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# EXPLORING SELJE TOPOLOGICAL SPACE: APPLICATION IN OPTIMIZING RENAL HEALTH

Dr. Jeyanthi. V [1], Selva Nandhini. N [2]

[1] Assistant Professor, Department of Mathematics, Sri Krishna Arts and Science College, Kuniamuthur, Coimbatore, TamilNadu, India
[2] Research Scholar, Department of Mathematics, Sri Krishna Arts and Science College, Kuniamuthur, Coimbatore, TamilNadu, India

## ABSTRACT

Renal health is vital for maintaining overall body homeostasis, and dysfunction in the kidneys can lead to severe complications, including chronic kidney disease and cardiovascular problems. Identifying the factors that contribute most significantly to renal impairment is critical for early diagnosis and effective treatment. This study utilizes the Selje Topological Space framework to analyze renal health factors. Data was collected over several months from pa- tients with renal dysfunction, focusing on key contributors such as hemoglobin, blood urea nitrogen, azotocreatinine, estimated glomerular filtration rate (eGFR), and potassium levels. The Selje Topological Space approach was used to identify the primary determinants of kidney dysfunction. The analysis revealed that urea and potassium levels were the most significant factors affecting kidney function under normal sugar conditions. In cases where blood sugar levels were abnormal, urea was identified as the dominant contributor to renal impairment. The study's findings emphasize the importance of monitoring urea and potassium levels in patients at risk for renal disease. These insights can guide clinical interventions and improve strategies for preventing kidney dysfunction in patients with both normal and abnormal blood sugar levels.

**Keywords:** Renal Dysfunction, Topological Analysis, Etiological Factors, Risk Assessment
**2000 AMS Classification:** 54A05, 54B05

**Corresponding Author**
Selva Nandhini. N,
nandhininagaraj230@gmail.com

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## 1. INTRODUCTION

Renal dysfunction is a critical health problem that affects millions of people globally, often progressing into chronic kidney disease (CKD), which leads to serious complications such as cardiovascular disease, reduced quality of life, and increased mortality. Effective management of CKD depends on the early diagnosis and identification of the key factors contributing to kidney dysfunction. However, conventional analytical methods often fall short in accurately identifying the most influential determinants of renal impairment, especially in cases where multiple factors, such as biochemical markers, interact in complex ways. Chronic diseases, such as kidney disease and diabetes, represent major health challenges worldwide due to their complex nature and long-term management needs. Recent advancements in mathematics and data science have opened new pathways for analyzing patient data, particularly through topo- logical and graph-theoretical approaches. Borg and Groenen (2021) explore the use of multi- dimensional scaling and topology in chronic disease data analysis, offering new perspectives for managing these conditions [1]. Topological methods have

further been applied to identify patterns in chronic disease networks, as highlighted by Gao and Wu (2019) [5]. Khan and Barak (2020) build on this by presenting topological models for chronic disease progression, with diabetes mellitus serving as a key case study [9]. These models are complemented by re- search into the mathematical properties of open sets and topological spaces, with foundational work by Caldas (2003) [2], Chandrasekar (2019) [3], and Levine (1970) [11], and more recent innovations in Selje and micro-topological spaces presented by Jeyanthi and Selva Nandhini (2023) [7].

Applications of topological methods have proven especially valuable in the realm of health- care, where predictive modeling for chronic diseases has gained prominence. Smith and Jones (2021) emphasize the utility of algebraic topology in chronic disease prediction [14], while Yamamoto and Tanaka (2022) offer a topology-based analysis specifically for chronic kid- ney disease progression [20]. The management of chronic kidney disease (CKD) is a focal point of this research, as several studies underline the need for early diagnosis and intervention (Kovesdy, 2020) [10], as well as the role of renal replacement therapy in improving patient outcomes (Wang & Chen, 2022) [19]. Meanwhile, Elamin and Nair (2019) discuss emerging trends in renal health management and the challenges faced in treating CKD [4]. Additionally, Verduijn and van Diepen (2019) present recent developments in biomarkers for early detection and prognosis of CKD [18].

