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Population Dynamics of Microorganisms by Means of Cellular Automatas

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Abstract | **Original Research Article**

In this project, the growth of a glioblastoma multiforme (GMB) was modeled by means of cellular automata, with the intention of corroborating whether with the conditions given in our model the growth of said tumor adheres to the Gompertz model, which is a model tested for the growth of small but very fast growing tumors. The results obtained by our cellular automata model closely adhere to the Gompertzian model, so we conclude that the model used by us to analyze the dynamic system that represents tumor growth is correct. The model has rules for evolution in discrete time steps, with which we can determine the required time in which the tumor acquires a size considered fatal in various medical studies. Our model was compared with others, resulting in ours being a better approximation to the Gompertz curve, with which we were able to conclude that our analysis and methodology are correct.

Keywords: Component; formatting; style; styling; insert.

I. INTRODUCTION

In this project, the growth of a cancerous tumor of the glioblastoma type, also known as glioblastoma multiforme (GMB), which is a very aggressive tumor that is generated either in the brain or in the spinal cord, was modeled. Glioblastoma can occur at any age, however it is more common in older adults. GMB is very difficult to treat and often to cure, so treatments focus on slowing down its evolution and reducing signs and symptoms.

With this work we intend to analyze its development in a way that is very close to reality once the tumor has been diagnosed, with the intention that it can be used for possible treatment techniques once the way in which it develops has been analyzed.

Mathematics is a discipline capable of expressing the creative power of the human being. That is why they currently play an important role in relating to other disciplines, this in order to achieve significant progress by combining one or more disciplines with mathematics. For this, mathematics has two important aspects known in popular dominoes as: Pure Mathematics and Applied Mathematics [1].

When mentioning the words pure Mathematics we refer to their most abstract aspect, it is where we find the art of said discipline, in which axioms, theorems, relationships, etc. can be found.

However, we cannot limit mathematics to its most abstract part, since there is a part of it that is of vital importance and without which it would be impossible to understand the behavior of most physical phenomena. That is why it is of vital importance that when studying any physical phenomenon, a good dose of mathematics be included in its repertoire of tools for the study of it [1].

In this work we will focus on the union of mathematics with other disciplines such as Biology and computer science. The union of them are the disciplines known as: computational biology and bioinformatics.

When we hear the terms Bioinformatics and Computational Biology it is clear that informatics and computation are implemented for both disciplines. However, these disciplines are applied to a greater extent in a more than in another Bioinformatics is a discipline that is more related to the biological field, in which computer technologies are used basically for calculation processes in the area of biology. However, computational biology not only uses computer technologies for calculation processes, but also branches of these such as programming, and statistical processes applied to a biological problem in order to achieve a better understanding of it.

Computational Biology deals with the development of algorithms and the use of computers to facilitate the knowledge of biology, and it can be considered an interdisciplinary subject in which subjects such as biology, computer science, chemistry, medicine, biochemistry, mathematics, systems engineering, physics or statistics come together.

Computational Biology is a more theoretical and much more science-based discipline. in the use of Mathematics and Statistics, and I believe that he understands to a greater degree the biological settings and processes. That is why Computational Biology is a very important part of this thesis project, since the very name of the thesis mentions that we will use cellular automata (mathematical models of which we will delve into later), in order to simulate and understand the population dynamics of certain microorganisms in order to better understand their population growth [2].

II. BACKGROUND

Many of the great advances in the medical area are a consequence of threats against people's health. Currently we find ourselves with new diseases that are increasingly dangerous and those that already exist have become more resistant to the treatments used or to the drugs used [3,4].

Resistance to multiple substances is a public health problem that has been observed worldwide after the appearance of antibiotics. The indiscriminate use of antibiotics and the environmental selective pressure exerted by antiseptics and disinfectants have generated a survival response in microorganisms, which enables them to efficiently evade the bactericidal action of some agents. At present, attempts are being made to determine whether there are shared mechanisms between antibiotics, antiseptics and disinfectants that allow bacteria and other microorganisms to activate genes that potentially express the five mechanisms proposed up to now as an evolutionary response to human intervention [4].

Much of the enormous scientific development in this field of study is thanks to the incursion of mathematics and its conjunction with other sciences such as computing. Together the two generate a powerful tool for the study and a better understanding of these diseases. Through computational modeling, in its different forms, either through simulations using discrete methods or using differential equations, and cellular automata, it has been possible to emulate in a way that is very close to reality the way in which tumors grow [5,6].

