

# Artificial Immune System Implementation for Predicting WM Presence from MYD88 and CXCR4

Nichenametla Rajesh<sup>1</sup>, Naresh Vurukonda<sup>2</sup>

Department of Computer Science and Engineering, Koneru Lakshmaiah Education Foundation, Vaddeswaram, Guntur, Andhra Pradesh, 522502, India <sup>1,2</sup>

Email-ID: nrajeshcse@kluniversity.in, naresh.vurukonda@kluniversity.in

## Abstract

Waldenstrom's Macroglobulinemia (WM) is a rare malignancy that affects human blood cells and spreads slowly. The development of WM occurs whenever the blood cells undergo genetic changes. Better therapies can be offered by the healthcare sector to get rid of the symptoms that cannot be cured. Everyone in the healthcare sector is aware that genetic abnormalities cause WM, but they are unsure of what causes the alterations. The risk factors that increase the number of WM's aberrant cells have been found. The greatest risk variables have a fatal impact on humans. The healthcare sector is working to save lives by offering better care. Only when WM is discovered earlier when it is treatable with better care and potent medications, is it very likely. For analysing the healthcare data associated with WM, a number of prior research studies have suggested both standard and unique software models and techniques. However, the accuracy is subpar and inefficient in terms of both time and money. To analyse the genomic dataset and detect Waldenstrom's Macroglobulinemia or its symptoms, this research explored this issue and suggested an Artificial Immune System (AIS) approach. Software written in Python is used to conduct the experiment and validate the findings. by contrasting the trial outcomes with other performance assessment techniques. The analysis reveals that the suggested AIS algorithm works better than the others.

**Keywords:** Artificial Immune System, Waldenstrom Macroglobulinemia, Blood Cancer, Data Analysis.

## I. Introduction

Human Immune System (HIS) which is complex in nature has the inherent ability to learn about pathogens that enter it and fight infection. This complex nature of HIS has been a source of inspiration for problem-solving in various domains, computer science, and engineering in particular. A majority of research works carried out these days using AI and molecular biology, revolve around the concept of genome mapping to cater to the needs of microbiologists. As these applications generate a large amount of DNA/RNA/mRNA sequences there is an urgent need for automatic computational models for increasing the speed and accuracy of the sequence in analysis. The task of identifying abnormal B-cells (WM affected) is a complex one for the reasons that follow, such as irregular coding, difficulty in recognizing and translating the sequences, redundancy, and complexity in understanding the patterns.

WM is a type of cancer that affects the B-Cells in the human body. It can be identified either using medical image diagnosis or genomic data analysis methods. This researcher in his previous research works has dealt with image processing methods for identifying the presence of WM but the accuracy was not so accurate and satisfactory. Since the presence of WM is better identified biologically,

the researcher has shifted to the genomic data analysis method for identifying WM cancer.

Most of the earlier research works published in the National Library of Medicine (NLB) have discussed clinical methods for identifying cancer that have made it difficult for candidates from non-clinical domains to comprehend. Though a major part of the research works proposed medical image processing methods for detecting and identifying WM cancer, very few had suggested biologically inspired algorithms for analyzing human genomic data. One of the popular and potentially effective biological-inspired algorithms is AIS. So, Artificial Immune System (AIS) algorithm has been used for analyzing the genomic data of WM patients. Consequently, the abnormal regions are identified in the human genome microarray data. The AIS algorithm mainly focuses on analyzing gene symbol, chromosomal location, GB-ACC, and cytoband for determining the presence of cancer. This paper contributes towards

- Preparing the data in a manner where the proposed algorithm can learn each entity.
- Implementing the AIS algorithm and experimenting with the prepared genomic data.

- Detecting WM cancer in terms of accuracy and comparing it with the other state-of-the-art methods to evaluate the performance.

Before implementing the AIS algorithms, a detailed literature survey is carried out to understand the issues and challenges present in WM detection.