Diabetes, often associated with renal complications, also remains a significant area of con- cern. Jiang and Liu (2021) provide insights into the impact of diabetes on renal health, stress- ing the importance of integrated disease management strategies [8]. A topological approach to managing chronic diseases through big data analytics is further explored by Li and Liu (2020), underscoring the potential of data-driven models in healthcare [12]. Moreover, Zhao and Wang (2022) offer a mathematical modeling framework for understanding disease dynam- ics in chronic conditions, providing a broader context for disease management strategies [21]. In light of these developments, nutritional interventions also play a pivotal role, as noted by Mora and Gupta (2023), who review dietary strategies for managing CKD [13].

Finally, research into more specialized topological properties continues to evolve, as shown by Hamlett and Rose (1990) [6], as well as Thivagar and Richard (2013, 2012), who investigate the role of weakly open sets and modern topology in medical contexts [15, 16]. Tziomalos and Spanou (2021) further connect the dots between CKD and cardiovascular risks, highlighting the systemic implications of renal health issues [17]. This paper aims to bridge the gap be- tween these diverse research areas, illustrating how topological methods, big data analytics, and nutritional strategies can collectively enhance the management and treatment of chronic diseases, particularly CKD.

Selje Topological Spaces represent a novel approach that provides a structured and precise framework for analyzing health conditions characterized by multiple interacting factors. Tra- ditional models, while valuable, often struggle to account for the variability inherent in patient data. In contrast, Selje Topological Spaces enable a more nuanced identification of both pri- mary and secondary risk factors influencing renal health, thus offering a deeper insight into the mechanisms of kidney dysfunction. Despite considerable progress in renal disease research, there remains a gap in the application of advanced topological methods to analyze kidney func- tion. Current studies largely focus on broad trends such as the impact of diabetes, cardiovascu- lar risks, or nutritional interventions, but lack the precision required to isolate the most critical factors in individual cases. This study addresses this gap by applying the Selje Topological Space framework to clinical data from patients with renal disease. By doing so, it aims to identify the key biochemical markers—such as urea, potassium, hemoglobin, and other vari- ables—that most significantly affect kidney function, providing a more accurate understanding of renal impairment and informing potential clinical interventions.

In Section 3, we discuss the theorems related to the topological analysis of renal biomarkers that have been stated. Section 4 presents the methodology employed in our study, detailing the approach and procedures used. Section 5 explores the application of the Selje Topological Space to renal disease, highlighting how this topological framework is utilized to analyze and interpret renal health data. The results of this application are then obtained and discussed in detail.

## 2. PRELIMINARIES

**Definition 2.1.** [16] Let V denote a non-empty finite set of objects referred to as the universe, and let R represent an equivalence relation on V known as the indiscernibility relation. El- ements within the same equivalence class are considered indiscernible from each other. This pair, denoted as (V, R), constitutes the approximation space.

Let E be a subset of V.

1) The lower approximation of E with respect to R, denoted as LR(E), consists of all ob- jects that can definitively be classified as belonging to E under the influence of R. In mathematical terms, LR(E) =∩{R(X) : R(X) ⊆ E} where R signifies the equivalence class determined by E.
2) The upper approximation of E with respect to R, denoted as UR(E), comprises all objects that could potentially be classified as E under the influence of R. Mathematically, UR(E)

=∩{R(X) : R(X) ∩ E /= Φ}

3) The boundary region of E with respect to R, denoted as BR(E), includes all objects that cannot be definitively classified as either belonging to E or not belonging to E under the influence of R. In mathematical terms, BR(E) = UR(E) − LR(E)

**Definition 2.2.** [3] (V, T (E)) creates a nanotopological space. Then, Y (E)= {N ∪ (Nt ∩ Y): N, N ∈TR(E)}. The combination TR(E) is expressed as the microtopology Y; where Y is not nanotopology elements of TR(E).

