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A Study of The Ability to Remove Textile Dye (Eosin Y) Using a Novel Nano Co-Polymer

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P A P E R I N F O

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nano co-polymer, eosin yellwoish, adsorption, remove of textile dye

Abstract

In this paper, a nanoparticle co-polymer was made through condensation polymerization, which releases water as a byproduct when one mole of glycerol and one mole of phthalic anhydride react at various temperatures and periods. DSC and FT-IR were used to analyze the nano co-polymer produced. Adsorbed textile dye Eosin yellowish from aqueous solutions was disclosed in this paper, along with measurements of the nano co-polymer's particle size by AFM and XRD. The nano co-polymer had a particle size of 69.42 nm according to the results of the AFM and 69.04 nm according to the results of the XRD. Three distinct concentrations of nano co-polymer (1 ppm, 3 ppm and 5 ppm) as well as three different temperatures (298 K, 308 K and 318 K) were examined for their effects on the adsorption process. It is clear that these variables are crucial to the process. The findings of the experiment revealed that the exothermic nature of the reaction was demonstrated by the fact that the amount of eosin yellow dye that could be adsorbed on the surface of this nano co-polymer decreased as temperature increased. The obtained findings confirmed the great efficacy of nano co-polymer in the removal of yellowish eosin textile dye.

1. INTRODUCTION

An important class of materials known as polymer nanocomposites exhibits unique physicochemical features that are not possible with the individual components functioning alone. As a result of their promising potential for a wide range of applications in environmental remediation and the solution to diverse environmental challenges, these nanocomposites have lately gained intense scientific interest. [1]

The contaminants (dyes, heavy metals, phenols, medicines, etc.) are prevalent and constitute a serious hazard to humans and other living things even in low quantities. [2] Industrial colors are often used in modern technology. Paper, skin, hair, food, cosmetics, and textiles may all be colored using dyes. [3] They are water-soluble. The treatment of industrial wastewater, which present environmental dangers, is the issue that is posing the greatest challenge to the sustainable growth of human civilization. [4-5] This is due to the fact that domestic industry's toxicity has made removing color from effluent or industry very popular. Sewage reclamation and recycling are the two key goals for preserving the world ecological and improving environmental quality. Some of the often used procedures include oxidation, adsorption, membrane filtration, coagulation and focculation, chemical precipitation, ion exchange, electrochemical removal, biosorption, and reverse osmosis. [6-8].

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2. LITERATURE SURVEY

Previous research focused on the use of a hybrid adsorption membrane technique (HAMT) to remove dye from both synthetic and natural wastewater. Three unique configurations were used to remove the dye methyl green (MG) from synthetic wastewater. By using the best circumstances, this technology demonstrated that it is quite effective in the real treatment of wastewater. [9]

In a different study, the Adsorption Kinetic and Isotherm Study focuses on the adsorption of the organic dye (methylene blue, MB) from aqueous media using natural Iraqi bentonite clay (NIBC) and examines the characteristics and applicability of the NIBC for toxic cationic dye removal. This study shows that the NIBC may be used to effectively remove MB from aquatic environments. [10]

3. CONTENTS AND METHODS

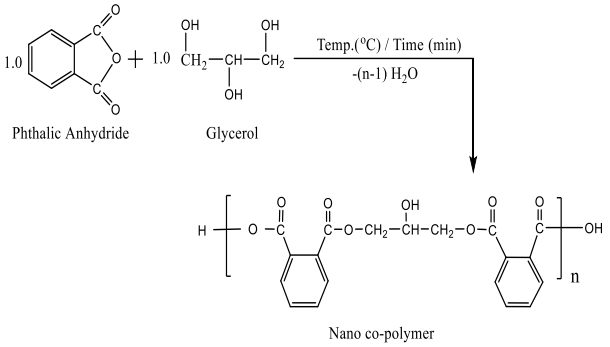
Previous research focused on the use of a hybrid adsorption membrane technique (HAMT) to remove dye from both synthetic and natural wastewater. Three unique configurations were used to remove the dye methyl green (MG) from synthetic wastewater. By using the best circumstances, this technology demonstrated that it is quite effective in the real treatment of wastewater. [9]

TABLE 1. Chemical material, purity and companies supply

Materials	Purities	Company
Glycerol	99.5%	BHD
Phthalic Anhydride	99%	ALPHA
Di methyl sulfoxide (DMSO)	99.5%	CDH
Ortho xylene	99%	MERCK
Eosin Yellowish	98%	MERCK

3.1. Preparation of Nano Co-polymer

In a 200 mL beaker, 30 mL of DMSO and (1.0 mole, 148 g) of phthalic anhydride were mixed. There was a thermometer with this beaker. Glycerol (1.0 mole, 92 g) was slowly added to the solution after it had been carefully warmed to 70 ºC and clear liquor had formed. After the mixture had been properly warmed to 100 ºC, 10 mL of o-xylene was dropped in stages of three drops into the reaction beaker to remove the water produced during esterification. The reaction beaker was then gradually heated. Heating was halted at 110 ºC after 45 minutes when the remaining water was evaporating to produce the nano co-polymer. The icy distilled water is then added, producing the suspension solution. The equation below shows how to filter the suspension solution, soak it in distilled water, and let it dry freely after allowing it to precipitate overnight.



3.2. Establishing the Calibration Curve

Various concentrations of eosin yellowish fluid (1, 3, and 5 ppm) were used to generate the titration curve, which showed the connection between absorbance and concentration. The maximum wavelength of the eosin yellowish dye (λ max = 516 nm) [11] was used to determine the absorbance of these concentrations, and the standard curve between absorption and concentration was then created, as seen in Figure 1.

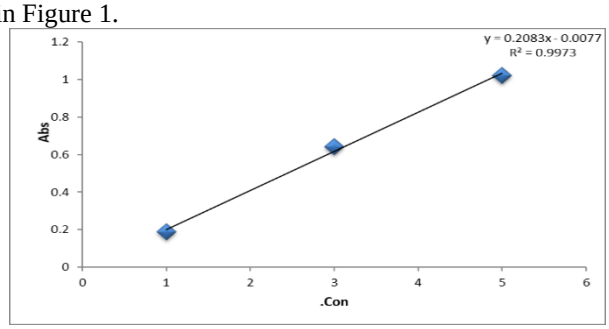


Figure 1. Standard curve between Eosin yellow dye concentration and adsorption.

3.3. Nano Co-polymer Adsorption Measurement

Eosin Y stock remedy By initially combining 0.5 g of the dye with a certain amount of distilled water, then adding another 1000 mL, you may create a concentration of 500 ppm. In volumetric flasks, 30 ml of each concentration of the dye (eosin Y) was placed in contact with the predetermined weight (0.2 g) of the adsorbent outer layer (nano co-polymer) after being appropriately diluted with 100 mL of distilled water. To create the diluted solutions with concentrations of (1, 3, and 5 ppm), this concentrated solution was employed. After the predetermined equilibrium time of 20 minutes, these flasks were placed in a shaking device with a temperature of 298 K. The amount of each solution at equilibrium Ce (mg/L) and the quantities of the adsorbate material Qe (mg/g) of the calibration curves were calculated using UV-Vis spectroscopy after the solutions had been filtered: [12]

$$Qe = (Co-Ce) .V_{sol} / Wt \ldots\ldots\ldots\ldots\ldots\ldots\ldots(1)$$

4. DESCRIPTION AND RESULTS

4.1. Assessment of Nano Co-Polymer

Utilizing FT-IR, DSC, AFM and XRD techniques, the nano co-polymer was investigated. Figure 2. displays extending band at (1668 cm^{-1}) assigned to the bond (C=O) ester, and a prominent sharp peak at (1069 cm^{-1}) of the ester bond (C-O). The FT-IR spectrum has a fragile broad band at (2500-3300 cm^{-1}) assigned to the bond (O-H) alcoholic, as well as an extended band at (3001 cm^{-1}) attributed to the bond (C-H) aromatic.

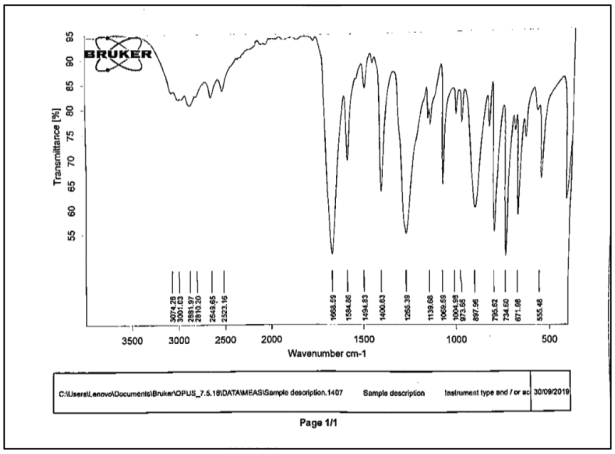
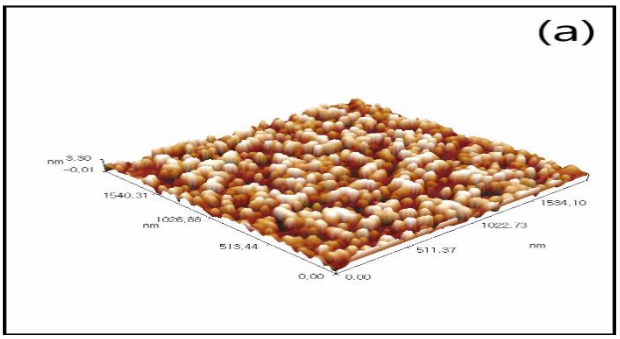


Figure 2. FT-IR of nano co-polymer

After esterification, the dimensions of the nano co-polymer's particles were measured using an atomic force microscope (AFM). The surface of the nano co-polymer had a roughness coefficient of 0.827 nm, and its square root was 0.955 nm. Additionally, 3.30 nm was the average particle height. The findings show that, as illustrated in Figure 3. the co-polymer nanoparticle's particle size was 69.42 nm.



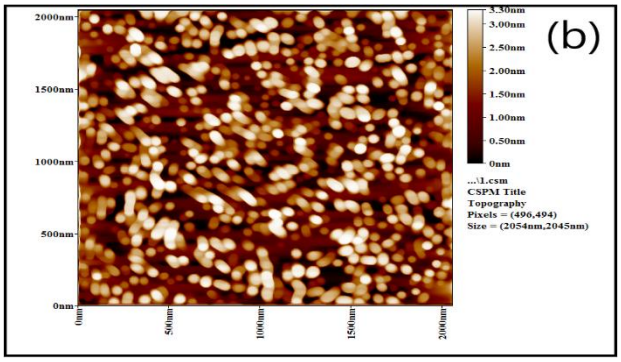


Figure 3. a) AFM image in 3D for nano co-polymer, b) AFM image in 2D of a nano co-polymer.

In Figure 4. the nanoparticle co-polymer exhibits peaks at 2θ values of 15.4°, 18.6°, 22.3°, 27.0°, 30.5°, and 37.0° in the x-ray diffraction (XRD) analysis. The creation of the novel co-polymer as a crystalline material with decreased amorphous carbon atoms was shown by these peaks. According to Bragg's Law [13], the average interplaner distance (d hkl) between atoms was 0.385 nm using Origin software:

$$n\lambda = 2d\sin\theta \ldots\ldots\ldots\ldots..(2)$$

The total average crystallite size was 69.04 nm relative to Scherrer's equation:

$$D = k\lambda / \beta\cos\theta \ldots\ldots\ldots.. (3)$$

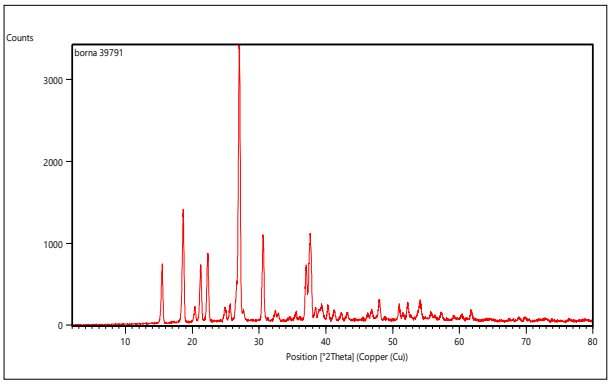


Figure 4. The nanoparticles co-polymer's x-ray diffraction.

Figure 5. displays the DSC thermal imagery for the nano co-polymer. The glass transition temperature (T_g) is represented by the first thermal transition at the peak (71.77 ^oC), the crystallization temperature (T_c) is denoted by the second transition at the peak (230.13 ^oC), and the melting temperature (T_{m1} and T_{m2}, respectively) is characterized by the third and fourth thermal transitions at the peaks (279.37 and 296.27 ^oC). [14]

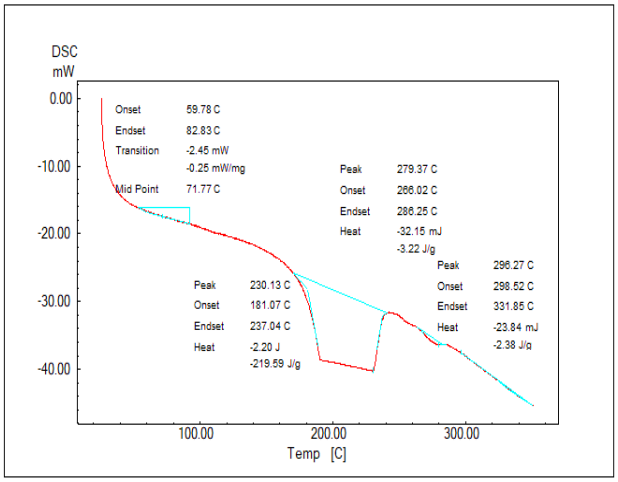


Figure 5. Nano co-polymer DSC thermo grams.

4.2. Eosin Y Dye Removal via Adsorption

As Table 2 reveals, it has been investigated how temperature within the thermal limits (298, 308, and 318 K) impacts the adsorption of eosin Y dye on the outer layer of nano co-polymer. According to the experimental findings, the reaction's exothermic character is demonstrated by the fact that the amount of eosin yellow adsorption on these nano co-polymers' outer layer decreased as the temperature increased. This might be an indication of a desorption process, which is described as the dissociation of the adsorbate granules on the outermost layer of the adsorbent and their return to the solution, lowering the speed of the diffusion process with raising the temperature. [15] as in Figure 6. a, b, and c.

TABLE 2. Effect of temperature on eosin yellow dye adsorption

Conc. (ppm)	Temp.	Nano Co-Polymer	
		Ce	*Qe*
1 ppm	298K	0.045	477.52
	308K	0.047	476.52
	318K	0.057	471.52
3 ppm	298K	0.057	1471.5
	308K	0.062	1469
	318K	0.068	1466
5 ppm	298K	0.066	2467
	308K	0.068	2466
	318K	0.071	2464.5

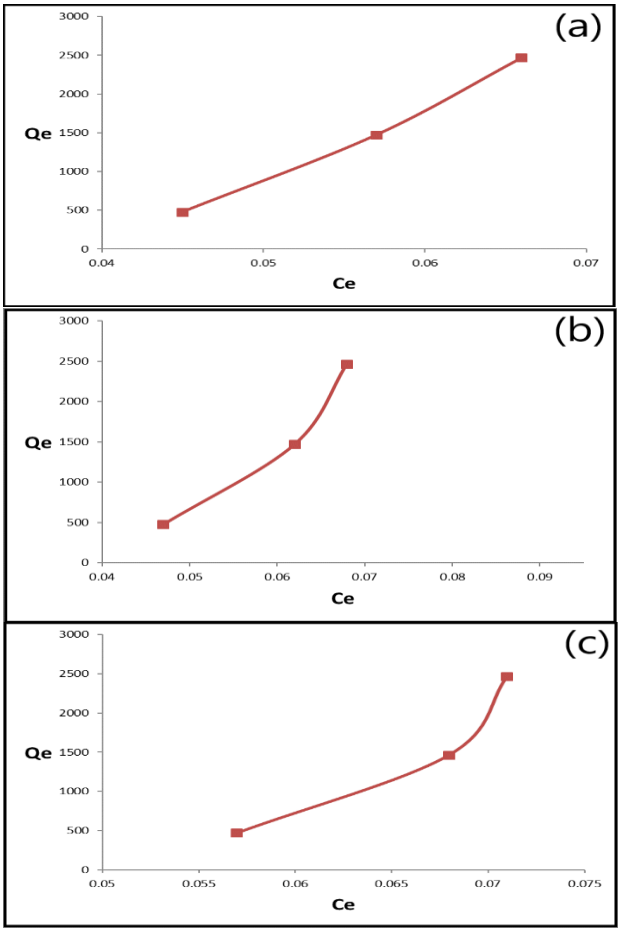


Figure 6. Impact of temperature on the adsorption of a nano co-polymer with (1, 3, and 5 ppm) of the eosin Y dye at: a) 298K, b) 308K, c) 318K

4.3. Isotherms of Adsorption

Adsorption isotherms were discovered when analyzing the eosin Y dye's adsorption on the outer layer of the nano co-polymer at 298 K and pH = 6.6. Given that the adsorbate particles can be positioned either vertically or obliquely on a surface, Figure 7. The demonstration of the nano co-polymer adsorption isotherms being of type (S1) which results from Freundlich's principles, shows that the material has a heterogeneous surface. [16]

TABLE 3. Eosin Y dye adsorpiton on nano co-polymer surface at 298 k

Conc. (ppm)	Temp.	Nano Co-Polymer	
		Ce	*Qe*
1 ppm	298K	0.045	477.52
3 ppm	298K	0.057	1471.5
5 ppm	298K	0.066	2467

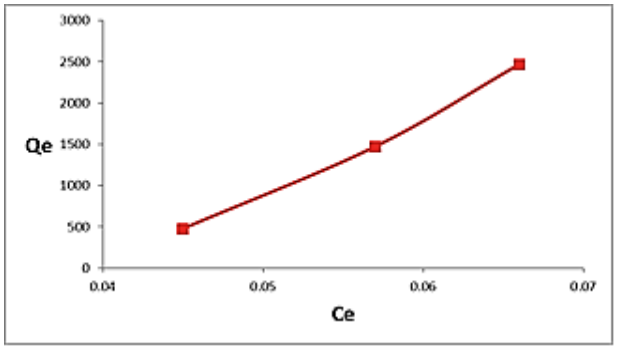


Figure 7. Eosin yellowish dye adsorption Freundlich isotherm on nano co-polymer surface

4.4. Freundlich equation for adsorption

The Freundlich equation is among the most significant isothermal equations for the explanation of the adsorption of solutions on substance surfaces[17] :

$$Qe = K_f \cdot Ce^{1/n} \quad \ldots\ldots\ldots\ldots\ldots\ldots(4)$$

where Ce is the equilibrium adsorbate substance concentration in the fluid (in mg/L). Qe stands for the equilibrium adsorbate concentration (mg/g). Kf, n: The adsorption amplitude and intensity are each represented by a Freundlich constant. The following results from calculating the logarithmic formula of equation:(4)

$$\text{Log } Qe = \text{Log } K_f + (1/n) \text{ Log } Ce \ldots\ldots.(5)$$

When we plot the connection throughout Log Qe and Log Ce, as seen in Figure 8. we get a straight line. The findings of the Freundlich test for the eosin yellowish adsorption on nano co-polymer at 298 k are shown in Tables 3 and 4. The values of Freundlich constants :

$K_f = 8.523$, $n = 4.329$, $R^2 = 0.994$.

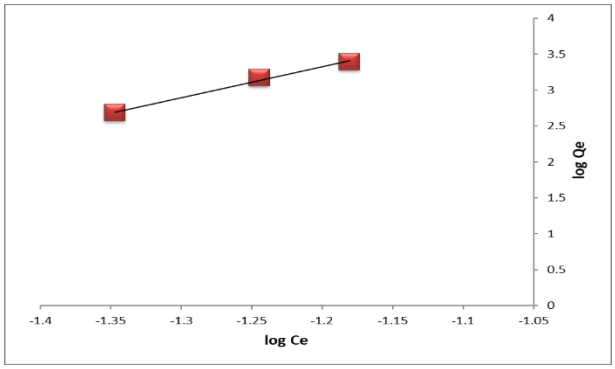


Figure 8. Eosin yellowish dye adsorption Freundlich isotherm on nano co-polymer surface at 298K

TABLE 4. Friendlich isotherm results

Conc. (ppm)	Temp.	Nano Co-polymer	
		log Ce	***log Qe***
1 ppm	298K	-1.347	2.679
3 ppm	298K	-1.244	3.1678
5 ppm	298K	-1.181	3.3922

5. CONCLUSION AND FUTURE

5.1. Conclusion

Under conditions with temperatures below 250 °C, the nano copolymer was created by phthalic anhydride and glycerol. AFM photos validated the polymer's nano structure, while FT-IR revealed the functional groups of the ester co-polymer, and XRD data suggested a crystalline structure. In batch mode, the nano co-polymer's adsorption of the yellowish eosin dye was investigated for the constant solution pH at room temperature and the starting dye concentration. The outcomes demonstrated that the ability of the novel nano co-polymer to remove eosin yellowish dye by adsorption increased with initial concentration and temperature.

5.2. Future

Apply different polymers and techniques. Figure out how many pigments will be absorbed to clean up the land, water, and air. Apply more hydroxyl compound removal techniques to provide the carboxylic compounds with more connections to other hydroxyl compounds

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Arabic Abstract

في هذا البحث، تم تصنيع مركب نانوي متعدد البلمرة عن طريق البلمرة التكثيفية، والتي تحرر الماء كناتج ثانوي عند تفاعل مول واحد من الجلسرين مع مول واحد من أنهيدريد الفثاليك عند درجات حرارة وأزمنة مختلفة. تم استخدام تقنيتي المسح الحراري التفاضلي (DSC) ومطيافية الأشعة تحت الحمراء لتحليل المركب النانوي المتعدد البلمرة الناتج. وتم الكشف عن امتزاز صبغة الأيوسين الصفراء من المحاليل المائية على هذا المركب النانوي، بالإضافة إلى قياس حجم جسيمات المركب النانوي باستخدام مجهر القوة الذرية(AFM) وحيود الأشعة السينية (XRD). وقد أظهرت نتائج كل من AFM و XRD أن حجم جسيمات المركب النانوي هو 69.42 نانومتر و 69.04 نانومتر على التوالي. تم دراسة تأثير ثلاثة تركيزات مختلفة للمركب النانوي (1 جزء في المليون، 3 جزء في المليون، و 5 جزء في المليون) وثلاثة درجات حرارة مختلفة (298 كلفن، 308 كلفن، و 318 كلفن) على عملية الامتزاز. ومن الواضح أن هذه المتغيرات ذات أهمية حاسمة في هذه العملية. وأظهرت نتائج التجربة أن الطابع الباعث للحرارة للتفاعل يتضح من انخفاض كمية صبغة الأيوسين الصفراء التي يمكن امتزازها على سطح هذا المركب النانوي مع زيادة درجة الحرارة. وأظهرت النتائج التي تم الحصول عليها كفاءة عالية للمركب النانوي على امتزاز وإزالة صبغة الأيوسين الصفراء.

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Mean Semi-Open Sets

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Abstract

This article introduces a new kind of mean sets that are also semi-open. Each such set contains a non-void proper semi-open set and also it is contained in another proper semi-open set. Within this work, we compare our new concepts against the corresponding concepts that were defined via open sets. We study some fundamental properties of mean semi-open sets and their complements and provide some new results. Moreover, we investigate the behavior of such sets when they are also minimal and maximal.

1. INTRODUCTION

Generalizing open sets is not a new research line in general topology. A vast number of studies and investigations were initiated to examine various kinds of open sets, ranging from semi-open , α-open and not ending with pre-open sets. Almost each such study investigated and explored various topological properties possessed by generalized open sets. For example, generalized interior and closure operators were defined depending on various generalized open sets. In [1], a study introduced a new kind of open set called minimal. Many significant results were explored. Then in [2], another study introduced and investigated maximal open sets. The complements of minimal and maximal open sets were investigated in [3]. Maximal and minimal clopen sets were introduced in [4]. Then many authors re-introduced minimal and maximal sets in terms of various generalized open sets such as [5] and [6]. Minimal soft sets have been re-investigated in [7]. Recently, minimal and maximal anti-open sets and their complements have been introduced in [8]. Mean open set was introduced in [9]. This kind of set appeared to possess many interesting properties that can be related to other kinds of sets, like minimal and maximal open sets. The study in [10] characterized mean open sets in connected T_1-spaces. Furthermore, the concept of mean sets has also been introduced on fuzzy open sets in [11].

In this work, we introduce mean sets in terms of semi-open sets. The duality of such sets is also introduced and explored in some detail.

This article consists of the following sections: Section 2 includes some basic but fundamental concepts. Section 3 is devoted to introducing mean semi-open sets and their complements and exploring some of their basic properties. Section 4 includes further results of mean semi-open sets.

2. PRELIMINARIES

Throughout this article, we use letter X to represent a topological space. An open set P in X is minimal open if the only open sets in P the sets ϕ and P itself. The dual of this set is the maximal open set, defined in [2]. An open set P is maximal in X if the only open sets that contain P are X and P itself. Suppose that P is a minimal open set then the complement of P (that is, $X - P$) is a maximal closed. In dual sense, the set

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$X - P$ is a minimal closed set whenever P is maximal open set. See also [3] for further reading.

Mean open sets have been introduced and investigated in [9] and [12]. A set O that is open in X is called mean if $R \subset O \subset Q$ where R and Q are open in X with $R \neq \emptyset$ and $Q \neq X$. Within this article, by $P \subset Q$ we mean that P is a subset of Q with $P \neq Q$. A semi-open set S of X is a set yields $B \subset P \subset \bar{B}$ where B is an open set of X and $\bar{B}$ is the closure of B, [13].

Let $SO(X) = \{P \subseteq X : P \text{ is semi} - \text{open in } X\}$.

The set $A^{o^s} = \cup\{P \in SO(X) : P \subseteq A\}$ represents the semi-interior of A. Now, a set F is semi-closed in X if $X - F \in SO(X)$. Consider the collection $SC(X) = \{F \subset X : F \text{ is semi} - \text{closed in } X\}$.

The set $\overline{A}^s = \cap\{F \in SC(X) : A \subseteq F\}$ represents the semi-closure of A. Further information can be found in [3].

Theorem 2.1. [13] Consider the product space of a topological space X and a topological space Y. If $P \in SO(X)$ and $Q \in SO(Y)$, then $P \times Q \in SO(X \times Y)$.

Let $P, Q \in SO(X)$ with $P, Q \neq X$. Then P is called minimal if the only semi-open set of P is either ϕ or P itself. On the other hand, Q is called maximal if the only semi-open set that contains Q is Q itself or X.

An easy observation is that minimal open sets are indeed minimal semi-open sets.

Lemma 2.2. [14] Let $P \in SO(X)$ and $Z \subset X$ be an open set. Then $P \cap Z \in SO(X)$.

Proposition 2.3. Let $P \in SO(X)$ and $Z \subset X$ be an open set.

1. Whenever P is minimal, then either $P \subset Z$ or $P \cap Z = \emptyset$.
2. Whenever P and Z are minimal, then either $P = Z$ or $P \cap Z = \emptyset$.

Proof. Suppose that $P \cap Z \neq \phi$. Since P is minimal semi-open and $P \cap Z$ is semi-open, Lemma 2.2. So, $P \cap Z = S$. Therefore, $P \subset Z$.

2. If $P \cap Z \neq \emptyset$, then by (1) we have $P \subset Z$. But the set Z is minimal with $P \cap Z \neq \emptyset$, then $P \cap Z = Z$. Thus, $Z \subset P$. Consequently, $P = Z$.

Theorem 2.4. [5] Let $P, Q \in SO(X)$. If P is maximal, then either $P \cup Q = X$ or $Q \subset P$. If both P and Q are maximal with $P \neq Q$. Then $P \cup Q = X$.

A semi-continuous is a map $h : X \to Y$, where X and Y are two topological spaces such that $h^{-1}(P)$ is semi-open in X for any open set P in Y.

3. MEAN SEMI-OPEN SETS

We present in this section mean semi-open sets and present few properties they have.

Definition 3.1. A semi-open set S of X is called mean semi-open if there exist $K, L \in SO(X)$ with $K \neq \emptyset$ and $L \neq X$ such that $K \subset S \subset L$.

Example 3.2. In the usual topological space $X = R$, let consider the set $S = [3,6)$ in X. Clearly S is a mean semi-open set since $K \subset S \subset L$ where $K = [4,5)$ and $L = [2,7)$ and $K, L \in SO(X)$.

Example 3.3. Consider the Euclidean space on $\mathbb{R}^2$. Let $S = \{(x, y) \in \mathbb{R}^2 : 0.2 \leq x < 0.8,\ 0.2 \leq y < 0.8\}$. It is clear that S is semi-open.

