

Modeling Tumor Growth

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Abstract

Our purpose for this project is to showcase some of the applications of differential equations. The method in which we sought to achieve our purpose is through the modeling of tumor growth. We provide some context on tumors and their formation to better understand what is to be modeled. Then described two models once commonly used to model tumor growth. We finally conclude by explaining why the models are not commonly used today.

Gompertz

Of the models in existence that serve to describe the growth behaviors of tumors, Gompertz model was arguably the most important and celebrated of the group. The model has three common representations, which hold the key assumption that the rate of growth will decrease exponentially as time increases. Its use was attributed to reports of the model being of good fit to empirical data collected on growth behaviors of some tumors, mainly benign tumors.

The Main differential equation:

$$\frac{dy}{dt} = yr \left(\ln \left(\frac{k}{y} \right) \right)$$

Its Solution:

$$y(t) = ke^{\ln\left(\frac{y_0}{k}\right)e^{-r}}$$

Von Bertalanffy Model

Austrian biologist Karl Ludwig von Bertalanffy (1901-1972) created a model to describe the growth of tumors.

The differential equation:

$$\frac{dL}{dt} = k(-L + L_\infty)$$

Its Solution:

$$L(t) = L_\infty \left[1 - e^{-k(t-t_0)} \right]$$

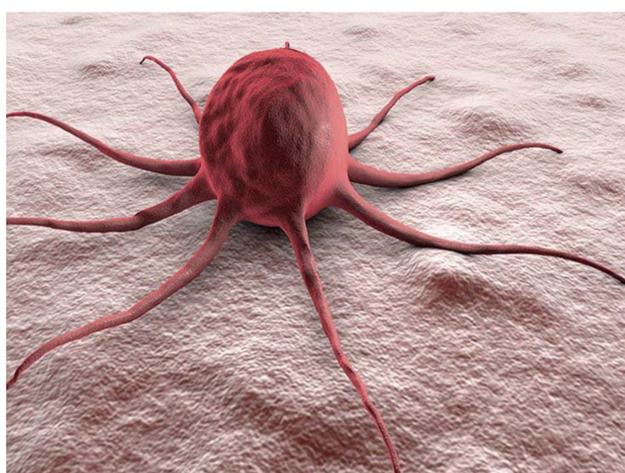
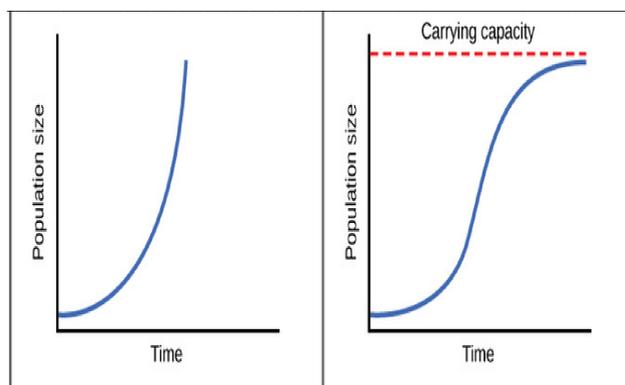
Conclusion

Currently, Gompertz and Bertalanffy models are not used to model tumors as there exist many different parameters and variables that the models can not keep up with. Now-a-days, the equations are created for on a case-by-case basis. There also exist incredibly intricate programs with complicated parameters that allow the modeling of the growth of only some tumors.

Context

A tumor, or Neoplasm, is tissue composed of abnormal cells that feature an accumulation of key mutations in genes that regulate cellular behaviors. Tumors fall into one of two classes: benign or malignant. Benign tumors are those that remain localized in one region as they cannot survive outside the capsule that surrounds them. Malignant tumors are invasive tumors that metastasize, i.e., smaller masses break off from the original mass and spread via the circulatory system to other parts of the body. Both classes of tumors are further classified by the type of tissue surrounding the given tumor.

Exponential Growth Logistic Growth



References

1. Boland, C. R. (2003). Tumor formation: Number of mutations required. In D. N. Cooper, *Encyclopedia of the human genome*. Hoboken, NJ: Wiley. Retrieved from http://db25.linccweb.org/login?url=http://search.credoreference.com/content/entry/wileyhg/tumor_formation_number_of_mutations_required/0
2. Neoplasm. (2016). In P. Lagasse, & Columbia University, *The Columbia encyclopedia*. New York, NY: Columbia University Press. Retrieved from <http://db25.linccweb.org/login?url=http://search.credoreference.com/content/entry/columency/neoplasm/0>
3. West, G. B., Brown, J. H., & Enquist, B. J. (2001). A general model for ontogenetic growth. *Nature*, 413(6856), 628.
4. <http://chemoth.com/tumorgrowth>

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Other References:

5. Martin, Mike. "The von Bertalanffy Model for Growth." The von Bertalanffy Model for Growth. N.p., n.d. Web. 18 Feb. 2017.
6. Murphy, Hope, et al. "Differences in Predictions of ODE Models of Tumor Growth: a Cautionary Example." BMC Cancer, BioMed Central, 26 Feb. 2016, bmccancer.biomedcentral.com/articles/10.1186/s12885-016-2164-x. Accessed 28 Feb. 2017.
7. Ramasawmy, Rajiv, et al. "Monitoring the Growth of an Orthotopic Tumour Xenograft Model: Multi-Modal Imaging Assessment with Benchtop MRI (1T), High-Field MRI (9.4T), Ultrasound and Bioluminescence." PLOS ONE, Public Library of Science, journals.plos.org/plosone/article?id=10.1371/journal.pone.0156162. Accessed 28 Feb. 2017.
8. Gerlee, Phillip. "The Model Muddle: In Search of Tumor Growth Laws." *Cancer Research*, American Association for Cancer Research, 15 Apr. 2013, cancerres.aacrjournals.org/content/73/8/2407. Accessed 28 Feb. 2017.