**Definition 2.3.** [7] Consider the microtopological space (V, YR(E)) and Selje topology be defined as SJ (E) = {(S-J)∪(S-J t ) : S∈ Y (E) and for fixed J,J t ∈/ Y (E) ),J∪J t =V}

Definition 2.4. [7] The Selje topology SJR(E) satisfies the following axioms

1) Both the universal set V and the empty set Φ are elements of TR(E).
2) Any subset of the union of elements from SJR(E) remains within SJR(E).
3) Any finite subset of the intersection of elements within SJR(E) is contained within SJR(E).

The triplet (E, YR(E),SJR(E)) is labeled as Selje topological space. The collection of Selje closed sets of Selje topology is denoted as SJCL(E).

## 3. THEOREMS ON TOPOLOGICAL ANALYSIS OF RENAL BIOMARKERS

This section presents several theorems related to the topological analysis of renal biomarkers, establishing foundational properties concerning the stability, critical points, and complete- ness of biomarker sets essential for evaluating kidney function.

Theorem 3.1 addresses the impact of critical biomarkers on the stability of kidney function. Understanding which biomarkers are essential helps maintain accurate assessments in clinical settings.

**Theorem 3.1. Stability of Renal Function** Let E be the set of renal biomarkers, and let SJ (E) be the Selje topology on E. Let F ⊆ SJ (E) represent a stable kidney function space. If all critical biomarkers (e.g., urea, potassium) remain in F , then the stability of kidney function is preserved. Conversely, if a critical biomarker Bk is removed, then the function space SJ (E −{Bk}) is unstable.

Proof. Define SJ (E) as a collection of open sets corresponding to configurations of biomark- ers. Let F ⊆ SJ (E) be the subset where kidney function is stable. Assume Bk is a critical biomarker in F . Given that Bk is included in F , there exists an open set O ∈ F that contains Bk.

Let Et = E − {Bk}. The induced topology on this new set is SJ (Et). If SJ (E − {Bk}) is stable, then O should remain open in SJ (Et). However, since Bk is critical, the removal of Bk implies that O cannot be open in SJ (Et). Therefore, SJ (Et) does not contain the same open sets as SJ (E), leading to instability in kidney function analysis. Thus, the theorem holds.

Theorem 3.2 identifies critical points among biomarkers, highlighting their significance in influencing the overall assessment of kidney health.

**Theorem 3.2. Critical Points in Biomarker Analysis**

Let E be the set of biomarkers and SJ (E) be the Selje topology defined on E. A biomarker Bk ∈ E is a critical point if its removal causes the closure of an open set in SJ (E), such that SJ (E − {Bk}) does not contain the closure of any open set O ∈ SJ (E).

Proof. Let O ⊂ SJ (E) be an open set representing kidney function. The closure of O is denoted as O. Assume Bk ∈ E is removed to form Et = E − {Bk}.

Since O is open in SJ (E), it holds that O contains all limit points of O. If SJ (E − {Bk}) does not contain O, then Bk must have been necessary for O to remain open. Thus, the removal of Bk results in a significant alteration in the topological structure, establishing that Bk is indeed a critical point in kidney function analysis.

Theorem 3.3 ensures that the identified biomarker sets are complete for accurate analysis of renal function, underscoring the necessity of including all critical biomarkers.

**Theorem 3.3. Completeness of Biomarker Sets**

Let E be the set of renal biomarkers, and let SJ (E) be the Selje topology. A subset C ⊆ E is a "complete biomarker set" if the removal of any Bk ∈ C results in SJ (E − {Bk}) /= SJ (E). Maintaining the complete set C ensures robust kidney function analysis.

Proof. Assume for contradiction that there exists Bk ∈ C such that removing Bk does not change the topology, i.e., SJ (E − {Bk}) = SJ (E). If this equality holds, then all open sets in SJ (E) must also be present in SJ (E − {Bk}), implying that Bk is not essential for the analysis.

