Scholar Publishing Group
International Journal of Multimedia Computing
https://doi.org/10.38007/IJMC.2022.030204
ISSN 2789-7168 Vol. 3, Issue 2: 23-28

Computer Simulation Modification and Simulation of the Mechanism of Immunosuppressive Tumors

Tran Anh Tuan and Emanuela Guido

University of Helsinki, Finland

Keywords: Body Immunity, Computer Simulation of Tumors, Tumor, Tumor Simulation

Abstract: In recent years, with the rapid development of medical imaging, computer and hemodynamics theory and technology, it has become a hotspot to use computer simulation technology to simulate the mechanism of immunosuppressive tumors. The simulation results tend to be real tumors of human body, and play an important role in revealing the mechanism of the occurrence and evolution of immunosuppressive tumors. Effect. In this paper, a computer simulation model for the growth of tumors in normal cell tissues is established by studying the mechanism of immunosuppressive effect on tumors.The simulation design of computer simulation of tumors is proposed.

1. Introduction

Objectively evaluate computer simulation technology, which realizes the close imitation of the attributes of things, that is to say, only achieve the relative simulation [1-3]. In addition, the use of computer simulation technology will be limited to a certain extent [4-5]. When using simulation technology, under certain target requirements, peel off a certain characteristic of the analysis object, and then build a model according to the analysis situation. After inputting data and simulation process, the results are obtained. Finally, through comparison and judgment, we can know whether the established model is in line with the actual situation [6-9]. Using the results of the simulation process, the model should be adjusted to ensure that the established model meets the needs of use [10-13] in the process of continuous simulation, analysis and adjustment. The validation model should be able to reflect a certain characteristic of the simulated object, to conform to the relationship between each component of the system, and to conform to the output of the system as a whole [14]. The combination of computer technology and simulation technology achieves the effective play of their functions. To meet the application requirements of simulation technology.

2. Morphological Structure of Tumors

2.1. Tumor Morphology

The gross morphology of tumors is varied, and it can reflect the benign and malignant of tumors

to a certain extent. Tumors are generally classified into benign and malignant tumors. The number and size of tumors vary. More than one, sometimes more than one. The size of tumors is related to the nature (benign, malignant), growth time and location of tumors. Tumors growing on the body surface or in larger cavities can sometimes grow very large, while tumors growing in closed narrow cavities are generally smaller. Those with large tumors usually grow slowly and are mostly benign. Malignant tumors grow rapidly and can cause adverse consequences in a short time, so they often grow little. The shapes of tumors are various, and the differences in shapes are closely related to the location of tumors, the origin of tissues, the way of growth and the benign and malignant tumors. Generally, the cut surface of tumors is gray-white or grey-red, depending on the amount of blood, whether there is bleeding, degeneration, necrosis, etc. Some tumors show different colors because they contain pigments. Therefore, we can infer what kind of tumors are based on the color of the tumors. It is related to the types of tumors, the ratio of the substance of tumors to stroma, degeneration and necrosis. Tumors with more stroma than parenchyma are generally softer; on the contrary, tumors with more stroma than parenchyma are generally harder. Tumor tissue is soft when necrosis occurs, but hard when calcification or ossification occurs. Lipoma is soft and osteoma is hard.

2.2. Histological Structure of Tumors

Tumors have various histological structures, but all tumors can be divided into parenchymal and interstitial components. The essence of tumors. Tumor parenchyma is the general name of tumor cells and the main component of tumors. It determines the biological characteristics of tumors and the specificity of each type of tumors. Usually, the histological origin of various tumors is identified according to their substantial morphology, and the classification, nomenclature and histological diagnosis of tumors are carried out. The benign and malignant degree of tumors and the malignant degree of tumors are determined according to their differentiation maturity and size of atypia; the stroma of tumors. The stromal components of tumors are not specific and play a supporting and nutritional role in the essence of tumors. Generally, it consists of connective tissue and blood vessels, and the stroma sometimes has lymphatic vessels. Tumors that usually grow faster tend to have richer interstitial blood vessels and fewer connective tissue; tumors that grow slowly tend to have fewer interstitial blood vessels. In addition, tumors are often infiltrated by monocytes such as lymphocytes, which is the body's immune response to tumors. In addition, fibroblasts and myofibroblasts can also be seen in the connective tissue of tumors. Myofibroblasts have the dual characteristics of fibroblasts and smooth muscle cells. These cells can produce collagen fibers and have contractile function, which may limit the infiltration of tumor cells. The proliferation of myofibroblasts can explain the retraction of breast cancer papillae, the stiffness and stricture of intestinal tract caused by esophageal and intestinal cancers.

