



## NONLINEAR CA BASED ASSOCIATIVE MEMORY MODEL FOR BONE CANCER PREDICTION

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### Abstract

Cancer prediction is a real time dynamic problem existing in the real world. The cause and reason for drastic spread of this disease is very difficult to understand. We have various forms and types of cancers, an abstract model to predict cancer is difficult. Although many papers are available to trace cancer, there is still room for evolving a new strategy for predicting cancer. We propose a novel Non Linear CA based associative memory which learns from various case studies analyzing the data and predicts the Bone Cancer. We have taken datasets from ICCR Datasets and processed them using Hybrid Unsupervised learning algorithm. Preliminary work was done and we have compared our work with some standard existing literature. The proposed classifier performance was found promising.

**Keywords:** *Cellular Automata, NLCA, Cancer*

## 1. INTRODUCTION

Cancer growth is an expansive term. It portrays the ailment that outcomes when cell changes cause the uncontrolled development and division of cells. A few kinds of malignant growth cause fast cell development, while others cause cells to develop and partition at a slower rate. Certain types of disease bring about unmistakable developments called tumors, while others, for example, leukemia, don't. A large portion of the body's cells have explicit capacities and fixed life expectancies. While it might seem like a terrible thing, cell passing is a piece of a characteristic and advantageous marvel called apoptosis. A cell gets directions to bite the dust so the body can supplant it with a more up to date cell that capacities better. Dangerous cells come up short on the parts that educate them to quit isolating and to pass on.

Therefore, they develop in the body, utilizing oxygen and supplements that would as a rule support different cell. Carcinogenic cells can shape tumors, hinder the invulnerable framework and cause different changes that keep the body from working consistently. Destructive cells may show up in one zone, at that point spread by means of the lymph hubs. These are groups of invulnerable cells situated all through the body.

## 2. LITERATURE SURVEY

We have done an extensive literature survey on the applicability of CA [6], [7], [8], [9] on cancer. We have also studied the standard literature for finding the reasons for cancer and the symptoms [1], [2], [3], [4], [5].

## 3. IMPLEMENTATION OF NON LINEAR CA BASED ASSOCIATIVE MEMORY

Algorithm 1

Input: Training Sequence  $S = \{ S_1, S_2, \dots, S_l \}$  /\* Antigens\*/

Maximum Number of Generations ( $MN_g$ )/\* Problem Specific\*/

Multiplying factor for cloning ( $\beta$ ) /\* Value between [0-1] \*/

Population size (nbpop)/\* Antibodies Population\*/

Threshold /\* Diversity of a node calculation \*/

Out Put: Transition Matrix (T), Complementary Matrix (U) and Class(C)

begin

Step 1: Generate nbpop antibodies for initial population (INP).

Step 2: Initialize the generation counter  $G_c = 0$ ,  $PPP = INP$ . ( Present

Population=Initial Population)

Step 3: Initialize the antibodies  $Ab_m$  with random population.

Step 4: Construct a set of antigen population  $Ag$ .

Step 5: Select an antigen  $Ag_j$  from  $Ag$  and compute the fitness of each

antigen  $Ag_j$  with the population in  $Ab_m$ .

Step 6a: Calculate the diversity at each node. If the diversity of the

node  $>$  threshold, stop splitting. If more number of

elements belong to a class then assign that class label to the

node .

Step 7: Increment the generation counter  $G_c$ .

Step 8: If  $G_c > MN_g$  go to step15.

Step 9: Select the m best antibodies.

Step 10: Apply Cloning as per equation

Step 11: Apply mutation as per equation

Step 12: Calculate the fitness of the newly formed rules.

Step 13:  $PP \leftarrow NP$  (New population is placed in present population)

Step 14: Store the antibody, values of F, U and corresponding class information for which fitness is more than 1.5.

Step 15: Sort the antibodies in descending order. Store top antibodies in  $Ab_m$ .

Step 16: Place the rest of antibodies in a set A. Randomly generate antibodies to create diversity call it as B. Compare the antibodies in set A, B and  $Ab_r$ . Place the best rules in  $Ab_r$  (Reserved Pool).

Step 17: For every  $G_c$  compare the antibodies in  $Ab_m$  and  $Ab_r$  and place the best in  $Ab_m$ .

Step 18: Stop.

Algorithm 1 shows the efficient implementation of AIS-MACA. The minimum number of generations  $MN_g$  required for predicting the class with the desired accuracy depends on the given problem (PCR & PR). The minimum number of generations that are required for prediction of PCR with maximum accuracy is 65. For AIS-PRMACA it is 70 and for IN-AIS-MACA it is 80. The population size of antibodies (nbpop) is fixed as 200. Multiplying factor for cloning ( $\beta$ ), is chosen as 0.5 for AIS-MACA, 0.6 for AIS-PRMACA and 0.5 for IN-AIS-MACA. The analysis on the parameters  $MN_g$ ,  $\beta$  and the number of fuzzy states chosen for addressing these problems are discussed.

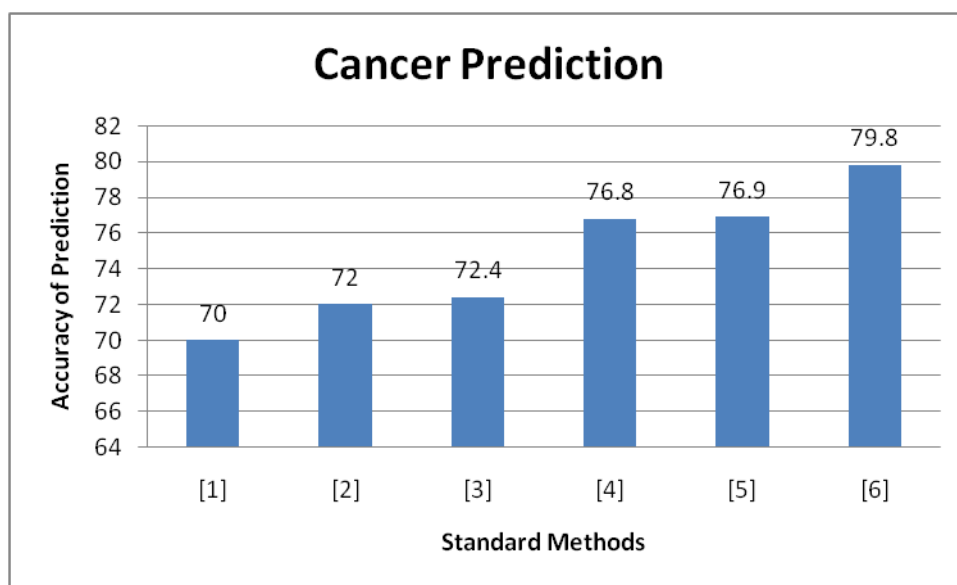


Fig 1: Comparison with Existing approaches

The inputs for our work is taken from ICCR Datasets [11] and extensive implementation on the trained and tested datasets are done. It is compared with the existing literature and found an accuracy of 79.8 for our method.

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