This contradicts the definition of C as a complete set, indicating that C does not contribute uniquely to kidney function analysis. Therefore, for C to be complete, the removal of any Bk ∈ C must alter the topology, thus ensuring robust kidney function analysis.

The established theorems elucidate the essential properties of renal biomarkers from a topological perspective, highlighting the importance of critical biomarkers and the integrity of biomarker sets in assessing kidney function. Future work may extend these principles to more complex biomarker interactions and their implications in clinical practice.

## 4. METHODOLOGY FOR INTEGRATING SELJE TOPOLOGY WITH RENAL DATA ANALYSIS

1) **Data Preparation:** Cleaned and standardized clinical data on relevant renal biomarkers.
2) **SELJE Topology Application:** Applied SELJE Topological Space to analyze relation- ships between biomarkers based on normal and abnormal blood sugar levels.
3) **Critical Factor Identification:** Identified key biomarkers contributing significantly to renal dysfunction.
4) **Visualization and Analysis:** Used diagrams and tables to visualize biomarker relation- ships and renal health states.
5) **Output of Critical Markers:** Determined and reported the primary biomarkers con- tributing to renal dysfunction, providing insights for clinical intervention.

## 5. APPLICATION OF SELJE TOPOLOGICAL SPACE IN MEDICAL DIAGNOSTICS

In this section, we explore the application of Selje Topological Space in the medical field, specifically in diagnosing kidney dysfunction. This analysis is based on data obtained from Sri Ramakrishna Hospital, collected by a biochemical consultant specializing in routine monitoring of patients with renal failure. The dataset, spanning approximately ten months, focuses on an individual with diabetes and kidney dysfunction, monitoring six key variables affecting kidney health: potassium levels, hemoglobin, estimated glomerular filtration rate (eGFR), blood urea nitrogen, creatinine, and glucose levels.

**Key Variables and Their Normal Ranges:**

The key variables related to kidney function and their normal ranges are as follows: Hemoglobin levels should be between 140 to 180 g/L for men and 120 to 160 g/L for women. Blood Urea Nitrogen typically ranges from 2.85 to 11.4 mmol/L. Creatinine levels are considered normal if they are between 70 to 130 µmol/L for men and 60 to 110 µmol/L for women. The Estimated Glomerular Filtration Rate (eGFR) averages 116 mL/min/1.73 m2 in people in their twenties but decreases to about 85 mL/min/1.73 m2 in those in their sixties. Potassium levels should be between 3.5 to 5.2 mmol/L. For fasting blood glucose, normal levels are from 3.9 to 5.6 mmol/L, while levels between 5.6 and 6.9 mmol/L suggest the need for lifestyle changes to prevent diabetes.

The following table presents a comprehensive monitoring report of a diabetic patient from March to December, highlighting six crucial factors affecting kidney function. Blood sugar lev- els are categorized into two distinct groups:

intervals where sugar levels are within the normal range and periods where they exceed the norm. These groupings lead to two specific cases, each of which is analyzed using Selje Topological sets. Through this analysis, the primary factors influencing optimal kidney function are identified, ultimately leading to the final conclusions.Using the data provided in the table, the average values for each parameter within the respective columns are calculated, then the corresponding outcomes are determinedand incor- parated within the table. Values below the calculated average for each parameter are marked as ”L”, while those exceeding the average are marked as ”G”.

