionally, various antihypertensive drugs, including monotherapy and combination therapies, often involving a RAAS blocker paired with another agent, are discussed. Furthermore, medications such as statins for lowering cholesterol, oral hypoglycemic agents for managing diabetes, insulin, glucocorticoids, sterilizing non-inflammatory medication, digitalis, inhaled nitrates, a protein called inhibitors, antiplatelet intermediaries, anticoagulant agents, immunosuppressive medication (such as opiates, ant proliferative players, and monoclonal immune systems), short- and persistent beta-agonists are, and other agents utilized for chronic lung conditions were also included in the study [11]. By generating peptide hormones, Activation of the brain

RAAS often results in increased blood volume and hypertension, affecting cognitive functions, memory, experience of pain, sexual behavior, and reactions to stress. Understanding the local activities of Ang II in the brain has been made possible by the use of genetically modified transgenic animal models [12]. Beyond their antihypertensive action, drugs that target RAAS, such as ACE inhibitors and AT1 antagonists, have demonstrated strong protective effects, protecting against high blood pressure and organ damage brought on by diseases like diabetes. It's interesting to note that ACE inhibitors' capacity to lower circulating Ang II levels is not directly correlated with their proven antihypertensive effects. The reason for this disparity is that the peptide is created locally in organs such as the kidney, vessels in the bloodstream, hearts, and brain's activity, where Ang II is generated. independently of plasma levels. The RAAS in these tissues is autonomic ally regulated and plays vital roles in their respective organs' functions and pathophysiology [13]. RAAS activity exhibits a dual nature in its production and action within the brain. Initially, angiotensinogen can be generated and released by astroglial cells through enzymatic processes, subsequently acting on specific receptors. Angiotensins, once extracellular, can then be replaced intracellularly on neurons or glial cells. In a secondary pathway, Ang II is produced within neurons from absorbed angiotensinogen, stored in vesicles, and released to act on receptors on postsynaptic cells. Within the brain, the RAAS influences various physiological mechanisms, including the autonomic nervous system, the hypothalamus-pituitary axis, and vasopressin regulation, impacting cardiovascular function and fluid-electrolyte homeostasis [14].

2. MATERIALS AND METHOD

Plasma Renin Activity (PRA): This gauges the blood's level of a protein called enzyme activity. By starting the system composed of renin, angiotensin, and (RAAS), which then in turn produces the powerful the dilation angiotensin II, the hormone a protein called plays a crucial role in controlling blood pressure. Angiotensin II Level: One hormone that is a component of the RAAS is angiotensin II. The heart rate rises as a result of the constricting blood vessels. Aldosterone Level: The hormone adrenaline, which the adrenal glands are able to generate, aids in controlling the body's brine and water homeostasis. thereby influencing blood pressure. It is also part of the RAAS and acts to increase blood pressure by promoting sodium reabsorption in the kidneys. Blood Pressure: When blood is pumped throughout the body by the heart, it exerts force on the artery walls. It is commonly expressed as a couple of numbers: the diastolic pressure, which is the amount of pressure the heart feels between circulates, and the systolic pressure, which is the tension the heart experiences during a heartbeat. It can be expressed in centimetres of mercury (mmHg). Hypertension Status: This indicates whether the individual has hypertension, which is defined as persistently elevated blood pressure. Hypertension is usually diagnosed when measurements of blood pressure are taken, they often come in at 130/80 mmHg or above. Method: The DEMATEL method is a distinctive approach that highlights issues, identifies their interconnections, and organizes them in a hierarchical structure to tackle problems effectively. It plays a crucial role in uncovering workable solutions by utilizing structural modeling techniques, primarily focusing on understanding the relationships between different organizational components. The method emphasizes the significance of dependencies and contextual factors, which can fundamentally influence the relationships among elements [18]. Modeling this structure involves creating a motivational diagram that takes on various forms to illustrate the interrelatedness and influence among relationships, aiming to provide insight and values for analyzing factors. It discerns causes from effects among systemic factors and visually depicts their relationships, categorizing all elements into cause and effect groups within the system structure. This approach offers researchers a framework to understand the complexities and relationships involved in addressing computer problems and other challenges [19]. This time, the accuracy of reflecting the character remains uncertain. Ultimately, within each integrated PPA, we employ our proprietary DEMATEL method twice, from varying perspectives, to derive conclusive outcomes. The DEMATEL methodology, formally known as Results connections among subsystems, facilitating the aggregation of collective knowledge—a potent technique. However, the inherent vagueness of real-world situations may not be adequately captured by rigid numerical values [20]. DEMATEL, which delves into equity and diversity in investment factors and their interrelations, scrutinizes connections both among investment factors and between them and ANP (Analytic Network Process). This section integrates these analyses, commencing with a thorough examination. It establishes network connections for each ANP factor, prioritizing them before further elaboration. Lastly, it presents a structured outline of the data collection process [21]. The DEMATEL system addresses a spectrum of complex factors,

