Ukrainian Society of Cell Biology

Institute of Cell Biology NAS of Ukraine

Ivano-Frankivsk National Medical University

6th Ukrainian Congress for Cell Biology with international representation

PROCEEDINGS

18-21 June **2019**, Yaremche, Ukraine

6th Ukrainian Congress for Cell Biology with international representation

Proceedings. – Yaremche, 2019. – 180 p.

Proceedings contain the materials of ***6th Ukrainian Congress for Cell Biology with international representation***, which was focused on novel insights in cell biology and biotechnology in Ukraine and abroad. The authors are solely responsible for the content of the abstracts.

Edited by:

Prof. Sibirny A.A.
Dr. Sci. Panchuk R.R.
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Desktop publishing, cover design by: **Rostyslav Panchuk**

MATHEMATICAL APPROACHES TO THE STUDY OF THE DISTRIBUTION OF OXYGEN IN HETEROGENEOUS TUMOR TISSUES

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Cancer is a complex process that involving many different cell types. Traditionally, mathematical models of cancer growth fall into two broad camps: descriptive and mechanistic (Araujo R.P. et al., 2004), (Kozusko F. et al., 2007). Descriptive models tend to focus on reproducing the gross characteristics of tumours, such as size and cell number. The mechanistic models focus on specific aspects of tumour progression (Batyuk L., Kizilova N., 2018), (Ponomarenko N., Batyuk L., et al. 2014). The mathematical model of cancer growth includes three steps. The first is the modeling of vasculature formation by sprouting angiogenesis. Second is fluid flow in interstitial space, and third is blood flow through vasculature and solute transport in interstitium. As known, tissue is heterogeneous, consists of cells and extracellular spaces. These heterogeneities affect the distribution of oxygen in the tissue. A model of tumor tissue composed of plane layers with different diffusion characteristics was proposed (Batyuk L.V., Kizilova N.N. et al., 2017). The Krogh tissue cylinder model of oxygen transport between blood capillaries and tissue has served as the foundation and starting point for many theoretical studies (Kreuzer F. et al. 1983). It has also been broadly used in physiological studies for estimating oxygen distribution in tissue. Tumors especially dangerous after they induce blood vessel growth. The new vessels of blood can not only carry oxygen and nutrition that further facilitate tumor growth but help also spreading for tumor cells to spread to other it's of the body. Thus tumor growth can be divided into two phases: avascular and vascular growth. We investigate how growth of tumor is related to the oxygen concentrations in the environment that tumor lives in. Oxygen concentration distribution is governed by the diffusion-consumption equation.

The above analysis is the preliminary step of the role of the parameters of the model upon the asymptotic behavior of the solutions. Using such a model, we tested two saturation hypotheses for avascular tumor growth separately. *In vivo,* it should be a combination of the mechanisms that all together contribute to the stop of growth, and we are on the way of putting them together.

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