## II. Literature Review

This section presents a detailed literature survey on the methods adopted so far in detecting and classifying WM cancer using image processing and genomic data analysis methods. For example, the Author in [1] explained the human immune system is a basis for data analysis methods. It helps to create an artificial immune system model to solve biological problems in terms of representing, comparing, and visualizing genomic datasets. The authors in [2] attempted to utilize the advanced merits of the deep learning algorithms for analyzing lymphoma and cancer of lymph nodes. The experimental results obtained 97.33% of accuracy in analyzing small sized lymphoma dataset. For a large size dataset, the author in [3] proposed an R-CNN algorithm for diagnosing lymph nodes. The R-CNN algorithm is faster than other algorithms and analyses the data region-wise using convolution neural networks. The training accuracy is compared with the testing accuracy.

Three different immune-based algorithms, such as negative selection, clonal selection, and immune network are used for analyzing the DNA/RNA/mRNA sequences and recognizing the normal and abnormal patterns [11]. These algorithms were compared with the AIS, and ANN for pattern recognition. The core functionalities of the computation like mechanisms used in the computation, basic components, etc. For example in CNN, MLP [12], associative memories [13], and SON [14] are used for pattern recognition. The neural networks are classified based on the basic units like artificial neurons, storage patterns, learning methods, and the architectures used for weight calculation, etc are used to increase the strength of the network [15].

From the statement, it has been identified that it is essential to choose the appropriate algorithm for pattern recognition according to the dataset. Thus, this paper is motivated to implement the AIS algorithm for genomic data analysis.

### Immune system

The Human Immune System (HIS) is made up of a large number of cells and substances that combine in various methods to identify and get rid of hazardous organisms (pathogens). HIS cells have receptors on their interfaces,

and several of them chemically attach to pathogens. Others attach to certain HIS cells or substances to simplify the complicated structure of signaling that facilitates the immune reaction. As a result, these relationships are localized since they rely on chemical coupling. The majority of HIS cells travel throughout the body through the lymphatic and blood streams, providing a dynamic system of scattered observation and reaction in the absence of systematic organization and centralized management. Pathogen identification and eradication are the results of billions of cells functioning according to small-scale, localized criteria. As a result, the HIS is durable to both the threats on the HIS and the malfunctioning of specific elements. The difficulty in identifying “self (elements of the body)” from “non-self (Pathogens)” is frequently used to illustrate the difficulties of diagnosing diseases. Many pathogens are not hazardous, and the body could be harmed by the act of HIS to get rid of them. In these situations, it would be healthy to remain dormant, making it more appropriate to state that the HIS struggles with differentiating between damaging non-self and everything else present in the body. We are firm in the notion that “self” refers to non-toxic components like all physiologically healthy body cells, while “nonself” refers to any pathogen that damages the body.

Once infections are discovered, the HIS must take some action to get rid of them. The units of the HIS that carry out this elimination of various pathogens in various methods are referred to as effectors. The HIS 's challenge with elimination is to select the appropriate effectors for the specific pathogen to be eradicated. Appropriate effectors need to be chosen for eradicating a particular type of pathogen and IS usually faces certain issues while choosing the correct effectors. In the absence of a healthy immune system, an artificial immune system must be built to protect against numerous diseases and pathogens. Artificial immune systems (AIS) are dynamic systems that use empirical immune processes, theories, and modeling as well as conceptual immunology to resolve issues.

### Artificial Immune System (IAS)

Artificial immune systems (AIS) are a family of rule-based, cognitively smart machine learning systems that draw inspiration from the functions and theories of the mammalian immune system. For application in problem-solving, the techniques are frequently based on the training and storage capabilities of the immune system. This section outlines the design of our Artificial Immune System (AIS). Since AIS is closely patterned after the biological immune system, we will also detail the corresponding biological systems that serve as the model's inspiration. The

immunologic data must be brief and incomplete; see [1] for a thorough introduction of immunity that is indeed understandable to non-immunologists.