distinguishing between sender and receiver systems within organizations. It swiftly proceeds to choose the most suitable information management tool, translating this into a strategic approach. Additionally, the ZOGP model aims to optimize businesses' limited resources, integrating various frameworks with defined priorities to enhance management systems and develop more effective plans. [22]. The DEMATEL methodology focuses on identifying cause-and-effect relationships to prevent potential adverse impacts on affected groups, particularly in the context of e-waste management. It suggests that particular attention should be directed towards addressing barriers that significantly influence effective Electronic waste management the decision makers Identify obstacles reduction is a priority should be given to ensure effective implementation, backed by robust legal frameworks and appropriate regulations [23]. In DEMATEL research, the focus lies on its specific applications, particularly in categorizing factors or criteria. This involves delineating important factors through clarifying causal relationships and assessing the degree of interrelationship. The method essentially aims to identify two types of relationships and criteria for analysing impact levels [24]. Traditional decision-makers tend to rely on their intuition and among other influencers, hoping for broad acceptance. Consequently, the latest evaluation serves as a guide, bringing their judgments closer to it. If the final evaluation aligns with their perspectives, they are likely to accept it readily; otherwise, they may reject it. In instances of conflicts like the one described Above, like DEMATEL of unstructured comparisons based methods significant roleis believed to hold [25]. The broad correlation between factors, analyzed in terms of cause and effect categories, is a well-recognized application area for DEMATEL. Consequently, each cited source in this paper regards DEMATEL as a key criterion in decision-making processes. To address conflicting evidence, DEMATEL can determine the significance of each source and its degree of importance. However, for optimal outcomes, it is essential to supplement DEMATEL with theoretical expansion [26], the DEMATEL technique, alongside various integrated methods such as Multi-Criteria Decision Making (MCDM) approaches. These tools are employed to assess staffing plans and establish causality between criteria, forging relationships through DEMATEL and a novel cluster-weighted system, where DEMATEL plays a pivotal role. The complexity in visualizing the structure of relationships and measuring the influence of criteria is addressed by employing DEMATEL. Buyukozkan and Ozturkcan have pioneered an approach that integrates Analytic Network Process (ANP) with DEMATEL, resulting in a technological breakthrough [27].

3. RESULTS AND DISCUSSION

TABLE 1. Renin-Angiotensin-Aldosterone System

	C1	C2	C3	C4	C5	Sum
Plasma Renin Activity	0	13	14	13	15	55
Angiotensin II Level	11	0	12	10	18	51
Aldosterone Level	2	1	0	25	4	32
Blood Pressure	18	11	13	0	12	54
Hypertension Status	12	14	13	10	0	49

Table 1 shows the DEMATEL Decision making trail and evaluation laboratory in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 sum this value.

Figure 1 shows the DEMATEL Decision making trail and evaluation laboratory in Renin-Angiotensin-Aldosterone System with respect to Plasma Renin Activity, Angiotensin II Level, Aldosterone Level, Blood Pressure and Hypertension Status sum this value.