Now, let $L = \{(x, y) \in \mathbb{R}^2 : 0 \leq x < 1,\ 0 \leq y < 1\}$ and $K = \{(x, y) \in \mathbb{R}^2 : 0.4 \leq x < 0.6,\ 0.4 \leq y < 0.6\}$. Clearly, $K \subset S \subset L$. Thus, S is mean semi-open.

One can easily notice that if an open set R is mean, then R is also a mean semi-open. However, in general, the converse may not be true. Furthermore, the union of mean semi-open sets need not be mean semi-open sets. The same may occur for the intersection case; see Example 3.4.

Example 3.4. Let $X = \{1,2,3,4,5\}$ be a set and $\tau = \{\phi, \{1\}, \{3,4\}, \{1,3,4\}, \{2,3,4,5\}, X\}$ be a topology on X. If we take the sets $S_1 = \{3,4,5\}$ and $S_2 = \{2,3,4\}$. It is clear that both S_1 and S_2 are mean semi-open, but both the sets $S_1 \cap S_2 = \{3,4\}$ and $S_1 \cup S_2 = \{2,3,4,5\}$ are not mean.

Definition 3.5. A mean semi-closed set C of X is a semi-closed set such that $D \subset F \subset H$ for some semi-closed sets D and H with $D \neq \emptyset$ and $H \neq X$

Example 3.6. Consider the topological space in Example 3.4. Notice that $SC(X) = \{\emptyset, \{2,3,4,5\}, \{1,2,5\}, \{2,5\}, \{1,2\}, \{1,5\}, \{2\}, \{1\}, X\}$. It is clear that $\{2,5\}$ is a mean semi-closed in X since $\{2\} \subset \{2,5\} \subset \{1,2,5\}$.

Theorem 3.7. Let $P \in SO(X)$. Then P is mean if and only if $X - P$ is mean semi-closed.

Proof. Assume that P is a mean semi-open.

So $K \subset P \subset L$ where $K, L \in SO(X)$ with $K \neq \emptyset$ and $L \neq X$. The complement of $K \subset P \subset L$ gives $X - L \subset X - P \subset X - L$. Clearly, $X - L$ is non-empty and distinct from $X - P$. Moreover, $X - K$ differs from $X - P$ and X. Therefore, $X - P$ is a mean semi-closed set.

Now, let $P \in SO(X)$ such that $X - P$ is mean semi-closed set.

Thus, $F \subset X - P \subset H$ where F and H are semi-closed sets differ from each other with $F \neq \emptyset$ and $H \neq X$.

Hence, $X - H \subset P \subset X - F$. But $\emptyset \neq X - H \neq P$ and $P \neq X - F \neq X$. Therefore, P is mean semi closed set.

Proposition 3.8. Assume that E be a minimal semi-open, L be a minimal open subsets of X with $E \neq$L. Let $R \in \{E, L\}$. Then either $R = \overline{R}^s$ or $X - \overline{R}^s$ is a mean semi-open set in X.

Proof. From Proposition 2.3, we have $E \cap L = \emptyset$. Hence $E \cap \overline{L}^s = \emptyset$. So $E \subset X - \overline{L}^s$. But both E and L are minimal semi-open, so $X - \overline{L}^s \neq \emptyset, X$.

If the set $X-\overline{L}^{s}$ is not mean semi-open set, then this set is either minimal semi open or maximal semi-open. Since, $E \subset X-\overline{L}^{s}$, hence $X-\overline{L}^{s}$ is not minimal.
Thus, $X-\overline{L}^{s}$ is maximal. Now, L is semi-open, by Theorem 2.4, we have either $L \subset X-\overline{L}^{s}$ or $L \cup \left(X-\overline{L}^{s}\right)=X$. The former case cannot occur, so we have $L \cup \left(X-\overline{L}^{s}\right)=X$. This implies that $L=\overline{L}^{s}$. If this case does not occur, then $\overline{L}^{s}$ is a mean semi-open.
We can proceed with a similar argument to the one above so we can get that either $E=\overline{E}^{s}$ or $X-\overline{E}^{s}$ is mean semi-open.

Proposition 3.9. Assume that P and Q be distinct maximal semi-closed subsets of X. Let $H \in \{P, Q\}$, then either $H=H^{s^{o}}$ or $X-H^{s^{o}}$ is a mean semi-closed set.

Proof. Since both P and Q are distinct maximal semi-closed sets, so their complement sets, $X-P$ and $X-Q$ are minimal semi-open sets with $X-P \neq X-Q$. Proposition 3.8 leads to either $G=\overline{G}^{s}$ or $X-\overline{G}^{s}$ is a mean semi-open set, where $G=X-P$ or $G=X-Q$.
In case that $G=X-P$, then clearly $P=P^{o^{s}}$, as $G=\overline{G}^{s}$, or since $X-\overline{G}^{s}$ is a mean semi-open set, we get that $X-\left(\overline{X-P}^{s}\right)=P^{o^{s}} \in SO(X)$ and it is a mean in X.
Hence, Theorem 3.7 implies that $X-P^{o^{s}}$ is a mean semi-closed set. Now, a similar argument can be applied for $X-Q$ to find that either $Q=Q^{o^{s}}$ or $X-Q^{o^{s}}$ is mean semi-closed by Theorem 3.7.

4. MORE RESEULTS on MEAN SEMI-OPEN SETS

Proposition 4.1. Suppose that $P, Q \in SO(X)$ are both maximal with $P \neq Q$. If $R \in SO(X)$ is a mean, then, $P \cap Q \neq \emptyset$.

Proof. By Theorem 2.4, $P \cup Q=X$. Since R is a mean set. So R can not be maximal or minimal semi-open.
So $R \notin \{P, Q\}$. On the other hand, $R \neq \emptyset, X$.
Theorem 2.4 leads to either $R \subset P$ or $R \cup P=X$ and $R \subset Q$ or $R \cup Q=X$.
Consequently, we have to check the following possible cases:
$R \subset P$ and $R \subset Q$.
Verification: Since $R \subset P, Q$ and never $R=P, Q$, so $R \subset (P \cap Q)$. Hence, $P \cap Q \neq \emptyset$.
$R \subset P$ and $R \cup Q=X$.
Verification: If $R \cap Q \neq \emptyset$, then $P \cap Q \neq \emptyset$. Assume now $R \cap Q=\emptyset$. But, $R \subset P$, so there exists an $x \in P-R$. But $R \cup Q=X$, so $x \in Q$. Therefore, $P \cap Q \neq \emptyset$.
$R \subset Q$ and $R \cup P=X$.
Verification: Similar proof to the previous one.
$R \cup P=X$ and $R \cup Q=X$.
Verification: Since $R \cup P=X$ and $R \cup Q=X$, so $R \cup (P \cap Q)=X$. Hence, if $P \cap Q=\emptyset$, then $R=X$. But $R \neq X$, therefore, $P \cap Q \neq \emptyset$.

Proposition 3.4. Consider the product space $X \times Y$ where X and Y be two spaces. Let S and T be a mean semi-open subsets of X and Y, respectively. Then $P \times Q$ is a mean semi-open set in $X \times Y$.

Proof. Consider the set $P \times Q$. By the hypothesis, we get that $\phi \neq K_X \subset P \subset L_X \neq X$ where $K_X, L_X \in SO(X)$. Similarly, $\phi \neq K_Y \subset Q \subset L_Y \neq Y$ where $K_Y, L_Y \in SO(Y)$. By Theorem 2.1, $\{K_X \times K_Y,\ L_X \times L_Y, P \times Q\} \subset SO(X \times Y)$. Since P and Q is a mean semi open set in X and Y, respectively. Consequently, $K_X \times K_Y \neq \phi$ and $L_X \times L_Y \neq X$. Therefore, $K_X \times K_Y \subset P \times Q \subset L_X \times L_Y$.

Proposition 4.3. Consider the following bijective map h from space X to space Y. If h is semi-continuous and P is a mean open set Y, then $h^{-1}(P) \in SO(X)$ and it is also mean.

Proof. Since P is a mean open subset of Y, then $\phi \neq K \subset P \subset L \neq Y$ where $K, L \subset Y$ and both K and L are open in Y. Semi-continuity of h implies that $h^{-1}(P), h^{-1}(K), h^{-1}(L) \in SO(X)$.
But h, is bijective, so
$\phi \neq h^{-1}(K) \subset h^{-1}(P) \subset h^{-1}(L) \neq h^{-1}(Y)=X$.
Hence, $h^{-1}(P)$ is mean semi-open set in X.

5. CONCLUSION

We present the notion of mean semi-open sets. Such sets are defined via semi-open sets. investigated The properties of such sets along with their complements namely mean semi-closed sets, are investigated. In addition, the study offer some interesting results regarding being mean semi-open sets maximal and minimal. Besides, other similar results for mean semi-closed sets are presented.

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Arabic Abstract

في هذه المقالة تقدم نوعا جديدا من مجموعات المتوسط المعرفة بدلالة المجموعات شبه المفتوحة. تحتوي كل مجموعة على مجموعة شبه مفتوحة فعلية غير فارغة وفي نفس الوقت توجد نلك المجموعة في مجموعة شبه مفتوحة فعلية أخرى. في هذا العمل نقارن المفاهيم التي قدمناه مع المفاهيم المناضرة لها والمعرفة بدلالة المجموعات المفتوحة. ندرس بعض خصائص متوسط المجموعات شبه المفتوحة وكذلك مكملاتها مع تقديم بعض النتائج الجديدو. بالاضافة الى ذلك، فإننا ندرس سلوك هذه المجموعات عندما تكون أيضا مجموعات صغرى و عندما تكون مجموعات كبرى.

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Measurement of *SLC5A5* Gene Expression in Patients with Hypothyroidism

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SLC5A5, TSH, Family History, Heart Disease, Diabetes, Triiodothyronine, Thyroxine.

A B S T R A C T

This study determines the amount of gene expression level of the *SLC5A5* gene in women with hypothyroidism (HD), as it is one of the key genes influencing the development of the condition. Additionally, some risk factors were examined, including diabetes, hypertension, heart diseases, prior thyroidectomy, and family genetic history, to assess the impact of these factors on the prevalence of HD. Peripheral blood (PB) samples were obtained from 50 women with HD who were followed up by medical diagnosis and whose ages ranged from (40 to 70) years. 50 samples of healthy women who do not suffer from chronic diseases were selected as a control group. Polymerase chain reaction (qPCR-RT) technique was used to quantify expression *SLC5A5* gene levels in the samples, as well as to measure some hormonal criteria for patients with thyroid disease to demonstrate their importance in diagnosing the disease. The level of thyroid stimulating hormone (TSH) and the thyroid hormones thyroxine (T4) and triiodothyronine (T3) were measured. The results showed no statistically significant on the expression of the *SLC5A5* gene in women with hypothyroidism compared with the healthy group. In contrast, the results of thyroid hormones showed a significant decrease in both triiodothyronine (T3) and thyroxine (T4) and non-significant effect of TSH. Additionally, the findings indicated that factors such as family history, diabetes, hypertension, and heart disease contribute to the development of HD. In conclusion, risk factors such as family history, diabetes, hypertension, and heart disease were found to play a role in the development of the condition, emphasizing the multifactorial nature of HD.

1. INTRODUCTION

Hypothyroidism (HD) is a disorder characterized by an inability of the thyroid gland to produce enough thyroid hormone that performs metabolic processes [1]. Untreated HD can contribute to high blood pressure, non-equilibrium, infertility, cognitive impairment, and neuromuscular dysfunction. Its prevalence increases with age and it affects females more than males [2].

HD may result from primary thyroid failure or insufficient production stimulation of the thyroid gland by the hypothalamus or pituitary gland [3]. Primary Thyroid insufficiency can cause congenital abnormalities, autoimmune destruction (Hashimoto's disease), and iodine deficiency, HD affects growth, development, and cellular functions [4]. Also, disorders include association with transient HD that includes postpartum thyroiditis, subacute thyroiditis and silent

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thyroiditis [5]. The reasons are usually the same. HD is usually present with other manifestations of hypothalamic or pituitary dysfunction, and they stand out low or abnormal levels of TSH compared to insufficient thyroid hormone [6]. Thyroid disorders include hyperthyroidism, thyroid nodules, and thyroid cancer, but the most common disease is HD [7].

Genes play a pivotal role in determining susceptibility to hypothyroidism, as mutations or multiple genetic variations across different loci in key genes contribute to an individual's risk of developing the condition. Among the most prominent genes associated with thyroid function are *Solute Carrier Family 5 Member 5* (*SLC5A5*), *NK2 Homeobox 1* (*NKX2-1*), *Thyroid Peroxidase* (*TPO*), and *Thyroid-stimulating Hormone Receptor* (*TSHR*). These genes are linked to essential thyroid functions, including thyroid differentiation, iodide regulation, thyroglobulin synthesis, iodide transport, and iodide deiodination [8,9]. The *SLC5A5* gene is specific to the thyroid gland that plays a key role in in regulating iodide transport to thyroid follicular cells, as it encodes the sodium-iodide symporter (NIS). This transporter moves iodide from circulating blood to follicular cells, enabling the thyroid gland to synthesize hormones [10,11]. Mutations in the SLC5A5 gene disrupt iodide transport, leading to impaired thyroid hormone synthesis and resulting in hypothyroidism [12].

2. MATERIALS and MEsTHODS

2.1. Sample Collection

The study had been conducted in Karbala Governorate from November 2023 to April 2024. A total of 100 blood samples were collected and divided into two groups. The first 50 samples were obtained from the outpatient clinics at Imam Hussein (PBUH) Medical City and had included individuals diagnosed with HD, while the second 50 samples were from healthy individuals (control group).

2.2. Estimation of Serum Thyroid Stimulating Hormone

TSH concentrations were measured quantitatively in the laboratory by an immunoassay. The ECLIA photochemical immunoassay is designed for use with the Cobas e 411. The kit is based on photoelectrochemical measurement. As with total T4, the method for measuring total T3 in serum is a competitive chemiluminescence immunoassay using the 100-sample test kit from Roche diagnostics.

2. 3. Determination of Serum TG Concentration

Serum TG levels were measured using a special kit linear chemicals cromatest/Spain TG - CHOL Kit.

2. 4. Molecular Detection

2.4.1. RNA Extraction

The most important solutions used in the extraction are shown in **Table 1** aacoring to manufactures company (Promega/ USA).

TABLE 1. Extraction steps

Sequence	Reagents and materials	Volumes
1	Transzol	600 µl
2	RNA Extraction Agent	0.2ml
3	Isopropanol	0.5 ml
4	Ethanol	1ml

The process of extracting RNA from frozen samples with transsol are extracted with pre-added begins with mixing the samples using a rotary mixer, then centrifuging to separate the components. After adding 0.2 ml of RNA Extraction Agent, the samples are centrifuged again to separate the layers, and the upper aqueous layer containing RNA is transferred to a new tube. Isopropanol is added to the samples and mixed well, then centrifuged to separate the precipitate. 75% ethanol is added, then centrifuged again to form a dry layer of RNA. The RNA is dissolved in a dissolution solution and stored at -70°C, and can be stored for a long time at 55-60°C for 10 minutes.

2.4.2. Estimation of RNA concentration and purity

The samples were diluted to a concentration of 20ng/µl and used in the reaction as in the equation:

$$M_1V_1 = M_2V_2$$

TABLE 2. Extraction steps

Component	Volume
qPCR Master Mix	10 µl
RT Mix Buffer at concentration	0,4 µl
CXR Reference Dye, 30µM	0.3µl
MgCl2, 25mM	1.6µl
Forward primer	2 µl
Reverse primer	2 µl

Adjust all add-ons, transfer samples to the device, and adjust the program as in Table (3).

TABLE 3. Extraction steps

Steps	Temp.(℃)	Durataon	Cycle
Reversetranscription	37°C	10 minutes	1
RTinactivation_Hot-start activation	95.0 °C	15 seconds	40

3.StepqPCR			
a.denature	95°C	10 Seconds	
b.Anneal-collect data	60°C	30Seconds	40
c.Extend	72°C	30 Seconds	
Dissociation	60-95C°		1

2.4.3. Specific Primers Sequence used for PCR amplification

The specific primers shown in **Table 4** were designed to determine the specific sequence of the genetic segments of the genes under study, of course, according to the standard specifications of the National Center for Biotechnology and Biotechnology (NCBI).

TABLE 4. Extraction steps

Primer	Direction	Sequence (5'→3')	References
Reference gene	Forward	Hs00950362_g1	Riming Liu.,et al.,2017)
SLC5A5	Reverse	Hs00166567_m1	

3. RESULTS

3.1. Change value for *SLC5A5* gene

The results of the statistical analysis in **Table 5** and Figure 1 revealed that the *SLC5A5* gene was not significant, i.e., it was unlikely to affect the HD **Table 5**.

TABLE 5. A comparison between the control group and the affected group in the fold change value for *SLC5A5* gene

Group	B actin	*SLC5A5*	ΔCt	ΔΔCt	Fold change
Control	20.287	30.109	9.822	-0.517	1.00 ±0.00
Patients	23.642	34.538	10.896	0.556	0.48 ±0.07
T-test	--	--	--	--	0.558 NS
P-value	--	--	--	--	0.1628

NS: Non-Significant.

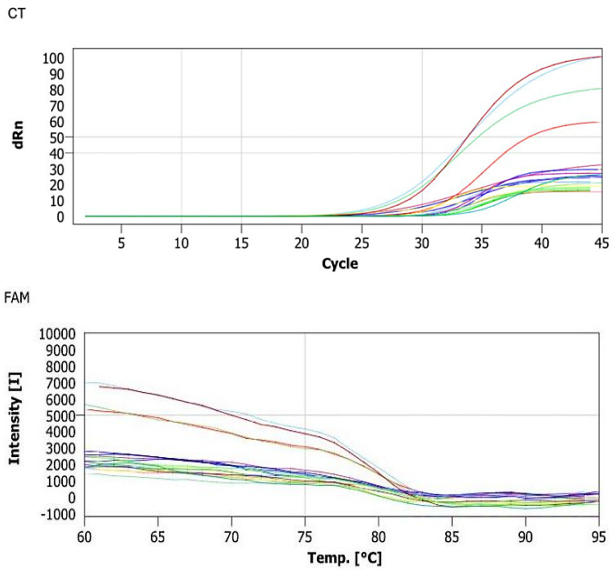

Figure 1. Exponential change curves of gene expression in the polymerase chain reaction assay for the SLC5A5 gene

3.2. Thyroid hormone tests

Table 6 shows no significant difference in TSH hormone levels between the healthy individuals and patients. However, the T3 and T4 levels were significantly lower in HD patients (P<0.01) compared to controls.

TABLE 6. Comparison between healthy group and patients in thyroid hormones.

Group	Means ±SE		
	TSH (Nmol/L)	T4 (ng/dl)	T3 (ng/dl)
Patients	11.56 ±0.62	108.57 ±1.97	1.119 ±0.03
Control	11.15 ±0.68	146.56 ±2.57	2.433 ±0.55
T-test	1.834 NS	6.424 **	1.089 **
P-value	0.659	0.0001	0.0019

**** (P≤0.01), NS: Non-Significant.**

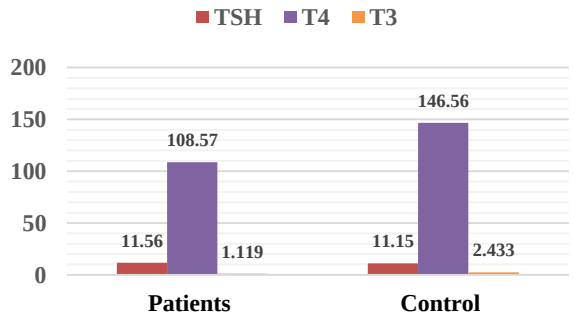

Figure 2. Comparison between healthy group and patients in thyroid hormones

3.3. Family history

The results in **Table 7** showed significant differences (P≤0.05) in the proportion of women with HD who had a family history of the condition. Additionally, the findings indicated that factors such as diabetes, hypertension, and heart disease contribute to the development of HD at a highly significant level (P≤0.01).

TABLE 7. Effect of risk factors on the development of hypothyroidism

Factors		No	Percentage (%)	P-value
Diabetes	Yes	37	74.00%	0.0007 **
	No	13	26.00%	
Pressure	Yes	36	72.00%	0.0019 **
	No	14	28.00%	
Heart disease	Yes	32	64.00%	0.0477 *
	No	18	36.00%	
Remove the gland	Yes	27	54.00%	0.571 NS
	No	23	46.00%	
Family genetic history	Yes	31	62.00%	0.0498 *
	No	19	38.00%	

* (P≤0.05), ** (P≤0.01).

4. DISCUSSION

HD is a prevalent endocrine disorder characterized by insufficient activity of thyroid hormones, which impacts a range of vital bodily functions. These functions include regulating energy metabolism, promoting growth, differentiation, and other physiological processes. Normal circulating levels of the thyroid hormones T3 and T4 are significantly reduced in HD [13].

The results of this study showed no statistically significant effect of the *SLC5A5* gene on patients with HD. These findings are consistent with the study by Geysels et al. [14], which demonstrated an absence of disease-causing variants in the coding region of the *SLC5A5* gene in three out of four patients with HD. However, our results contrast with those of Kostopoulou et al. [15], who showed that the *SLC5A5* gene is involved in thyroid dysgenesis and dyshormonogenesis. Other causes of congenital HD, such as iodothyronine transporter defects and resistance to thyroid hormones, have also been associated with genetic mutations. The reason for the lack of effect may be related to a specific sample of the population that has certain genetic patterns or environmental factors, which affects the importance of this gene in data analysis.Thus, we recommend conducting future studies on this aspect.

In contrast, HD caused a significant decrease in the levels of thyroid hormones T3 and T4 and non-affect the level of TSH. These results are consistent with Al-Farttoosi et al., [16], Hashim et al., [17], Naji [18] and Ateah et al., [14]. The decrease in hormone secretion in the results of the study is attributed to iodine deficiency or autoimmune diseases such as Hashimoto disease, that lead to produce autoantibodies, which in turn attack the thyroid cells and interfere with their ability to produce hormones [19].

Additionally, significant differences were observed in the percentage of women with HD who had a family history of the condition. In addition, the results indicated that factors such as diabetes, high blood pressure, and heart disease contribute to the development of HD, and these results are consistent with a study Hassen and Ahmed [20] which showed that family history increases the risk of hypothyroidism compared to people who do not have a family history of the disease. In terms of diabetes, the study agrees with [21] who proved that 11% of cases of HD are caused by diabetics, because diabetes cause metabolic imbalance and insulin resistance, which may affect thyroid function. Although high blood pressure and heart disease are not a direct cause of hypothyroidism, high blood pressure often coincides with other metabolic disorders and can stress the body's hormone regulation systems, indirectly affecting the thyroid gland [22].

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Arabic Abstract

هدفت هذه الدراسة إلى تحديد مقدار مستوى التعبير الجيني لجين *SLC5A5* لدى النساء المصابات بقصور الغدة الدرقية (HD)، حيث أنه أحد الجينات الرئيسية المؤثرة على تطور الحالة. بالإضافة إلى ذلك، تم فحص بعض عوامل الخطر، بما في ذلك مرض السكري وارتفاع ضغط الدم وأمراض القلب واستئصال الغدة الدرقية السابق والتاريخ الوراثي العائلي، لتقييم تأثير هذه العوامل على انتشار قصور الغدة الدرقية. بالإضافة إلى ذلك، تم فحص بعض عوامل الخطر، بما في ذلك مرض السكري وارتفاع ضغط الدم وأمراض القلب واستئصال الغدة الدرقية السابق والتاريخ الوراثي العائلي، لتقييم تأثير هذه العوامل على انتشار قصور الغدة الدرقية. تم الحصول على عينات الدم المحيطي (PB) من 50 امرأة مصابة بقصور الغدة الدرقية تم متابعتهن بالتشخيص الطبي وتراوحت أعمارهن من (40 إلى 70) عامًا. تم اختيار 50 عينة من النساء الأصحاء اللاتي لا يعانين من أمراض مزمنة كمجموعة سيطرة، تم استخدام تقنية تفاعل البوليميراز المتسلسل (qPCR-RT) لتحديد مستويات التعبير عن جين *SLC5A5* في العينات، وكذلك لقياس بعض المعايير الهرمونية لمرضى قصور الغدة الدرقية لإثبات أهميتها في تشخيص المرض وهي قياس مستوى هرمون تحفيز الغدة الدرقية (TSH) وهرمونات الغدة الدرقية الثيروكسين (T4) وثلاثي يودوثيرونين (T3). أظهرت النتائج عدم وجود فرق معنوي في مستوى التعبير لجين *SLC5A5* لدى النساء المصابات بقصور الغدة الدرقية مقارنة بالمجموعة السيطرة. بالمقابل أظهرت النتائج انخفاض كبير في هرمونات الغدة الدرقية لكل من ثلاثي يودوثيرونين (T3) والثيروكسين (T4) وعدم وجود فرق معنوي في هرمون TSH. بالإضافة إلى ذلك، أشارت النتائج إلى أن عوامل مثل التاريخ العائلي ومرض السكري وارتفاع ضغط الدم وأمراض القلب تساهم في تطور قصور الغدة الدرقية. وفي الختام، وجدا أن عوامل الخطر مثل التاريخ العائلي، ومرض السكري، وارتفاع ضغط الدم، وأمراض القلب تلعب دورًا في تطور الحالة.

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Identification of B1 and SAG3 of Toxoplasma Gondii Genes in Breast Cancer Tissues Among Females in Kerbala Province, Iraq

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P A P E R I N F O

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Keywords:
Breast carcinoma, FFPE, tissues, PCR, T.gondii, females

Abstract

The present investigation was done in November 2023. One hundred fifty specimens of malignant Breast cancer tissue, covered with paraffin wax and diagnosed by a specialist, have been gathered along with fifty specimens of benign tumour tissue. The control set was comprised of specimens taken from the histopathologist lab at Imam AL- Hussein Medical City (IHMC), the Al-Kafeel Spec Iality Hospital Lab (KSHL), and the Specialized Al-Sajjad Laboratory (SSL) for histopathology and tumour identification. The investigation contained two sets of specimens. The initial cohort comprised 66 females with breast carcinoma who did not receive chemotherapy, while the subsequent cohort included 84 females with breast carcinoma who did take chemotherapy. PCR technique was utilized on DNA from specimens of tissue afflicted by breast carcinoma across all studied sets, focusing on the amplification of the B1 and SAG3 genes.This work attempted to ascertain the prevalence of toxoplasmosis in patients with breast carcinoma. The toxoplasmosis incidence in patients with breast carcinoma was 11.3%, and the infection rate in normal participants was 4 percent. The data indicated that the infection rate among females who underwent therapy was higher than that of untreated females, attributable to their weakened immunity and increased infection risk, resulting in a 3.7 % greater probability of patients females acquiring breast carcinoma in comparison to normal females.

1. INTRODUCTION

Toxoplasmosis was a zoonotic disease resulting from *Toxoplasma gondii (T.gondii)*, an obligate intracellular protozoan that infects people as well as animals that are warm-blooded as intermediate hosts, with different feline family members serving as intermediate and final parasite hosts [1]. Toxoplasmosis infection is worldwide in its spread among humans, as it varies from one region to another, and about a 3rd the world's population is exposed to infection with the parasite [2]. The infection is often symptomatic in immune-competent persons. However, it can be severe and perilous for those who are immunocompromised, such as pregnant females and those with AIDS. Research identified the potential of the *T.gondii* parasite to induce dysplasia in the reproductive system and aberrant adhesions inside the uterus, leading to infertility in females [3].

Cancer is an abnormal and random growth of cells that originate from a single cell with

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malfunctioning regulatory mechanisms and whose growth exceeds and does not keep pace with the growth of the normal tissue from which it originated. These cells continue to hyperdrive even after the stimulus or stimulus that provoked these changes has disappeared [4].There are many kinds of cancer and the most important one is breast carcinoma; it is one of the most common kinds in females and it has been diagnosed repeatedly among female all over the world. It is considered the second leading to cancer-related deaths [5]. It has been observed that the incidence of the number of infections in Iraq with this kind of cancer has increased in the latest years from 30/100,000 to 40/100,000 in the period between 2006 and 2012 [6]. Breast carcinoma ranks first among the malignant tumors that affect society. In Iraq, it represents 19.5 percent of all cancer cases and 34.3 percent of cancers that affect females. In 2016, more than 897 females died from this disease, which is the first cause of cancer-related deaths among Iraqi females [7]. T.gondii is a possible cause of cancer and it has a function in the development and induction of malignant diseases. This is explained by several theories, including preventing apoptosis and enhancing the movement of dendritic cells and macrophage cells. Another theory detected that *T.gondii* works to accumulate oncogenic mutants due to the disruption of traditional barriers [8]. Studies have proven a potential relationship between malignant and toxoplasmosis diseases, including brain and oral cancers [9]. Furthermore, in Iran, *T. gondii* DNA was discovered in breast carcinoma tissues fixed with paraffin and formalin wax [10]. The genetic and diagnosis description of the *T.gondii* is essential in clinical management, epidemiological investigation, and control of parasites in animals and humans [11]. Polymerase chain reaction (PCR) has been widely utilized in detecting Toxoplasmosis infection since it was first utilized by [12] to target the *B1* gene [13]. PCR also utilizes traditional sequences targeting single-copy genes such as *SAG1*, *SAG2*, *SAG3*, and *GRA1* in humans and animals [14].