### Problem Definition

In the IS, covalent linkages that develop among protein molecules serve as the foundation for all distinctions into self and non-self. To maintain flexibility, protein links are modeled as binary threads of a specified size ( $l$ ). The IS must use proteins to identify self against non-self; AIS solves a related issue, which we characterize as below. The totality,  $U$ , which is made up of all threads of length  $l$  is divided into two disjoint subgroups that we refer to as self,  $S$ , and nonself,  $N$ . It can be represented as  $U=S \cup N$  and  $S \cap N$ . A detection or classification operation lays AIS and it needs to determine whether a random thread from  $U$  is normal (related to self) or abnormal (related to nonself).

A false positive happens if a self-thread is identified as abnormal, and a false negative happens if a non-self-thread is labelled as normal. These are two different types of classification errors that AIS could generate. Comparable mistakes are made by the HIS as well. A false negative error happens if the HIS is unable to identify and eliminate infections, and a false positive error happens if the HIS commences an assault on the body (a process called an auto-immune reaction). Both types of errors are hazardous to the body, thus the HIS has developed to reduce them; likewise, AIS seeks to reduce both types of errors. Figure 1 is a 2D illustration of a thread environment. Figure-1 shows how the immune identification mechanism classifies threads as either normal (related to self) or abnormal (related to non-self) to capture the border within the two groups.

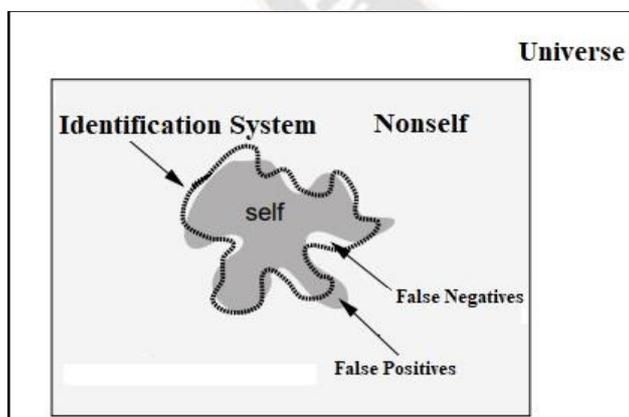


Figure-1. Thread Environment

Self and non-self could not be distinct if real-world issues are transferred to this concept since some threads may characterize both self and non-self. The classification of such threads as either of the type may cause inevitable errors

under this circumstance. This case is not one we take into account. It highlights the need of selecting the appropriate feature for the application area. It is crucial to select proteins that could be utilized to accurately distinguish the self and non-self.

### DETECTORS

Numerous types of cells and substances that have been found and scientifically examined make up innate immune systems. By adopting a single fundamental kind of detector that is based on the lymphocyte type of immunological cells, we can optimize our approach. This detector incorporates characteristics of antibodies, T-cells, and B-cells. In that, it is made up of a large number of portable detectors that move throughout a dispersed area and AIS is equivalent to the HIS. We use a graph  $G = (V; E)$  to represent the dispersed surroundings; every vertex ( $v \in V$ ) has a small cluster of detectors (referred to as a detection unit), and detectors move from one node to the subsequent along the edges. The graph design also introduces the concept of location. At the identical node, detectors can communicate with each other. This location concept is helpful.

Since lymphocytes have millions of similar receptors on their membrane, they are referred to as monoclonal. Such receptors attach to areas on pathogens termed epitopes. Because adhesion is based on chemical composition plus charges, receptors are expected to attach to a small number of epitopes of a similar type. The sensitivity among the receptor or epitope increases with the probability of binding formation. In AIS, chemical coupling among epitopes or receptors is treated as a roughly thread match. All epitopes and receptors are modelled as binary threads of constant size  $l$ . Every detector has a binary thread that correlates to its receptors.

Hamming length or edit length are two viable rough matching criteria, however, we had chosen the more immunologically appealing  $r$ -contiguous bits criterion [2]. If two threads share  $r$ -contiguous bits, they are considered to be equal and are shown in Figure-2. Indicating the dimension of the subgroup of threads that a unique detector may match, the number  $r$  serves as a cut-off and establishes the specificity of the detector. For example, if the value of  $r$  equals  $l$ , the match is entirely accurate and the detector will exactly match one string (itself). When  $r$  equals 0, the matching is completely generic and the detector matches with every single thread with length  $l$ .