TABLE 2. Normalization of Direct Relation Matrix

	C1	C2	C3	C4	C5
Plasma Renin Activity	0	0.236363636	0.254545455	0.236363636	0.272727273
Angiotensin II Level	0.2	0	0.218181818	0.181818182	0.327272727
Aldosterone Level	0.036363636	0.018181818	0	0.454545455	0.072727273
Blood Pressure	0.327272727	0.2	0.236363636	0	0.218181818
Hypertension Status	0.218181818	0.254545455	0.236363636	0.181818182	0

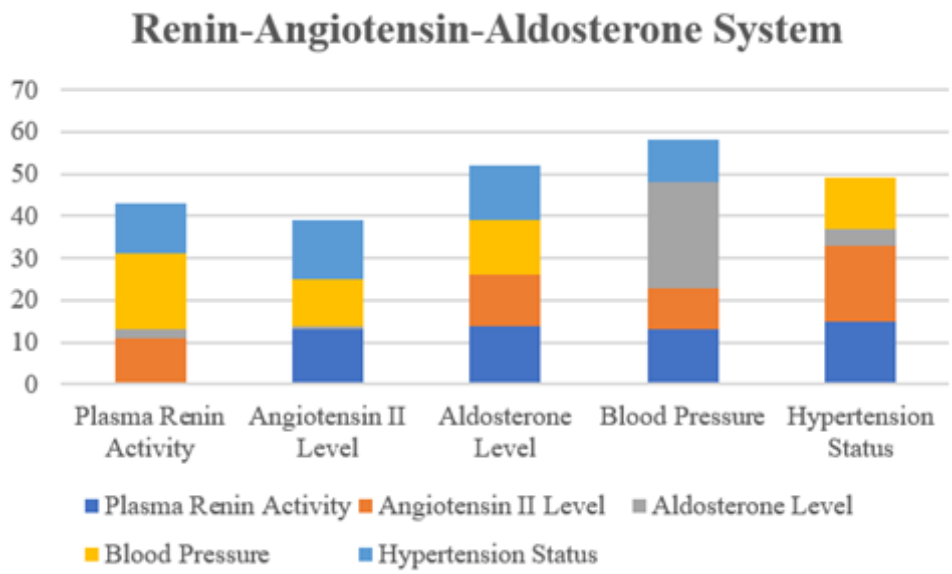

FIGURE 1. Renin-Angiotensin-Aldosterone System

Table 2 normalizing the direct relationship matrix within the Renin-Angiotensin-Aldosterone System concerning C1, C2, C3, C4, and C5, it is evident that all diagonal values across the dataset become zero.

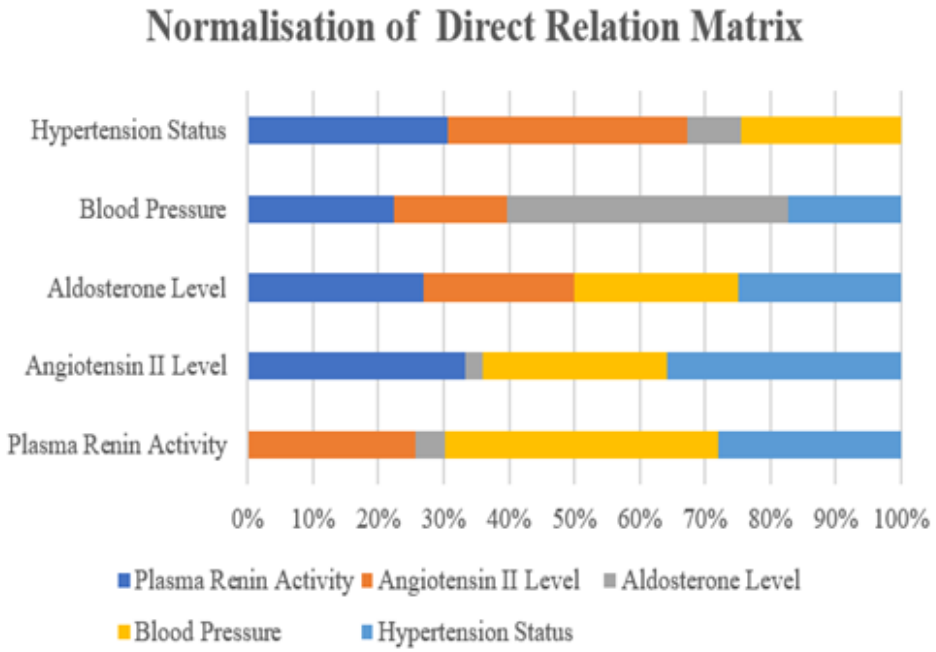

FIGURE 2. Normalization of direct relation matrix

Figure 2 shows that the Normalizing of the direct relation matrix in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 the diagonal value of all the data set is zero.