2. MATERIALS and METHODS

2.1 Specimens Collection

A total of 150 specimens of aggressive breast carcinoma tissue, embedded in paraffin wax and identified by a specialist, were gathered, along with fifty specimens of benign tumor tissue. They were deployed as a control set. The specimens were obtained from the IHMC, KSHL, and the SSL tissue cutting lab. The investigation comprised two sets of specimens. The initial set comprised 66 females with breast carcinoma who were not given chemotherapy. On the other hand, the second set consisted of 84 females with breast carcinoma who had undergone chemotherapy. PCR technology was utilized on DNA from breast carcinoma-affected tissue specimens across all studied sets, focusing on amplifying the *B1* gene. *SAG3*.

2.2 Genomic and Extraction

Deoxyribonucleic acid was isolated from cancerous breast tissue specimens. Five sections, each 10 microns thick, were removed from tumor tissue of breast encased in paraffin wax to isolate the DNA genomes utilizing the DNA Genomic Mini Kit Tissues Protocol, mainly developed for this procedure. I used the Korean commercial kit manufactured by Favorgen for extracting DNA from cancerous breast tissue specimens.

2.3 The Utilized Primer In PCR

Particular primers for the *B1* gene have been obtained from [15] and for the *SAG3* gene from [16], manufactured by the Korean business Bioneer, as shown in **Table 1**.

TABLE 1. Sequence of primers used in molecular studies

Primers sequence (5`-3`)			PCR product size
Gene *B1*	Primer Forward	GGAACTGCATCCGTTCATGAG	194bp
	Primer Reverse	TCTTTAAAGCGTTCGTGGTC	
Gene *SAG3*	Primer Forward	CGCGACAC AAGCTGCGATAG	1158bp
	Primer Reverse	TTAGGCAGCCACATGCACAA	

The PCR was conducted utilizing a commercial 20µ Reaction kit from the Korean business Addbio, following the manufacturer's instructions (Table 2).

TABLE 2. Components of the Master Blend for PCR

Components	Volume (µL)
Polymerase enzyme Taq DNA Polymerase	1U
Each: d NTP(d ATP, CTP,d GTB,dTTP A mixture of nitrogenous bases	400 µL
Buffer solution	10 µL
Loading dye	30 µL
MgCl2	3 µL

2.4 Preparing the Polymerase Chain Reaction (PCR) Mixture

This combination was created according to the manufacturer's specifications in Table (2). All components listed in the Table have been put in specialized tubes. The tubes have been sealed and subjected to the microcentrifuge at the highest speed for thirty seconds. The tubes have been moved to the thermal cycler of PCR.

TABLE 3. PCR Mixture

PCR Blend	Volume (µL)
Extraction of DNA	2
Primer Forward	2
Reverse Primer	2
Free DNAase water	6.5
Master mixing	12.5
Total volume	25

2.5 PCR Program PCR Thermo cycle

The PCR was conducted applying a thermal cycler from the Chinese business Biobase, resulting in the amplification of the target genes B1 and SAG3 to sizes of 1158 bp and 194 bp, respectively, as illustrated in Tables 4 and 5.

TABLE 4. The *B1* gene utilized program in the PCR machine

Step	Temp (C°)	Duration	Number of Cycles
DNA Initial Denaturation	95	3 min	35
Denaturation		3s	
Annealing	60	30 min	
Extension	72	1	
Final extension		5	

TABLE 5. Thermal cycling program for the *SAG3* gene

Step	Temp (ºC)	Duration	Number of Cycles
DNA Initial Denaturation	95	5 min	35
Denaturation		35s	
Annealing	60	30 min	
Extension	72	55s	
Final extension		5min	

Five microliters from PCR products were visualized utilizing a UV Transilluminator. The remaining 20 microliters from the PCR product were subjected to DNA sequences to identify genetic variation. The favorable PCR *SAG3* gene products were shipped in an ice bag to Macrogen Company in Korea for DNA sequencing on an AB DNA sequencing machine through DHL. Molecular Evolutionary Genetics Analysis version 6.0 was utilized to calculate evolutionary distances by employing the "Maximum Composite Likelihood technique utilizing the phylogenetic Tree (UPGMA) Method and the Multiple Sequence Alignment Analysis of the Incomplete *SAG3* Gene Based on the Clustal W Alignment Analysis" by Utilizing Software (Mega 6.0). The collection of *T.gondii* sequences in Genbank of *T.gondii* sequences was performed utilizing "NCBI (https://www.ddbj.nig.ac.jp/ddbj/updt-form-e.html)" to compare local SAG3 sequences with other global sequences. The main inclusion criterion was linear DNA sequences of the SAG3 marker with a broad geographical representation from different regions with definitive or intermediate hosts of similar size. The size of the *SAG3* marker is 1158 bp. Phylogenetic analysis utilizing multiple sequence alignments, especially when the sequences are not highly conserved, necessitates the removal of poorly matched locations and divergent sections, as these regions are unlikely to be homologous and may be saturated by repeated replacements.

TABLE 6. Investigation sets based on infection

	Investigation sets				Total		X^2p-magnitude	OR (95 percent CI)	RR (95 percent CI)
Toxoplasma. test	patients		Control						
	N	percent	N	percent	N	percent			
Ve+Toxo.	17	11.3percent	2	4.0 percent	19	9.5 percent	2.346 0.126 Ns	3.07 (0.68-3.77)	2.83 (0.68-11.84)
Ve- Toxo.	133	88.7percent	48	96.0 percent	181	90.5 percent		Ref.	

2.6 Sectioning

The wax molds containing the tissues were placed in the microtome manual tissue cutting equipment, with the desired thickness set at about 3 to 5 micrometers, to produce strip sections of the tissue samples. The strips are arranged on a black plate for selection beside 1.5 ml Eppendorf tubes [17].

2.7 Tissue staining

The tissue slices have been stained with hematoxylin-eosin according to the techniques outlined in [18]. The paraffin-embedded and formalin-fixed cancerous breast tissues were sectioned to 4-5 microns, affixed to glass slides, and allowed to dry at normal temp for one day.

2.8 Statistical analysis

The categories of factors are expressed as percentages and numbers and evaluated utilizing Fisher's exact or chi-square tests, as suitable [19], [20]. Continuous factors are expressed as average±standard deviation and evaluated utilizing Student's t-test. Risk variables for breast carcinoma were evaluated applying a model of logistic regression and reported as odds ratios (OR) with corresponding confidence intervals (CI)95 percent. All assessments have been performed utilizing SPSS version 28. Differences with p-magnitudes below 0.05 have been considered statistically significant.

3. RESULTS AND DISCUSSION

This study detected 150 females diagnosed with cancer of the breast, with a mean age ranging from 25 to 56 years. 17 females infected with *T.gondii* have been identified as illustrated in Table 1:

Toxoplasmosis incidence increased to almost 40 percent during the occupation of Iraq, in contrast to 2 percent in the 1980s [21]. In 2016, 335 patients infected with Toxoplasma were documented throughout all Iraqi governorates [22]. The frequency of toxoplasmosis varies for several reasons, including climatic conditions differences and cultural patterns throughout various areas of Iraq [23]. Research in Iraq detected that the prevalence of breast carcinoma among females was 33.81%. Relative to other Arab country, the breast carcinoma incidence was lower in certain countries, including Bahrain, Jordan, and Kuwait.

Conversely, the rate was elevated in other Arab nations, including Qatar, the UAE, Oman, and Saudi Arabia, as well as in bordering Arab countries like Turkey and Iran [24], [25]. Limited information exists on the incidence of T. gondii infection in immunocompromised individuals undergoing neoplastic disease treatment or immunosuppressive therapy in Iraq [26]. Recent research detected that the incidence toxoplasmosis rate among patients with breast carcinoma was 77.50 percent [27]. Chronic inflammation often induces carcinogenesis and can expose a person to cancer.

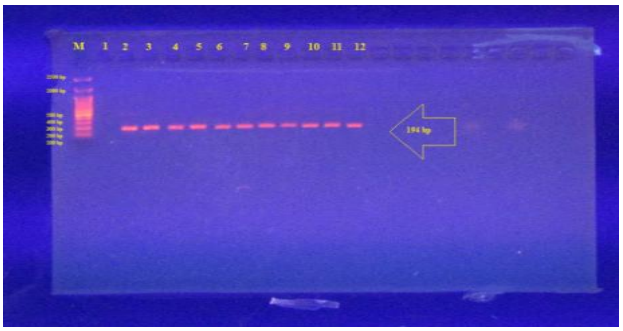

Figure 1. *B1* gene DNA Detection

T.gondii B1 gene PCR product Electrophoresis was conducted on an agarose gel at a voltage=60 for thirty minutes, employing the fluorescent red dye Ethidium Bromide. The specimens exhibited a band corresponding to 194 base pairs of DNA isolated from patients with cancerous breast tissues.

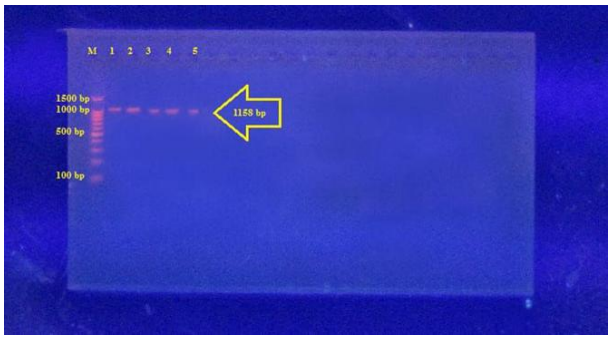

Figure 2. *SAG3* gene DNA Detection

T.gondii parasite (*SAG3* gene) PCR product Electrophoresis was conducted on an agarose gel at a voltage=60 for thirty minutes, employing the fluorescent red dye Ethidium Bromide. The specimens exhibited a band corresponding to 1158 base pairs of DNA isolated from patients with cancerous breast tissues.

TABLE 7. Relationship of toxoplasmosis with cancer kind

cancer kind	***T.gondii***				**Total**		X^2 **p-magnitude**	**OR (95 percent CI)**
	Ve+		**Ve-**					
	N	percent	N	percent	N	percent		
Invasive ductal carcinoma	16	94.1percent	126	94.7percent	142	94.7percent	0.011 0.915	0.89 (0.103-7.70)
Lobular ductal carcinoma	1	5.9percent	7	5.3percent	8	5.3percent		Ref.
Total	17	100percent	133	100percent	150	100percent		

The present investigation detected the highest infection rate with the parasite *T.gondii*, as shown in Table 2. The findings revealed that all kinds of breast carcinoma are susceptible to infection with *T.gondii*,

and there is no specific kind of infection. The findings detected that the kind most affected by females is invasive ductal carcinoma because it is the most popular kind of breast carcinoma.

TABLE 8. Relationship of *T.gondii* with breast carcinoma grade

cancer grade	***T.gondii***				**Total**		***X*² p-magnitude**	**OR (95 percent CI)**
	Ve+		**Ve-**					
	N	percent	N	percent	N	percent		
Grade 1	0	0.0 percent	9	6.8 percent	9	6.0percent	1.452 0.484	-
Grade2	9	52.9 percent	73	54.9 percent	82	54.7percent		Ref.
Grade3	8	47.1 percent	51	38.3 percent	59	39.3percent		0.79 (0.28-2.17)
Total	17	100 percent	133	100 percent	150	100percent		

The present investigation also detected the grades of breast carcinoma in *T.gondii* patients, and the statistical findings detected which grade of cancer is at risk for infection with *T.gondii*, as shown in Table (3).

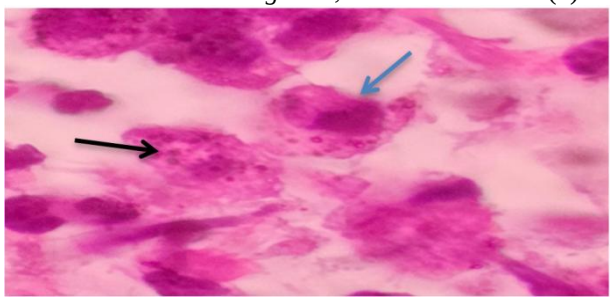

Figure 3. A tissue section from breast carcinoma is affected by the *T.gondii* parasite, the Cytoplasm of macrophage cell *s, and the T.gondii* parasite (1000x H&E).

The current research findings align with a survey conducted to ascertain the existence of the parasite in 900 patients in China, as reported by [28], [29], where the parasite's DNA was obtained from the cancer

tissues of 510 patients, and the *T. gondii* B1 gene was amplified utilizing a nested PCR technique. The survey investigation identified an overall infection incidence of 35.56 percent among patients of cancer categorized by organ kind [30]. Patients with lung cancer had the most remarkable prevalence rate of the *T. gondii* parasite at 60.94percent, then patients with cervical cancer at 50 percent, patients with brain cancer at 42.31 percent, and patients with endometrial cancer at 41.67percent. The infection of *T. gondii* in patients with cancer was significantly associated with soil exposure and the consumption of undercooked meat [31]. The infection rate in the breast tissues was reduced in the present study due to the potential non-uniform distribution of the parasite throughout the sampled tissues or biopsies. Several studies indicate that the hormonal system in tissues contaminated with the parasite *T.gondii* significantly contributes to the parasite's stability by affecting its tissue activities and enabling it to exploit host cells for its benefit. The parasite *T.gondii* can manipulate the immune system of infected individuals, utilizing hormonal alterations to evade immune detection by binding to specific receptors on the parasite, thereby inhibiting the action of host-secreted antibodies.

Furthermore, the immune reaction to *T.gondii* might contribute to neurological and histological alterations. *T.gondii* identified the uneven necrosis distribution in infected tissues, and there is definitive evidence of the parasite's presence in neurons. It induces infection by the *T.gondii* parasite [32]. In tissue, injury induces the synthesis of various inflammatory and cytokines mediators that facilitate inflammation in neighboring blood vessels, fibrous tissue, and immune cells, resulting in proliferation and hypertrophy of the tissues [33]. Proliferating epithelial cell layers and faulty cuboidal or basal cells in prostate tissue during the rapidly expanding phase of chronic toxoplasmosis inside host cells [34].

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Arabic Abstract

تم إجراء هذه الدراسة في نوفمبر 2023، وشملت جمع 150 عينة من أنسجة سرطان الثدي الخبيث المغطاة بشمع البارافين، والتي تم تشخيصها من قبل أخصائي، بالإضافة إلى 50 عينة من أنسجة أورام حميدة. تم الحصول على عينات مجموعة التحكم من مختبرات مدينة الإمام الحسين الطبية(IHMC) ، ومختبر مستشفى الكفيل التخصصي(KSHL) ، ومختبر السجاد التخصصي (SSL) لتحليل الأنسجة والخلايا وتحديد الأورام. شملت الدراسة مجموعتين: الأولى تضمنت 66 سيدة مصابة بسرطان الثدي ولم يتلقين العلاج الكيميائي، بينما تضمنت الثانية 84 سيدة تلقين العلاج الكيميائي. تم استخدام تقنية PCR على الحمض النووي المستخرج من أنسجة المرضى في جميع المجموعات، مع التركيز على تضخيم الجينات B1وSAG3لتحديد انتشار مرض التوكسوبلازما. أظهرت النتائج أن معدل الإصابة بالتوكسوبلازما بين مرضى سرطان الثدي بلغ 11.3%، مقارنة بـ 4% بين الأفراد الأصحاء. كما أشارت البيانات إلى أن معدل الإصابة كان أعلى بين السيدات اللواتي خضعن للعلاج الكيميائي، بسبب ضعف المناعة وزيادة خطر العدوى، مع زيادة بنسبة 3.7% في احتمالية إصابة المريضات بسرطان الثدي مقارنة بالمشاركات الأصحاء.

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New Type of Generalized Continuous Functions in Proximity Topological Space

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P A P E R I N F O

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Abstract

The paper deals with the importance of the continuum theory as a mathematical concept and its great role in solving many problems. Since the term proximity is a modern term, we were able in this research to answer a very important question: Is it possible to study types of continuity in proximity space like the types found in the usual topological space? In this research, we were able to find a type of continuity (δ_ω −continuous) and obtain many results, relying on proximity theories and the different definitions of proximity space, and we were able to link them to the new definition that was established.

1. INTRODUCTION

Continuity is almost as old as general topology. Both notions are firstly mentioned by Fr`echet, topological structure in 1906 [2]. The importance of continuity in general topology is that the continuous image of a connected set is connected which is important in digital image [2, 4]. In 1909, Riesz introduced in his "theory of enchantment" proximity structures [1,4]

Then, specifically in1952 Efremovič rediscovered the subject (see Nainpally and Warrack, 1970; Engelking, 1977) [1,4]. They (Riesz and Efremovič) defined and developed the axioms of relationship between sets in a metric space by stating "A is near to B i.e. $A\delta B$) if and only if $D(A,B) = \inf\{d(x,y): x \in A, y \in B\} = 0$ [4]. Further, the notion of δ −neighborhood started from, Efremovič recast his axiomatization in term of strong inclusion. Lodato [8,9] and [6,7] Leader have worked with weaker axioms those of Efremovič proximity space that enable them to define an arbitrary topology on the underlying set. It must be mentioned that L.A. Al Swidi, has presented many studies and researches in the field of studying proximity space [5,12].

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Generally speaking, [3] topology representsit (K. Kuratowski) a closeness between points and sets, while proximity (V.A. Efremovič) is a closeness between sets. Topology less than proximity which have more structure ones carry but carry less structure than metric ones[2].Thus a notion of proximal continuity has been proposed, with the property of preserving the closed of two sets. Simple continuity is weaker than the proximity continuity since it preserves nearness of a point and a set [2]. Furthermore, [3] proximity relations are important solution problems based on human perception.

This paper is an attempt to present a new type and a new definition of continuity function in proximity space.

2. PRELIMINARIES OF PROXIMITY SPACE

In this section, proximity space will be introduced in addition to presenting important definition and proposition.

2.1 Definition [5, 6, 12]

On the family $P(\chi)$ a relation δ called a proximity on χ of if it satisfies the following conditions all subsets of a set χ:

$(P1)$ If A δ Γ, then Γ δ A;

$(P2)$ A δ (Γ ∪ H) if and only if either A δ Γor $A\ \delta\ H$;

$(P3)$ $\chi\ \overline{\delta}\ \emptyset$;

$(P4)$ $\{x\}\ \delta\ \{x\}$ for each $x \in \chi$;
$(P5)$ if $A\bar{\delta}\ \Gamma$, then there exist $\mathrm{E} \in P(\chi)$ such that $A\bar{\delta}\ \mathrm{E}$ and $\chi - \mathrm{E}\ \bar{\delta}\ \Gamma$, $(i.e\ \forall \mathrm{E}, A\delta \mathrm{E}\ or\ \Gamma\delta\chi - \mathrm{E} \Longrightarrow A\delta\Gamma$ (see [12]). We called the pair $(\chi;\ \delta)$ a proximity space. If $(P4)$ is instead of $(P'(4))\{\mathrm{P}\}\delta\ \{o\}$ if and only if $\mathrm{P} = o$, then the realtion δ is called a separated proximity, $(\chi;\ \delta)$ called a separated proximity space.

2.2 Example [5, 6]

Discrete and indiscrete proximity were defined as

(i) If we defined $A\delta_1\Gamma$ if and only if $A \cap \Gamma \neq \emptyset$, then δ_1 is discrete proximity on χ.

(ii) If $A\delta_2\Gamma$ for every $\mathrm{A} \neq \emptyset \neq \Gamma$ and $A, \Gamma \subseteq \chi$, then δ_2 is indiscrete proximity on χ.

2.3 Definition [5]

If δ_1 and δ_2 are two elements of class P of all proximities that are defined on a set χ,inclusion is defined as

$\delta_1 > \delta_2$ if and only if $A\ \delta_1\Gamma$ implies $A\ \delta_2\Gamma$ .In such a case we say $that\ \delta_1$ is finer $then\ \delta_2$, or $\delta_2$is coarser than δ_1.

2.4 Proposition [4, 5, 12]

Let $(\chi;\ \delta)$ be a proximity space. Then
(a) if $A\ \delta\ \Gamma$ and $\Gamma\ \delta\ C$, then $A\ \delta\ C$;
(b) if $A\ \bar{\delta}\ \Gamma$ and $C \subseteq \Gamma$, then $A\bar{\delta}C$;
(c) if there exists a point $\mathrm{P} \in X$ such that $A\ \delta\ \{\mathrm{P}\}$ and $\{\mathrm{P}\}\ \delta\ \Gamma$, then $A\ \delta\ \Gamma$;
(d) if $A \cap \Gamma \neq \emptyset$, then $A\ \delta\ \Gamma$;
(e) $A\bar{\delta}\emptyset$ for every $A \subseteq \chi$;
(f) if $A\ \delta\ \Gamma$, then $A \neq \emptyset$ and $\Gamma \neq \emptyset$.

2.5 Proposition [7]

The axiom (P5 in Definition 1.1] is equivalent to any of the following statements:

(i) If $A\bar{\delta}B$, then there are sets M and N such that $A\bar{\delta}M, B\bar{\delta}N$;

(ii) If $A\bar{\delta}B$, then there are sets M and N such that $A\bar{\delta}\chi - M, X - N\bar{\delta}B$ and $M\bar{\delta}N$;

(iii) If $A\bar{\delta}B$, there are two sets M and N such that $A\bar{\delta}\chi - M$,and $B\bar{\delta}\chi - N, M \cap N = \emptyset$.

2.6 Definition [2, 6]

Let (χ, δ) be a proximity space, then for all $A, B \subset \chi$, B a proximity or δ -neighborhood of A and denoted that relation as $A \ll B$if and only if $A\bar{\delta}\chi - B$.

2.7 Theorem [2]

Let (X, δ) be a proximity space. Then the relation$\ll$ satisfies the following properties:
$(O1)$ $\chi \ll \chi$;
$(O2)$ If $\mathrm{A} \ll B$, then $\mathrm{A} \subset B$;
$(O3)$ $\mathrm{A} \subset B \ll C \subset D$ implies $\mathrm{A} \ll D$;
$(O4)$ $\mathrm{A} \ll B$ implies $\chi - B \ll \chi - \mathrm{A}$;
$(O5)$ $\mathrm{A} \ll B_k$ is true for $k = 1, 2, \ldots, n$ if and only if $\mathrm{A} \ll \cap_{k=1}^{n} B_k$;
$(O6)$ If $A \ll B$, then there exists a set $N \subset X$ such that $A \ll N \ll B$. This is implies $A \ll int\ N \subset cl\ N \ll intB \subset clB$. (see Proximity Approach to Problem in Topology and Analysis). If δ is a separated proximity, then
$(O7)$ $\{\mathrm{P}\} \ll \chi - \{c\}$ if and only if $\mathrm{P} \neq c$.

2.8 Corollary [6]

If $A_\pi \ll B_\pi$,$\pi = 1, 2, \ldots n$, *then*

$$\bigcap_{\pi=1}^{n} A_\pi \ll \bigcap_{\pi=1}^{n} B_\pi \quad and \quad \bigcup_{\pi=1}^{n} A_\pi \ll \bigcup_{\pi=1}^{n} B_\pi$$

2.9 Remark [6]

The family all δ-neighborhoods of a set ω in proximity space(χ, δ) is $\mathcal{F}(\omega)$and δ-neighborhoods in general is not open set with respect to this topology.

2.10 Proposition [7,6]

Let (χ, δ) be a proximity space. Then
$(\iota 1)$ $\Gamma \in \mathcal{F}(\mathrm{A})$ implies $A \subset \Gamma$;
$(\iota 2)$ $\Gamma \in \mathcal{F}(A)$ implies $X - A \in \mathcal{F}(\chi - \Gamma)$;
$(\iota 3)$ If $A \subset \Gamma$, then $\mathcal{F}(A) \subset \mathcal{F}(\Gamma))$;
$(\iota 4)$ $\mathcal{F}(A \cup \Gamma) = \mathcal{F}(A) \cap \mathcal{F}(\Gamma)$;
$(\iota 5)$ If $\Gamma \in \mathcal{F}(A))$, then there exists a $C \in \mathcal{F}(A)$ such that $\Gamma \in \mathcal{F}(C))$;
(f) $\mathcal{F}(A) \cap \mathcal{F}(\Gamma)) \subset \mathcal{F}(A \cap \Gamma)$, where $\mathcal{F}(A) \cap \mathcal{F}(\Gamma) = \{C \cap D : C \in \mathcal{F}(A), D \in \mathcal{F}(\Gamma)\}$.

3. TOPOLOGY GENERATED BY a PROXIMITY

In this part, we will consider the topology on χ induced by a proximity on X, and we will study some definitions and elementary properties.

3.1 Theorem [2, 9, 10]

The family T_δ in a proximity space (χ, δ) is called a topology on the set χ.

3.2 Remark [5]: If (χ, δ_J) is a proximity space, then it has a unique topology T_{δ_J}generated by δ_J.

3.3 Definition [5]

Let (χ, δ) be a proximity space. A subset $F \subset \chi$ is to be closed in χ if and only if $\mathrm{P}\ \delta\ F$ implies $\mathrm{P} \in F$. By T_δ denotes the family of complements of all the sets defined in such a way.

3.4 Proposition [7,5]

If G is a subset of a proximity space (χ, δ), then G is called open in topology T_δ if and only if $\{\mathrm{P}\}\ \bar{\delta}\ \chi - G$ for every $\mathrm{P} \in G$.

3.5 proposition [7,6]

For any two proximity relations δ_1, δ_2 in X, if $\delta_1 < \delta_2$, then $\mathrm{T}_{\delta_1} \subset \mathrm{T}_{\delta_2}$.

3.6 Proposition [7,11]

If A and Ω are subsets of a proximity space (χ, δ), then $\mathrm{A}\ \bar{\delta}\ \Omega$ implies:

(i) $\bar{\Omega} \subset \chi - \mathrm{A}$; and

(ii) (ii) $\Omega \subset int\ (\chi - \mathrm{A})$.

3.7 Proposition [4,11]

If $\bar{A}$ and int(A) detnote, respectively, the closure and the interior of the set A of a proximity space (X, δ) with respect to the topology T_δ, then

(i) $A \ll \Omega$ implies $\bar{A} \ll \Omega$

(ii) $A \ll \Omega$ implies $A \ll \text{int}(\Omega)$.

3.8 Theorem [11]

Let $\emptyset \neq Y \subset \chi$ *and* (χ, δ) be a proximity space, for $A, B \subset Y$ let $A\delta_Y B$ if and only if $A\ \delta B$.Then (Y, δ_Y) is a proximity space.

3.9 Proposition [5]

Let (χ, δ)be a proximity space, $\emptyset \neq Y \subset \chi$, then $\mathcal{F}_Y(A) = \{\Omega \subseteq Y, A \ll_Y \Omega\} = \mathcal{F}_\chi(A) \cap \{Y\}$.

3.10 Definition [6]

Let (χ, δ) be a proximity space, and let non empty set $Y \subset \chi$, the restriction on Y *of* the proximity δ and is denoted by δ_Y is defined on the subset Y of the set χ.The ordered pair (Y, δ_Y) is called the proximity subspace of the proximity space (X, δ).

4. Proximally of continuous functions

In this part, we will discuss the most important definition and special feature of continuity in proximity space.

4.1 Definition [1, 2, 3]

Let (χ, δ_χ) and $(\Upsilon, \delta_\Upsilon)$ be two proximity spaces. The mapping $f : \chi \to \Upsilon$ is said to be proximally or δ-continuous if $A\delta_\chi \eta$ implies $f(A)\delta_\Upsilon f(\eta)$ for every two sets $A, \eta \subset \chi$.

4.2 Proposition

If f is $\delta -$ continuous and onto function, then inverse image of each $T_{\delta_Y} -$ open set is $T_{\delta_\chi} -$open set.

4.3 Proposition [5]

Let a mapping $f : \chi \longrightarrow Y$, where (Y, δ) is a proximity space and let a relation on $P(\chi)$ of the set χ in the following way:

$A\delta^* \eta$ if and only if $f(A)\ \delta\ f(\eta)$

The inverse image of the proximity δ is relation δ^* defined in such a way and denoted by $f^{-1}(\delta)$.

4.4 Proposition [1, 4]

If $f : \chi \to Y$ and δ *is* a proximity on the set Y, then $f^{-1}(T_\delta) = T(f^{-1}(\delta))$.