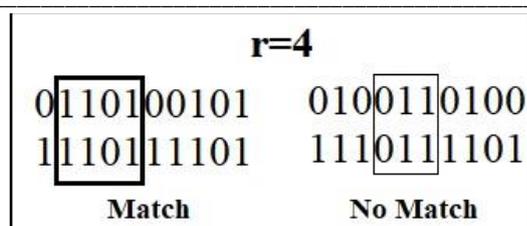


Figure-2. Matching Using Contiguous Match Rule

In the above figure, the matching is found for the value of  $r$  equal to 3 instead of 4. Due to the trade-off between the number of detectors utilized and the specificity, the incomplete matching rules with cutoff, such as  $r$ -contiguous bits, have this effect. The number of detectors needed to get a specific degree of detection grows as the specificity of the detectors rises. The ideal  $r$  is the one that provides better classification while requiring the fewest possible detectors.

If lymphocytic receptors attach to epitopes, a lymphocyte is stimulated. The process of activation alters the lymphocytic state and stimulates the sequence of responses. These responses may create the removal of pathogens. The stimulation of a lymphocyte will be achieved if the quantity of the receptors attached to the epitopes is more than the cutoff. A lymphocyte must attach enough receptors in a short time frame since the chemical interactions among receptors as well as epitopes are not long-lasting. This could be designed with activation cutoffs where at least  $T$  strings should match over a specific period. The detector is allowed to consolidate matches and the time decay should match over a short duration of time. This design provides the probability of attachment between the receptor and the epitope. If the detector is stimulated, the count of the match is reset to zero.

### The B cell model

The human DNA pattern specification, a variety of intermediate expressions (such as the nuclear RNA and the mRNA), a genome library, and the antibody are all available in the B cell. Additionally, it keeps track of the B cell's degree of stimulation and its connections with both sister cells and daughter cells in addition to the parent B cells that may be present.

There are two approaches to making a novel antibody. The initial technique aims to replicate the gene transcription, folding, and translation processes that take place in the B cells of the body's immune system. The B cell is given access to a genome library, and the gene selecting procedures are launched. From matured mRNA, the antibody's paratope is produced. To accomplish the above step, the mRNA thread is replicated in a similar manner (for

instance  $G \rightarrow C$  and  $C \rightarrow G$ ,  $T \rightarrow A$  and  $A \rightarrow T$ ) (We are aware that this is not a true representation of mRNA translating, which requires identifying the appropriate amino acids to integrate into proteins with the nucleotide triplet sequence).

The second technique employs an antigen's string definition as the model for the mRNA thread. Then, symmetrically, this is replicated as earlier. If the immune system component produces fresh B cells after each cycle, the first approach is applied. When no B cell would attach the present antigen, the second approach is employed to initialize the B cell population.

### The Immune network

The present set of B cells that have been created as well as the connections between such B cells are actively maintained in the immune system memories, which are organized like a network. Figure-3 demonstrates a B cell network memory case. A freshly generated B cell is put next to such B cells for which it has an affinity in immune-based storage. The two B cells with which the fresh B cell exhibits the strongest affinity are then identified, which is how the B cell gets incorporated into the network. It is then connected to such two B cells as well as any additional B cells connected to them. This eventually allows the formation of B cell-containing areas inside the network that can handle related issues. Some bridges connect those areas, indicating shared features between various issue areas.

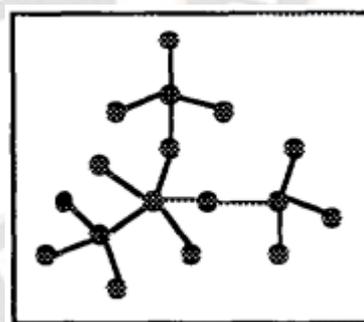


Figure-3. Interpretation Of An Immune Network

### An Antibody Model

The antibody has a paratope in the AIS that serves as a design for the layout it will employ to bind to the antigen. The depiction we have selected makes use of the letters A, T, G, C, and the wildcard X. It was discovered that this wild card, which complements all nucleotides, is essential. For the below antigens, for instance, we would seek to discover a typical antibody:

TATAATGCCGTATA  
 TATAATCGGCTATA  
 TATAATGATCTATA

We can create a B cell containing the wild card as the antibody (Given below):

TATAATXXXXTATA

We could not create a B cell that could attach all three in the absence of it. This is crucial because our ability to distinguish whether new DNA segments are promoter positives or negatives depends on our ability to find shared characteristics across the antigens.