Table 3 Shows the Calculate the total relation matrix in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 the diagonal is Calculate the Value.

Figure 3Shows the Calculate the total relation matrix in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 Status the diagonal is Calculate the Value.

TABLE 3. Calculate the Total Relation Matrix

	C1	C2	C3	C4	C5
Plasma Renin Activity	0	0.236363636	0.254545455	0.236363636	0.272727273
Angiotensin II Level	0.2	0	0.218181818	0.181818182	0.327272727
Aldosterone Level	0.036363636	0.018181818	0	0.454545455	0.072727273
Blood Pressure	0.327272727	0.2	0.236363636	0	0.218181818
Hypertension Status	0.218181818	0.254545455	0.236363636	0.181818182	0

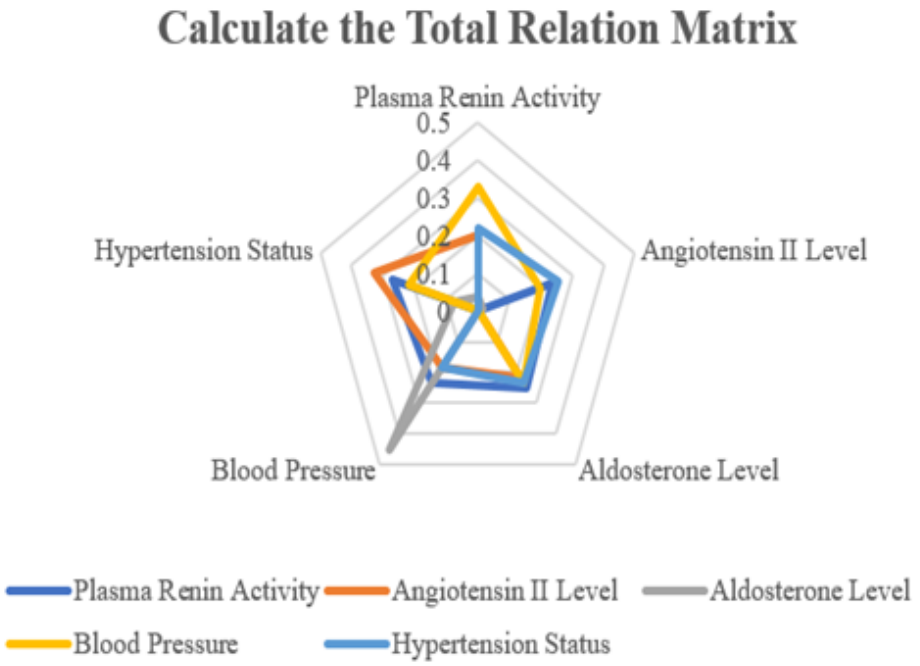

FIGURE 3. Calculate the Total Relation Matrix

Table 4 Shows the T= Y(I-Y)-1, I= Identity matrix in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 is the common Value.

Table 5 Shows the Y Value in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 is Calculate the total relation matrix Value and Y Value is the same value.

Table 6 Shows the I-Y Value in Renin-Angiotensin-Aldosterone System with respect to C1, C2, C3, C4 and C5 table 4 T= Y(I-Y)-1, I= Identity matrix and table 5 Y Value Subtraction Value.

Table 7 the statement, it appears to discuss the calculation of the (I-Y)-1Value within the Renin-Angiotensin-Aldosterone System concerning C1, C2, C3, C4, and C5. Table 6 likely displays the inverse matrix (Minverse) utilized in the process.

Table 8 The total correlation matrix (T) is derived by subtracting the identity matrix from the direct correlation matrix, and each value is then multiplied by its inverse

Figure 4 The total correlation matrix (T) is derived by subtracting the identity matrix from the direct correlation matrix, and each value is then multiplied by its inverse.

