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Modified Gompertz equation for electrotherapy murine tumor growth kinetics: Predictions and new hypotheses

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The aim of this lecture is to describe the complete growth kinetics of unperturbed and perturbed tumors through use of the modified Gompertz equation in order to generate useful insight into the mechanisms that underpin this devastating disease. Electrotherapy effectiveness at different doses has been demonstrated in preclinical and clinical studies; however, several aspects that occur in the tumor growth kinetics before and after treatment have not yet been revealed. Mathematical modeling is a useful instrument that can reveal some of these aspects. The complete tumor growth kinetics for control and treated groups are obtained by interpolation and extrapolation methods with different time steps, using experimental data of fibrosarcoma Sa-37. In the modified Gompertz equation, a delay time is introduced to describe the tumor's natural history before treatment. Different graphical strategies are used in order to reveal new information in the complete kinetics of this tumor type. The model, at this stage, shows different aspects that agree with those reported theoretically and experimentally. Tumor reversibility and the proportionality between regions before and after electrotherapy are demonstrated. In tumors that reach partial remission, two antagonistic post-treatment processes are induced, whereas in complete remission, two unknown antitumor mechanisms are induced. The modified Gompertz equation is likely to lead to insights within cancer research. Such insights hold promise for increasing our understanding of tumors as self-organizing systems and, the possible existence of phase transitions in tumor growth kinetics, which, in turn, may have significant impacts both on cancer research and on clinical practice.

Biography

Luis Enrique Bergues Cabrales has studied electrotherapy in cancer for 13 years, during which time he has authored more than 40 peer-reviewed reports. He has served on the editorial boards for the *Applied Mathematics and Computation, Physica Medica: European Journal of Medical Physics, IEEE Transactions on Biomedical Engineering, Journal of Biophysics and Structural Biology, Journal of Public Health and Epidemiology, Cuban Medical Magazine and Bioelectromagnetics.* At present, he is proposed for reviewer of five oncology magazines of EEUU. Cabrales is membership of the Scientific Advisory Committees of Cuban Bioengineering, Cuban Mathematical and Cuban Physics, and he has served on numerous review committees.

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Anticancer gene-engineered mesenchymal stem cell-mediated cytotoxic effects on pancreatic cancer cells: The complementary action of TRAIL and PTEN

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Pancreatic cancer is one of the most refractory malignancies. A major obstacle in the development of an effective therapy is the absence of sufficient tumor specificity. Thus, it is critical to explore effective strategies that can specifically target tumor tissue and distinctively attack malignant cells. Mesenchymal stem cells (MSCs) can serve as ideal carriers in cancer therapy owing to their tumor-oriented homing capacity and the feasibility of autologous transplantation. Anticancer gene-engineered MSCs specifically target tumor sites and can produce anticancer agents locally and constantly. In the present study, MSCs were engineered with anticancer genes TRAIL (tumor necrosis factor-related apoptosis-inducing ligand) and PTEN (phosphatase and tensin homolog) respectively. Human pancreatic cancer cell lines HP62 and Panc-1 were used as target cells. The expression of TRAIL and PTEN in MSCs was assessed by enzyme-linked immunosorbent assay and western blot analysis. Different patterns of death receptor expression in HP62 (DR4+, DR5+) and Panc-1(DR4-, DR5-) represent the heterogeneity of pancreatic cancer. Cell viability was assessed using image and a real-time monitoring system (xCelligence real-time cell analyzer, RTCA) during direct and indirect co-cultures. The results showed MSCs' intrinsic inhibition of pancreatic cancer cells and differentiated cytotoxic effects of MSC^{TRAIL} and MSC^{PTEN} on HP62 and Panc-1 cells. The complementary action of TRAIL and PTEN under this experimental condition suggests that multiple targeted strategies should be considered with regard to such highly heterogeneous malignancies.

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