

HYBRID NON LINEAR CA FOR BONE CANCER PREDICTION

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ABSTRACT

Cancer prediction is a difficult problem to predict in the real world. The reason and purpose behind extraordinary spread of this malady is exceptionally hard to get it. We have different structures and sorts of tumors, a unique model to foresee malignancy is troublesome. Albeit numerous papers are accessible to follow malignant growth, there is still space for advancing another procedure for anticipating disease. We propose a novel Hybrid Non Linear CA based acquainted memory which gains from different contextual investigations breaking down the information and predicts the Bone Cancer. We have taken datasets from ICCR Datasets and handled them utilizing Hybrid Unsupervised learning calculation. Fundamental work was done and we have contrasted our work and some standard existing writing. The proposed classifier execution was discovered promising.

KEYWORDS: Cellular Automata, NLCA, Cancer.

INTRODUCTION

Disease development is a far reaching term. It depicts the disease that results when cell changes cause the uncontrolled improvement and division of cells. A couple of sorts of threatening development cause quick cell improvement, while others cause cells to create and parcel at an all the more moderate rate. Specific sorts of illness achieve unquestionable improvements called tumors, while others, for instance, leukemia, don't. An enormous segment of the body's cells have express limits and fixed futures. While it may appear to be an awful thing, cell passing is a bit of a trademark and invaluable wonder called apoptosis. A cell gets bearings to fail miserably so the body can supersede it with a more state-of-the-art cell that limits better. Perilous cells miss the mark on the parts that instruct them to stop disconnecting and to pass on.

In this way, they create in the body, using oxygen and enhancements that would govern in doubt bolster various cells. Cancer-causing cells can shape tumors, ruin the insusceptible structure and cause various changes that shield the body from working reliably. Damaging cells may appear in one zone, by then spread by methods for the lymph center points. These are gatherings of insusceptible cells arranged all through the body.

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].

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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 (β) /* 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 step 15.

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: $PP < NP$ (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
(β), 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 , β and the number of
fuzzy states chosen for addressing these
problems are discussed.

RESULTS AND COMPARISON

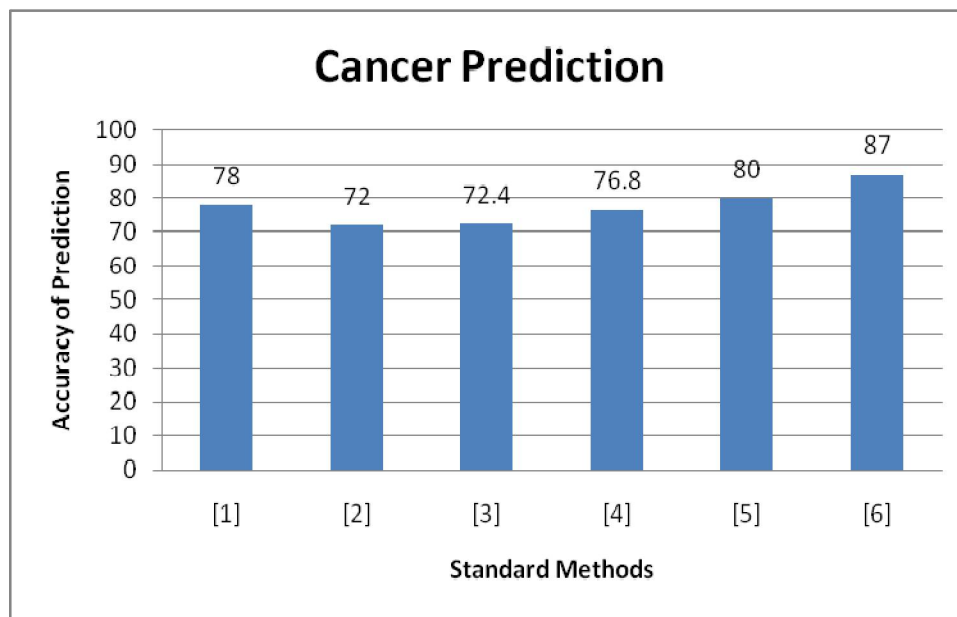


Figure 1. Comparison with Existing approaches

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 87% for our method.

CONCLUSION

We have developed a dynamic classifier augmented with CA to predict traces of cancer. Hybrid non linear CA was used to detect the traces of cancer. The classifier is tested under various inputs from various sources including the cited source. The results are now getting benched marked and future extension could be adaptability of these methods to various other forms of cancers.

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