TABLE 4. $T = Y(I - Y)^{-1}$, I = Identity Matrix

1	0	0	0	0
0	1	0	0	0
0	0	1	0	0
0	0	0	1	0
0	0	0	0	1

TABLE 5. Y Value

0	0.2363636	0.2545455	0.2363636	0.2727273
0.2	0	0.2181818	0.1818182	0.3272727
0.0363636	0.0181818	0	0.4545455	0.0727273
0.3272727	0.2	0.2363636	0	0.2181818
0.2181818	0.2545455	0.2363636	0.1818182	0

TABLE 6. Y Value

1	-0.2363636	-0.2545455	-0.2363636	-0.2727273
-0.2	1	-0.2181818	-0.1818182	-0.3272727
-0.0363636	-0.0181818	1	-0.4545455	-0.0727273
-0.3272727	-0.2	-0.2363636	1	-0.2181818
-0.2181818	-0.2545455	-0.2363636	-0.1818182	1

TABLE 7. Y Value

2.266810376	1.3311832	1.6701608	1.8230059	1.573094
1.356832502	2.0753918	1.5579793	1.6844189	1.5300815
0.891317503	0.7679939	1.9503595	1.4083938	0.9435603
1.51316077	1.3021557	1.6513058	2.623427	1.5313193
1.325747542	1.2370029	1.5222068	1.6363877	2.2341408

TABLE 8. Total Relation Matrix (T)

	Total Relation Matrix (T)					R_i
	C1	C2	C3	C4	C5	
	1.266810376	1.3311832	1.6701608	1.8230059	1.573094	7.664254
	1.356832502	1.0753918	1.5579793	1.6844189	1.5300815	7.204704
	0.891317503	0.7679939	0.9503595	1.4083938	0.9435603	4.961625
	1.51316077	1.3021557	1.6513058	1.623427	1.5313193	7.621369
	1.325747542	1.2370029	1.5222068	1.6363877	1.2341408	6.955486
C_i	6.353868693	5.713728	7.352012	8.175633	6.812196	

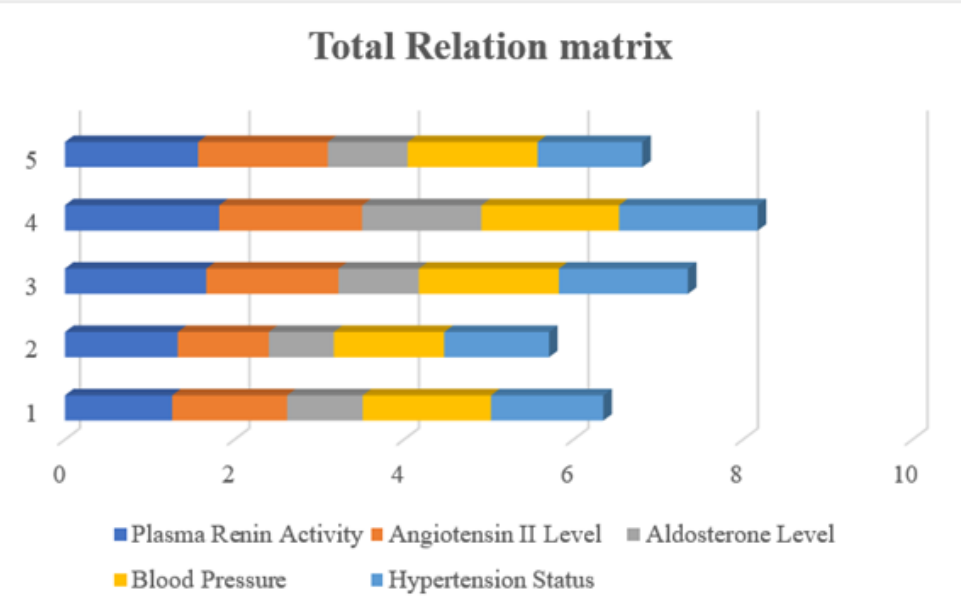

FIGURE 4. Total Relation matrix (T)

TABLE 9. Renin-Angiotensin-Aldosterone System R_i and C_i Values

	R_i	C_i
Plasma Renin Activity	7.6642542	6.3538687
Angiotensin II Level	7.2047041	5.7137275
Aldosterone Level	4.9616249	7.3520122
Blood Pressure	7.6213686	8.1756333
Hypertension Status	6.9554858	6.8121959

Table 9 The Renin-Angiotensin-Aldosterone System (RAAS) comprises Plasma Renin Activity, Angiotensin II Level, Aldosterone Level, Blood Pressure, and Hypertension Status. Within this system, Plasma Renin Activity exhibits the highest value for Ri, while Aldosterone Level demonstrates the lowest. Conversely, Angiotensin II Level displays the highest value for Ci, whereas Hypertension Status indicates the lowest value.