2.3 atypical Tumors

Tumor tissues differ in cell morphology and histological structure from the normal tissues from which they originated. This difference is called heteromorphism. Atypia is the morphological manifestation of abnormal differentiation of tumors. Small heteromorphism indicates high differentiation and large heteromorphism, indicating low differentiation. Differentiating the size of this atypia is the main histological basis for the diagnosis of tumors and for the determination of benign and malignant tumors. The atypia of benign tumour cells is not obvious, and they are generally similar to their origin tissues. Malignant tumors often have obvious atypia. Malignant tumors composed of undifferentiated cells are also called anaplastic tumors. Anaplasia refers to the lack of differentiation and marked atypia of malignant tumors. Anaplastic tumors have obvious

pleomorphism, and the size and shape of tumor cells vary greatly from one another, so it is often impossible to determine the origin of their tissues. Anaplastic tumors are usually highly malignant.

3. The Mechanism of Immunosuppressive Tumors

3.1. Tumor Immune Escape

Tumor immune escape refers to the phenomenon that tumor cells can survive and proliferate in vivo by evading the recognition and attack of immune system through various mechanisms. The body's immune system has the function of immune surveillance. When malignant cells appear in the body, the immune system can recognize and specifically remove these "non-self" cells through the immune mechanism to resist the occurrence and development of tumors. However, malignant cells can escape immune surveillance by various mechanisms in some cases, and rapidly proliferate in vivo to form tumors. That is to say, on the one hand, the body can resist the occurrence of tumors through natural and acquired immunity; on the other hand, cancer cells can escape the recognition and attack of immunity through a variety of mechanisms. The occurrence and prognosis of tumors depend on the overall role of these two aspects.

3.2. Role of Immunosuppressive Cells in Anti-Tumor Immune Response

Macrophages. Macrophages can be classified into classically activated macrophages and alternatively activated macrophages according to their phenotypes and functions. Substituted activated macrophages are abundant in tumor tissues, also known as tumor-associated macrophages (TAM s). TAM can prevent T cells from attacking tumor cells, secrete growth factors to nourish tumor cells, promote angiogenesis of tumor tissues, and further promote the metastasis and proliferation of tumor cells.

3.3. Tumor Immune Escape Mechanism

Low immunogenicity. Some tumors do not have proteins that are not present in normal cells by MHC molecules, so they are normal for the immune system. Others have lost one or more MHC molecules, and most of them do not express the costimulatory proteins needed to activate naive T cell maturation; they are recognized as autoantigens. Tumor antigens present without costimulatory signals make T cells that respond to them tolerate the antigen; antigen modulation. The antigens initially expressed in tumors can be recognized and attacked by the immune system. However, there are antibody-induced antigen internalization and mutation of antigen itself in tumors, which lead to the decrease or even disappearance of these antigens. It is now believed that genetic instability in cancer cells results in a balance period of antigen reduction (which should refer to the slow or even stagnant growth of tumors inhibited by the immune system). When the immune system fails to fight against tumors, the immune system may no longer be able to eradicate tumors, leading to the growth of tumors. When a tumor is attacked by lymphocytes that respond to a specific antigen, any tumor cell that does not express the antigen will have the advantage of selectivity; tumor-induced immunosuppression. Tumors usually produce immunosuppressive molecules, such as TGFbeta, IL-10, IDO or PD-L1, which can directly inhibit the immune response, or recruit regulatory T cells capable of secreting immunosuppressive cytokines by themselves; tumors induce immune regions. Tumor cells can secrete a variety of molecules, such as collagen, forming a physical barrier around the tumor, preventing lymphocytes and APC antigen presenting cells from entering the tumor area.