4.5 Corollary [5]

If $f : \chi \to Y$ and if δ_1 and δ_2 are the proximities on Y for which $\delta_1 < \delta_2$, then $f^{-1}(\delta_1) < f^{-1}(\delta_2)$ hold.

4.6 Proposition [1, 2]

A mapping $f : \chi \to Y$ of a proximity (χ, δ_χ) into a proximity space $(Y, \delta_Y)$is δ-continuous if and only if for every two sets, $K \subset Y$, $R\bar{\delta}_Y K$ implies $f^{-1}(R)\ \bar{\delta}_\chi f^{-1}(K)$.

4.7 Corollary [5]

Let $f : \chi \to \Upsilon$ be a mapping from a set χ on a proximity space $(\Upsilon, \delta_\Upsilon)$, then$\delta_\chi = f^{-1}(\delta_\Upsilon)$) is the coarsest proximity on χ for which f is a δ-continuous mapping.

4.8 Corollary [5]

Let δ_1 and δ_2 be two proximities χ. The identity mapping i: $(\chi, \delta_1) \to (\chi, \delta_2)$from the set χ is a δ-continuous mapping if and only if $\delta_1 > \delta_2$.

5. δ_ω −CONTINUOUS FUNCTION

5.1 Definition

Let $f : (\chi, \delta_\chi) \to (Y, \delta_Y)$ be a mapping, the f is said to be δ_ω −continuous if and only if for all $\theta \in \chi, and$ for all $V \subset Y$, $f(\theta)\bar{\delta}_Y Y - V$, there exist $U \subset \chi, \theta\bar{\delta}_\chi \chi - U$, $f(U)\bar{\delta}_Y Y - V$.

5.2 Example

Let $f : (\chi, \delta_\chi) \to (Y, \delta_Y)$ such that $\chi = \{1,2\}, Y = \{3,4\}$ be a mapping, defined as follow $f(1) = 3, f(2) = 4$ for all $\theta \in \chi$, and the proximity relation defined on χ, Y respectively as follows:

δ_χ: $AS_\chi B \leftrightarrow A \cap B \neq \emptyset$ and δ_Y: $A\delta_Y B \leftrightarrow A \cap B \neq \emptyset$, then

δ_X={(χ,χ),(χ,{1}),(χ,{2}),({1},{1}),({2},{2}),({1},χ),({2}, χ)}, and

δ_Y = $\{(Y, Y), (Y, \{3\}, (Y, \{4\}), (\{3\}, Y), (\{4\}, Y), (\{3\}, \{3\}), (\{4\}, \{4\})\}$.

Now if $x = 1, f(\theta) = f(1) = 3$, then

(i) if $V = \{3\}, f(1) = 3\bar{\delta}_Y Y - \{3\} = \{4\}$,

there exist $U = \{1\}, \{1\}\bar{\delta}_\chi X - \{1\} = \{2\}$, and

$f(U) = f(\{1\}) = 3\bar{\delta}_Y Y - \{3\}$={4}.

(ii) If $V = \{4\}$, then $f(1) = 3\delta_Y Y - \{4\} = \{3\}$,

Let $x = 2, f(x) = f(2) = 4$, then

(iii) If $V = \{3\}, f(2) = 4\delta_Y Y - \{3\} = \{4\}$,

If $V = \{4\}$, then $f(2) = 4\bar{\delta}_Y Y - \{4\} =$ {3}, there exist $U = \{2\}, 2\bar{\delta}_\chi \chi - \{1\}$,

$f(U) = f(\{2\}) = 4\bar{\delta}_Y Y - \{4\}$={3}, that's imply f is δ_ω −continuous function.

5.3 Remark

Notes that f in above example is proximity continuous function ($\delta -$ continuous) since $\{3\}\bar{\delta}_Y\{4\}$ and $f^{-1}\{3\}\bar{\delta}_\chi f^{-1}\{4\}$.

5.4 Remark:

If f: $\left(\chi, T_{\delta_\chi}\right) \to \left(Y, T_{\delta_Y}\right)$ such that $T = T_{\delta_\chi}$ and f is δ_ω −continuous function, then f is continuous function since if f is δ_ω −continuous function, then [by Definition 5.1] for all $x \in \chi, V \subset Y$, such that $f(x)\bar{\delta}_Y Y - V, f(x) \ll_Y V$, there exist $U \subset X, x\bar{\delta}_\chi \chi - U, x \ll_X U$ and $f(U)\bar{\delta}_Y Y - V$, $f(U) \subset V$ imply $f$is continuity function.

But conversely, is not true as that example

5.5 Example:

Let: $(\chi,\delta_\chi)\to(Y,\mathrm{T}_{\delta_Y})$, $\mathrm{T}_\chi=\mathrm{T}_{\delta_{I\chi}}$ is indiscrete proximity topological space defined on a set $\chi=\{\sigma,\mu\}$ and $\mathrm{T}_{\delta_{DY}}$ is discrete proximity topological space and $\mathrm{T}_Y=\mathrm{T}_{\delta_{DY}}$ defined on a set $Y=\{\rho,\vartheta,\alpha\}$ such that $f(\sigma)=f(\mu)=\alpha$.
If $x=\sigma, f(\sigma)=\alpha$ and $V=\alpha$, then $f(\sigma)=\alpha\bar{\delta}_Y Y-\{\alpha\}=\{\rho,\vartheta\}$ since $\mathrm{T}_{\delta_{DY}}$ is discrete proximity topological space, there isn't not exist $U\subset\chi$ such that $x\bar{\delta}_\chi\chi-U$, hence f is not δ_ω −continuous function.

5.6 Remark

If $f:(\chi,\delta_\chi)\to(Y,\delta_Y)$ is δ −continuous function, then f is δ_ω −continuous function, since if for all $x\in\chi, V\subset Y$, $f(x)\bar{\delta}_Y Y-V, f(x)\ll V$[by Definition2.6], then there exist $h\subset Y$such that $f(x)\ll h\ll V$ [by Theorem 2.7 (O6)], put $h=f(U)$, then $f(\theta)\ll f(U)\ll V$ imply $f(U)\ll V$, $f(U)\bar{\delta}_Y Y-V$ [by Definition2.6], and to prove $\theta\bar{\delta}_\chi\chi-U$ since $f(\theta)\bar{\delta}_Y Y-f(U)$ [by Definition 2.5] $f(\theta)\ll f(U)$, $f$is δ −continuous, then $f^{-1}(f(\theta))\bar{\delta}_\chi f^{-1}(Y-f(U))$[by Proposition 3.5] imply $\theta\bar{\delta}_\chi\chi-U, U\subseteq\chi$, then f is δ_ω −continuous function.

5.7 Proposition

$f:(\chi,\delta_\chi)\to(\gamma,\delta_\gamma)$ is δ_ω −continuous if and only if for all $x\in\chi$ and for all $V\subset\gamma, f(x)\ll V$, there exist $U\subset\chi, x\ll U, f(U)\ll V$.

Proof

Let $f:(\chi,\delta_\chi)\to(\gamma,\delta_\gamma)$ is δ_ω −continuous from proximity space χ to proximity space γ then for all $x\in\chi$, and for all $V\subset\gamma$, $f(x)\bar{\delta}_\gamma\gamma-V$ iff $f(x)\ll V$ [by Definition2.6], there exist $U\subset, x\bar{\delta}_\chi\chi-U$ iff $x\ll U, f(U)\bar{\delta}_\gamma\gamma-V$ and $f(U)\ll V$ [by Definition2.6].

5.8 Proposition

Let $f:(\chi,\delta_\chi)\to(Y,\delta_Y)$ is onto δ_ω −continuous function, then $f^{-1}:(Y,\delta_Y)\to(\chi,\delta_\chi)$ is δ_ω −continuous function.

Proof

Let $y\in Y$ and $V\subset\chi, f^{-1}(y)\bar{\delta}_\chi\chi-V$ since f is onto, then there exist $x\in\chi$ such that $f^{-1}(y)=x$, $x\bar{\delta}_\chi\chi-V$. Since f is δ_ω −continuous function, then for all $x\in\chi, H\subset Y, f(x)\bar{\delta}_Y Y-H$, there exists $U\subset\chi$, $U=V, x\bar{\delta}_\chi\chi-U, x\bar{\delta}_\chi\chi-V\ f(U)\bar{\delta}_Y Y-H$.

5.8 Proposition

Let$f:(\chi,\delta_\chi)\to(Y,\delta_Y)$, then f is δ_ω −continuous function if f for all $x\in\chi$, and for all $V\subset Y, V\in\mathcal{F}(f(x))$, there exist $U\subset\chi$ such that $U\in\mathcal{F}(\{x\}), V\in\mathcal{F}(f(U))$.

Proof:

Let $f:(\chi,\delta_\chi)\to(Y,\delta_Y)$ is δ_ω −continuous function, then for all $x\in\chi$, and for all $V\subset Y, f(x)\bar{\delta}_Y Y-V$ that mean $f(x)\ll V$ [by Definition 2.6] imply $V\in\mathcal{F}(f(x))$ [by Remark 2.9], there exist $U\subset X$ such that $x\in X, x\bar{\delta}_\chi\chi-U, U\in\mathcal{F}(\{x\}), f(U)\bar{\delta}_Y Y-V, V\in\mathcal{F}(f(U))$[Remark 2.9]. Conversely, let for all $\in X$, $V\subset Y$, $V\in\mathcal{F}(f(x))$[by Remark 2.9], then $f(x)\bar{\delta}_Y Y-V$there exist $U\subset X$ such that $U\in\mathcal{F}(\{x\}), x\bar{\delta}_\chi\chi$-$U$, $V\in\mathcal{F}(f(U))$ hence $f(U)\bar{\delta}_Y Y-V$, then f is δ_ω −continuous function

5.9 Proposition

If $f:(\chi,\delta_\chi)\to(Y,\delta_Y)$ is δ_ω −continuous function from (χ,T_{δ_χ}) to proximity space (Y,T_{δ_Y}) then $f$is continuous with respect topology T_{δ_χ} and $T_{\delta_Y}, (T=T_\delta)$

Proof:

Let f is δ_ω −continuous function from proximity space X to proximity space Y then for all $x\in X, and$ for all $V\subset Y$, $f(x)\bar{\delta}_Y Y-V$,that mean $f(x)\ll V$ [by Definition 2.6] and V is T_{δ_Y} −open set [by proposition 3.3], there exist $U\subset X$, $x\bar{\delta}_\chi\chi-U$[by proposition 3.3]U *is* T_{δ_Y} −open set [by proposition 3.3],) that mean $x\ll U$ [by Definition 1.6] and $f(U)\bar{\delta}_Y Y-V$ and $f(U)\ll V, then\ f(U)\subset V$ [by theorem 2.7(2)] thus f is continuous function.

5.10 Remark

The Converse of [proposition 5.9] is not true since if $\chi=R, d(x,y)=|x-y|, \delta_\chi=\delta_d$, let take $A=N$, $B=\left\{\frac{(n+1)}{2n},\ n\in N\right\}$, $A\delta_\chi B\ if\ and\ only\ if\ d(A,B)=0$, where $d(A,B)=\inf\{d(x,y): x\in A, y\in B\}$ and $T_{\delta_\chi}=T_{\delta_Y}$and let $A=\bar{A}, B=\bar{B}, A\bar{\delta}_Y B\iff\bar{A}\cap\bar{B}=\emptyset$ and since $\delta_\chi<\delta_Y$, then f is not δ_ω −continuous function.

5.11 Remark

If $f:(\chi,\delta_{I\chi})\to(Y,\delta_Y)$, then f is not δ_ω −continuous function because, if f is any function from $(\chi,\delta_{I\chi})$ to any proximity space (Y,δ_Y) and for all $x\in\chi$, $V\subset Y$ such that $f(x)\ \bar{\delta}_Y Y-V$, $\delta_{I\chi}$ is indiscrete proximity space, then [by Example 2.2] for all $A,B\subset\chi, A\delta_\chi B$ if and only if $A\neq\emptyset, B\neq\emptyset$ and $\{x\}\neq\emptyset$ imply is near from any nonempty subset of X, there is not exist $U\subset X$ and $x\ \bar{\delta}_{IX}X-U$, finally f is not δ_ω −continuous function.

5.12 Proposition

I$f:(\chi,\delta_\chi)\to(Y,\delta_Y)$ is constant function from proximity space (χ,δ_χ) to proximity space (Y,δ_Y), then $f$is δ_ω −continuous function.

Proof:

Let f is constant function and for all $x\in X$, $f(x)=\alpha,\alpha\in Y$and let $V\subset Y$such that $f(x)=\alpha\ \bar{\delta}_Y Y-V$, then for all $U\subset\chi$ and since f is constant $f(U)=f(x)=\alpha$, then $f(U)\bar{\delta}_Y Y-V$, f is δ −continuous since for all $A\delta_\chi B\to f(A)\delta_Y f(B)$ and $\{x\}\delta_Y\{x\}$ for all $x\in X$ [by Definition 2.1 P4], To prove $x\bar{\delta}_\chi X-U$, since $f(U)\bar{\delta}_Y Y-V$, $f$is δ −continuous $f^{-1}(f(U))\ \bar{\delta}_Y\ f^{-1}(Y-V)$ [by

Proposition 3.5], $f^{-1}(f(x))\bar{\delta}_Y X - f^{-1}(V)$, $x\ \bar{\delta}_\chi \chi - f^{-1}(V)$, put $f^{-1}(V) = U$ that's imply $x\ \bar{\delta}_\chi \chi - U$, hence f δ_ω −continuous function.

5.13 Proposition

If $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$is δ_ω −continuous function,$A \subseteq X$, then $f_{/A}:(A,\delta_A) \to (Y,\delta_Y)$ is δ_ω −continuous function.

Proof

If $x \in A$ and $V \subset Y$ so $x \in X$ and because $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous function then for all $x \in X, V \subset Y, f(x)\bar{\delta}_Y Y - V$there exist $U \subset \chi$, $x\bar{\delta}_\chi \chi - U$, then $x \ll_\chi U$ hence, $x\bar{\delta}_A \chi - U$[by Theorem 3.9] and $\mathcal{F}_A(\{x\}) = \mathcal{F}_X(\{x\}) \cap A$ [by proposition 2.7],$A \cap U \in \mathcal{F}_A(\{x\}), \{x\} \ll_A A \cap U, \{x\}\ \bar{\delta}_A\ A - (A \cap U)$, since $A - (A \cap U) \subset \chi - U$, since$f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous and $A \cap U \subset U$,then$f(U))\bar{\delta}_Y Y - V$, hence $f(A \cap U))\bar{\delta}_Y Y - V$ which mean $f_{/A}:(A,\delta_A) \to (Y,\delta_Y)$ is δ_ω −continuous function.

5.14 Proposition

If $f:(X,\delta_X) \to (Y,\delta_Y)$ is $\delta -$ continuous such that δ_X, δ_Y are discrete relations, then f is δ_ω −continuous function.

Proof

Let $x \in \chi$ and $V \subset Y, f(x)\ \bar{\delta}_Y Y - V$, so because f is $\delta -$ continuous, then $x\ \bar{\delta}_\chi \chi - f^{-1}(V)$, let $f^{-1}(V) = U$, then $x\ \bar{\delta}_X \chi - U$. To prove $f(U)\ \bar{\delta}_Y Y - V$,if $f(U)\delta Y - V$, then $f(f^{-1}(V)\delta_Y Y - V$ and then $V\delta Y - V$ but $V \cap (Y - V) = \emptyset$ and this contradiction, then f is δ_ω −continuous function.

5.15 Proposition

Let $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous function and $\mathrm{K} \subset Y$, $(\mathrm{K},\delta_\mathrm{K})$is proximity subspace of proximity space$(Y,\delta_Y), then\ f:(\chi,\delta_\chi) \to (\mathrm{K},\delta_\mathrm{K})$ is δ_ω −continuous function.

Proof:

let $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous function., then for all $x \in \chi, V \subseteq Y, f(x)\bar{\delta}_Y Y - V$, since $(\mathrm{K},\delta_\mathrm{K})$ is proximity subspace of Y, then $V \subset \mathrm{K} \subset Y$, then $\mathrm{K} - V \subset Y - V$, also [by Theorem 3.8] $f(x)\bar{\delta}_\mathrm{K}\mathrm{K} - V$, and since f is $\delta\omega$ −continuous, then there exist $U \subset X, x\bar{\delta}_X X - U, f(U)\bar{\delta}_Y Y - V$ imply $f(U)\bar{\delta}_\mathrm{K}\mathrm{K} - V$ [by proposition 1.4(b)] hence $f:(\chi,\delta_\chi) \to (\mathrm{K},\delta_\mathrm{K})$ is δ_ω −continuous function.

4.16 Remark

Let $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous function and $(H,\delta_{/H})$ is proximity subspace of proximity space (χ,δ_χ) and $(K,\delta_{/K})$ is proximity subspace of (Y,δ_Y), then $f:(H,\delta_{/H}) \to (K,\delta_{/K})$ is δ_ω −continuous function since if $x \in H$ and let $V \subset K$, $f(x)\bar{\delta}_{/K}K - V, f(x) \ll_K K$.

To prove $f:(H,\delta_{/H}) \to (K,\delta_{/K})$ is δ_ω −continuous function. Since f is δ_ω −continuous, then there exist $U \subset \chi$ such that $x \ll_\chi U$ [by Definition 5.1] and since $H \subset \chi, H - U \subset \chi - U, x\ \bar{\delta}_H H - U$ and $f(U)\bar{\delta}_Y Y - V$ but $V \subset K \subset Y$, $K - V \subset Y - V$, then $f(U)\bar{\delta}_{/K}K - V$, hence $f:(H,\delta_{/H}) \to (K,\delta_{/K})$ is δ_ω −continuous function.

5.17 Proposition

Let $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$ is δ_ω −continuous function, then there exist two disjoint sets $C, D \subseteq Y$such that $C^c \ll f(\varphi)^c$.

and $D^c \ll V$, for all $\varphi \subset X$, $x\bar{\delta}_\chi \chi - \varphi$ and $V \subset Y$, $f(x)\bar{\delta}_Y Y - V$.

Proof

Since f is δ_ω −continuous then for all $x \in X$, and for all $V \subset Y$, $f(x)\bar{\delta}_Y Y - V$there exist $\varphi \subset X, x\bar{\delta}_\chi \chi - \varphi$,$f(\varphi)\bar{\delta}_Y Y - V$ and [by proposition 2.4(ii)] there exist two disjoint sets $C, D \subseteq Y$ [by Proposition 2.5(iii)], $f(\varphi)\bar{\delta}_Y Y - C, f(\varphi) \subset C, C^c \subset f(\varphi)^c$,hence $C^c \ll f(\varphi)^c$ and $Y - V\bar{\delta}_Y Y - D$, then $Y - D\bar{\delta}_Y Y - V$ imply $D^c \subset V, D^c \ll V$.

5.18 Proposition

Let $f:(\chi,\delta_\chi) \to (Y,\delta_Y)$is δ_ω −continuous, then for all $x \in X, V \subset Y$, then the following is satisfies:

1. $\overline{V^c} \subseteq \chi - f(\varphi)$ $[\overline{V^c} \subseteq (f(\varphi))^c$,
2. $V^c \subseteq (f(\varphi)^c)^\circ$ $[V$ is open],
3. $\overline{f(\varphi)}\ \bar{\delta}_Y Y - V$ for some $\varphi \subseteq \chi$ and for all $V \subseteq Y$,
4. $f(\varphi) \ll int(V)$ $[f(\varphi) \ll \overline{(V^c)^c}]$.

Proof:

1. Since $f$is δ_ω −continuous, then for all$x \in \chi, V \subseteq Y, f(x)\bar{\delta}_Y Y - V$ there exist $\varphi \subset \chi$ such that$x \in \chi$, $x\bar{\delta}_\chi \chi - \varphi$ and $f(\varphi)\bar{\delta}_Y Y - V$[by Proposition 3.4] $\overline{Y - V} \subset \chi - f(\varphi)$, then $\overline{V^c} \subset (f(\varphi))^c$.

2. By [Proposition 3.4] $\chi - V \subset int\ (\chi - f((\varphi))$, then $V^c \subseteq (f(\varphi)^c)^\circ$,
3. Since $f(\varphi) \ll V$[ByProposition 3.6(i)],$\overline{f(\varphi)} \ll V$ and $\overline{f(\varphi)}\ \bar{\delta}_Y Y - V$ [By Proposition 3.6(ii)].
4. Since $f(\varphi) \ll V, then\ f(\varphi) \ll int(V)$ and since [$int\ A = (\overline{A^c})^c$] then $[f(\varphi) \ll \overline{(V^c)^c}]$.

5.19 Proposition

Let δ_χ,δ_Y be two proximity relations on the set χ, $f:(\chi,\delta_X) \to (Y,\delta_Y), \chi = Y$ is identity function of a set χ, then f is δ_ω −continuous function if and only if $\delta_\chi > \delta_Y$.

Proof:

Let f is δ_ω −continuous, then for all $x \in \chi$, and for all $V \subset Y$, $f(x)\ \bar{\delta}_Y Y - V$ there exist $U \subset \chi$, $x\bar{\delta}_\chi \chi - U, x \in U$, then $f(x)\bar{\delta}_Y f(\chi - U) = f(\chi) - f(U)$ but $f(\chi) = Y$ [f is identity], let $f(U) = V$ hence$f(x)\bar{\delta}_Y Y - V$ for all $x \in \chi$, then $\delta_\chi > \delta_Y$,

Conversely, let $\delta_\chi > \delta_Y$ and for all $x \in \chi, V \subset Y$, $f(x)\bar{\delta}_Y Y - V$ but $f(x) = x, f^{-1}(Y) = \chi, x\bar{\delta}_\chi \chi - f^{-1}(V)$, now let $f^{-1}(V) = U \subset \chi$, hence there exist $U \subset \chi, x\bar{\delta}_\chi \chi - U, x \in U$ and $f(U) = \cup \{f(x), x \in U\}$, then f *is* δ_ω −continuous function.

5.20 Proposition

If $f: (\chi, T_{\delta_\chi}) \to (Y, T_{\delta_Y})$is onto δ_ω −continuous, then for every open set V in T_{δ_Y} there exist open set U in T_{δ_χ} such that $f(U) \subset V$.

Proof

Let f is δ_ω −continuous , $V \in T_{\delta_Y}$ since f is onto then for all $y \in V \subset Y, y\bar{\delta}_Y Y - V$ since f is onto there exist $x \in \chi$, such that $y = f(x)$ and since $V$is open, then $y = f(x)\ \bar{\delta}_Y Y - V$[by Proposition 3.4] and since f is δ_ω − continuous function, then there exist $U \subset \chi$ such that $x\bar{\delta}_\chi \chi - U$, $x \in U$ imply U is open [by Proposition 2.3] and $f(U)\bar{\delta}_Y Y - V$, hence $f(U) \subset V$.

5.21 Remake

If $f: (\chi, T_{\delta_\chi}) \to (Y, T_{\delta_Y})$ is open function, then not necessary f is δ_ω −continuous function as explained in that example.

5.22 Example

Let $f: (\chi, T_{\delta_\chi}) \to (Y, \delta_Y)$ such that f is identity function, $\chi = \{m, j, k\} = Y$, T_{δ_χ} is normal proximity topological space, $T_{\delta_\chi} = \{X, \emptyset, \{m\}, \{j, k\}\}$and δ_Y is discrete relation defined on Y, clearly $f$is open function but is not δ_ω −continuous since if $x = j, f(j) = j$, let V={j}, $f(j)\bar{\delta}_Y Y - \{j\} = \{m, k\}$since $\{j\} \cap \{m, k\} = \emptyset$ [by Example 1.2 (i)], there exist $U = \{j, k\} \subset X$, $j\bar{\delta}_\chi \chi - U = \chi - \{j, k\} = \{m\}$ but $f(\{j, k\})\delta Y - \{j\} = \{m, k\}$, then f is not δ_ω −continuous.

5.23 Proposition

Let $f: (\chi, \delta_\chi) \to (Y, \delta_Y)$ δ_ω −continuous, then the following statements are holds for all $x \in \chi, U, \mathrm{E} \subset \chi$and $V, H \subset Y$:

i. There exist nonempty set E subset of χsuch that $x \ll_\chi \chi - \mathrm{E} \ll_\chi \mathrm{U}$,
ii. There exist nonempty set Hsubset of Y such that, $f(U) \ll_Y Y - H \ll_Y V$.

Proof:

i. since f is δ_ω −continuous, then for all $x \in \chi, V \subset Y, f(x)\bar{\delta}_Y Y - V$, there exist $U \subset \chi, x\bar{\delta}_X \chi - U$, then there exist $\mathrm{E} \subset \chi$ [by Definition 2.1.P5] such that $\{x\}\bar{\delta}_\chi \mathrm{E}$, $x \ll_X \chi - \mathrm{E}$ [by Definition 2.6] and $\chi - \mathrm{E}\ \bar{\delta}_\chi\ \chi - \mathrm{U}$ imply $\chi - \mathrm{E} \ll \mathrm{U}$ [by Definition 2.6], then $x \ll_\chi \chi - \mathrm{E} \ll_X \mathrm{U}$ [by Definition 2.6].
ii. since f is δ_ω −continuous, then for all $x \in \chi, V \subset Y, f(x)\bar{\delta}_Y Y - V$, there exist $U \subset \chi, x\bar{\delta}_\chi \chi - U$, $f(U)\bar{\delta}_Y Y - V$, then there exist $\emptyset \neq H \subset Y$, $f(U)\bar{\delta}_Y H$ and $f(U) \ll_Y Y - H$ [by Definition 2.6] and $Y - H\bar{\delta}_Y Y - V, Y - H \ll_Y V$[by Definition 2.6] imply $f(U) \ll_Y Y - H \ll_Y V$.

6. CONCLUSION

δ_ω −continuous is a new type of continuity in the space of topological proximity. The lesson of continuity of each element of domain depending on definition of the neighborhood in the space of topological proximity.

This kind has got qualification of special condition and it is related to or connected with continuity in topological space and continuity within the topologicl proximity space.

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13. YKM Altalkany, L.A. Al Swidi, Focal Function in i −Topological Space via Proximity Space.

Abstract Arabic

نظراً لأهمية الاستمراريه كمفهوم رياضي ودورها الكبير في حل الكثير من المشاكل الرياضيه ولكون فضاء القرب من المصطلحات الحديثه تمكنا في هذا البحث من الاجابه على سؤال مهم جدا هو هل يمكن وضع ودراسة أنواع من الاستمراريه كالانواع الموجودة في الفضاء التبولوجي الاعتيادي؟ وقد تمكنا في هذه الورقه من إيجاد نوع من الاستمراريه أطلق عليه ($\delta_\omega - \text{continuous}$) واستطعنا الحصول على العديد من النتائج معتمدين بذلك على نظريات القرب وخواص فضاء القرب المختلفه والحالات الخاصه للتبولوجي المتولد بواسطة فضاء القرب.

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A Review of The Relationship Between B Cell Phenotype and Amino Acids in the Development of Chronic Hepatitis B Progression

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A B S T R A C T

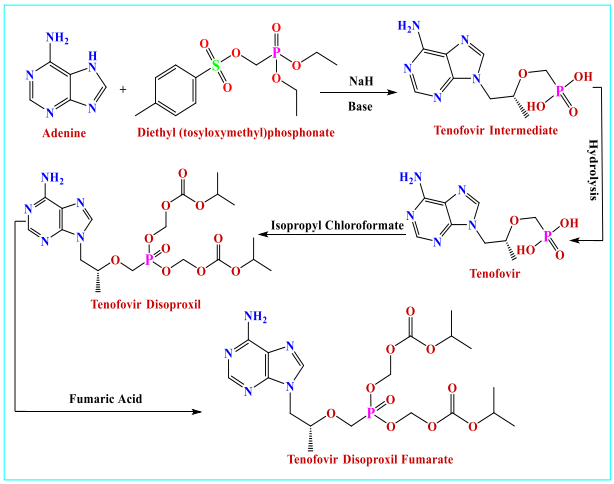
Chronic Hepatitis B (CHB) is still a major global health problem, even though there are vaccines and some antiviral treatments. The virus can stay in the liver for a long time because of its strong genetic material, which helps it escape current treatments. This study is a literature review that looks at how B cells—important immune cells—change during chronic Hepatitis B infection. It focuses on special types of B cells, like atypical memory B cells (AtM B cells) and regulatory B cells (Bregs), and how they may weaken the body's ability to fight the virus.
The review highlights gaps in current knowledge and suggests that future research should explore new ways to treat Hepatitis B by targeting these B cells. Better understanding of how B cells work in HBV infection may lead to more effective treatments and possibly a functional cure.