III. OBJECTIVES

Evaluate the growth of the population of microorganisms pathogenic to humans under ideal reproduction conditions, by means of cellular automata in different dimensions and under different border conditions.

1. Specific objectives

- Modeling epidemics and population dynamics through cellular automata.
- Apply the properties of the ACs, under different boundary conditions and different types of neighbors.
- Devise a CA model to model the dynamics of a population under ideal breeding conditions.
- Analyze the population growth model.

2. METHODOLOGY

To carry out this thesis project, a series of steps must be carried out that are listed below:

1. Analysis of the literature

The state of the art of the problem is analyzed and the bases for the realization of the project are established. Based on the information collected, the general and particular objectives of the project are set.

2. Problem Statement

The idea of the investigation is formally structured and refined.

3. Feasibility and verification of the model

It is used to make decisions regarding the evaluation of the project.

It is formulated with information that has the least possible uncertainty in order to measure the chances of success or failure of the research project.

4. Model tests.

With this practice, you will have the certainty that the most important requirements for carrying out the research project are included in the test.
The pseudo code that will be used in the programming of the model is established.

5. Model programming.

The model is programmed using the pseudo code created during model testing. It is in this step where the data and specifications of the previous steps are combined to obtain the objectives established in the research project.

6. Analysis of results.

The results obtained by the model are compared with the models of the state of the art, making sure that the expected objectives for the research project have been achieved.

IV. RESULTS

We are going to present the results that we obtained with our model, the improvements that we made to it, referring to this, the differences that we made in comparison with various studies already done on which we focused for the realization of this project.

3. Changes and improvements.

For the realization of this project there were different changes, however among the most important is the change of geometry in the cells that represent our AC. In previous studies, a simple geometry is used, to put it in some way, they are square cells, with which it cannot have a maximum of eight neighbors if the Moore neighborhood is taken. In our model, the maximum number of neighbors can vary in each run, since we generate our cells randomly, seeding a given number of cells in a finite space, even after that number of cells are randomly placed in our space, we use the Voronoi tiling to define its boundaries in terms of the other cells.

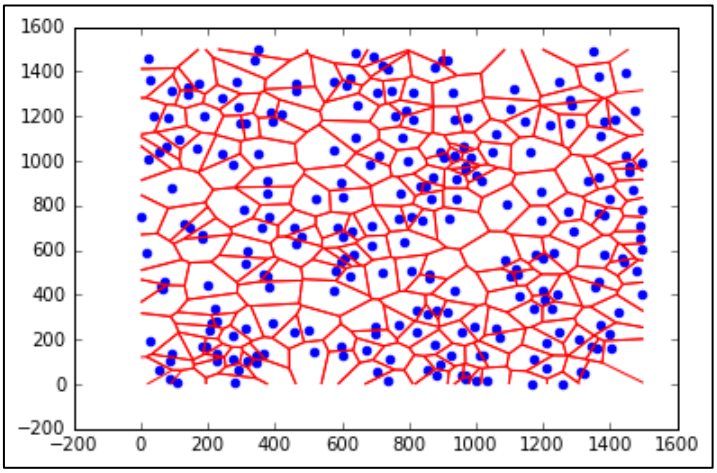

Figure 1: Configuration of our AC with 250 random cells

That is why our cells are left with a polygonal geometry, a sample of a run of our model gave us the following configuration of our CA shown in figure 1. Figure 2 shows another example with a greater number of cells in our space, in order to observe that the greater our number of cells, the number of possible neighbors between them increases. The example shown in figure 2 is some of the configurations that were run for the development of this thesis process.