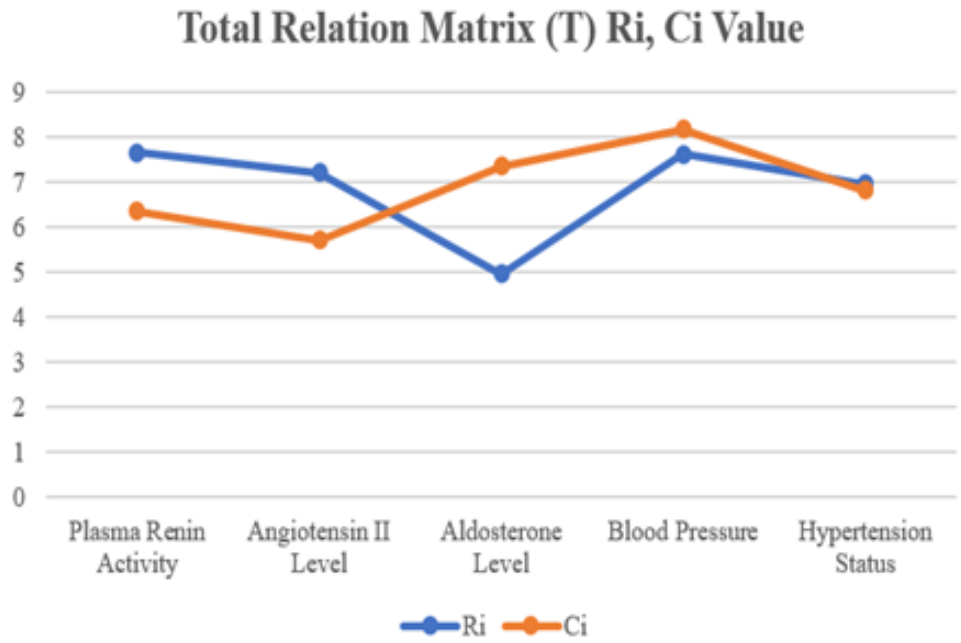

FIGURE 5. Total Relation Matrix (T) Ri, Ci Value

Figure 5the process involves computing Ri+Ci and Ri-Ci to determine cause and effect relationships. In the Renin-Angiotensin-Aldosterone System, Plasma Renin Activity and Hypertension Status exhibit the highest values for causality. Conversely, in the same system, Angiotensin II Level, Aldosterone Level, and Blood Pressure demonstrate the lowest values for effect.

TABLE 10. Calculation of $R_i + C_i$ and $R_i - C_i$ to Get the Cause and Effect

	$R_i + C_i$	$R_i - C_i$	Rank	Identity
Plasma Renin Activity	14.018123	1.3103855	2	cause
Angiotensin II Level	12.918432	1.4909766	4	cause
Aldosterone Level	12.313637	-2.3903873	5	effect
Blood Pressure	15.797002	-0.5542647	1	effect
Hypertension Status	13.767682	0.1432899	3	cause

Table 10 the process involves computing Ri+Ci and Ri-Ci to determine cause and effect relationships. In the Renin-Angiotensin-Aldosterone System, Plasma Renin Activity and Hypertension Status exhibit the highest values for causality. Conversely, in the same system, Angiotensin II Level, Aldosterone Level, and Blood Pressure demonstrate the lowest values for effect.

TABLE 11. *T* Matrix Value

1.2668104	1.3311832	1.670161	1.823006	1.573094
1.3568325	1.0753918	1.557979	1.684419	1.530082
0.8913175	0.7679939	0.9503595	1.408394	0.9435603
1.513161	1.3021557	1.651306	1.623427	1.531319
1.3257475	1.2370029	1.522207	1.636388	1.2341408

Table 11. the T matrix computes the mean of the matrix and determines its threshold value (alpha), which is 1.376298. If the value in the T matrix exceeds the threshold, it is highlighted in bold.