4. Computer Tumor Simulation

4.1. Principle and Model of Numerical Simulation

The commonly used methods for numerical simulation of human hemodynamics are finite difference method, finite volume method and finite element method, among which FEA is more commonly used. This method is proposed by a scholar. Its main idea is to divide the large computational area into several small units, and take a point on each unit as a node, then integrate the governing equation to obtain the discrete equation, and then solve it. The solution obtained by this method is an approximate solution. At present, in the finite element simulation of tumors, the geometric parameters of aneurysms of patients are obtained by imaging method to establish the morphological model of tumors, then the boundary conditions and initial conditions of the model are set by the finite element model of tumors, then the flow field is calculated and analyzed, and finally the data are obtained and visually displayed,As shown in Figure 1, a computer simulation of tumors is presented.

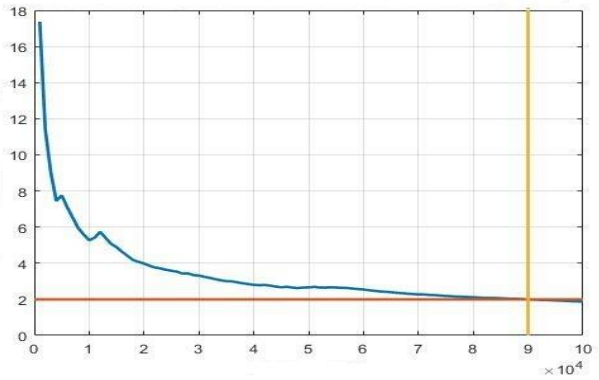

Figure 1. Computer simulation of tumors

4.2. simulation Model

Two kinds of models are used in our simulation, one is the initial model of various cell structures, the other is the mathematical model that drives the growth and division of cancer cells. The initial model uses two-dimensional Voronoi structure as cell tissue model. The method of forming two-dimensional Voronoi model by cell growth method on computer is summarized as follows: N points are randomly scattered on the plane of x3y (generated by 23N random numbers), their numbers are recorded in the order of their generation, and stored in a queue (FIFO). These points are taken from the queue in turn as nuclei to cause them to grow in all directions, one unit length at a time. In the program, the nucleus number of the cell is marked on the top, bottom, left and right of the point. When two different numbers of cells meet, they stop growing, so that different numbers of cells automatically form boundaries. The above growth process is cycled in turn until all points in the plane have attribution. In the above process, a two-dimensional cellular structure is discretized into a plane composed of x3y lattice points. Each cell in the plane contains an average of x3y/N points (also known as small cells). So far we have a two-dimensional array with each element value: {1,2,N} tumor cells are usually placed in the center of the simulation model, which can be easily selected in the two-dimensional array, and their numbers are placed in another array of cancer cells, whose growth rate is different from that of normal cell tissues in the course of subsequent growth and division. In our model, we initially used one (or several) cancer cell, which is at the center of the cell tissue. Cell reproduction can be divided into two stages: growth stage and

mitotic stage.

4.3. Simulation System Design

Just like we simulate two-dimensional soap bubbles. The class structure encapsulation feature of the simulation system is very convenient for our design. The system adopts the structure of multi-document and multi-view, and can open multiple documents at the same time. The data of the same document can be multi-view. Document-view structure separates data storage from operation and user's observation of data. There are two kinds of core objects in this simulation system. One is the environment plane of cell growth and division, which is used to visually express the initial structure and evolution pattern of two-dimensional cells. In the program, the edges and areas of each cell on the plane are calculated, so that many meaningful statistical data can be obtained, such as the distribution of cell edges, area, perimeter, boundary and neighbors of each cell. The other is the dynamic structure describing the internal process of cell growth and division. When dealing with the dynamic characteristics of cells, many data will be generated and updated in the system. For dynamic memory allocation and data storage, the system is accomplished by dynamically allocated Oblist linked list objects.

5. Conclusion

The body's immune system plays a dual role in the occurrence and development of tumors. On the one hand, the body can resist the occurrence of tumors through innate and acquired immunity; on the other hand, tumor cells can escape the recognition and attack of the immune system by forming special immunosuppressive microenvironment and other mechanisms to generate immune escape.

Funding

This article is not supported by any foundation.

Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

References

[1]Lee J H, Han I H, Choi B K, et al. Virtual Preoperative Simulation for Excision of Spinal Tumors: Surgeon Processing of Medical Computer-Assisted Design Software:. Korean Journal of Spine, 2017, 14(4):170-174. https://doi.org/10.14245/kjs.2017.14.4.170

[2]Das S, Paul S. Computer Simulation Studies of the Mechanism of Hydrotrope-Assisted Solubilization of a Sparingly Soluble Drug Molecule. Journal of Physical Chemistry B, 2016, 120(14):3540-3550. https://doi.org/10.1021/acs.jpcb.5b11902

[3]Basler L, Andratschke N, Ehrbar S, et al. Modelling the immunosuppressive effect of liver SBRT by simulating the dose to circulating lymphocytes: an in-silico planning study. Radiation Oncology, 2018, 13(1):10. https://doi.org/10.1186/s13014-018-0952-y

[4] *Park S W, Deyoung A D, Dhumal N R, et al. Computer Simulation Study of Graphene Oxide Supercapacitors: Charge Screening Mechanism. Journal of Physical Chemistry Letters, 2016, 7(7):1180-1186. https://doi.org/10.1021/acs.jpclett.6b00202*

[5] *Welter M, Rieger H. Computer Simulations of the Tumor Vasculature: Applications to Interstitial Fluid Flow, Drug Delivery, and Oxygen Supply. Systems Biology of Tumor Microenvironment. 2016. https://doi.org/10.1007/978-3-319-42023-3_3*

[6] *Li H, Lan L, Sennett C, et al. SU-E-J-248: Contributions of Tumor and Stroma Phenotyping in Computer-Aided Diagnosis. Medical Physics, 2015, 42(6Part11):3323-3323. https://doi.org/10.1118/1.4924334*

[7] *Welter M, Rieger H. Computer Simulations of the Tumor Vasculature: Applications to Interstitial Fluid Flow, Drug Delivery, and Oxygen Supply. Advances in Experimental Medicine & Biology, 2016, 936:31. https://doi.org/10.1007/978-3-319-42023-3_3*

[8] *Ewing A D, Houlahan K E, Hu Y, et al. Combining tumor genome simulation with crowdsourcing to benchmark somatic single-nucleotide-variant detection. Nature Methods, 2015, 12(7):623-630. https://doi.org/10.1038/nmeth.3407*

[9] *Azarnoush H, Alzhrani G, Winkler-Schwartz A, et al. Neurosurgical virtual reality simulation metrics to assess psychomotor skills during brain tumor resection. International Journal of Computer Assisted Radiology & Surgery, 2015, 10(5):603-618. https://doi.org/10.1007/s11548-014-1091-z*

[10] *Lashin S A, Mamontova E A, Matushkin Y G, et al. Mechanisms of the formation and propagation of sociobiological interactions: a computer simulation study. Russian Journal of Genetics Applied Research, 2015, 5(6):672-678. https://doi.org/10.1134/S2079059715060040*

[11] *Sun Q, Tang P, Zhao L, et al. Mechanism and structure studies of cinnamaldehyde/cyclodextrins inclusions by computer simulation and NMR technology.. Carbohydrate Polymers, 2018:S0144861718304417. https://doi.org/10.1016/j.carbpol.2018.04.055*

[12] *Minkin V I, Starikova A A. Computer simulation of the isomerization mechanism and spectral characteristics of spiro[1,3,4]oxadiazines. Russian Chemical Bulletin, 2016, 65(1):40-46. https://doi.org/10.1007/s11172-016-1262-x*

[13] *Kasaie P, Kelton W D, Ancona R M, et al. Lessons Learned from the Development and Parameterization of a Computer Simulation Model to Evaluate Task Modification for Healthcare Providers.. Academic Emergency Medicine, 2017. https://doi.org/10.1111/acem.13314*

[14] *Stishkov Y K, Bogdanov D V. Computer simulation of the injection mechanism of initiation of EHD flows in liquids with a higher level of low-voltage conductivity. Surface Engineering & Applied Electrochemistry, 2017, 53(1):27-33. https://doi.org/10.3103/S1068375517010148*