NOMENCLATURE	
ccDNA	closed circular DNA
CHB	Chronic Hepatitis B
TLR	Toll-like receptor
Bregs	regulatory B cells
CD	Cluster differentitation
NUC	Nucleos(t)ide analogue
NK	Natural killer cell
DLBCL	diffuse large B cell lymphoma
IL-10	interleukin-10
HCV	hepatitis C virus
AtM B cells	Atypical memory B cell

1. INTRODUCTION

One of the most serious threats to public health is HBV inspite of the availability of vaccines and antiviral medications [1-3). It is the cause of liver cancer, cirrhosis, and chronic viral hepatitis [4, 5]. Even after long-term antiviral therapy, the viral genome (the genetic material of the virus) remains in infected liver cells, meaning that the virus is not completely eliminated. When the viral genome integrates with the host genome (the genetic material of the liver cells), the stability of the cells' genome changes. This integration stops the virus from actively replicating, but it can affect the host cell, making it more difficult to eliminate the infection completely [6]. All viral gene products in HBV come from tightly closed circular DNA (cccDNA) present in the nucleus of infected liver cells. This DNA is a stable and persistent form of the viral genome, acting as a "mini-chromosome".This means that it carries all the information needed for viral replication and the production of viral proteins. cccDNA presents a

significant challenge in treatment because it remains in the nucleus of cells even after antiviral therapy, making it a continuous source of viral replication and protein production [7]. The persistence of this cccDNA is the basis for the ongoing chronic viral hepatitis [2]. Some studies have attempted to target the viral reverse transcriptase enzyme, but they have not been effective in eliminating it. As a result, researchers have explored therapeutic strategies through antiviral immune responses, aiming to eliminate or inactivate the DNA [8]. B cells have shown a role in controlling acute hepatitis B virus infection by producing antibodies against the viral surface antigen HBsAg, which is considered a therapeutic target for chronic infection [9]. However, they display signs of functional impairment and an overabundance of AtM B cells, which have high expression of inhibitory receptors such as PD-1. Consequently, this lowers B cells' ability to defend against the virus [10, 11]. Bregs have also been identified as important modulators of the immune response to HBV through Toll-like receptor (TLR) signaling [12, 13]. Although Bregs are involved in regulating immune responses and maintaining tolerance, their precise role in HBV infection remains unclear [14, 15]. The research suggests that HBcAg-specific B cells are more abundant and more capable of maturing into functional, antibody-producing cells than HBsAg-specific B cells in CHB patients, potentially playing a significant role in immune defense [16, 17]. HBcAg-specific B cells not only differ in their appearance and surface markers but also in their genetic activity, suggesting they play a unique and specialized role in the immune response against HBV compared to other memory B cells.This highlights their potential importance in HBV immunity. Additionally, elevated serum HBsAg levels are associated with increased immune inhibition and weakened HBV-specific CD4+ T cell responses, which can contribute to viral persistence and chronic infection [18]. These findings suggest that monitoring HBsAg levels may provide insights into the immune potential of HBV-infected patients and guide therapeutic interventions [19]. There are several drugs that are used for patients with viral hepatitis, including (Entecavir, Tenofovir, Interferon, Lamivudine, and Adefovir) [20, 21]. Each type has its own advantages, but the best of these drugs is Tenofovir disoproxil fumarate [TDF], which is considered safer for the kidneys and bones [22, 23]. It is prepared as follows: by reacting adenine with diethyl (tosyloxymethyl) phosphonate in the presence of a strong base such as sodium hydride (NaH). After removing the protecting groups (deprotection), the diethyl ester is converted to phosphonic acid via ester hydrolysis. Reaction with isopropyl chloroformate (Disoproxil Protection): Tenofovir reacts with isopropyl chloroformate to form tenofovir disoproxil, which improves bioabsorption. The final formulation is fumarate salt formation (the active pharmaceutical formula) [24-26]. As in Scheme [1]:



Scheme 1. Tenofovir Disoproxil Fumarate.

With an emphasis on the information gaps and prospective treatment approaches, this study attempts to investigate the role of B cell shape and function in chronic hepatitis B. This review looks at how B cells' phenotypic alterations affect the immune response to HBV. It aims to provide a thorough understanding of how these cells contribute to the infection's persistence and investigate new avenues for developing a functional cure.

2. RESEARCH METHODOLOGY

A nonsystematic narrative review was conducted to explore the role of B cell morphology and function in chronic hepatitis B [CHB]. The search was performed over a two-month period using specific keywords, including "Chronic Hepatitis B," "B Cell Morphology," "B Cell Function," "AtM B cells," and "regulatory B cells." Four reputable databases—Web of Science, PubMed, Google Scholar, and Scopus—were utilized to identify relevant literature. The initial search yielded numerous publications, which were then screened by reviewing titles and abstracts to select studies that specifically addressed the morphological and functional changes of B cells in CHB patients. Only studies that directly reported on B cell alterations in the context of chronic hepatitis B were included. To ensure the inclusion of relevant studies, the following selection criteria were applied:

- Inclusion criteria: Studies that focused on B cell morphology and function in CHB patients.
- Time frame: Studies published from 2015 to 2024 were reviewed to include the most recent findings.
- Study types: Both clinical studies and laboratory-based studies were considered,

excluding general literature reviews that did not present original data.

Full texts of the selected studies were carefully reviewed, and data were extracted and synthesized to identify consistent morphological and functional changes observed in CHB patients. These findings were then summarized and synthesized narratively. The potential for bias in study selection was considered, especially in relation to study outcomes and the inclusion of studies with specific findings.

3. DISCUSSION

3.1. Docking Molecular

The molecular docking study was conducted using the Biovia and PyRx software, using previously optimized structures and their optical analogues. The crystal structure of the protein [1qgt) was downloaded from the Protein Data Bank (PDB),27, 28 water molecules were removed, and polar hydrogen atoms were added using the PyRx computer program to demonstrate the binding strength. As **TABLE 1** shows the binding energy of the compound to the amino acids that make up the protein chain (1qgt). It also shows the upper and lower limits of the root mean square deviation (RMSD). We then used Biovia Discovery Studio to create 2D and 3D images of the amino acids bound to the drug, showing the type of bonds for each ligand. **Figure 1** illustrates the binding of the compound to the amino acids that make up the protein chain (1qgt). Among these acids are the amino acid threonine (THR), with sequence numbers (B:33) and (C:128), the amino acid serine (SER), with sequence numbers (C:121), and the amino acid leucine (LEU), with sequence numbers B:140. These acids are linked by hydrogen bonds and are colored dark green. Leucine (LEU), with sequence numbers (B:30), and isoleucine (ILE), with sequence numbers (B:105), are linked by alkyl bonds and appear light pink. The amino acids proline (PRO) with the sequence number (C:129), tyrosine (TYR) with the sequence number (C:132), and phenylalanine (PHE) with the sequence number (B:23) are linked by a (Pi-Alkyl) bond in light pink, while the rest of the amino acids appear in light green when they are molecularly bound to the protein (1qgt) by van der Waals forces. (SER- THR- VAL- PRO- TRP- PHE- ILE- TYR- ARG). **Figure 2** represents the area where hydrogen bonds were formed when the compound bonded with the amino acids of the protein (1qgt).

TABLE 1. The bonding energy of composite with amino acid

Ligand	Binding Affinity (kcal/mol)	Mode	RMSD lower bound	RMSD upper bound
1qgt_Fragment_uff_E=817.23	-8.4	0	0.0	0.0
1qgt_Fragment_uff_E=817.23	-7.6	1	1.487	2.126
1qgt_Fragment_uff_E=817.23	-7.2	2	1.97	3.364
1qgt_Fragment_uff_E=817.23	-7.0	3	4.009	8.145
1qgt_Fragment_uff_E=817.23	-6.9	4	4.361	8.411
1qgt_Fragment_uff_E=817.23	-6.5	5	16.797	19.985
1qgt_Fragment_uff_E=817.23	-6.5	6	4.09	8.206
1qgt_Fragment_uff_E=817.23	-6.4	7	16.028	19.194
1qgt_Fragment_uff_E=817.23	-6.4	8	17.401	20.613

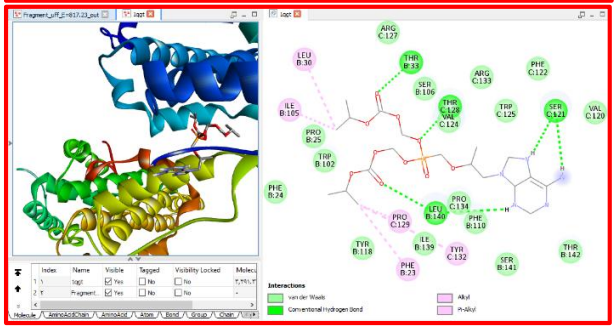


Figure 1. Shows the association of the compound with the amino acids in the protein (1qgt).

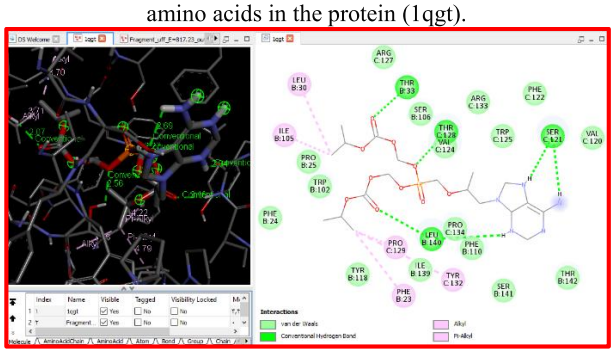


Figure 2. shows the type and length of bonds between the drug and the amino acids of the protein (1qgt).

Hydrogen bonds play an important role in the effectiveness of tenofovir as an inhibitor of hepatitis B cells. Their influence on biological activity can be summarized as follows: The presence of hydrogen bonds in tenofovir enhances its inhibitory activity against hepatitis B cells by improving binding to target enzymes,29 increasing cell permeability, and inducing cell death. Therefore, designing a drug (tenofovir) with optimal hydrogen bonding properties may lead to the development of more effective and safer anti-hepatitis therapies.30, 31.

3.2. B Cell Alterations a in Chronic Hepatitis B

3.2.1.Disruption of the CD39/CD73/Adenosine Pathway

In patients with chronic hepatitis B (CHB), B cells undergo important changes, especially in the CD39/CD73/adenosine pathway, which plays a major role in regulating immune activation. Studies have shown that patients with high levels of HBV-DNA, positive HBeAg, and elevated HBsAg tend to have reduced expression of CD39 and CD73 on circulating B

cells (27). This reduction disrupts the adenosine-mediated immune regulation, leading to excessive B cell activation and contributing to disease progression. Targeting this pathway may help restore immune balance and improve treatment outcomes.

3.2.2. Impaired Antibody Production and Functional Deficiencies

B cells in CHB also show reduced antibody production and signs of general dysfunction. This is accompanied by altered expression of important surface markers, such as a decrease in activation markers [28]. These defects limit the body's ability to fight the virus effectively. Understanding and correcting these changes could support the development of treatments that enhance B cell responses.

3.2.3 Modulation by Nucleos(t)ide Analogue Therapy

Treatment with nucleos(t)ide analogues (NUCs) not only reduces viral load but also affects B cell behavior. NUC therapy has been associated with a shift of B cells from an activated to a more resting phenotype. This change correlates with improved HBV-specific T cell responses and better immune function overall [29]. These findings suggest that NUCs may indirectly improve B cell function and contribute to viral control.

3.2.4. Cytokine Signaling and Immune Cell Interactions

B cells in CHB interact with various other immune cells and are influenced by cytokine signaling. For example, although PD-1 expression is increased on CD8+ CXCR5+ T cells, this is associated with improved B cell support and HBV-specific cytokine production [30). Additionally, cytokines such as IL-12 can influence T helper (Th) cell responses, which in turn impact B cell activity. NK cells also play a regulatory role: in CHB patients, they tend to show an inflammatory profile, but this is improved with NUC therapy, enhancing their regulatory interactions with both B and T cells [31]. These complex networks are illustrated in Figure 1.

The phenotypic and functional changes observed in B cells during CHB reflect a broader pattern of immune system disruption. Understanding how these changes affect disease progression and interact with other components of the immune system is essential for developing more effective treatments. One current limitation is the lack of direct comparison across different studies, which makes it difficult to assess the strength and consistency of the evidence. Future research should focus on uncovering the molecular mechanisms behind B cell alterations and comparing study outcomes to strengthen the scientific understanding of B cell involvement in CHB.

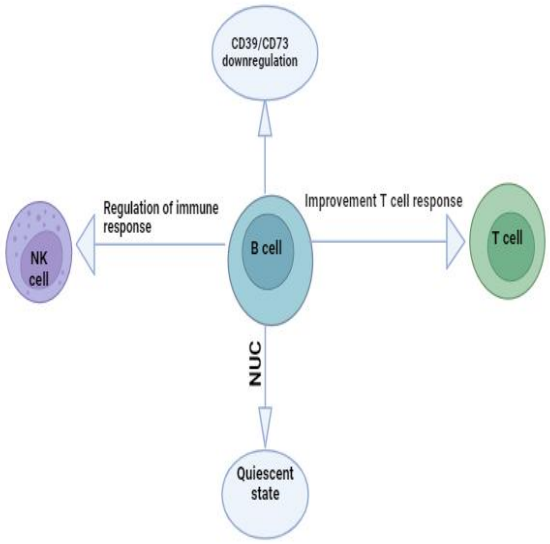


Figure 3. The diagram illustrates the regulatory role of B cells in modulating immune responses. It highlights the interactions between B cells, NK cells, and T cells, including CD39/CD73 downregulation, enhancement of T cell responses, and the induction of a quiescent state, potentially influenced by nucleos(t)ide analogues (NUC).

3.3. Physiological Alterations in B Cells Throughout CHB Stages

CHB infection is characterized by progressive changes in B cell functionality that impact the disease's progression and management. This discussion synthesizes findings from recent studies to elucidate these changes across different stages of CHB.

- **Early Stages and Acute Infection:**

In the initial stages of HBV infection, B cells are pivotal in the immunological response, engaging in antibody production and antigen presentation. However, chronic HBV infection often disrupts these functions. Early during chronic infection, B cells exhibit phenotypic and functional abnormalities, such as impaired production of HBsAg-specific antibodies and changes in cell surface markers [32].

- **Progression and Chronic Infection:**

As the disease progresses, the disruption in B cell functions becomes more pronounced. The expression of key molecules such as CD39 and CD73, which are crucial for modulating immune responses via the adenosine pathway, is often reduced in B cells from patients with high HBV DNA levels and active hepatic inflammation (33). This reduction in CD39/CD73 expression contributes to enhanced B cell activation and disease progression. Furthermore, there is an accumulation of AtM B cells and Bregs, which are associated with a compromised immune response.

These cells contribute to immune evasion and persistent viral replication by impairing effective B cell responses against HBV.

- **The Influence of Nucleos(t)ide Analog Therapy:**

Nucleos(t)ide analogue (NUC) therapy has a significant impact on B cell functionality. Treatment with NUCs can shift B cells from an activated state to a more quiescent phenotype, which is associated with improved HBV-specific T cell responses and overall immune function [34]. This modulation of B cell activity by NUCs highlights the potential for therapeutic strategies that target B cell dysfunction to enhance antiviral responses and improve clinical outcomes.

- **Advanced Stages and Complications:**

In advanced stages of chronic hepatitis B, particularly when associated with conditions like diffuse large B cell lymphoma (DLBCL), B cells undergo significant alterations. The presence of HBV can exacerbate B cell dysfunction, contributing to poor treatment outcomes and an increased risk of lymphoma [35]. HBV-associated DLBCL presents unique clinical features and necessitates careful monitoring for HBV reactivation during treatment.

- **Future Directions and Therapeutic Opportunities:**

Recognizing the specific processes underlying B cell malfunction in chronic HBV infection is crucial for developing targeted therapies. Innovative strategies are needed to address the diverse roles of B cells in HBV pathogenesis, including novel immune therapies aimed at restoring B cell function and improving antiviral responses (36). B cell dysfunction throughout the stages of CHB is marked by distinct phenotypic and functional changes that influence disease progression and therapeutic responses. Addressing these alterations through targeted treatments could improve the administration of chronic HBV infection and enhance patient outcome.

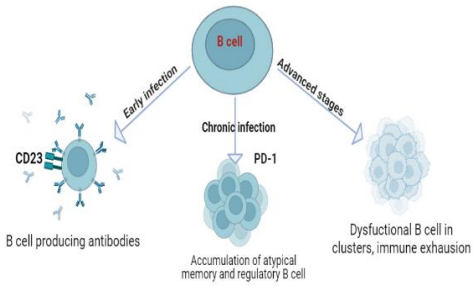


Figure 4. The figure illustrates the progression of B cell involvement during hepatitis B virus (HBV) infection across different stages of the disease. In the early infection phase, B cells engage in antibody production and antigen presentation as part of the immune response. As the infection becomes chronic, there is an accumulation of AtM B cells and Bregs cells, which contribute to immune evasion and persistent viral replication. In the advanced stages of infection, further B cell dysfunction occurs, often leading to complications and an exacerbated disease state.

3.4. Impact of Regulatory B Cells on Immune Dysfunction and Vaccine Efficacy in Chronic Hepatitis B

3.4.1. Role of Regulatory B Cells (Bregs) in Weakening Immune Response

In chronic hepatitis B (CHB), the immune system becomes unbalanced due to complex interactions between different immune cells. One important change is the increase of regulatory B cells (Bregs), especially those that produce the anti-inflammatory molecule IL-10. These Bregs can suppress the immune response by reducing the activity of HBV-specific T helper cells, which are important for fighting the virus. This weakens the body's ability to clear the infection and makes it harder to develop effective vaccines [37].

3.4.2. Bregs and Poor Vaccine Response

High levels of a specific type of Breg (CD24^hi^CD38^hi^) have been found in people who do not respond well to the hepatitis B vaccine. These people often have lower levels of protective antibodies (anti-HBs). This suggests that Bregs might be one reason why some individuals fail to develop immunity after vaccination [38].

3.4.3. Comparing HBV and HCV Infections

In hepatitis C (HCV), Bregs are also increased and linked to higher viral load and liver damage. However, unlike HBV, HCV does not cause a major increase in Tregs or Bregs. Instead, it may lead to more IL-10 production in B cells. This shows that Bregs work differently in different viral infections [39].

3.4.4. Functional Impairment of B Cells in CHB

Even though some types of B cells are more common in CHB, they often don't work properly. Studies show that these B cells have a reduced ability to make antibodies against HBV. This poor function helps the virus stay in the body and makes it harder to find a lasting cure [40]

3.5. Atypical Memory B Cells in Chronic Hepatitis B: Impaired Function and Contribution to Disease Pathogenesis

3.5.1. What Are Atypical Memory B Cells?

Atypical memory B cells (AtM B cells) are a special type of B cell that appear in many chronic infections, including chronic hepatitis B (CHB). They are different from normal memory B cells. AtM B cells have high levels of inhibitory markers like PD-1 and FCRL5 and low levels of markers such as CD21 and CD27 (41, 42). These changes show that the cells are

not working normally and may be constantly exposed to the virus (43, 44).

3.5.2. Dysfunction in CHB

In people with CHB, AtM B cells do not function well. They have weak B cell receptor (BCR) signaling and produce fewer antibodies. They also respond poorly to cytokines, which are signals that help immune cells communicate [45, 46]. Even when the virus seems under control, these cells can still be found, suggesting they may help keep inflammation going and contribute to long-term disease [47, 48).

AtM B cells may also respond to other viruses or vaccines. This means they are involved in more than just hepatitis B and can influence the overall immune response. Their behavior can affect how the body reacts to vaccines and other infections [49, 50]. As shown in Table 1, AtM B cells and regulatory B cells (Bregs) have different roles and features in chronic HBV infection. AtM B cells are an important part of the immune system in CHB. Their unique features may influence how the disease develops and how effective treatment will be.

TABLE 2. Comparison of AtM B cells and Regulatory B Cells (Bregs) in CHB infection

Feature	AtM B cells	Bregs
Phenotypic Characteristics	High expression of PD-1, low expression of CD21 [51-53].	Express CD19, CD24, and CD38, high secretion of anti-inflammatory cytokines like IL-10 [54].
Functional Role	Impaired ability to produce effective antibodies, dysfunction in response to HBV [55, 56].	Regulate immune responses by secreting IL-10, reduce the activity of other immune cells [37].
Impact on Disease Progression	Weak immune response, contributing to the persistence of the infection [57].	Help maintain immune balance but may hinder effective immune responses against the virus [58].
Response to Treatment	Less responsive to immunotherapies due to high expression of inhibitory receptors	May respond to immunotherapies, but could dampen inflammation without clearing the virus

appears to be a key factor in the persistence of chronic HBV infection. The regulatory functions of B cells, especially via IL-10 production and interactions with T helper cells, play a significant role in the immune evasion strategies utilized by HBV. These insights into the immune regulation of chronic HBV suggest potential avenues for therapeutic intervention, particularly by targeting Bregs to modulate the immune response and enhance antiviral efficacy. Further research into the exact mechanisms of B cell dysregulation in chronic HBV is needed to fully understand their role in disease progression and to identify strategies for restoring normal immune function.

4. CONCIUSION

In conclusion, the pathogenesis of Chronic Hepatitis B (CHB) is intricately linked to the dysregulation of B cell function, with both atypical memory B cells (AtM B cells) and regulatory B cells (Bregs) playing pivotal roles in sustaining viral persistence and immune dysfunction. While current antiviral therapies have made strides in controlling the virus, they fall short in addressing the underlying immune dysregulation, particularly the dysfunctional B cell response. This underscores the need for novel therapeutic approaches aimed at restoring B cell functionality, focusing on inhibiting the immune-suppressive roles of AtM B cells and Bregs. Future research should focus on better understanding the molecular mechanisms driving these B cell abnormalities and their interactions within the broader immune network. Such efforts will be crucial in developing strategies that not only target the virus more effectively but also promote immune system restoration, paving the way for a more durable and functional cure for CHB.

5. Recommendations for Future Research:

1. Clinical Trials for B Cell–Targeted Therapies: Future studies should test therapies that target B cell dysfunction in CHB. Clinical trials could explore whether modifying the activity of AtM B cells or Bregs can help restore immune balance and control HBV replication.

2. Study B Cell Function Over Time: Long-term (longitudinal) studies should follow patients from acute to chronic HBV infection. This could identify when B cell dysfunction begins and the best time for intervention.

3. Focus on PD-1 and FCRL5 Pathways: More research is needed on the molecular pathways that cause the build-up of AtM B cells. Studying PD-1 and FCRL5 signaling might help in designing drugs that fix or reverse this dysfunction.

4. Improve Vaccine Responses in CHB Patients: Since Bregs may suppress vaccine responses, studies should explore how reducing Breg activity could make the HBV vaccine more effective—especially for people with chronic infection.

Therapeutic Implications

- **Targeting AtM B Cells:** Blocking inhibitory receptors such as PD-1 or FCRL5 may help reactivate functional antibody responses and improve viral clearance.

- **Using TLR Agonists:** Stimulating Toll-like receptors (TLRs) might help overcome immune exhaustion and boost antiviral immunity.
- **Modulating Bregs:** Therapies that reduce IL-10–producing Bregs could improve both natural immune responses and vaccine effectiveness in CHB patients.

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Arabic Abstract

يُعد التهاب الكبد المزمن (CHB) من التحديات الصحية العالمية الكبيرة، على الرغم من توافر اللقاحات والعلاجات المضادة للفيروسات. تشير البيانات المتعلقة بعوامل الإصابة ومعدلات الوفيات إلى أن التهاب الكبد الفيروسي يشكل العامل الرئيس في تفشي هذا المرض. وتُعزى قدرة الفيروس على البقاء والتطور إلى حالة مزمنة إلى قدرته على الاستمرار في البقاء نشطًا أو شبه نشط داخل خلايا الكبد، نتيجة لمقاومته للمادة الوراثية التي تمكنه من التهرب من العلاجات المتوفرة حاليًا. في ظل هذه التحديات، بدأت الأبحاث في البحث عن طرق علاجية بديلة تستهدف الفيروس من داخل جسم العائل. لقد تم التعرف على دور خلايا "ب" في مكافحة العدوى من خلال توليد الأجسام المضادة ضدHBcAg ، حيث يُعتبر التخلص من هذه الأجسام المضادة مؤشراً على فعالية العلاج. ومع ذلك، أظهرت الدراسات وجود خلايا الذاكرة غير النمطية (AtM B cells) التي تحمل مستقبلات مثبطة مثلPD-1 ، وهو ما يساهم في خلل في وظيفة جهاز المناعة، مما يقلل من فعالية الدفاعات المناعية ضد الفيروس ويستمر في الحفاظ على وجوده داخل الجسم. بالإضافة إلى ذلك، على الرغم من أن التأثير الدقيق للخلايا التنظيمية ب (Bregs) على فيروس التهاب الكبد B لا يزال غير واضح بشكل كامل، إلا أن دورها في تعديل الاستجابات المناعية من خلال إشارات مستقبلات التدفق الشبيه بالنوكليوتيد (TLR) بدأ يلقى اهتمامًا متزايدًا.

تهدف هذه الدراسة إلى استكشاف التحولات الفينوتيبية والوظيفية التي تطرأ على خلايا "ب" في سياق التهاب الكبد المزمنB ، مع تسليط الضوء على الفجوات المعرفية في هذا المجال واقتراح استراتيجيات علاجية مستقبلية. إلى جانب تعزيز الفهم حول ديناميكيات خلايا "ب" في العدوى بفيروس التهاب الكبدB ، تسعى هذه الدراسة إلى استكشاف أساليب مبتكرة للوصول إلى علاج وظيفي من خلال فحص التغيرات الفينوتيبية وتأثيراتها على تقدم المرض وفعالية العلاج.

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Environmental Stress Response and Toxin-Antitoxin Systems Influence bacterial Persister Formation

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P A P E R I N F O

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Abstract

Bacterial respond to extreme environment stress let them to survive and grow. One bacterial strategy to survive is persister state. Persistence enables a subset of small cells population to become a dormant, non-replicative state and avoid stress. Bacterial persister has been linked to a number of biological processes. The toxin-antitoxin system(TA) is one of the most significant persistence mechanisms in bacteria. Thus, it has been determined that bacterial toxin–antitoxin complexes are a suitable target for treatment. Through two gene, protein is encoded. toxin that targets a vital cellular function besides an antitoxin that suppresses the activation of the toxin. Toxin expression can have a Bactericide effect; they could be regarded as "intracellular molecular bombs" that may cause their host cells to be eliminated. Therefore, current review discusses the functions of TA modules in development persister cell and the effects of environmental stress on persister cells.

1. INTRODUCTION

More effective antimicrobial medicines. For microorganisms to survive and compete in harsh conditions, Metabolic changes are necessary, for example, when antibiotics are present or the host is sick. TA Systems that contain a toxin that has the ability to modify metabolism and a nearby antitoxin that counteracts it are important components of the bacterial stress defense. [1] A toxin section and its corresponding antitoxin section make up the ubiquitous gene of toxin-antitoxin modules, which are loci found in bacteria. Antitoxin neutralizes the toxin's toxicity under normal physiological conditions. A large number of the toxins are proteinaceous and interfere with DNA replication or translation. However, antitoxins immediate interaction, mostly neutralizes its cognate toxin., but also aids in the organization of the TA module with the assistance of other signaling elements. TAs are several groups the antitoxin is categorized based on its molecular bases and how it interacts with its cognate toxin [2]. These "two" gene loci are arranged in operons, and the two (antitoxin and toxin–antitoxin complexes) closely control their expression [1]. Persistence is condition that arises when tiny subpopulations of bacterial cells momentarily exhibit a recognizable, also not inherited and stress-resistant phenotype [3]. Physiological programs conferring increasing fitness, also, are one of the essential properties of a few metabolic states known as persister that may rise as a result of toxin activity [4]. One method of preventing phage infection is Persistence, bacteriostatic efficacy inhibiting the making of mature visions during bacterial cloning [5]. However, absent plasmids TA-encoding, the accumulation of stable toxins causes the intracellular level of antitoxins to rapidly drop, which ultimately results in significant metabolic restriction [6]. Because TAs lessen the competitiveness of cells that did not inherit TA-encoded plasmids, they generate a significant selective pressure for their maintenance [7]. The current review discusses the effects of environmental stress on persister cells and the functions of TA modules in formation of persisted cell.

2. BACTERIAL PERSISTENCE

2.1 Bacterial persistence Ancient History

Staphylococcus aureus was found to exhibit persister cells. First, when discovered that penicillin could break down ninety-nine present of the target bacteria by Hobby and colleagues (1942). "One in a million" according to Bigger that *Staphlococcus aureus* cells are still alive during exposure to penicillin. Two years after this discovery, he called these cells persisters

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[8]. Additionally, the subpopulation of bacterial cells survived while the member of alive bacteria was regrown and subjected to antibiotics [8]. Before making this discovery, researchers believed that the populations of bacteria in planktonic cultures were uniform [9]. Bacterial phenotypes were recognized to be different within the same population because of the development and accessibility of instruments to study bacterial cell physiology [10]. Interestingly, a different bacterial phenotype known as "viable but non-culturable" (VBNC) was found by Xu and associates in 1982. Where the total number of cells remined unchanged in direct "fluorescent microscopic analysis", that is a subpopulation stayed viable but could not grow on the offered medium, in the (culture-based method), which including plate counting and most probable number determination. Escherichia coli was exposed to stressful situations (5–25% NaC1) during 2 weeks showed a rapid decrease in the total bacterial count when E. coli was exposed to stressful conditions (5–25% NaC1) for two weeks [11]. The recognizing of living cells that might be cultivated also retrieved under normal growing conditions. This served as foundation for experimental microbiology prior to this discovery, which was in line with ideas put forth by Robert Koch more than 150 years earlier [11].