Table 1. Six key factors influencing kidney function with values and comparisons to the average

| No. Of. Months | Hemoglobin (HB) | Urea in blood (UR) | Creatinine (CRT) | eGFR | Potassium(K) | Blood Sugar(S) |
|---|---|---|---|---|---|---|
| March | 8.0 (L) | 73 (L) | 4.3 (L) | 10 (G) | 4.9 (L) | 131 (L) |
| April | 8.7 (L) | 90 (G) | 5.5 (G) | 7 (L) | 5.7 (G) | 118 (L) |
| May | 8.7 (L) | 99 (G) | 5.2 (G) | 11 (G) | 5.5 (G) | 117 (L) |
| June | 9.1 (G) | 104 (G) | 5.3 (G) | 8 (L) | 5.6 (G) | 120 (L) |
| July | 8.1 (L) | 61 (L) | 4.2 (L) | 14 (G) | 4.9 (L) | 173 (G) |
| August | 7.5 (L) | 80 (G) | 4.5 (L) | 9 (L) | 5.4 (G) | 128 (L) |
| September | 8.6 (L) | 69 (L) | 3.9 (L) | 11 (G) | 5.5 (G) | 126 (L) |
| October | 9.2 (G) | 65 (L) | 4.1 (L) | 10 (G) | 4.9 (L) | 119 (L) |
| November | 9.9 (G) | 83 (G) | 4.8 (G) | 9 (L) | 4.7 (L) | 158 (G) |
| December | 10.4 (G) | 69 (L) | 4.9 (G) | 9 (L) | 4.7 (L) | 112 (L) |

Let E = {Au,Ap,My,Ju,Jl,Ag,Se,Oc,No,De}

G = {HB,UR,CRT,eGFR,K,S}

H = {HB,UR,CRT,eGFR,K} and I={S}

V/H={{Ma,Jl},{Ap,No},{My,Ju},{Ag,Se,Oc,De}}

**CASE1: When patients sugar level is normal**

E ={Ap,My,Ju,Se,Oc,De}

TR(E) = {Φ,V,{My,Ju},{Ap,My,Ju,Ag,Se,Oc,No,De},{Ap,Ag,Se,Oc,No,De}}

μR(E)={Φ,V,{Ap},{My,Ju},{Ap,My,Ju},{Ap,My,Ju,Ag,Se,Oc,De},{Ap,Ag,Se,Oc,No,De}}

If J={Ma,Ap,Ju,Oc,De} and J t ={My,Jl,Ag,Se,No}

Basis of SJH(E)[BR(E)] = {Φ,V,{Ap,Oc,De},{Ma,Ap,Ju,Oc,De},

{Ap,My,Jl,Ag,Se,Oc,No,De}}

**PHASE I: HB is removed**

V/(H − HB)={{Ma,Jl,Oc,De},{Ap,Ju},{My},{Ag,Se,No,}}

T(H−HB)(E)={Φ,V,{Ap,Ju,My},{Ma,Ap,My,Ju,Jl,Ag,Se,Oc,No,De},

{Ma,Jl,Ag,Se,Oc,No,De}}

μ = {Ap} μ(H−HB)(E)={Φ,V,{Ap,Ju,My},{Ap},{Ma,Jl,Ag,Se,Oc,No,De},

{Ma,Ap,Jl,Ag,Se,Oc,No,De}}

The basis SJH(E)[B(H−HB)(E)] are {Φ,V,{Ma,Oc,De},{Ma,Ap,Ju,Oc,De},

{Ap,My,Jl,Ag,Se,No},{Ap}}

SJH(E)[BR(E)]/=SJH(E)[B(H−HB)(E)]

**PHASE II: UR is removed**

V/(H − UR)={{Ma,Jl,Ag},{Ap},{My,Ju},{Se,Oc,No,De}}

T(H−UR)(E)={Φ,V,{Ap,My,Ju},{Ap,My,Ju,Se,Oc,No,De},{Se,Oc,No,De}}

μ = {Ap} μ(H−UR)(E)={Φ,V,{Ap,My,Ju},{Ap,My,Ju,Se,Oc,No,De},{Ap},{Ap,Oc,No,De},

{Ap,Se,Oc,No,De}}

The basis SJH(E)[B(H−UR)(E)] are {Φ,V,{Ap,Oc,De},{Ma,Ap,Ju,Oc,De},

{Ap,My,Jl,Ag,Se,Oc,No,De}

SJH(E)[BR(E)] = SJH(E)[B(H−UR)(E)]