### An Antigen Model

We employ the DNA segments to instruct the AIS (Towell, Shavlik & Noordewier 1990). Those variants have a length of 57 nucleotides. A few are promoter-negative, whereas others have recognized Escherichia coli (prokaryotic) gene promoters. The nucleotides in the promoter, which span from 50 nucleotides forward of the protein-coding area to 7 nucleotides within the protein-coding area, are coordinated so that the translation beginning location is at the end of the segment. Figure-4 in the section below offers a few examples.

threshold. The B cell will compute its level of stimulation after calculating the binding intensity. Here, every action is discussed in detail. Every component between the antigen and the antibody that correlates in a complementing way is counted by the matching algorithm. Additionally, continuous match locations are given more importance in the matching algorithm. If there are 4 matching components in a continuous area, the particular location would receive a score of 2 to the power of 4. (i.e. 2<sup>4</sup>).

Figure-5 shows what happens when an antibody and an antigen are "matched." The figure illustrates that there are 12 matching components. Moreover, every match location's value (for instance, the six components that match at the pattern's top) should include this number. As a result, the example's ultimate match score equals 98.

Antigen	c	g	c	t	t	g	c	g	t	t	c	g	g	t	g	
Antibody	g	c	x	x	a	c	a	c	a	c	g	c	t	a	c	
Evaluation	2	2	1	1	2	2	0	2	2	0	2	2	0	2	2	
Length							6					2				
Match value											22+2 <sup>6</sup> +2 <sup>2</sup> +2 <sup>2</sup> +2 <sup>2</sup> => 98					

Figure-5. Computing A Match Value

A measure of the effective bonding between two molecules is called the binding value, which is generated using the match score. An antibody should attach to an antigen for the bonding to be durable, which means that the match score should be higher than a specific level before the initiation of bonding. The AIS itself establishes this threshold. It accomplishes it by changing the threshold's value in relation to the present mean binding value by a factor of 0.5, i.e., by either raising or lowering the threshold by the difference between the two thresholds. This strategy is a modification of the matching method employed by Hightower, Forrest, and Perelson (1993), where we have expanded to add a wild card "X" for justification. But it was crucial that we would not simply produce a batch of B cells where its antibodies were all wild cards (those antibodies may attach to whatever theoretically). As a result, we assigned a score of 1 to a "wild card match" as opposed to a score of 2 for a "complete match," which seemed to function effectively.

### B-Cell Simulation Modeling

According to the immunological network hypothesis, a B cell's level of stimulation depends on how strongly its antibody attaches to both the antigen and adjacent B cells. In AIS, an antigen (Complementary to B cells) and B cells (Similar to it) stimulate the B cell. Also, B cells are suppressed by certain other B cells (Complementary to it). The stimulation intensity is determined using the method from (Farmer, Packard, & Perelson 1986). The resultant

+, S10,	tactagcaatcacgcttgcgcttcggttgtaagtatgtataatgcgcggttgcgt
+, AMPC,	tgctatcctgacagttgtcacgctgattggtgctgtacaatctaacgcacgcgcaa
+, AROH,	gtactagagaactagtgcattagcttattttttgttatcatgctaaccaccggcg
+, DEOP2,	aattgtgatgtgtatcgaagtgtgttcggagtagatgtagaataactaacaactc
- , 1218,	ttcgtctccgcgactacagatgagatgctgagtgcttccgcttactggattgtcacca
- , 668,	catgtcagcctcgacaacttgataaatgcttcttctgtagacgtgcctacgcgctt
- , 413,	aggaggaactacgcaaggttggaacatcggagagatgccaccgacgcacctgcacg
- , 991,	tctcaacaagattaaccgacagatcaactcctggtgagtgagcttcaacttgagga