2.2. VBNC Cells and Persister Cells

Persister & VBNC are cells found in a dormant condition, where a non-growing condition of hibernation the cells have been encouraged to enter in. The extent to which cells are led to enter the persisted as well as VBNC condition is thought to be largely determined by the length and severity of the relevant stress exposure [12]. Furthermore, three distinctive phases: initiation, resting, and revival have been classified to bacterial dormancy by Lennon & Jones (2011)

2.2.1 Initiation phase

Changes in environmental state the bacteria enter a persisted stage, for example temperature [13], osmotic pressure, oxygen concentration, pH level outside the optimal growth state. Nutrient starvation can be one of the factors that causes microorganisms to go into dormancy, primarily in the natural environment, which is an entry into the initiation phase. [14]

2.2.2 Resting phase

Microorganisms go through a rest period after commencement. During this phase, cells react to adverse circumstances by changing their metabolic processes and, on occasion, by changing their phenotype or structure, cell size decreases, these phenotypic changes include changes to the 18 concentrations of proteins, lipids, fatty acids, and nucleic acids; modifications to general stoichiometry; and rises in the quantities of survival-related storage compounds. Signoretto and associates (2002) monitored the *E. coli* during stress exposure entry into a persister state, there has been evidence that persister cells contain less ribosomes than non-persisted cells [15].

2.2.3 Revival phase

Bacterial strains differ in how long the resting stage lasts, ranging from a few days to years. However, as soon as favorable conditions and reduced stress allow for ongoing growth, cells invariably enter the revival stage. The first recorded instance of revival was when VBNC Salmonella cells were reported to have returned in 1984 by Roszak and associates, when bacterial cells emerge from hibernation and regain the capacity to establish colonies on nutritive media, they undergo intricate cellular transformations. Restoring metabolic competence, lowering oxidative stress, and controlling toxin-antitoxin ratios are the goals of these modifications [12].

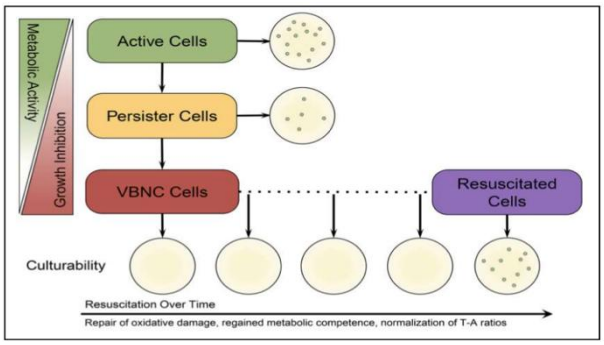

Figure 1. The VBNC cell concept and the encouragement of persister [12].

3. CLASSIFICATION of TOXIN ANTITOXIN SYSTEM

The TAs can be classified according to antitoxins interact with toxins or how antitoxins inhibit toxins [16]. The toxins are short RNA in type VIII TA modules, while proteins within type I to type VII modules [17]. In the case of type, I, type III and type VIII TA modules, antitoxins are small noncoding RNAs while in type II, type IV, type V, type VI and type VII TA modules are small proteins, **Table 1** [18].

4. FORMING PERSISTER CELL and THE ROLES of TA MODULES

Antibiotic-related research revealed that a tiny percentage of cells remained latent and exhibited a mechanism distinct from conventional antibiotic resistance [19]. "Persistence" refers to the small, genetically homogeneous cells' capacity to withstand stress by briefly becoming a dormant state. The primary traits of persistence are decreased cell growth and metabolic activity [20]. This "persister cells" consider phenotypic variants but not Mutants in genes [21]. The important roles that TA modules play in persister cell development have been unequivocally established. [22]. remains unknown TA systems are crucial for the persistence of bacteria. Other investigations confirm the importance of "Toxin Antitoxin systems" for persistence of bacteria [23]. in "dinJ/yafQ" Toxin Antioxin module has been implicated in cephalosporin and aminoglycoside antibiotic tolerance in another investigation [24]. According to a study on uropathogenic strains of E. coli, there is more evidence linking "type II" TA modules to the persistence of bacteria [25]. Before being used as antimicrobial targets, it is critical to ascertain if TA activation affects persister production because the overall mechanism of persistence is yet unknown. It has been demonstrated that persistence in E. coli is mediated by TA systems other than the "type II" TA modules. Although employing TAs as antibacterial targets may result in persistence, these systems can be also used in anti persister tactics. [26]. "Conlon and colleagues" [27] think about how a substance that can target dormant cells can destroy the persister cell. Protease "ClpP" is activated by acyldepsipeptide antibiotics (ADEP4) [28] then demonstrated that by damaging more than four hundred protein, that force the cells to (self-digest), thus becomes totally non-nominated and kills persisters [29]. Bacterial genomes have been shown to include over 90 Type (II) Toxin Antitoxin modules. In addition, it had been demonstrated that these modules are crucial in regulating persisted cell development [2]. TA modules are thought to play a role in persister cell production, but the exact mechanisms are still unknown. [30] Furthermore, HipA-hipB, the persisted phenomenon, and the first TA module are among the most well-known type-II TA modules. [31] It had been demonstrated that a rise in persistence in Escheria coli "K-12" is caused by the hipA and hipB genes. Both genes are found in the hip operon; a very stable toxin are encoded by the "hipA" gene, while the unstable antitoxin encoded by "hipB" gene. HipA forms the stable, non-toxic complex HipBA by forming a strong bond with hipB. However, when they attach to the DNAThrough an indirect method, HipB and maybe HipA can inhibit the hip operon's transcriptional activity. [32], colleagues & Rotem discovered, when the "hipB" (antitoxin gene) was cancelled in the Escherichia coli, the cells entered the VBNC condition, which stops the cells from forming colonies on medium, as a result of "hipA" overexpression [33]. HipA's toxicity and its direct and indirect regulatory effects on the expression of other proteins have been connected to its function in persister cell development. In 2018, Goormaghtigh & colleagues investigated how the presence of ampicillin and ofloxacin affected the generation of persisters after deleting ten Type-II TA modules. in comparison to the "wild-type" strain, they discovered that the deletion of 10 TA modules had no effect on the development of persister cells, indicating that Toxin A ntitoxin modules donot promote formation persister cell [34]. In summary, TA modules may contribute to persister cell formation; however, a thorough understanding is needed because it is difficult to analyze the connections between persistence and TA modules because of the complexity and diversity of TA modules as well as the ways in which

TABLE 1. Biological characteristics of types I to VIII toxin-antitoxin (TA) systems

TA types	Toxin	Antitoxin	Examples	Mode of action of antitoxin
I	Protein	RNA	*hok*/*sok*; SymE/SymR	Antitoxin binds to toxin mRNAs, thereby inhibiting toxin expression
II	Protein	Protein	*ccdA*/*ccdB*; *higB*/*higA*	Antitoxin and toxin form a protein–protein complex to neutralize its toxicity
III	Protein	RNA	*toxI*/ToxN; *antiQ*/AbiQ	Antitoxin binds directly to toxin, thereby inhibiting toxicity
IV	Protein	Protein	CbeA/CbtA; AbiEi/AbiEii	Antitoxin binds to the targets of toxin, thereby protecting such targets
V	Protein	Protein	GhoT/GhoS	Antitoxin is an RNase that specifically cleaves toxin mRNA
VI	Protein	Protein	SocA/SocB	Antitoxin acts as a proteolytic adaptor that promotes toxin degradation
VII	Protein	Protein	Hha/TomB; TglT/TakA	Antitoxin is an enzyme that neutralizes toxin via post-translational modification
VIII	RNA	RNA	SdsR/RyeA; CreT/CreA	RyeA and SdsR form a duplex that can be degraded; CreA forms a complex with Cascade and guide it to repress CreT transcription

they are expressed in bacteria. There is a knowledge vacuum about the potential effects of the various types of TA systems on bacterial persistence because most research has focused on Type-II TA.

5. EFFECT of ENVIRONMENTAL STRESS on PERSISTER CELL

5.1 Thermal Impact

Increase Antitoxin Degradation to TA Transcription. Several conditions that promote TA transcription heat -shock donot halt "protein synthesis", in contrast to chlor and SHX medications. The stress accelerating the breakdown of antitoxins must be the cause of the observed transcriptional activation in these circumstances. They investigated antitoxin stability after a transition from 30C to 45C using pulse chase analysis in order to test this theory. In fact, in line with earlier research, the heat -shock markedly accelerated the amount of "YefM" breakdown. [35]. Thus, heat shock does not inhibit translation, or enhanced breakdown of "YefM" & the ensuing alleviation of autorepression stimulate antitoxin transcription, which in turn should increase the quantities of antitoxin proteins.

5.2 Osmosis

One of fundamental part of cell functions is "Osmosis"; osmotic pressure influences the rate of cell growth, turgor and transport phenomena across the cell & its surroundings [36]**.** Study the effect of osmolytes & pH in *E. coli* strain "MG1655" which induce persistence state. The "LB broth" was used (no NaCl) in cell cultures to avoid any further osmotic effects that may occur. A study has demonstrated that the absence of "NaCl (1%)" from the standard LB broth has no significant effect on cell viability, cell growing average.

5.3 Alkaline and acidity

According to [37], they use a plasmid- encoded "fluorescent pH sensor", microfluidics, and time-lapse imaging to assess the intracellular pH dynamics of susceptible Escherichia coli, VBNC, and persister cells after being treated with ampicillin. They discovered that low degree of intracellular "pH" in persisters cell compared to cells of susceptible &VBNC. even before giving antibiotics. Tryptophanase activity, which is encoded by tnaA, is linked to the reported changes in pH control in persister Escherichia. coli cells, according to later research into the molecular mechanisms behind these variations. In fact, we found that the intracellular pH did not differ between susceptible, "VBNC", persister *E.coli* cells on DtnaA strain. In "DtnaA" strain lowered important "pH" homeostasis ways in addition to tryptophan metabolism, according to whole-genome transcriptome study. These pathways included the reaction to various carboxylic acid catabolism processes, oxidation reduction**,** and pH, in contrast to expression levels in the parental strain.

6.CONCLUSION

There is more than evidence that refer to contribution of the environmental stress and TA system in formation of persister cell. As a result, the bacterial cell will experience a transitory phenotypic transformation that allows surviving via a persistence occurrence in the presence of stress that led to limited availability and accessibility to the 'cellular target'.

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Arabic Abstract

تستجيب البكتيريا للإجهادات البيئية المختلفة مما يساعدها على البقاء والنمو. إحدى الاستراتيجيات التي تعتمدها البكتيريا للبقاء هي حالة "المثابرة". هذه المثابرة تمكّن مجموعة من خلايا البكتيريا الصغيرة من الدخول في حالة خاملة غير تكاثرية مما يساعدها على تجنب الإجهاد. تم ربط البكتيريا المثابرة بعدد من العمليات البيولوجية. ويعد نظام السم-المضاد أحد أهم آليات المثابرة في البكتيريا. ولذلك، تم تحديد أن انظمة السم-المضاد البكتيرية تعد هدفًا مناسبًا للعلاج. يحتوي النظام على جينين يشفران بروتينا، السم الذي يستهدف وظيفة خلوية أساسية، والمضاد الذي يثبط نشاط السم. يمكن أن يؤدي التعبير عن السم إلى تأثيرات قاتلة على البكتيريا؛ حيث يمكن اعتبارها "قنابل جزيئية داخل الخلايا" قد تؤدي إلى القضاء على الخلايا المضيفة. لذلك، تناقش المراجعة الحالية وظائف انظمة السم-المضاد في تكوين خلايا المثابرة وتأثيرات الإجهاد البيئي على خلايا المثابرة.

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Analysis of Large Networks Using Statistical and Mathematical Techniques: An Applied Study Using MATLAB

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P A P E R I N F O

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Network analysis, degree centrality, median centrality, closeness centrality.

A B S T R A C T

This paper presents an analytical study of networks using mathematical and statistical techniques, with a focus on practical applications using MATLAB. The study aims to understand the internal structure of networks and analyze the nodes with the highest influence using centrality factors such as degree centrality, median centrality, closeness centrality, the experience conducted with MATLAB-supported applications for network representation and extraction of statistical indicators.

1. INTRODUCTION

Networks have become a fundamental component of a wide range of systems. The growth and complexity of these networks has created an urgent need for advanced analytical methods to understand their structure and function.

Large-scale networks are characterized by numerous nodes and complex interconnections. Network analysis requires advanced mathematical and statistical techniques that enable the definition of essential components, the display of hidden structures, and the discovery of elements which may not be noticed directly.[10]

Network analysis incorporates the concept of node centrality, which reflects the importance or influence of each individual node within a network. Various centrality measures—such as degree centrality, closeness centrality, betweenness centrality, pagerank, and eigenvector centrality—offer diverse perspectives on a node's role in information flow, communication, and control within a system. These measures are active in fields such as computer science .

One of the key aspects of network analysis is community detection, which contains identifying sets of nodes that are more densely connected than the rest of the network. Algorithms such as the Louvain method are significantly used for big data due to their efficiency and scalability.

In this study, we present an overall approach to analyzing large networks by combining mathematical concepts with practical applications using the MATLAB.The MATLAB provides a powerful set of built-in functions that make it suitable for simulating, analyzing, and interpreting network structures. By using algorithms to data network's structures, this study aims to identify impact nodes for the underlying network.

2. PREVIOUS STUDIES

The developing of extensive networks has get significant academic interest across a common of fields. Some studies have used mathematical and statistical methods for evaluating the architecture and functionality of such networks, spatially in the discovering of influential nodes and the study of community structures.

Estrada and Rodríguez-Velázquez explained the notion of subgraph centrality, which matures the significance of a node based on its sharing in different subgraphs of the network, thereby

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providing more deep insights than traditional centrality metrics [1]. Klein used a comparison analysis of conventional centrality indicators, including degree, closeness, and betweenness centrality, accentuating their mathematical underpinnings and applicability across various contexts [3].

Latora, Nicosia, and Russo presented a thorough comprehensive study on complex network theory, showing both foundational principles and practical applications such as centrality analysis and community detection, often supplemented by MATLAB code exemplifications [2]. Gómez developed the discourse by applying centrality analysis to business networks, confirming the workable importance of recognize axial nodes in real world data [4].

In the scope of wireless sensor networks, Mbiya, et al. proposed an active routing algorithm that authority centrality mensuration to promote communication pathlane. Their method showing practical efficacy through simulations executed using MATLAB [5].

An important progression in community detection was introduced by Blondel et al. through the Louvain algorithm, that activity reveals hierarchical modular structures within extensive networks, rendering it appropriate, for large data [6].

Newman provided an elaborate mathematical methods for understanding, various network attributes such as modularity and centrality [7]. Additionally, Wasserman et al. understood, the foundational essentials for social network analysis, presenting rigorous statistical and mathematical approaches for examining network [8].

These previous studies constitute the bedrock of research in network science and provide a robust foundation for the present researches, which builds upon these approaches employing applied tools in MATLAB for the analysis of overall and intricate networks.

3. RESEARCH METHODOLOGY

The research is divided into two main parts:

3.1 Theoretical part

This part focuses on the mathematical and statistical ideas used to analyze large networks. It covers the definition of a network and its basic elements, and describes the significance of centrality measures such as degree , closeness , betweenness centrality. It also labels detecting communities' techniques within networks, with a focuse on the benefit of the Louvain algorithm in analyzing big data.

This section puts the theoretical base for the future application of analytical tools using MATLAB.

The Louvain algorithm is a fast and efficient method for subdividing large networks into communities. It is based on enhancing the modularity measure, which measures the density of links within each community compared to the links among communities.

Node Degree is a concept used in network analysis (whether computer, social, or otherwise) to indicate the number of direct connections a node has with other nodes.[4]

1. Importance of Node Degree

- In computer networks:
  - Node degree reflects the number of devices or paths connected to a router or access point.
  - Nodes with a high degree may be critical points in the network.
- In social networks:
  - Node degree represents the number of connections (friends or followers) an individual has.

It is used to analyze influence or importance in a network.

- In transportation networks:

The degree of a node determines the number of roads or paths leading to a given point.

2. Types of Node Degree:

- Node degree in an undirected network:
  - The number of edges connected to the node.

  For example: If a node is connected to three other nodes, its degree is 3.
- Node degree in a directed network:
- In-degree: The number of edges terminating at the node.
- Out-degree: The number of edges starting from the node.

  Total degree = In-degree + Out-degree.

3. Equations for Calculating Node Degree

- Undirected Networks:

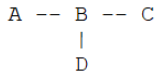
\text{Degree} = \text{Number of Edges Associated with the Node}

- Directed Networks:

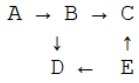
\text{In-Degree} = \text{Number of Edges Entering the Node}

\text{Out-Degree} = \text{Number of Edges Outgoing from the Node}

4. Practical Example
Undirected Network:



Node Degree:
A: 1
B: 3
C: 1
D: 1
Directed Network:



In and Out Degree:
A: In=1, Out=1
B: In=1, Out=2
C: In=1, Out=0
D: In=1, Out=1
E: In=1, Out=1

Closeness Centrality
is a measure in network analysis used to determine the "closeness" of a node. It is based on the total distance between a given node and all other nodes in the network. Simply put, it measures how quickly a node can reach other nodes in the network.[5]
The closeness centrality of a given node is given by the following relationship:

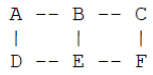
$$c(v) = \frac{1}{\sum_{u \neq v} d(v,u)} \qquad (1)$$

Where:
- d(v,u): is the shortest distance between nodes v and u.
- The sum is calculated for all other nodes u in the network.

Concept:
- Proximity to a node: If a node is close to most other nodes (i.e., the distances between it and other nodes are short), its centrality will be high.
- Far from other nodes: If a node is far from other nodes (i.e., the distances are long), its closeness centrality will be low.

The Importance of Closeness Centrality
- Social Networks:
  - Helps identify individuals who can spread information or influence others most quickly, because they are close to the majority of people in the network.
- Transport Networks:
  - Used to identify the most proximate stations or nodes that allow easy access to the rest of the network.
- Internet Networks:
  - Can be used to identify nodes or devices that may be central or vital for accessing data or other users.

Example of Closeness Centrality
Let's assume a simple network



If we want to calculate the closeness centrality of node "B":
The distances from "B" to other nodes are:
B to A: Distance 1
B to C: Distance 1
B to D: Distance 2
B to E: Distance 1
B to F: Distance 2
Total sum of distances: 1 + 1 + 2 + 1 + 2 = 7
The closeness centrality of B is . C(B)= 1 /7
The smaller this value, the less central the node B is in the network in terms of its closeness.

Important points:
- Nodes with high closeness centrality can be considered "centers of influence" in the network, because they are able to quickly reach most other nodes.
- In very large or disconnected networks, closeness centrality may not always be useful, as the distances between some nodes may be infinite.

Betweenness Centrality
is a measure used in network analysis to identify nodes that lie in the middle of many paths between other nodes in the network. This type of centrality reflects a node's ability to control the flow of information or resources between distant nodes.[6]
Definition of Betweenness Centrality
The betweenness centrality of a given node is measured based on the number of times the node lies on the shortest paths between two pairs of other nodes in the network. The more paths a node lies on between other nodes, the higher its centrality.
Mathematical Formula
To calculate the betweenness centrality of a node, the following formula is used:

$$C_B(V) = \sum_{s \neq v \neq t} \frac{6(s,t \setminus v)}{6(s,t)} \qquad (2)$$

Where:

- CB(V) is the betweenness centrality of node V.
- σ(s,t) is the number of shortest paths between nodes t and s.
- σ(s,t\v) is the number of shortest paths between t and s that pass through node v.

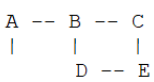
Concept

- Brokerage: If a node lies on many paths between other nodes, it is a "broker" in the network. Nodes with high brokerage centrality influence the flow of information or connections between nodes.
- Shortest paths: These are paths with the fewest edges between any pair of nodes.

Importance of Brokerage Centrality

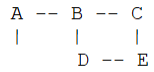
- Social Networks:
  - In social networks, nodes with high brokerage centrality are well-positioned to influence the flow of information between individuals. For example, an individual who resides at the center of a social network and has many connections with other individuals in different locations may be a "broker" in the network.
- Transportation Networks:
  - In transportation networks, nodes with high brokerage centrality can serve as vital hubs for communication between different locations. Disabling these nodes may reduce communication between other areas.
- Internetworks:
  - In the internet, nodes with high brokerage centrality can serve as key gateways between different sub networks.

A Practical Example of Betweenness Centrality:
Suppose we have a small network containing nodes A, B, C, D, and E.



To calculate betweenness centrality for node B:

The shortest paths between pairs in the network are:
From A to C: The path is A → B → C.
From D to C: The path is D → B → C.
From A to E: The path is A → B → E.
From D to A: The path is D → B → A.
In this example, node B is located on many paths between other nodes. Therefore, betweenness centrality for B will be high.



- Advantages of betweenness centrality
  - It helps identify nodes that can significantly influence the transmission of information in a network.
  - It can be used in social analysis to understand the people who connect different groups in a social network.
  - In technological networks, it helps identify the devices or servers that play a vital role in the transmission of data across the network.

Intermediateness centrality expresses the extent of a node's influence in connecting different parts of a network. Nodes that are located on many of the shortest paths between other nodes have high intermediation centrality, making them important in facilitating or controlling the flow of information in the network.

- Community detection

Community detection is a fundamental concept in network analysis. It is used to identify groups or "clusters" within a network in which nodes are more closely connected to each other than to the rest of the network. These clusters are typically defined as groups of nodes that interact or are more densely connected to each other than to other nodes outside the group.[7]

- Why is community detection important?

Cluster detection is useful for understanding the structure of a network and discovering patterns that may not be apparent when looking at the network as a whole. For example:
• In social networks, it can help identify groups or teams within the network, such as friends or individuals with similar interests.
• In internet networks, it helps segment the network into groups of devices or servers that engage in intensive communication.
• In transport networks, it helps identify hubs or key points that connect different parts of the system.
The algorithm employs in two iterative stages:

Locally improving modularity by moving nodes to clusters that increase modularity. Merging clusters into a new network and repeating the process. It is simple, efficient, and able of analyzing large networks, but it may have problems detecting small clusters.

3.2 Practical part

In practical part, MATLAB was used to measure the three factors, such as calculating centrality (degree, closeness, mediation)[11] a s shown in figure 1 :

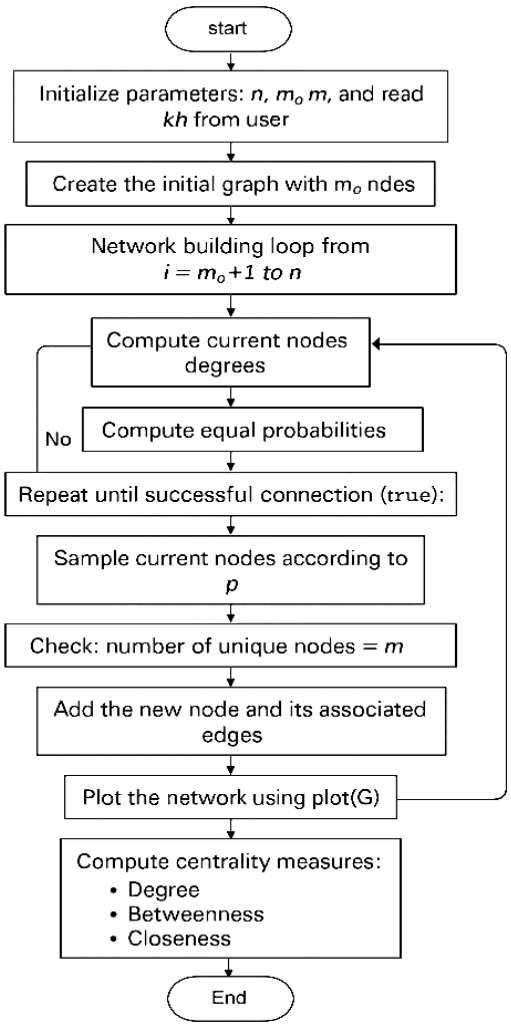


Figure 1. flowchart of measure the three factors

Such as using Louvain Algorithm , it employs in two iterative stages:

- Locally improving modularity by moving nodes to clusters that increase modularity.
- Merging clusters into a new network and repeating the process.

It is simple, efficient, and able of analyzing large networks, but it may have problems detecting small clusters.

```
A -- B -- C
     |
     D
```

4. RESULTS AND DISCUSSION

The analysis of the experimental network revealed that certain nodes play a essential role in the network structure. For example, nodes with high centrality degrees were the most impact in data flow, while betweenness centrality helped identify nodes that represent critical crossing points between network communities.

The Louvain algorithm was employed to detect clusters, demonstrating high effectiveness in uncovering the structure of community within the network.

The impact Factor is similar if the number of cluster is 3 ,it appeared that all clusters were equals.

TABLE 1. no. of nodes each factors

Degree Centrality	3	6	7
Betweenness Centrality	6	3	7
Closeness Centrality	6	3	7

Due to the small number of nodes, nodes 3, 6, and 7 exhibited similar importance, though some variations were observed.

it shown it's shown in next figure 2:

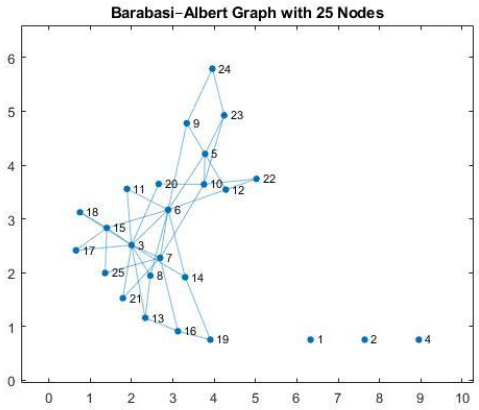


Figure 2. Barabasi-Albert graph with 25 nodes

In the second experiment took 15 nodes and distributed them into Five clusters and the result is as Fellows:

TABLE 2. no. of nodes each factors

Degree Centrality	8	7	1	6	14

Betweenness Centrality	8	7	6	1	17
Closeness Centrality	8	6	1	7	13

It is evident that the ranking of influential nodes varies depending on the centrality metric used. example 14 is existant in Degree Centrality and not in the others.

and the same applies to 17 and 13 for Betweenness Centrality and Closeness Centrality respectively .

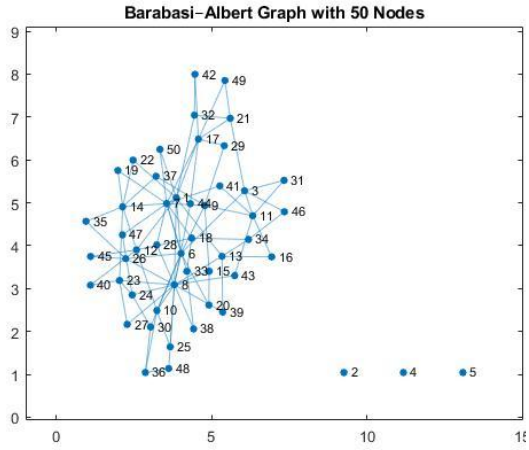


Figure 3. Barabasi-Albert graph with 50 nodes

In the third experiment seven nodes were used and distributed as follows:

TABLE 3. no. of nodes each factors

Degree Centrality	9	11	6	4	23	7	8
Betweenness Centrality	9	11	6	4	7	23	8
Closeness Centrality	9	11	6	4	7	8	5

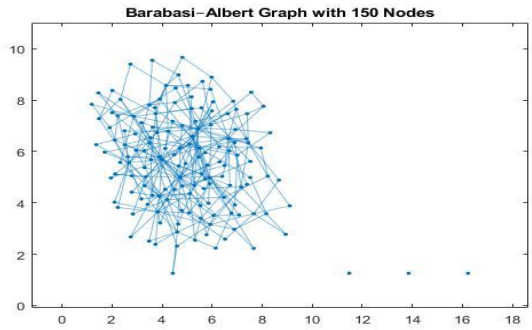


Figure 4. Barabasi-Albert graph with 50 nodes
There are different points.

Example 7 is in Degree Centrality and it is not in Betweenness Centrality and Closeness Centrality Also the Node 23 and 5 for Betweenness Centrality and Closeness Centrality respectively.