Once we change the type of geometry of the cells of our automaton, we focus on the generation of states and, in turn, the transition rules between them. In our automaton we established three types of states for each cell:

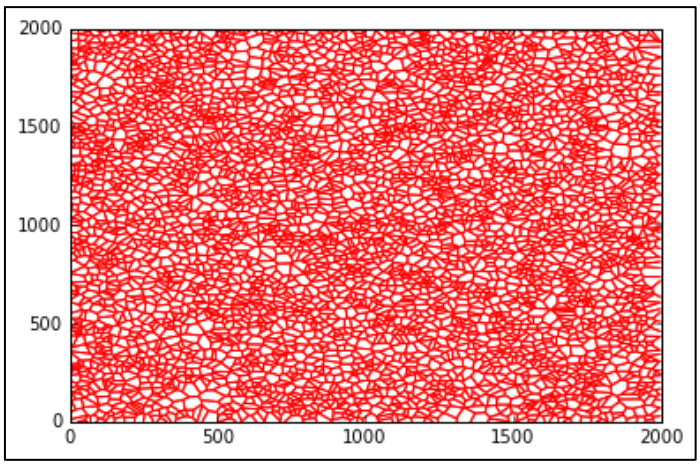

Fig. 2: Initial configuration of our AC with 4500 cells located randomly in space of 1500 X 1500

Automaton rules

- State 0: This state represents healthy cells in our model.
- State 1: This state represents cells that are infected and capable of proliferating.
- State 2: This state represents necrotic cells and already unable to continue infecting other cells.

To achieve the transition between the three different states of our cells, they are carried out based on four main parameters. These four parameters are:

- Rt: represents the tumor radius that each of the infected cells capable of proliferating possesses, this parameter is introduced arbitrarily in our model. However, this remains constant during all the iterations of time spent in our model.

- r: refers to the position of each infected cell relative to the center of our given space.
- p0: represents a probability of infection which is introduced by us into our model.
- p1: represents a probability, which changes in each iteration following the equation 1.

$$p_1 = p_0\left(1 - \frac{r}{R_t}\right) \quad (1)$$

Once we know these parameters, we can follow the rules by which the states of our cells in the AC change. The rules we use in our model are the following:

- The healthy cells represented with state 0, change to state 1 or become infected as long as the distance to any infected cell is less than Rt and in turn the probability p1 is less than or equal to some value that we will give before starting the run.
- Infected cells represented with state 1 cease to be proliferative or cells represented with state 2 when all their neighbors are already proliferating cells and it is where these cells become necrotic cells represented with state 2.
- The results we obtained with our model will be presented below.

We ran our code with the following values:

- Cell number in our AC was 4500.
- The size of the space where we sowed our cells was 1500X1500.
- The tumor radius used Rt = 29.
- A probability p0 = 0.75.
- Number of iterations t = 200.
- Number of iterations t = 200.

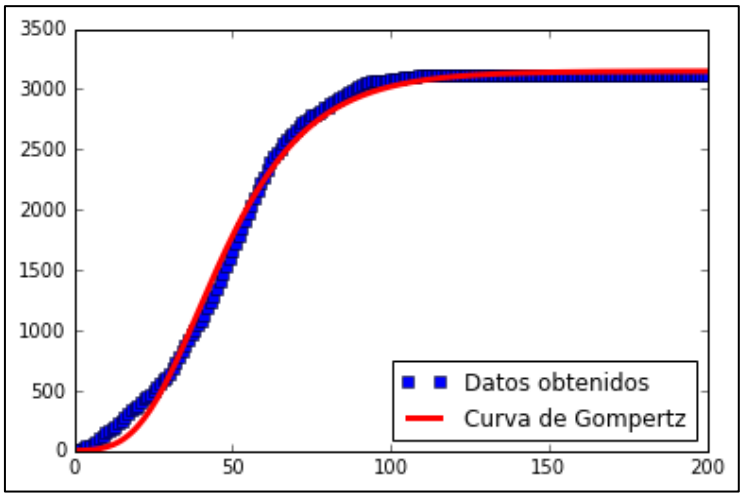

Fig. 3: Results obtained with Rt = 29, p0 = 0.75, for proliferating cells

With these input values we obtained the graphs shown in figure 3, in which it shows us the values obtained from proliferative cells during the 200-time steps and compares this in turn with the Gompertz curve. This in order to demonstrate that our model adheres to the Gompertz model. With the same values, the graph obtained for the necrotic cells is shown. In the graph shown in figure 4, it can be seen that our results adhere to the Gompertz model in the same way. What can be clearly observed in both graphs is that our model effectively matches the Gompertz model, which is one of the models best adapted to tumor growth.