Figure-4. Some Examples Of Promoters With Positive And Negative DNA Segments

52 of the 53 positive samples comprised the antigen population. A, G, C, and T are thus the components of every antigen (Wild card is absent). Our AIS uses a very basic antigen model. An antigen component stands for every potential antigen. This component has a unique epitope, or antigen-representing thread, that antibodies will try to bind. The antigen population component loads the antigens onto the AIS after they have been specified in an independent ASCII file. This component creates antigen instances from a collection of lists that it receives from a data file.

### The Behavior Of The Artificial Immune System

#### Antibody/Antigen Association

A match score is generated by determining how strongly the antibody and antigen coincide during the immune reaction. The antibody could attach the antigen when the match score is greater than a predetermined

stimulation intensity must be greater than a specific threshold for the B cell to produce copies of itself. Those clones activate a hypermutation operator and so they are introduced to the immunological network.

### Somatic Hypermutation Modeling

To apply somatic hypermutation to a newly formed B cell, we adopt the method described by Farmer, Packard, and Perelson (1986). The three kinds of mutation used by the AIS are simple substitution, substring regeneration, and multi-point mutation. The specific mutation that is used is selected at random.

Every component of the antibody is synthesized individually at a time during multi-point mutation. The component is mutated when an arbitrarily produced value exceeds the mutation threshold. This indicates that to substitute the actual component, a value of A, T, G, C, or X is created at random. Two locations in the paratope of the antibody are randomly chosen for substring regeneration. Subsequently, any of the constants A, T, G, C, or X, which are selected at random, is substituted for every component within those two locations. The simple substitution function chooses a different B cell to utilize as a resource of fresh components for the present B cell using the roulette wheel algorithm (Goldberg 1989). The operator then chooses a paratope from the initial antibody or an antibody from the recovered B cell. The novel “mutated” B cells belong to the immunological system when they could attach to the antigen available or when an affinity could be identified for them anywhere in the system, regardless of the mutation operator used.

### Diversity Maintenance

Every time the primary algorithm iterates, the lowermost 5% of the B cell number is eliminated. Equivalent numbers of new B cells are taken by the immune system to replace them. As a result, a network is formed where the population of B cells is constantly changing in terms of both quantity and kind. This promotes the heterogeneity of the system’s B cells. The overall functionalities of the AIS algorithm are given in the form of an algorithm and flowchart to understand in a better manner and will be implemented in any computer programming language to experiment and verify the performance.

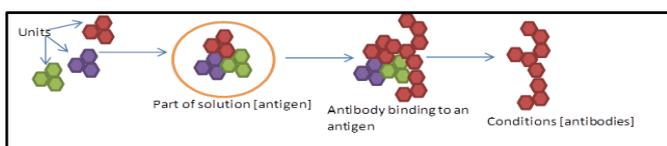


Figure-6. Building Blocks, Partial Solutions And Conditions Mapped To The AIS Metaphor

### Artificial Immune System

If a population of responses is important, whether throughout the search or as a result, AISs are important. The issue must also involve some kind of "matching" concept. Because AISs are fundamentally evolving algorithms, they are more suited for recurring optimizations as compared to one-off issues that shift over time and have to be addressed repeatedly. To execute the AIS, four basic considerations must be made: the indoctrination design, the likeness measurement, the choice, and the modification. Once the model has been formed, locate the likeness measure, then select and modify the best option [10].

### Algorithm of AIS

- The AIS operation is typically described as sequential procedures:
- Defining N as the total number of sequences and S denote the string.
- K is defined as the size of the sequence taken.
- The target pattern (TP) is designated as SK.
- SK is mapped over K using the pattern matching technique.
- If SK is found in the string Si, then i will be updated in the target array.
- If not SK is not found in the string Si, we need to clone the string using reverse mutation and inverse position mutation techniques.
- The above-mentioned procedures are repeated till the length of the string and length of K.