We can notice here that if network is bigger, then the difference will increase because the factors differ according to the mathematical rules of each Factor which determines the importance points.

the sort is important and it is shown in table 4:

TABLE 4. no. of nodes each experience

Experience	No. of nodes	No. of points	Factors		
			Degree Centrality	Betweennness Centrality	Closeness Centrality
1	25	3	3,6,7	6,3,7	6,3,7
2	50	5	8,7,1,6, 14	8,7, 6, 1, 17	8,6, 1, 7, 13
3	150	7	9,11, 6, 4,23,7, 8	9,11, 6,4,7 ,23,8	9,11,6,4,7,8 ,5

5. CONCLUSION

This study highlights the importance of using mathematical and statistical ways in analyzing big networks, focusing on MATLAB tools which provide a productive environment for pursuance algorithms and information analysis. Core nodes within the network were identified through different centrality metrics, and cluster discovery algorithms were successfully implemented.

Future work

The results of this study can be used in many practical fields. We can use this algorithm to measure large networks, and employ it in social network analysis, to identify the most influential individuals or entities on social media networks to target marketing campaigns, for example.

Practical application using MATLAB

This chapter explains how to apply theoretical concepts to analyze large networks utilizing the MATLAB environment. It includes representing a random network, calculating various centrality matters, and exploring clusters within the network by pursuance an algorithms utilizing MATLAB tools.

Representation of a Random Network

A random network of many nodes is made using the Barabási–Albert model or a random connection matrix.

Example: To create a network of 100 nodes with a 5% connection probability ,using MATLAB code:

```
n = 100;
p = 0.05;
A = rand(n) < p;
A = triu(A, 1);
A = A + A';
G = graph(A);
plot(G);
title('Random network representation');
```

Calculating Centrality Measures

using MATLAB functions to calculate the centrality of nodes:

-Degree Centrality
-Closeness Centrality
-Betweenness Centrality
-PageRank and Eigenvector

```
deg = centrality(G, 'degree');
close = centrality(G, 'closeness');
btwn = centrality(G, 'betweenness');
ev = centrality(G, 'eigenvector');
pr = centrality(G, 'pagerank');
```

Ploting the network with highlighted influential nodes

The significance of this study lies in its practical applicability across various domains.

The techniques covered in this paper can be extended to real data networks in diverse fields such as cybersecurity and public health.

By using the computational methods of MATLAB, the study provides an efficient framework for researchers to explore and analyze large data.

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Arabic Abstract

تقدم هذه الورقة دراسة تحليلية للشبكات باستخدام تقنيات رياضية وإحصائية، مع التركيز على التطبيقات العملية باستخدام MATLAB. تهدف الدراسة إلى فهم البنية الداخلية للشبكات وتحليل العقد ذات التأثير الأعلى باستخدام عوامل المركزية، مثل مركزية الدرجة، ومركزية الوسيط، ومركزية القرب، بالإضافة إلى الخبرة المكتسبة باستخدام تطبيقات MATLAB لتمثيل الشبكات واستخراج المؤشرات الإحصائية.

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New Subclasses of Regular and Bi-univalent Functions Based on Horadam polynomials

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A B S T R A C T

In this paper, new subclasses of bi-univalent functions associated with Horadam polynomials are introduced and investigated. Additionally, the researchers find estimates of the first two coefficients of functions in these subclasses. Moreover, they obtained the Fekete-Szegö inequalities for these function classes. Besides, pertinent links of the results are provied with those considered in previously investigations.

1. INTRODUCTION

Let f be function of the form

$$f(z) = z + \sum_{k=2}^{\infty} a_k z^k \ . \qquad (1)$$

which belongs to A where A is the class of regular functions defined on the disk

$$U = \{z \in \mathbb{C} : |z| < 1\},$$

with $f(0) = f'(0) - 1 = 0$ and let S be the subclass of A consisting of the form (1) which are also univalent in U. The Koebe's Covering Theorem (see [1]) states for each $f \in S$ the image $w = f(z), z \in U,$, in the w-plane contains the disk$\{w : |w| < 1/4\}$. From this theorem, every function $f \in S$ has an inverse f^{-1}which holds

$$f^{-1}\big(f(z)\big) = z, (z \in U)$$

and,

$$f\big(f^{-1}(w)\big) = w \left(|w| < r_0(f), r_0(f) \geq \frac{1}{4}\right),$$

where

$$g(w) = f^{-1}(w) = w - a_2 w^2 + (2a_2^2 - a_3)w^3 - (5a_2^3 - 5a_2 a_3 + a_4)w^4 + \cdots$$

A function $f \in \mathbb{A}$ is called bi-univalent in U if both f and f^{-1} are univalent in U.The class of all bi-univalent functions in U is denoted byΣ. This class was introduced by Lewin [2] and showed that $|a_2| \leq 1.51$ for the function in the class Σ. Recently, Brannan and Clunie [3] conjectured that $|a_2| \leq \sqrt{2}$. Netanyahu in [4] proved that $|a_2| = \frac{4}{3}$. Several researchers have examined these subclasses of bi-univalent regular function and found estimates of the initial coefficients for functions in different subclasses [5-10].

A function $f \in \mathbb{A}$ is called a λ–pseudo starlike function in U if the following inequality holds are true (see [11]):

$$\mathcal{R}\left[\frac{z\big(f'(z)\big)^{\lambda}}{f(z)}\right] \geq 0, (z \in U), \lambda \geq 1.$$

For two regular functions f_1 and f_2, the function f_1 subordination to f_2 in the disk U, is written as follows:

$$f_1(z) \prec f_2(z), \quad (z \in U),$$

if there is a regular function k with $k(0) = 0$ and $|k(z)| < 1$, such that

$$f_1(z) = f_2\big(k(z)\big), (z \in U).$$

In particular, when f_2 is univalent in U,

$f_1 \prec f_2 \quad (z \in U) \iff f_1(0) = f_2(0)$ and $f_1(U) = f_2(U)$.

Lately, the polynomial defined below has been included in the topic of the geometric function theory of complex

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analysis. The Horadam polynomials are defined by the recurrence relationship as follows [12].

$$\hbar_n(r) = \wp r \hbar_{n-1}(r) + q\hbar_{n-2}(r) \;\; r \in \mathbb{R}, n > 2, n \in \aleph, \qquad (2)$$

with

$$\hbar_1(r) = a,\ \hbar_2(r) = br \text{ and } \hbar_3(r) = \wp br^2 + aq \text{ for some } \wp. q, a, b \in \mathbb{R}.$$

The characteristic equation of the iteration relation (2) is

$$t^2 - \wp r t - q = 0.$$

The real roots of this equation are

$$\alpha_1 = \frac{\wp r + \sqrt{(\wp r)^2 + 4q}}{2}, \alpha_2 = \frac{\wp r - \sqrt{(\wp r)^2 + 4q}}{2}.$$

It should be noted that for specific values of parameters, the Horadam polynomial $\hbar_n(x)$ leads to different polynomials. To learn more about the details and special cases of the Horadam polynomials (see [13–14]). In [14], the Horadam polynomials $\hbar_n(r)$ are generated by:

$$g(r, z) = \sum_{n=1}^{\infty} h_n(r) z^{n-1} = \frac{a + (b - ap)rz}{1 - prz - qz^2}. (3)$$

The Horadam polynomials $\hbar_n(r)$ have recently been used in a similar context by Srivastava et al. [15]. Subsequently, many authors used a Horadam polynomial (see [16–19]).

The aim of the present work is to define two subclasses of the function classΣ using the Horadam polynomials $\hbar_n(r)$ and estimate the bounds of the coefficients a_2, a_3 and the Fekete–Szegö for functions of the subclasses presented throughout this work.

2. COEFFICIENT ESTIMATES FOR SUBCLASS $G_\Sigma(\lambda, \wp, q, r, g)$

We start with introducing the function subclass $G_\Sigma(\lambda, \wp, q, r, g)$ using the following definition..

Definition (2.1): Let $G(\lambda, \Sigma, \wp, q, r, g)$ be the class of the function $f \in \Sigma$ given by (1) under the next subordination

$$\left[\frac{zf'(z)}{f(z)} + \frac{zf''(z)}{f'(z)} - \frac{\lambda z^2 f''(z) + zf'(z)}{\lambda zf'(z) + (1-\lambda)f(z)}\right] \prec g(r, z) - a, (z \in U),$$

$$\left[\frac{wg'(w)}{g(w)} + \frac{wg''(w)}{g'(w)} - \frac{\lambda w^2 g''(w) + wg'(w)}{\lambda wg'(w) + (1-\lambda)g(w)}\right] \prec g(r, w) - a, (w \in U),$$

and for $g = f^{-1}, 0 \le \lambda \le 1$.

Remark(2.2): Setting $\lambda = 0$ in the class $G(\lambda, \Sigma, \wp, q, r, g)$, we obtain $X(\Sigma, \wp, q, r, g)$.

Remark(2.3): Setting $\lambda = 1$ in the class $G(\lambda, \Sigma, \wp, q, r, g)$, we obtain $T(\Sigma, \wp, q, r, g)$.

At first, we state and prove the next result**.**

Theorem (2.4): Let $f \epsilon G_\Sigma(\lambda, \wp, q, r, g)$. Then

$$|a_2| \le \frac{|br|\sqrt{|br|}}{\sqrt{|(br)^2\,(1-4\lambda+(\lambda+1)^2) - (bpr^2+qa)(2-\lambda)^2|}}, \quad (4)$$

$$|a_3| \le \frac{|br|^2}{(2-\lambda)^2} + \frac{|br|}{2(3-2\lambda)}, \qquad (5)$$

$$|a_3 - \varepsilon a_2^2| \le \begin{cases} \frac{|br|}{2(3-2\lambda)}, & if \\ |\varepsilon - 1| \le \frac{|(br)^2\,(1-4\lambda+(\lambda+1)^2) - (bpr^2+qa)(2-\lambda)^2|}{2(3-2\lambda)(br)^2}, & \\ \frac{|(br)^3\,(1-\varepsilon))|}{|(br)^2\,(1-4\lambda+(\lambda+1)^2) - (bpr^2+qa)(2-\lambda)^2|}, & if \\ |\varepsilon - 1| \ge \frac{|(br)^2\,(1-4\lambda+(\lambda+1)^2) - (bpr^2+qa)(2-\lambda)^2|}{2(3-2\lambda)(br)^2} & \end{cases}$$

Proof: Since $f \epsilon G_\Sigma(\lambda, \wp, q, r, g)$, there are holomorphic functions π, υ belong to $\mathbb{A}$ and $\pi, \upsilon: U \to U$ given

$\pi(z) = \pi_1 z + \pi_2 z^2 + \pi_3 z^3 + \cdots \quad (z \in U)$,

$\upsilon(w) = \upsilon_1 w + \upsilon_2 w^2 + \upsilon_3 w^3 + \cdots, (w \in U)$,

such that $\pi(0) = \upsilon(0) = 0$ *and* $|\pi(z)| < 1, |\upsilon(w)| < 1, (z, w \in U)$ and we can write

$$\left[\frac{zf'(z)}{f(z)} + \frac{zf''(z)}{f'(z)} - \frac{\lambda z^2 f''(z) + zf'(z)}{\lambda zf'(z) + (1-\lambda)f(z)}\right] = g\big(r, \pi(z)\big) - a,$$

$$\left[\frac{wg'(w)}{g(w)} + \frac{wg''(w)}{g'(w)} - \frac{\lambda w^2 g''(w) + wg'(w)}{\lambda wg'(w) + (1-\lambda)g(w)}\right] = g\big(r, \upsilon(w)\big) - a. \quad (6)$$

Or, in equivalent way,

$$\left[\frac{zf'(z)}{f(z)} + \frac{zf''(z)}{f'(z)} - \frac{\lambda z^2 f''(z) + zf'(z)}{\lambda zf'(z) + (1-\lambda)f(z)}\right] = \hbar_2(r)\pi_1 z + [\{\hbar_2(r)\pi_2 + \hbar_3(r){\pi_1}^2\} +]z^2 + \cdots, \quad (7)$$

and

$$\left[\frac{wg'(w)}{g(w)} + \frac{wg''(w)}{g'(w)} - \frac{\lambda w^2 g''(w) + wg'(w)}{\lambda wg'(w) + (1-\lambda)g(w)}\right] = \hbar_2(r)\upsilon_1 w + [\{\hbar_2(r)\upsilon_2 + \hbar_3(r){\upsilon_1}^2\}]w^2 + \cdots. \quad (8)$$

From (7) and (8), it follows that

$$(2-\lambda)a_2 = \hbar_2(r)\pi_1, \qquad (9)$$

$$(6-4\lambda)a_3 - (5-(\lambda+1)^2){a_2}^2 = \{\hbar_2(r)\pi_2 + \hbar_3(r){\pi_1}^2\}, (10)$$

$$-(2-\lambda)a_2 = \hbar_2(r)\upsilon_1, \quad (11)$$

$$(7 - 8\lambda + (\lambda+1)^2){a_2}^2 - (6-4\lambda)a_3 = \{\hbar_2(r)\upsilon_2 + \hbar_3(r){\upsilon_1}^2\}. \quad (12)$$

From (9) and (11), it follows that

$$\pi_1 = -\upsilon_1 \quad (13)$$

$$2(2-\lambda)^2{a_2}^2 = {\hbar_2}^2(r)\,(\pi_1^2 + \upsilon_1^2). \quad (14)$$

Adding (10) and (12), we obtain that

$$2[1 - 4\lambda + (\lambda+1)^2]{a_2}^2 = \hbar_2(r)(\pi_2 + \upsilon_2) + \hbar_3(r)(\pi_1^2 + \upsilon_1^2) \qquad (15)$$

Using (14) in (15), we conclude that

$$2\left[(1 - 4\lambda + (\lambda+1)^2) - \frac{\hbar_3(r)(2-\lambda)^2}{{\hbar_2}^2(r)}\right]{a_2}^2 = \hbar_2(r)(\pi_2 + \upsilon_2), \qquad (16)$$

$$a_2^2 = \frac{{\hbar_2}^3(r)(\pi_2+\upsilon_2)}{2[{\hbar_2}^2(r)\,(1-4\lambda+(\lambda+1)^2) - \hbar_3(r)(2-\lambda)^2]} \quad (17)$$

From (2) and (17), we have the required inequality (4).

Subtracting (12) from (10), we have

$$a_3 = {a_2}^2 + \frac{\hbar_2(r)(\pi_2 - \upsilon_2)}{4(3-2\lambda)}. \qquad (18)$$

In view of (13) and (14), equation (18) becomes

$$a_3 = \frac{\hbar_2{}^2(r)\,(\pi_1^2+v_1^2)}{2(2-\lambda)^2} + \frac{\hbar_2(r)(\pi_2-v_2)}{4(3-2\lambda)}.$$

By using $|\pi_i| < 1$, $|v_i| < 1$ and (2), we have the required inequality (5).

From (18), for $\varepsilon \in \mathbb{R}$, we write

$$a_3 - \varepsilon a_2{}^2 = \frac{\hbar_2(r)(\pi_2-v_2)}{4(3-2\lambda)} + (1-\varepsilon)a_2{}^2 \quad (19)$$

By substituting (17) in (19), we obtain that

$$a_3 - \varepsilon a_2{}^2 = \frac{\hbar_2(r)(\pi_2-v_2)}{4(3-2\lambda)} + \frac{(1-\varepsilon)\,\hbar_2{}^3(r)(\pi_2+v_2)}{2[\hbar_2{}^2(r)\,(1-4\lambda+(\lambda+1)^2)-\hbar_3(r)(2-\lambda)^2]} = \hbar_2(r)\left[\left(\Upsilon(\varepsilon,r) + \frac{1}{4(3-2\lambda)}\right)\pi_2 + \left(\Upsilon(\varepsilon,r) - \frac{1}{4(3-2\lambda)}\right)v_2\right],$$

where

$$\Upsilon(\varepsilon,r) = \frac{(1-\varepsilon)\,\hbar_2{}^2(r)}{2\,[\hbar_2{}^2(r)\,(1-4\lambda+(\lambda+1)^2)-\hbar_3(r)(2-\lambda)^2]}.$$

Thus, we deduce that

$$\left|a_3 - \varepsilon a_2{}^2\right| \leq \begin{cases} \frac{\hbar_2(r)}{2(3-2\lambda)}, & 0 \leq \Upsilon(\varepsilon,r) \leq \frac{1}{4(3-2\lambda)}, \\ 2|\,\hbar_2(r)|\Upsilon(\varepsilon,r), & |\Upsilon(\varepsilon,r)| \geq \frac{1}{4(3-2\lambda)}\,. \end{cases}$$

From here with consideration of (2), it clearly shows that the proof of Theorem 2.4 is completed.

Corollary (2.5): If the function $f(z)$ in $X(\Sigma, p, q, r, g)$ given by (1), then

$$|a_2| \leq \frac{|br|\sqrt{|br|}}{\sqrt{|2(br)^2 - 4(bpr^2+qa)\,|}},$$

$$|a_3| \leq \frac{|br|^2}{4} + \frac{|br|}{6},$$

$$\left|a_3 - \varepsilon a_2{}^2\right| \leq \begin{cases} \frac{|br|}{6}, & if \\ |\varepsilon - 1| \leq \frac{|2(br)^2 - 4(bpr^2+qa)\,|}{6(br)^2}, & \\ \frac{|(br)^3\,(1-\varepsilon))|}{|2(br)^2 - 4(bpr^2+qa)\,|}, & if \\ |\varepsilon - 1| \geq \frac{|2(br)^2 - 4(bpr^2+qa)\,|}{6(br)^2}\,. & \end{cases}$$

Corollary (2.6): If the function $f(z)$ in $T(\Sigma, p, q, r, g)$ given by (1), then

$$|a_2| \leq \frac{|br|\sqrt{|br|}}{\sqrt{|(br)^2 - bpr^2+qa\,|}},$$

$$|a_3| \leq |br|^2 + \frac{|br|}{2},$$

$$\left|a_3 - \varepsilon a_2{}^2\right| \leq \begin{cases} \frac{|br|}{2}, & |\varepsilon - 1| \leq \frac{|(br)^2 - (bpr^2+qa)\,|}{2(br)^2}, \\ \frac{|(br)^3\,(1-\varepsilon))|}{|(br)^2 - (bpr^2+qa)\,|}, & |\varepsilon - 1| \geq \frac{|(br)^2 - (bpr^2+qa)\,|}{2(br)^2}. \end{cases}$$

3. COEFFICIENT ESTIMATES FOR SUBCLASS $\mathcal{M}_\Sigma(\mu, \lambda, \rho, p, q, r, g)$

Definition (3.1): A function $f \in \Sigma$ of the form (1) is in $\mathcal{M}_\Sigma^q(\mu, \lambda, \rho, p, q, r, g)$ if it satisfies conditions

$$(1-\mu)\left[\rho\left(\frac{zf''(z)}{f'(z)} + 1\right) + (1-\rho)f'(z)\right] + \mu\left[\frac{z\left(f'(z)\right)^\lambda}{f(z)}\right] \prec g(r,z) + 1 - a\,, (z \in U),$$

$$(1-\mu)\left[\rho\left(\frac{wg''(w)}{g'(w)} + 1\right) + (1-\rho)g'(w)\right] + \mu\left[\frac{w\left(g'(w)\right)^\lambda}{g(w)}\right] \prec g(r,w) + 1 - a,\ (w \in U),$$

for $g = f^{-1}$, $0 \leq \mu \leq 1$, $0 \leq \rho \leq 1$ and $\lambda \geq 1$.

Remark (3.2): For $\mu = 0$, the class $\mathcal{M}_\Sigma(\mu, \lambda, \rho, p, q, r, g)$ is shortened to the class $\mathcal{G}_\Sigma^*(\alpha, x)$ presented and investigated in [20]. In particular, in [20], for $\rho = 0$, we have

$$\mathcal{M}_\Sigma(0, \lambda, 0, p, q, r, g) \coloneqq \mathcal{H}_\Sigma(x),$$

also, for $\rho = 1$, we have

$$\mathcal{M}_\Sigma(0, \lambda, 1, p, q, r, g) \coloneqq \mathcal{K}_\Sigma(x).$$

Remark (3.3): For $\mu = a = 1, p = b = 2, q = -1$ and $r \to t$, then $\mathcal{M}_\Sigma(\mu, \lambda, \rho, p, q, r, g)$ reduced to the class $\mathcal{LB}_\Sigma(\lambda, t)$ presented and investigated in [21].

Remark (3.4): For $\mu = 1$, we have the class $\mathcal{H}_\Sigma(\lambda, \rho, p, q, r, g)$.

Remark (3.5): For $\lambda = 1$, we have the class $\mathcal{N}_\Sigma(\mu, \rho, p, q, r, g)$.

Theorem (3.6): If $f \epsilon \mathcal{M}_\Sigma(\mu, \lambda, \rho, p, q, r, g)$, then

$$|a_2| \leq \frac{2|br|\sqrt{|br|}}{\sqrt{\left|\begin{matrix}(br)^2\,\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)+2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\} \\ -2(bpr^2+qa)\{2+\mu(2\lambda-3)\}^2\end{matrix}\right|}}, \quad (20)$$

$$|\,a_3| = \frac{br}{3(1-\mu)(\rho+1)+\mu(3\lambda-1)} + \frac{(br)^2}{\{2+\mu(2\lambda-3)\}^2}, \quad (21)$$

$$|\,a_3 - \varepsilon a_2{}^2| \leq \begin{cases} \frac{|br|}{3(1-\mu)(\rho+1)+\mu(3\lambda-1)} & if \\ |\varepsilon - 1| \leq \frac{|\boldsymbol{\theta_1}-\boldsymbol{\theta_2}|}{2(br)^2[3(1-\mu)(\rho+1)+\mu(3\lambda-1)]}, & \\ \frac{|1-\varepsilon\ |\,|br|^3}{|\boldsymbol{\theta_1}-\boldsymbol{\theta_2}|} & if \\ |\varepsilon - 1| \geq \frac{|\boldsymbol{\theta_1}-\boldsymbol{\theta_2}|}{2(br)^2[3(1-\mu)(\rho+1)+\mu(3\lambda-1)]} & \end{cases}$$

Where $\boldsymbol{\theta_1} = (br)^2\{\mu(2\lambda^2 - 4\lambda + 1) - 4\rho(1-\mu) + 2(1-\mu)(\rho+3) + \mu(2\lambda^2 + 2\lambda - 1)\}$

$\boldsymbol{\theta_2} = 2(pbr^2 + qa)\{2 + \mu(2\lambda - 3)\}^2$

Proof: Since $f \epsilon \mathcal{M}_\Sigma^q(\mu, \lambda, \rho, p, q, r, g)$, there are regular functions π, v belong to $\mathbb{A}$ and $\pi, v: U \to U$ given

$$\pi(z) = \pi_1 z + \pi_2 z^2 + \pi_3 z^3 + \cdots, \quad (z \in U),$$

$$v(w) = v_1 w + v_2 w^2 + v_3 w^3 \ldots, \quad (w \in U),$$

Such that $\pi(0) = \upsilon(0) = 0\ and\ |\pi(z)| < 1, |\upsilon(w)| < 1, (z, w \in U)$ satisfying

$$(1-\mu)\left[\rho\left(\frac{zf''(z)}{f'(z)}+1\right)+(1-\rho)f'(z)\right]+\mu\left[\frac{z\left(f'(z)\right)^{\lambda}}{f(z)}\right]=g\left(r,\pi(z)\right)+1-a,$$

$$(1-\mu)\left[\rho\left(\frac{wg''(w)}{g'(w)}+1\right)+(1-\rho)g'(w)\right]+\mu\left[\frac{w\left(g'(w)\right)^{\lambda}}{g(w)}\right]=g\left(r,\upsilon(w)\right)+1-a,$$

$$(1-\mu)\left[\rho\left(\frac{zf''(z)}{f'(z)}+1\right)+(1-\rho)f'(z)\right]+\mu\left[\frac{z\left(f'(z)\right)^{\lambda}}{f(z)}\right]=$$

$$1+\hbar_2(r)\pi_1 z+[\{\hbar_2(r)\pi_2+\hbar_3(r){\pi_1}^2\}+]z^2+\cdots, \quad (22)$$

$$(1-\mu)\left[\rho\left(\frac{wg''(w)}{g'(w)}+1\right)+(1-\rho)g'(w)\right]+\mu\left[\frac{w\left(g'(w)\right)^{\lambda}}{g(w)}\right]=$$

$$1+\hbar_2(r)\upsilon_1 w+[\{\hbar_2(r)\upsilon_2+\hbar_3(r){\upsilon_1}^2\}+]w^2+\cdots. \quad (23)$$

From (22) and (23), it follows that

$$\{2+\mu(2\lambda-3)\}a_2=\hbar_2(r)\pi_1, \quad (24)$$

$$\{3(1-\mu)(\rho+1)+\mu(3\lambda-1)\}a_3+\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)\}{a_2}^2=\hbar_2(r)\pi_2+\hbar_3(r){\pi_1}^2, \quad (25)$$

$$-\{2+\mu(2\lambda-3)\}a_2=\hbar_2(r)\upsilon_1, \quad (26)$$

$$\{2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\}{a_2}^2-\{3(1-\mu)(\rho+1)+\mu(3\lambda-1)\}a_3=\hbar_2(r)\upsilon_2+\hbar_3(r){\upsilon_1}^2. \quad (27)$$

The equations (24) and (26), lead to

$$\pi_1=-\upsilon_1. \quad (28)$$

Squaring and adding (24), (26), we have

$$2\{2+\mu(2\lambda-3)\}^2{a_2}^2=\hbar_2^2(r)({\pi_1}^2+{\upsilon_1}^2). \quad (29)$$

Add (25) to (27), we conclude that

$$\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)+2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\}{a_2}^2=\hbar_2(r)(\pi_2+\upsilon_2)+\hbar_3(r)({\pi_1}^2+{\upsilon_1}^2). \quad (30)$$

By substituting (29) in (30), we obtain that

$$\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)+2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\}{a_2}^2=\hbar_2(r)(\pi_2+\upsilon_2)+\hbar_3(r)\frac{2\{2+\mu(2\lambda-3)\}^2}{\hbar_2^2(r)}{a_2}^2,$$

$${a_2}^2=\frac{\hbar_2^3(r)(\pi_2+\upsilon_2)}{\theta_3-\theta_4} \quad (31)$$

From (2) and (31), we have the required inequality (20).

In order to find a_3, we subtract (27) from (25) to obtain

$$[6(1-\mu)(\rho+1)+2\mu(3\lambda-1)]({a_2}^2-a_3)=(\pi_2-\upsilon_2)\hbar_2(r)+({\pi_1}^2-{\upsilon_1}^2)\hbar_3(r). \quad (32)$$

In view of (28) and (32), we have

$$a_3=\frac{(\pi_2-\upsilon_2)\hbar_2(r)}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}+{a_2}^2. \quad (33)$$

In view of (28) and (29), equation (33) becomes

$$a_3=\frac{(\pi_2-\upsilon_2)\hbar_2(r)}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}+\frac{\hbar_2^2(r)({\pi_1}^2+{\upsilon_1}^2)}{2\{2+\mu(2\lambda-3)\}^2}.$$

Now, by using equation (2) and applying $|\pi_i| \le 1, |\upsilon_i| \le 1$, we deduce that

$$|a_3|=\frac{|br|}{3(1-\mu)(\rho+1)+\mu(3\lambda-1)}+\frac{(br)^2}{\{2+\mu(2\lambda-3)\}^2}.$$

For $\varepsilon \in \mathbb{R}$, using (33), we conclude that

$$a_3-\varepsilon{a_2}^2=\frac{(\pi_2-\upsilon_2)\hbar_2(r)}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}+(1-\varepsilon){a_2}^2. \quad (34)$$

By substituting (31) in (34), we have

$$a_3-\varepsilon{a_2}^2=\frac{(\pi_2-\upsilon_2)\hbar_2(r)}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}+\frac{(1-\varepsilon)\hbar_2^3(r)(\pi_2+\upsilon_2)}{\theta_3-\theta_4}.$$

Where $\theta_3=\hbar_2^2(r)\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)+2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\}$

$$\theta_4=\hbar_2^2(r)\{\mu(2\lambda^2-4\lambda+1)-4\rho(1-\mu)+2(1-\mu)(\rho+3)+\mu(2\lambda^2+2\lambda-1)\}$$

Simplifying the above equation, we obtain that

$$a_3-\varepsilon{a_2}^2=\hbar_2(r)\left\{\left[\frac{1}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}+\vartheta(\varepsilon,r)\right]\pi_2+\left[\vartheta(\varepsilon,r)-\frac{1}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}\upsilon_2\right]\right\},$$

where

$$\vartheta(\varepsilon,r)=\frac{(1-\varepsilon)\hbar_2^2(r)}{\theta_3-2\hbar_3(r)\{2+\mu(2\lambda-3)\}^2}.$$

Thus, we conclude that

$$|a_3-\varepsilon{a_2}^2| \le \begin{cases} \dfrac{\hbar_2(r)}{3(1-\mu)(\rho+1)+\mu(3\lambda-1)}, & if \\ 0 \le |\vartheta(\varepsilon,r)| \le \dfrac{1}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)}, & \\ 2|\hbar_2(r)||\vartheta(\varepsilon,r)|, & if \\ |\vartheta(\varepsilon,r)| \ge \dfrac{1}{6(1-\mu)(\rho+1)+2\mu(3\lambda-1)} & \end{cases}$$

By using (2), it shows clearly the proof of Theorem 3.6 is completed.