Comparison

As mentioned before, our model was based on the article "Fateme Pourhasanzade, Seyed Hojjat Sabzpoushan, Ali Mohammad Alizadeh, and Ebrahim Esmati. A stochastic cellular automata model of tumorimmune interaction. Computational Research Progress in Applied Science Engineering ", that is why in this section we will contrast the results they obtained with ours. What is observed with the naked eye, if we compare the graphs shown in figure 5, it is clear that our model adheres in a better way to the Gompertz model, which, as previously mentioned, adheres in a very similar way to what was observed in documented clinical cases. Figure 5.a of figure 5 shows the results they obtained with their model in terms of the number of infected and necrotic cells. In the article they mention that their model adheres to Gompertz, however, if we look at the curves in both red and blue in figure 5a in figure 5, both curves are clearly not they adhere very well to the Gompertz model. However, the results that our model yielded are shown in figures 5b and 5c of figure 5, where figure 5b shows us our results in terms of to infected cells, like figure 5a which are our results obtained in terms of necrotic cells, the comparison of our results is observed in comparison with the Gompertz model, and it is clear that practically our results they stick in a better way than what was obtained by the Iranian model.

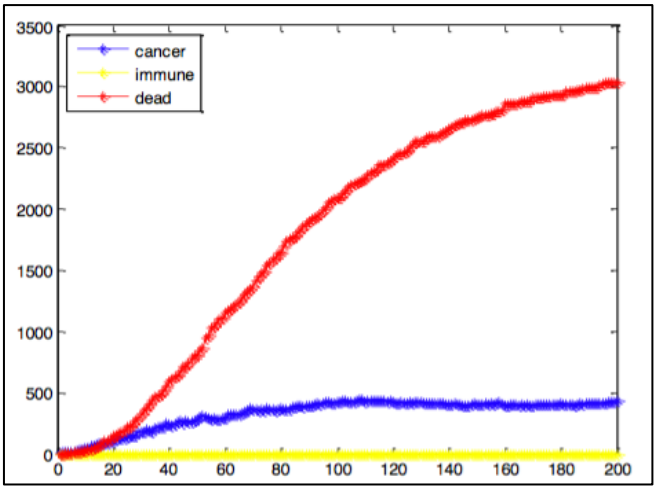

Fig. (a): Article Model

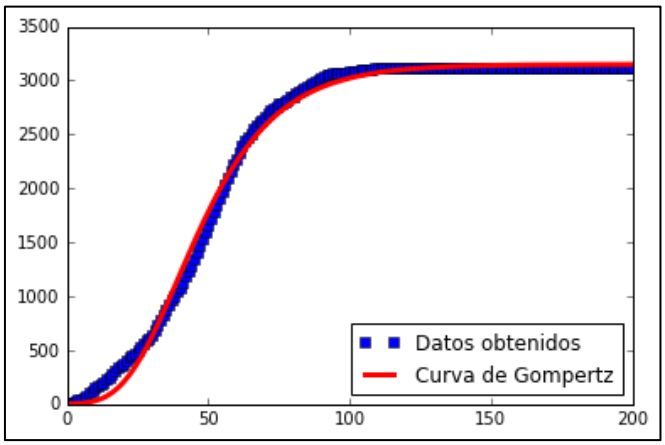

Fig. (b): Results of our model: infected cells

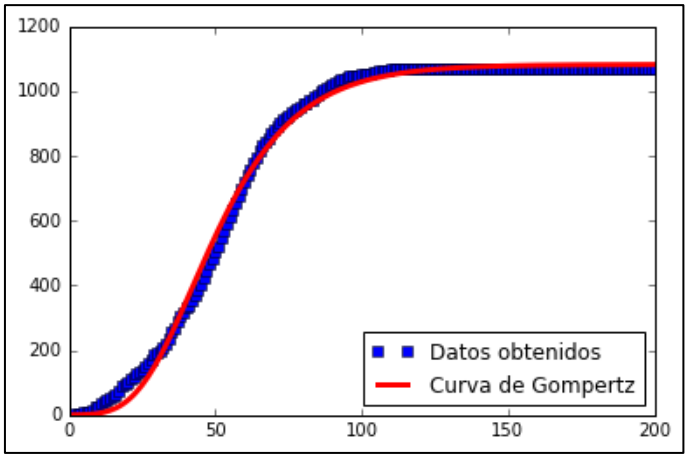

Fig. (c): Results of our model: necrotic

Cells Fig. 5. In this comparison it is clearly observed that our obtained results shown in 5b and 5c fit better than the model obtained in the article on which we based 5a.

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