### Pseudo Code for AIS

- 1 Let N be the total number of sequences for every individual
- 2 Let S be the string
- 3 Designate the Target pattern (TP) as SK
- 4 Map SK over K (pattern matching technique)
- 5 If (SK= Si)
- 6     Update i in the target array
- 7 If (SK≠ Si)
- 8     Clone the string (Reverse Mutation and Inverse Position Mutation techniques)
- 9 Repeat 4

The algorithm provides a step-by-step overview of how to solve an issue. To create a computer program in any programming language and arrive at the result, pseudo-code

is utilized. The schematic representation of the algorithm is shown in Figure-2. The flowchart shows the general operation of AIS.

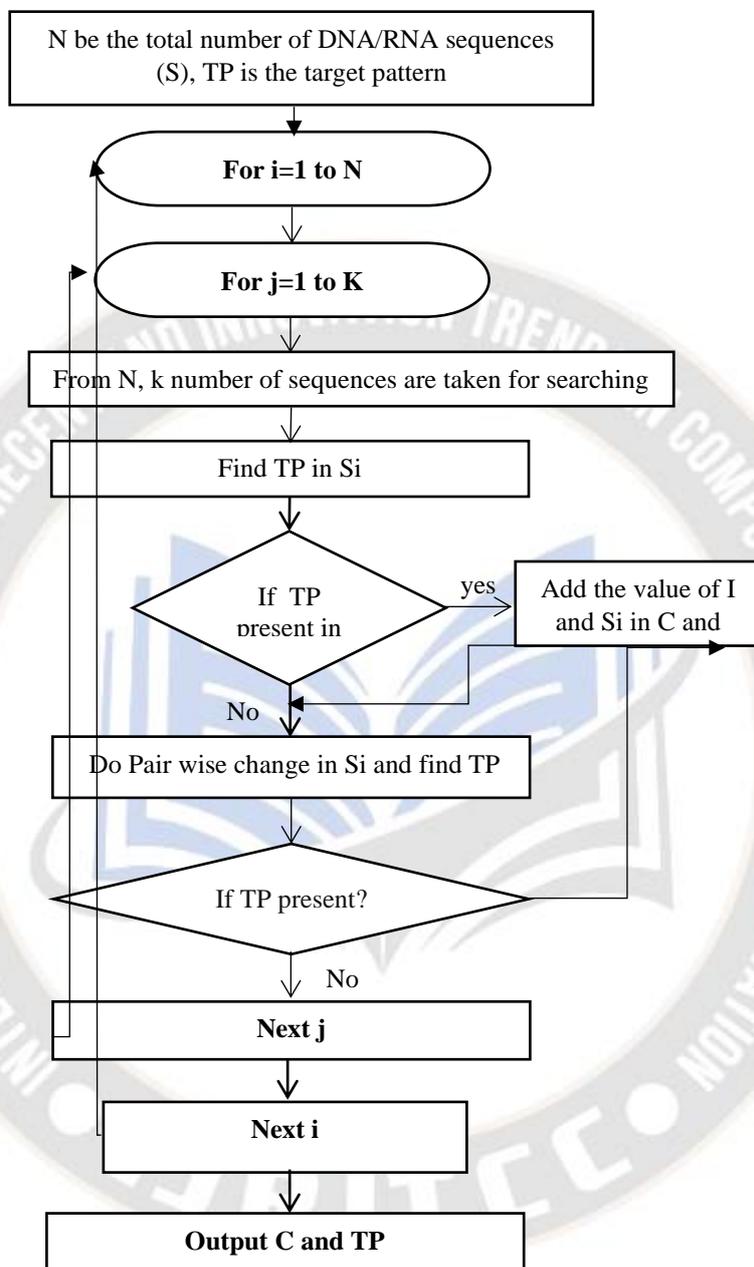


Figure-7. AIS Flowchart

### III. Experimental setup

The experimental setup consists of a laptop with an i7-7<sup>th</sup> generation processor, 8GB ram, 1TB storage, and 1650GPU. The experiment is run in the latest version of python with the required packages to run the MAS5.0 in the system. The open source dataset is loaded from the 1TB storage which is a solid-state drive. The following dataset is used to create an automatic immune system by analyzing the