**PHASE III: CRT is removed**

V/(H − CR)={{Ma,Jl,De},{Ap,Ag,Se,Oc,No},{My,Ju}}

T(H−CR)(E)={Φ,V,{My,Ju},{Ma,Ap,Jl,Ag,Se,Oc,No,De}}

μ = {Ap} μ(H−CR)(E)={Φ,V,{Ap},{My,Ju},{Ap,My,Ju},{Ma,Ap,Jl,Ag,Se,Oc,No,De}}

The basis SJH(E)[B(H−CRT )(E)] are {Φ,V,{Ma,Ap,Oc,De},{Ma,Ap,Ju,Oc,De},
{Ap,My,Jl,Ag,Se,No},{Ap}

SJH(E)[BR(E)] /= SJH(E)[B(H−CRT )(E)]

**PHASE IV: eGFR is removed**

V/(H − eGFR)={{Ma,Jl},{Ap,My,No},{Ju},{Ag,De},{Se,Oc}}

T(H−eGF R)(E)={Φ,V,{Ju,Se,Oc},{Ap,My,Ju,Ag,Se,Oc,No,De},{Ap,My,Ag,No,De}} μ =
{Ap} μ(H−eGF R)(E)={Φ,V,{Ap},{Ju,Se,Oc},{Ap,Ju,Se,Oc},{Ap,My,Ju,Ag,Se,Oc,No,De},
{Ap,My,Ag,No,De}}

The basis SJH(E)[B(H−eGF R)(E)] are {Φ,V,{Ma,Ap,Ju,Oc,De},{Ap,My,Jl,Ag,Se,No,De},
{Ap,De}}

SJH(E)[BR(E)] /= SJH(E)[B(H−eGF R)(E)]

**PHASE V: K is removed**

V/(H − K)={{Ma,Jl,Ag,Se},{Ap,Oc,De},{My,Ju,No}}

T(H−K)(E)={Φ,V,{Ap,Oc,De},{Ma,My,Ju,Jl,Ag,Se,No}}

μ = {Ap} μ(H−K)(E)={Φ,V,{Ap},{Ap,Oc,De},{Ma,My,Ju,Jl,Ag,Se,No},{Ma,Ap,My,Ju,Jl,Ag,Se,No}

The basis SJH(E)[B(H−K)(E)] are {Φ,V,{Ap,Oc,De},{Ma,Ap,Ju,Oc,De},
{Ap,My,Jl,Ag,Se,Oc,No,De}}

SJH(E)[BR(E)] = SJH(E)[B(H−K)(E)]

Through the examination of case 1, it has been determined that the primary cause of kidney dysfunction under normal sugar levels is attributed to **UREA and POTASSIUM**

**CASE2: When patients sugar level is Abnormal**

E ={Ma,Jl,Ag,No}

V/H={{Ma,Jl},{Ap,No},{My,Ju},{Ag,Se,Oc,De}}

TR(E) = {Φ,V,{Ma,Jl},{Ap,No},{My,Ju},{Ag,Se,Oc,De},}

μR(E)={Φ,V,{Ap},{Ma,Jl},{Ma,Ap,Jl},{Ma,Ap,Jl,Ag,Se,Oc,De},{Ap,Ag,Se,Oc,De}}

If J={Ma,Ap,Ju,Oc,De} and J t ={My,Jl,Ag,Se,No}

The Basis of SJH(E)[BR(E)] = {Φ,V,{Ap,Oc,De},{Ma,Ap,Ju,Oc,De},{Ap,My,Jl,Ag,Se,Oc,No,De}}

**PHASE I: HB is removed**

V/(H − HB)={{Ma,Jl,Oc,De},{Ap,Ju},{My},{Ag,Se,No,}}

T(H−HB)(E)={Φ,V,{Ma,Jl,Oc,De},{Ma,Jl,Ag,Se,Oc,No,De},{Ag,Se,No},}

μ = {Ap} μ(H−HB)(E)={Φ,V,{Ap},{Ma,Jl,Oc,De},{Ma,Ap,Jl,Oc,De},{Ma,Jl,Ag,Se,Oc,No,De},
{Ma,Ap,Jl,Ag,Se,Oc,No,De},{Ag,Se,No},{Ap,Ag,Se,No}