Corollary (3.7): If $f \epsilon \mathcal{H}_{\Sigma}(\lambda,\rho,\mathcal{p},q,r,g)$, then

$$|a_2| \le \frac{2|br|\sqrt{|br|}}{\sqrt{\left|2\lambda(br)^2(2\lambda-1)-2(bpr^2+qa)(2\lambda-1)^2\right|}},$$

$$|a_3| \le \frac{br}{(3\lambda-1)}+\frac{(br)^2}{(2\lambda-1)^2},$$

$|a_3-\varepsilon a_2{}^2| \leq$

$$\begin{cases} \frac{|br|}{(3\lambda-1)} \text{ if} \\ |\varepsilon-1| \leq \frac{|2\lambda(br)^2(2\lambda-1)-2(\not{p}br^2+q_a)(2\lambda-1)^2|}{2(br)^2(3\lambda-1)}, \\ \frac{|1-\varepsilon\ |\,|br|^3}{|2\lambda(br)^2(2\lambda-1)-2(\not{p}br^2+q_a)(2\lambda-1)^2|}, if \\ |\varepsilon-1| \geq \frac{|2\lambda(br)^2(2\lambda-1)-2(\not{p}br^2+q_a)(2\lambda-1)^2|}{2(br)^2(3\lambda-1)}. \end{cases}$$

Corollary (3.8): If $f\epsilon\mathcal{N}_{\Sigma}(\mu,\rho,\not{p},q,r,\not{g})$, then

$$|a_2| \leq \frac{2|br|\sqrt{|br|}}{\sqrt{|(br)^2\{2\mu-4\rho(1-\mu)+2(1-\mu)(\rho+3)\}-2(bpr^2+q_a)(2-\mu)^2|}},$$

$$|a_3| \leq \frac{br}{3(1-\mu)(\rho+1)+2\mu}+\frac{(br)^2}{(2-\mu)^2}$$

$|a_3-\varepsilon a_2{}^2|$

$$\leq \begin{cases} \frac{|br|}{3(1-\mu)(\rho+1)+2\mu}, \quad if \\ |\varepsilon-1| \leq \frac{|\theta_5-2(\not{p}br^2+q_a)(2-\mu)^2|}{2(br)^2[3(1-\mu)(\rho+1)+2\mu]}, \\ \frac{|1-\varepsilon\ |\,|br|^3}{|\theta_5-2(\not{p}br^2+q_a)(2-\mu)^2|}, if \\ |\varepsilon-1| \geq \frac{|\theta_5-2(\not{p}br^2+q_a)\{2+\mu(2\lambda-3)\}^2|}{2(br)^2[3(1-\mu)(\rho+1)+2\mu]}, \end{cases}$$

where $\theta_5 = 2\lambda(br)^2(2\lambda-1)$.

4. CONCLUSIONS

In this work, new subclasses of bi-univalent functions defined by means of the Horadam polynomials $\hbar_n(r)$ are studied. The central purpose of this study is that bounds for the initial coefficients are established. Furthermore, the researchers solve Fekete-Szego¨ functional problems for functions in$G_{\Sigma}(\lambda,\not{p},q,r,\not{g})and\mathcal{M}_{\Sigma}^{q}(\mu,\lambda,\rho,\not{p},q,r,\not{g})$ in the present work. In the form of corollaries, several exceptional and unique cases of the key theorems are illustrated.

5. REGULAR AND Bi-UNIVALENT FUNCTIONS AND APPLICATIONS

Recent RCS studies discuss the hidden body's response to electromagnetic. Cloaking of electromagnetic has gained interest in the scientific field, especially amongst scientists who are interested in materials-artificial composites which have properties of exotic electromagnetic. In the mathematical sense, in complex plane cloak of two dimensions and cloaked object can be considered as simple connected regions. There are equivalence between the above regions and conformal maps of the unit circle according to the theorem of Riemann Mapping. Suppose that cloaked object and cloak are respectively denoted by the functions $g(z)$ and q(z), then we obtain $g(z) \prec$ q(z).

For the cloak, it is better to be a three dimensional since the cloak relies on the cloaked body and the rays which are reflected by the body could be cloaked because of not consisting all reflected rays.

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Arabic Abstract

في هذا البحث، نُقدّم ونبحث فئات فرعية جديدة من الدوال ثنائية التكافؤ المرتبطة بمتعددات حدود هورادام. بالإضافة إلى ذلك، نجد تقديرات لأول معاملين للدوال في هذه الفئات الفرعية. كما حصلنا على متباينات فيكيتي-سيغو لهذه الفئات من الدوال. كما نُقدّم روابط وثيقة الصلة لنتائجنا بتلك التي نُوقشت في الأبحاث السابقة.

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On skew C_1C_2-Symmetric operators

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A B S T R A C T

Let C_1 and C_2 be conjugation operators, both of which are antilinear, isometric, and involution mappings, defined on a separable complex Hilbert space $\mathcal{H}$. This paper introduces the concept of skew C_1C_2-symmetric operators ($skew\ C_1C_2$-S.O). A bounded linear operator A on $\mathcal{H}$ is classified as a $skew\ C_1C_2$-S.O. if it satisfies the condition ($C_1A = -A^* C_2$), or equivalently, ($A = -C_1 A^* C_2$). We examine and analyze several fundamental properties of such operators and provide a concrete example to illustrate this notion.

1. INTRODUCTION

An algebra to all bounded linear operator specified on a separable complex Hilbert space $\mathcal{H}$ is represented by the notation B($\mathcal{H}$). A conjugation operation on $\mathcal{H}$ is antilinear operator $C: \mathcal{H} \to \mathcal{H}$ that fulfills for any $x, y \in \mathcal{H}$ and property of involution ($C^2 = 1$), and $\langle Cx, Cy\rangle = \langle x, y\rangle$. The research of complex symmetric operation was started in 2005 by [1]. According to their definition, an operator $A \in$ B($\mathcal{H}$) is considered C-symmetric if $CA = A^*C (A = CA^*C)$; it is complex symmetric; it is C-symmetric with regard to some C [1,2].

The idea of symmetric matrices in linear algebra are generalized by complex symmetric operation. Since for $x, y \in \mathcal{H}$, the matrix of C-symmetric operator A with regard to $\{e_n\}$ is symmetric. This because if C is a conjugation on $\mathcal{H}$, then there is an orthonormal basis $\{e_n\}$ of $\mathcal{H}$ in order to $Ce_n = e_n$ to all n [1]. The opposite is also true. In other words, A is complex symmetric if there is an orthonormal basis such that A has a symmetric matrix representation [1]. If there is a conjugation $C\ on\ \mathcal{H}$ in order to $CA = -A^*C (A = -CA^*C)$, then an operator $A \in$ B($\mathcal{H}$)is skew complex symmetric.

M. putinar, and W.Wogen, in different combinations, conducted a general investigation of such operators in [1-12]. The idea of a complex symmetric operation was expanded by Dakheel and Ahmed [13] in 2022. They defined a C_1C_2-S.O. as one in which an operator $A \in$ B($\mathcal{H}$) has certain conjugations C_1 and C_2 on $\mathcal{H}$ such that $C_1A = A^*C_2 (A = C_1A^*C_2)$.

The definition of skew complex symmetric operation is expanded in this work to be as follows: if $C_1A = -A^*C_2 (A = -C_1A^*C_2)$, then a bounded linear operator. We look at basic characteristics of these operators and arrive at the following conclusion: there are two orthonormal basis of $\mathcal{H}$ relative to that's A acknowledges a symmetric matrix illustration if A is a skew C_1C_2-S.O. . Additionally, we examine the matrix of skew C_1C_2-S.O. and demonstrate a few of its uses. Additionally, we looked at the skew C_1C_2-S.O. tensor product, direct sum, and tensor sum.

2. Main Results

This part deals with to introduce the concept of skew C_1C_2-S.O. , which serve as a generalization of skew complex symmetric operation. Furthermore, we examine fundamental properties of this concept and investigate its theoretical implications.

Definition 2.1 : put $\mathcal{H}$ be separable, complex Hilbert space and let C_1 and C_2 be conjugate linear operators acting on $\mathcal{H}$ ($C_1 \neq C_2$) that are both involution ($C_1^2 = C_2^2 = I$) and isometric ($\langle C_1x, C_1y\rangle = \langle y, x\rangle$ and $\langle C_2s, C_2k\rangle = \langle k, s\rangle$ to $all\ x, y, s$ and k in $\mathcal{H}$). A B.L.O. A on $\mathcal{H}$ is

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claimed to be C_1C_2-S.O. if met the condition C_1A = -A* C_2 equivalently expressed as (A = -C_1 A*C$_2$).

Example 2.2: Let $\mathcal{C}_1 = \begin{bmatrix} C_1 & 0 \\ 0 & C_2 \end{bmatrix}$ and $\mathcal{C}_2 = \begin{bmatrix} C_1 & 0 \\ 0 & -C_2 \end{bmatrix}$ are conjugations on $\mathcal{H} \oplus \mathcal{H}$ such that C_1, C_2 are conjugations operator on $\mathcal{H}$ and let $\mathcal{S}$ be skew complex symmetric operator such that $C_1\mathcal{S} = -\mathcal{S}^*C_1$. Then A = $\begin{bmatrix} \mathcal{S} & 0 \\ 0 & 0 \end{bmatrix}$ is skew C_1C_2-S.O. .

Remarks 2.3: For Conjugations C_1 and C_2, the following statements are holds:

1. Every skew complex symmetric operator is skew C_1C_2-S.O..
2. Put A is skew C_1C_2-S.O. , then A= -C_2A$^*C_1$.
3. Put A is skew C_1C_2-S.O. , then so is A*.
4. Put A is skew C_1C_2-S.O. , then so is A^{-1}.
5. The set of skew C_1C_2-S.O. is a subspace of B($\mathcal{H}$) for the same conjugations C_1 and C_2.

Proof:

1. Let A be a skew C_1C_2-S.O. such that C_1 A = -A*C$_2$. Assume that C_1 = C$_2$, hence A is skew complex symmetric operator.
2. Assume that A is skew C_1C_2-S.O. satisfying the condition A = - C_1 A*C$_2$ equivalently expressed as (-C_1 A C$_2$= A*), to demonstrate that A= - C$_2$ A$^*C_1$ we have:

   <-C$_2$ A* C_1w, z > = <C$_2z$, -C$_2$ C$_2$ A$^*C_1$ w > = < -A C$_2$ z, C_1 w >= < C_1C_1w, -C$_1$ A C$_2$ z > = < w, A* z > = <A w, z > , for all w, $z \in \mathcal{H}$.

3. Suppose that A skew C_1C_2-S.O. , such that C_1 A = -A*C$_2$ (A* = -C_1 AC$_2$).

   To show that C$_1$A* = -AC$_2$:

   < C_1 A* w, z > = < C_1 z, C_1 C_1 A* w > = < C_1z, A* w >= < C_1 z, -C_1 A C$_2$ w > = < -C_1 (C_1 A C$_2$ w), C_1 C_1 z > = < -AC$_2$ w, z > , for all w, $z \in \mathcal{H}$.

4. Let A = - C$_1$ A* C$_2$, to show that A^{-1} = -C$_1$ (A^{-1})*C$_2$:

   < -C_1 (A^{-1})*C$_2$ w, p > = < C_1 p, -C_1 C_1 (A^{-1})* C$_2$ w > = < C_1p, - (A^{-1})* C$_2$ w >= < -A^{-1} C_1 p, C$_2$ w > = < C$_2$ C$_2$ w, -C$_2$ A^{-1} C_1 p >= < w, -(C_1A C$_2$)$^{-1}$ p >= < w, (A*)$^{-1}$ p > = < w,- (-A^{-1})* p > = < A^{-1} w, p > , for all w, $p \in \mathcal{H}$.

5. The proof requires two steps:

i. if A$_1$, A$_2$ are skew C_1C_2-S.O. , then so is A$_1$+ A$_2$.

ii. if A skew C$_1$C$_2$-symmetric and $\mathscr{b} \in \mathbb{C}$, then $\mathscr{b}$ A is skew C$_1$C$_2$-symmetric.

For the first portion, observe that:

Since A$_1$, A$_2$ are skew C$_1$C$_2$-symmetric, it follows that C_1 A$_1$ = -A$_1^*$C$_2$ and C_1 A$_2$ = - A$_2^*$C$_2$.

To prove that A$_1$+A$_2$ is skew C_1C_2-S.O. , C_1 (A$_1$+A$_2$) = C_1 A$_1$ + C_1 A$_2$ = -A$_1^*$C$_2$ -A$_2^*$C$_2$ = - (A$_1$+A$_2$)* C$_2$. Thus (1) holds. For ii, since A skew C$_1$C$_2$-symmetric, it follows that C_1 A = -A*C$_2$ and $\mathscr{b} \in \mathbb{C}$.

To show that $\mathscr{b}$ A skew C_1 C$_2$-symmetric for the same conjugations C_1, C$_2$, C_1 ($\mathscr{b}$A) = $\overline{\mathscr{b}}$ C_1 A = - $\overline{\mathscr{b}}$ A*C$_2$ =- ($\mathscr{b}$A)* C$_2$.

The following proposition gives useful characterizations of skew C_1C_2-S.O. .

Proposition 2.4: take A in B($\mathcal{H}$) then an operator A in B($\mathcal{H}$) is skew C_1C_2-S.O. iff there are two orthonormal bases of $\mathcal{H}$ with that's A has symmetric matrix illustration.

Proof: Let A be skew C_1C_2-S.O. such that C_1 A= -A*C$_2$ and let $\{u_n\}$ and $\{v_n\}$ are two orthonormal bases such that C$_1u_n$= u_n and C$_2$ v_m = v_m for all $n, m \in \mathbb{N}$. The matrix of skew C_1C_2-S.O. A with respect to $\{u_n\}$ and $\{v_n\}$ is skew symmetric, to show that:

$$
\begin{aligned}
[A]_{ij} &= \langle Av_j, u_i \rangle \\
&= \langle -C_1 A^*C_2 v_j, u_i \rangle \\
&= \langle -C_1 A^* v_j, u_i \rangle \\
&= -\langle C_1 u_i, C_1 C_1 A^* v_j \rangle \\
&= -\langle C_1 u_i, A^* v_j \rangle \\
&= -\langle u_i, A^* v_j \rangle \\
&= -\langle Au_i, v_j \rangle \\
&= -[A]_{ji}, \text{ for } 1 \le i \le n, 1 \le j \le m.
\end{aligned}
$$

Conversely, let $\{u_n\}$ and $\{v_m\}$ be two orthonormal bases such that C$_1u_n$= u_n and C$_2$ σ_m = σ_m for all $n, m \in \mathbb{N}$. Define the conjugations C$_1$ and C$_2$ by C1($\sum_n a_n u_n$)= $\sum_n \overline{a_n} u_n$, C2($\sum_m c_m \sigma_m$)= $\sum_m \overline{c_m} \sigma_m$.

By hypothesis, the matrix ensures that $<A u_n, \sigma_m> = - <A\sigma_m, u_n>$ for all $n, m \in \mathbb{N}$, to show that A is skew C_1C_2-S.O. in order to $- C_1A^* C_2 = A$.

$$\begin{aligned} < - C_1 A^* C_2 \sigma_m, u_n> &= < - C_1 A^* \sigma_m, u_n> \\ &= < C_1 u_n, - C_1 C_1 A^* \sigma_m> \\ &= < C_1 u_n, - A^* \sigma_m> \\ &= < u_n, - A^* \sigma_m> \\ &= < -A u_n, \sigma_m > \\ &= - < A u_n, \sigma_m > \\ &= < A \sigma_m, u_n>. \end{aligned}$$

Proposition 2.5: If $\{A_n\}$ be a sequence of skew C_1C_2-S.O. with the same conjugations C_1 and C_2 in order to $\lim_{n\to\infty} ||A_n - A|| = 0$, then A is also skew C_1C_2-S.O..

Proof: Let $\{A_n\}$ be a sequence of skew C_1C_2-S.O. operator such that $C_1A_n = -A_n^* C_2$ ($A_n = - C_1A_n^*C_2$) for the same conjugations C_1 and C_2 with $\lim_{n\to\infty} ||A_n - A|| = 0$, we must prove that $A= - C_1A^* C_2$:

$$\begin{aligned} || -A- C_1A^* C_2|| &\le || -A - C_1A_n^* C_2|| + || C_1A_n^* C_2 - C_1A^* C_2 || \\ &\le || -A + A_n || + ||C_1|| \, || A_n^* - A^*|| \, ||C_2|| \end{aligned}$$

Since $||C_1|| = ||C_2||=1$, then

$$\begin{aligned} &\le || A_n - A|| + || A_n^* - A^* || \\ &\le || A_n - A|| + || A_n - A|| \\ &\le 2 \, || A_n - A|| \end{aligned}$$

Which tends to zero as $n\to \infty$. Hence skew C_1C_2-S.O..

The proper notion of equivalence for skew C_1C_2-S.O. is unitary equivalence as the following shows:

Proposition 2.6: If $A_1 \in B(\mathcal{H}_1)$ is skew C_1C_2-S.O. and $U: \mathcal{H}_1 \to \mathcal{H}_2$ is unitary operator, then there exists $A_2 \in B(\mathcal{H}_2)$ is skew C_3C_4-S.O. such that $A_2 = UA_1 U^*$, $C_3 = UC_1U^*$, $C_4 = UC_2U^*$.

Proof:

Since A_1 is skew C_1C_2-S.O. such that $C_1A= -A^*C_2$, then we have:

$$\begin{aligned} C_3A_2 &= (UC_1U^*)(UA_1U^*) \\ &= (U-A_1^*U^*)(UC_2U^*) \\ &= - (UA_1U^*)^*C_4 \\ &= - A_2^* C_4. \end{aligned}$$

Proposition 2.7: assume that the Cartesian decomposion of $A = \mathcal{X} + i\mathcal{Y}$ if and only if both X and Y are skew C_1C_2-S.O. with regard to identical conjugations C_1 and C_2, then A is skew C_1C_2-S.O.

Proof: Let A be skew C_1C_2-S.O. such that $C_1A = -A^*C_2$ with $A = \mathcal{X} + i\mathcal{Y}$ and $\mathcal{X} = \frac{1}{2}(A + A^*)$ and $\mathcal{Y} = \frac{1}{2i}(A - A^*)$.

To show that $\mathcal{X}$ and $\mathcal{Y}$ are skew C_1C_2-S.O. with the same conjugation C_1, C_2, then we have:

$$\begin{aligned} C_1 \mathcal{X} &= C_1 (\tfrac{1}{2}(A + A^*)) \\ &= \tfrac{1}{2}(C_1A + C_1A^*) \\ &= \tfrac{1}{2}(-A^*C_2 - AC_2) \\ &= - \tfrac{1}{2}(A^* + A) C_2 \\ &= - \mathcal{X}^* C_2. \end{aligned}$$

Similarly, we deduse that $\mathcal{Y}$ is also skew C_1C_2-S.O.. Conversely, since $\mathcal{X}$ and $\mathcal{Y}$ are skew C_1C_2-S.O. operators with respect to the same conjugations C_1 and C_2, then we obtain directly $C_1A = -A^*C_2$.

3.Tensor product and direct sum of skew C_1C_2-S.O.

This part deals with the necessary condition for the one rank operator on $\mathcal{H}$ to be skew complex C_1C_2-symmetric. Moreover, we discuss the tensor product and direct sum of skew complex C_1C_2-*S.O.*.

This part starts by the subsequent lemma [14]:

Lemma 3.1: Let C_1 and C_2 be a conjugations on $\mathcal{H}$ and x, y $\in \mathcal{H}$. Then $C_1(x\otimes y)C_2 = C_1x \otimes C_2y$ on $\mathcal{H}$.

The next proposition shows that when the finite rank operator becomes skew C_1C_2-S.O..

Proposition 3.2: If A is constant multiple of $-C_1x\otimes -C_2y$, then A is skew C_1C_2-S.O..

Proof

By previous lemma, we have $C_1(x\otimes y)C_2 = C_1x \otimes C_2y$ for conjugations operators $C_1$and C_2 on $\mathcal{H}$. Then we have:

$C_1AC_2 = C_1(-C_1x\otimes -C_2y)C_2 = - (y\otimes x) = -A^*$. Hence, A is skew C_1C_2-S.O..

Proposition 3.3: If A_1 is complex skew C_1C_2-S.O. on $\mathcal{H}_1$ and A_2 is skew C_3C_4-S.O. on $\mathcal{H}_2$ for some conjugations C_1, C_2, C_3 and C_4, then $A_1\otimes A_2$ is skew $(C_1\otimes C_3)(C_2 \otimes C_4)$-symmetric on $\mathcal{H}_1 \otimes \mathcal{H}_2$.

Proof: Since A_1 is skew C_1C_2-S.O. on $\mathcal{H}_1$ and A_2 is skew C_3C_4-S.O. on $\mathcal{H}_2$, then $C_1A_1 = - A_1^*C_2$ and $C_3A_2 = -A_2^*C_4$.

Now, to show that $A_1\otimes A_2$ is skew $(C_1\otimes C_3)(C_2 \otimes C_4)$-symmetric operator:

$$\begin{aligned} (C_1\otimes C_3)(A_1\otimes A_2) &= C_1A_1 \otimes C_3A_2 \\ &= - A_1^*C_2 \otimes -A_2^*C_4 \\ &= - (A_1^*\otimes A_2^*)(C_2\otimes C_4) \\ &= - (A_1 \otimes A_2)^* (C_2 \otimes C_4). \end{aligned}$$

Hence, we get what we want.

Proposition 3.4: If $\mathcal{M}$ is skew C_1C_2-S.O. on $\mathcal{H}_1$ and $\mathcal{B}$ is skew C_3C_4-S.O. on $\mathcal{H}_2$ for some conjugations C_1, C_2, C_3 and C_4 , then $\mathcal{M} \oplus \mathcal{B}$ is skew $(C_1\oplus C_3)(C_2\oplus C_4)$-symmetric on $\mathcal{H}_1 \oplus \mathcal{H}_2$.

Proof:

Let $\mathcal{M}$ be a skew C_1C_2-S.O. on $\mathcal{H}_1$ and $\mathcal{B}$ be a skew C_3C_4-S.O. on $\mathcal{H}_2$.

$(C_1 \oplus C_3)(\mathcal{M} \oplus \mathcal{B}) = C_1\mathcal{M} \oplus C_3\mathcal{B}$
$= -\mathcal{M}^* C_2 \oplus -\mathcal{B}^* C_4$
$= -(\mathcal{M}^* \oplus \mathcal{B}^*)(C_2 \oplus C_4)$
$= -(\mathcal{M} \oplus \mathcal{M})^*(C_2 \oplus C_4)$.

Proposition 3.5: If $\mathcal{M}$, $\mathcal{P}$ are a skew C_1C_2-S.O. on $\mathcal{H}$ and $\mathcal{E}_1$, $\mathcal{E}_2$ are skew C_3C_4-S.O. on $\mathcal{H}$, then $(\mathcal{M} \oplus \mathcal{P}) \otimes (\mathcal{E}_1 \oplus \mathcal{E}_2)$ is skew $(C_1 \otimes C_3)(C_2 \otimes C_4)$-symmetric operator on $\mathcal{H} \otimes \mathcal{H}$.

Proof:

$(C_1 \otimes C_3)[(\mathcal{M} \oplus \mathcal{P}) \otimes (\mathcal{E}_1 \oplus \mathcal{E}_2)] = (C_1 \otimes C_3)[\mathcal{M} \otimes \mathcal{E}_1 + \mathcal{P} \otimes \mathcal{E}_1 + \mathcal{M} \otimes \mathcal{E}_2 + \mathcal{P} \otimes \mathcal{E}_2]$
$= (C_1 \otimes C_3)(\mathcal{M} \otimes \mathcal{E}_1) + (C_1 \otimes C_3)(\mathcal{P} \otimes \mathcal{E}_1) + (C_1 \otimes C_3)(\mathcal{M} \otimes \mathcal{E}_2) + (C_1 \otimes C_3)\mathcal{P} \otimes \mathcal{E}_2)$
$= (C_1\mathcal{M} \otimes C_3\mathcal{E}_1) + (C_1\mathcal{P} \otimes C_3\mathcal{E}_1) + (C_1\mathcal{M} \otimes C_3\mathcal{E}_2) + (C_1\mathcal{P} \otimes C_3\mathcal{E}_2)$
$= (-\mathcal{M}^* C_2 \otimes \mathcal{E}_1^* C_4) + (-\mathcal{P}^* C_2 \otimes -\mathcal{E}_1^* C_4) + (-\mathcal{M}^* C_2 \otimes -\mathcal{E}_2^* C_4) + (-\mathcal{P}^* C_2 \otimes -\mathcal{E}_2^* C_4)$
$= (-(\mathcal{M}^* \otimes \mathcal{E}_1^*)(C_2 \otimes C_4)) + (-(\mathcal{P}^* \otimes \mathcal{E}_1^*)(C_2 \otimes C_4)) + (-(\mathcal{M}^* \otimes \mathcal{E}_2^*)(C_2 \otimes C_4)) + (-(\mathcal{P}^* \otimes \mathcal{E}_2^*)(C_2 \otimes C_4))$
$= -((\mathcal{M}^* \otimes \mathcal{E}_1^*) + (\mathcal{P}^* \otimes \mathcal{E}_1^*) + (\mathcal{M}^* \otimes \mathcal{E}_2^*) + (\mathcal{P}^* \otimes \mathcal{E}_2^*))(C_2 \otimes C_4)$
$= -((\mathcal{M}^* \oplus \mathcal{P}^*) \otimes (\mathcal{E}_1^* \oplus \mathcal{E}_2^*))(C_2 \otimes C_4)$
$= -((\mathcal{M} \oplus \mathcal{P}) \otimes (\mathcal{E}_1 \oplus \mathcal{E}_2))^*(C_2 \otimes C_4)$.

Proposition 3.6: If $\mathcal{M}_1$ and $\mathcal{M}_2$ are skew C_1C_2-S.O. on $\mathcal{H}$ with $C_1 \otimes C_2 = C_2 \otimes C_1$, then $\mathcal{M}_2 \boxplus \mathcal{M}_2$ is a skew $(C_1 \otimes C_2)(C_2 \otimes C_2)$-symmetric operator on $\mathcal{H} \otimes \mathcal{H}$.

Proof:

$(C_1 \otimes C_2)(\mathcal{M}_1 \boxplus \mathcal{M}_2)^*(C_2 \otimes C_2) = (C_1 \otimes C_2)(\mathcal{M}_1^* \otimes I + I \otimes \mathcal{M}_2^*)(C_2 \otimes C_2)$
$= [(C_1 \otimes C_2)(\mathcal{M}_1^* \otimes I) + (C_1 \otimes C_2)(I \otimes \mathcal{M}_2^*)](C_2 \otimes C_2)$
$= ((C_1 \otimes C_2)(\mathcal{M}_1^* \otimes I) + (C_2 \otimes C_1)(I \otimes \mathcal{M}_2^*))(C_2 \otimes C_2)$
$= (C_1\mathcal{M}_1^* \otimes C_2 + C_2 \otimes C_1\mathcal{M}_2^*)(C_2 \otimes C_2)$
$= (C_1\mathcal{M}_1^* \otimes C_2)(C_2 \otimes C_2) + (C_2 \otimes C_1\mathcal{M}_2^*)(C_2 \otimes C_2)$
$= C_1\mathcal{M}_1^* C_2 \otimes I + I \otimes C_1\mathcal{M}_2^* C_2$
$= -\mathcal{M}_1 \otimes I + I \otimes -\mathcal{M}_2$
$= -(\mathcal{M}_2 \boxplus \mathcal{M}_2)$.

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Arabic Abstract

لتكن C_1 و C_2 مؤثرات مترافقة معرفة على فضاء هلبرت العقدي القابل للفصل $\mathcal{H}$. في هذا البحث، قدمنا مفهوم المؤثر المتناظر من النمط skew C_1C_2 على أنهُ : المؤثر المُقيد الخطي المُعرف على فضاء هلبرت العقدي القابل للفصل أذا تحقق ($A=- C_1 A^*C_2$) $C_1A=-A^*C_2$. أيضاً، تمت دراسة و مناقشة العديد من الخواص و أيضاً أعطاء بعض الأمثلة التي تخص هذا النوع من المؤثرات.

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