medical data of the patients. The Genetic data of the patients are analyzed and real-time monitoring is done to check the variations in the gene to come up with remedies automatically. For comparing the proposed method the obtained dataset consists of the results calculated with the MAS5.0 method. It is considered a benchmark and the proposed method is compared with it. The sample taken for comparison is MC1336\_GEP from the GSM318148. The details of the patient corresponding to this sample data are



indicates a corresponding class of the sequence (see Figure-8). For simple understanding, there are only two different classes are obtained from the entire dataset, such as normal and WM-abnormal.

Table-1. Normal Vs. Abnormal Number of Data

# of sequences	Normal	WM-Abnormal
4000	2696	1211
8000	3775	4060
12000	5410	6366
16000	7086	8606
20000	8564	11031
22283	9170	12656

Table-1 shows the number of normal and WM predicted IDs from the sample. On the total 22283 IDs, the total WM affected IDs are 12656 and the normal IDs are 9170. This is the result obtained through the MAS5.0 method. It is provided by default in the dataset. We can see the number of IDs that are positive and negative with a consistent increase in the IDs.

Table-2. Accuracy of the AIS Pattern Matching Algorithm

No of ID	Normal Predicted	WM predicted	Accuracy
4000	2498	1122	92.6278
8000	3573	3843	94.6608
12000	5067	5962	93.6654
16000	6581	7992	92.8765
20000	8003	10309	93.4563
22283	8616	11892	93.9675

Table-2 shows the predictions made by the proposed algorithm and the accuracy of the prediction. The accuracy is calculated by comparing it with the predictions given in the algorithm. The results obtained to provide more than 90% accurate predictions. The provided results can be utilized to create an artificial immune system.

Table-3. Time Consumed By The Proposed Method

No of ID	Time (ms)
4000	3425
8000	7653
12000	10987
16000	14532
20000	17689
22283	20382

Table-3 gives the time consumed by the proposed algorithm to process and give the results. The time is given in ms. when the algorithm is compared with similar methods like

MAS5.0 and RCA the proposed algorithm is very much faster in computing the IDs.

Table-4. Confusion Matrix

Class	Positive	Negative
True	11892	8616
False	763	553

Table-4 shows the confusion matrix for the proposed algorithm. The wrong predictions are relatively lesser than the correct predictions. It is also comparable to the previous methods. Through this confusion matrix, the accuracy, sensitivity, and specificity of the algorithm are calculated.

Table-5. Performance Comparison

Methods	Accuracy	Sensitivity	Specificity
MAS5.0	99.05	0.98	0.94
Proposed AIS	93.96	0.95	0.91

Table-5 shows the performance evaluation by comparing the sensitivity, specificity, and accuracy obtained using various earlier methods and the proposed AIS method. The accuracy of the benchmark is 99.05%, whereas the proposed AIS obtained 93.96%. The obtained accuracy is closer to the benchmark and hence is considered a better method for small-size DNA data analysis.

#### IV. Conclusion

This paper proposed a biologically inspired natural algorithm for analyzing genomic data for identifying and detecting WM cancer through B-cell immune system. A learning methodology is used for analyzing the taxonomy of the immune system for creating a classifier for DNA/RNA sequences. The AIS is one of the best learning models which can learn the HIS thoroughly and predict the sequences having WM cancer. It is done by comparing the cancer pattern in the DNA sequences using AIS and outputting the DNA sequences and a number of sequences having the cancer pattern. Since the AIS can evolve the antibodies, determining the emerging patterns is easy and fast. AIS performs like a neural network and it provides 93.96% of accuracy in classifying the abnormal sequences from the dataset.

#### V. Future Work

The AIS algorithm provided high accuracy only for a limited amount of data. But the dataset that needs to be analyzed is crowdsourced. Thus, it is necessary to analyze the data using a deep learning model.

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