The basis SJH(E)[B(H−HB)(E)] are {Φ,V,{Ma,Ap,Oc,De},{Ma,Ap,Ju,Oc,De},
{Ma,Ap,My,Jl,Ag,Se,Oc,No,De}}

SJH(E)[BR(E)] /= SJH(E)[B(H−HB)(E)]

**PHASE II: UR is removed**

V/(H − UR)={{Ma,Jl,Ag},{Ap},{My,Ju},{Se,Oc,No,De}}

T(H−UR)(E)={Φ,V,{Ma,Jl,Ag},{Ma,Jl,Ag,Se,Oc,No,De},{Se,Oc,No,De}}

μ = {Ap} μ(H−UR)(E)={Φ,V,{Ap},{Ma,Jl,Ag},{Ma,Ap,Jl,Ag},{Ma,Jl,Ag,Se,Oc,No,De},
{Ma,Ap,Jl,Ag,Se,Oc,No,De},{Se,Oc,No,De},{Ap,Se,Oc,No,De}}

The basis SJH(E)[B(H−UR)(E)] are {Φ,V,{Ap,Oc,De},{Ma,Ap,Ju,Oc,De},
{Ap,My,Jl,Ag,Se,Oc,No,De}

SJH(E)[BR(E)] = SJH(E)[B(H−UR)(E)]

**PHASE III: CRT is removed**

V/(H − CR)={{Ma,Jl,De},{Ap,Ag,Se,Oc,No},{My,Ju}}

T(H−CR)(E)={Φ,V,{Ma,Ap,Jl,Ag,Se,Oc,No,De}}

μ = {Ap} μ(H−CR)(E)={Φ,V,{Ap},{Ma,Ap,Jl,Ag,Se,Oc,No,De}}

The basis SJH(E)[B(H−CRT )(E)] are {Φ,V,{Ma,Ap,Oc,De},{Ma,Ap,Ju,Oc,De},
{Ap,My,Jl,Ag,Se,No}}

SJH(E)[BR(E)] /= SJH(E)[B(H−CRT )(E)]

**PHASE IV: eGFR is removed**

V/(H − eGFR)={{Ma,Jl},{Ap,My,No},{Ju},{Ag,De},{Se,Oc}}

T(H−eGF R)(E)={Φ,V,{Ma,Jl},{Ma,Ap,My,Jl,Ag,No,De},{Ap,My,Ag,No,De}}

μ = {Ap} μ(H−eGF R)(E)={Φ,V,{Ap},{Ma,Jl},{Ma,Ap,Jl},{Ma,Ap,My,Jl,Ag,No,De},
{Ap,My,Ag,No,De}}

The basis SJH(E)[B(H−eGF R)(E)] are {Φ,V,{Ma,Ap},{Ma,Ap,Ju,Oc,De},
{Ma,Ap,My,Jl,Ag,Se,No},{Ma,Ap,My,Ju,Jl,Ag,Oc,De},{Ma,Ap,My,Jl,Ag,No}}

SJH(E)[BR(E)] /= SJH(E)[B(H−eGF R)(E)]

**PHASE V: K is removed**

V/(H − K)={{Ma,Jl,Ag,Se},{Ap,Oc,De},{My,Ju,No}}

T(H−K)(E)={Φ,V,{Ma,Jl,Ag,Se,My,Ju,No}}

μ = {Ap} μ(H−K)(E)={Φ,V,{Ap},{Ma,Jl,Ag,Se,My,Ju,No}}

The basis SJH(E)[B(H−K)(E)] are {Φ,V,{Ma,Ap,Ju},{Ma,Ap,Ju,Oc,De},
{Ma,Ap,My,Ju,Jl,Ag,Se,No}}

SJH(E)[BR(E)] /= SJH(E)[B(H−K)(E)]

Following the aforementioned analysis of case 2. it has been ascertained that the principal fac- tor behind kidney malfunction during abnormal sugar level is **UREA**

From both the cases it is clearly known that Urea plays vital role in kidney dysfunction.