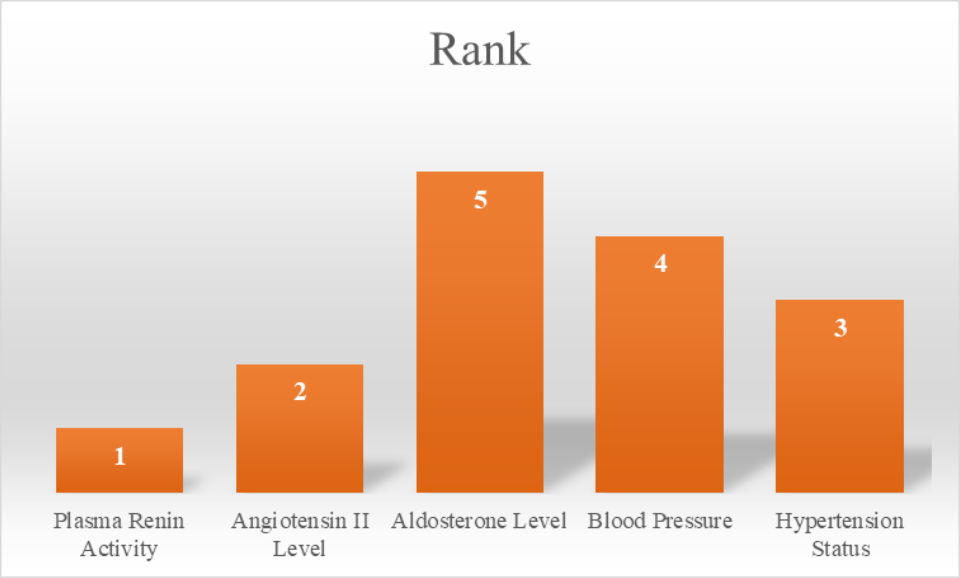

FIGURE 6. Shown the Rank

Figure 6 The DEMATEL analysis indicates that Plasma Renin Activity ranks highest, while Aldosterone Level ranks lowest in the RAAS.

4. CONCLUSION

The renin-angiotensin-aldosterone system (RAAS) controls both blood pressure and cardiac function. When dysregulated, it can contribute to conditions like hypertension, cardiovascular, and renal diseases. Targeting the RAAS with medications can help improve these conditions. The RAAS maintains fluid balance and electrolyte levels, crucial for regulating blood pressure through a complex network involving the endocrine system and vascular resistance. It also influences kidney function, including sodium and water absorption, directly impacting systemic blood pressure. Under normal conditions, the RAAS responds to decreased blood volume by activating juxtaglomerular cells in the kidney, leading to the secretion of renin The primary pathology in cases of chronic hypertension, kidney, and heart failure is attributed to a dysfunctional to sustained high blood pressure, necessitating its regulation. Typically prescribed for managing high blood pressure and heart failure, ACE inhibitors serve as key medications. The Renin-Angiotensin-Aldosterone System (RAAS) plays a pivotal role in regulating blood pressure, electrolyte equilibrium, and fluid levels within the body. Initiated by decreased blood flow to the kidneys or low sodium levels, the enzyme renin is released, Angiotensinogen conversion into angiotensin from promoting entering Angiotensin-converting enzyme The enzyme, or ACE, then This changes the precedent powerful vasoconstrictor called angiotensin II. In addition to narrowing blood arteries and increasing blood pressure, angiotensin II also causes the adrenal cortex to generate more aldosterone Because of the way that aldosterone affects the kidneys, blood circulation and blood pressure rise as potassium elimination is enhanced and salt and water reabsorption is promoted. This gauges the blood's level of a protein called enzyme activity. By starting the system composed of renin, angiotensin, and (RAAS), which then in turn produces the powerful the dilation angiotensin II, the hormone a protein called plays a crucial

role in controlling blood pressure. hormone stimulated by angiotensin II, promotes sodium retention in the kidneys and increases water retention, further elevating blood pressure. Furthermore, other factors such as hormones (e.g., adrenaline, vasopressin), endothelial function, and blood viscosity also contribute to blood pressure regulation. Plasma Renin Activity, Angiotensin II Level, Aldosterone Level, Blood Pressure, Hypertension Status: Diagnosed and Medication: ACE Inhibitor (Enalapril). The DEMATEL analysis indicates that Plasma Renin Activity ranks highest, while Aldosterone Level ranks lowest in the RAAS.

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