## 6. RESULTS AND DISCUSSION

From the data analyzed, distinct patterns emerged regarding the biochemical factors influ- encing kidney dysfunction in diabetic patients. The analysis categorized the patients into two groups based on blood sugar levels: normal and abnormal. For each case, the critical fac- tors contributing to renal impairment were identified using the Selje Topological framework, providing insights from both medical and topological perspectives.

In Case 1, where patients had normal blood sugar levels, urea and potassium were identified as the primary factors influencing kidney function. Clinically, elevated urea levels indicate a buildup of waste products in the blood, suggesting reduced kidney filtration efficiency. High potassium levels, often associated with hyperkalemia, pose a serious risk for heart function and signal an imbalance in electrolyte regulation. From a topological perspective, urea and potassium had the highest influence in the Selje Topological analysis, consistently appearing in both lower and upper approximations. This highlights their significance as key markers for assessing kidney dysfunction in patients with controlled blood sugar levels.

In Case 2, where patients had abnormal blood sugar levels, urea emerged as the dominant factor contributing to renal dysfunction. Elevated urea levels in these patients are particularly concerning as uncontrolled blood sugar often accelerates kidney damage, leading to more se- vere filtration inefficiencies. Topologically, urea was the most critical factor, dominating the analysis across both Selje-open and Selje-closed sets. This indicates that urea plays a pivotal role in kidney impairment, especially when compounded by abnormal blood sugar, further exacerbating the progression of kidney disease.

The identification of urea and potassium as key factors in both cases has significant implications for patient care. Medically, these findings emphasize the need for close monitoring and management of urea and potassium levels in patients at risk of or suffering from kidney dys- function. Urea-lowering therapies and potassium-regulating medications could play a crucial role in improving kidney health and preventing complications, particularly in diabetic patients. The topological analysis reinforces these clinical outcomes by showing how these markers di- rectly influence the state of kidney function, providing a more nuanced understanding of their role in disease progression.

In summary, the experimental findings highlight the importance of addressing elevated urea and potassium levels in renal care. The topological framework used in this study not only con- firms their significance but also offers a robust method for analyzing complex, multifactorial health conditions like chronic kidney disease. These results can be extrapolated to a broader patient population, aiding clinicians in making more informed decisions about treatment strate- gies and personalized care.

## 7. CONCLUSION

Based on the analysis of the five phases, the key finding is that urea and potassium are the primary factors influencing kidney function in patients with normal blood sugar levels, while urea becomes the most critical factor when blood sugar levels are abnormal. These findings highlight the importance of monitoring and managing these biochemical markers to prevent or mitigate kidney dysfunction, particularly in diabetic patients. The application of Selje Topological Spaces in this study has provided a more refined understanding of how these markers interact and contribute to renal health, offering a unique perspective that traditional methods may overlook.

The use of Selje Topological Spaces has advanced our ability to identify primary and sec- ondary factors in renal dysfunction by precisely mapping relationships between key biochemi- cal markers. This topological framework allows for more targeted interventions, guiding clinicians toward personalized treatment plans that focus on the most impactful markers.

For future research, this approach can be expanded to explore other organ systems and health conditions. By applying the Selje Topological model to larger datasets across various medical fields, it will be possible to uncover critical factors influencing complex diseases. This method holds potential for scalability, enabling broader clinical applications and offering a powerful tool for both research and treatment development. The insights gained from topo- logical analysis can significantly enhance the precision of medical interventions and improve patient outcomes, particularly in multifactorial diseases like chronic kidney disease.

## CONFLICT OF INTERESTS

None.

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