



SIMIODE

Systemic Initiative for Modeling Investigations
and Opportunities with Differential Equations

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Article Review and Annotation

BioModels Database. 2015. The European Bioinformatics Institute. <http://www.ebi.ac.uk/biomodels-main/> . Accessed 15 March 2015.

“Part of the European Molecular Biology Laboratory at the Wellcome Trust Genome Campus, Hinxton, Cambridgeshire, CB10 1SD, UK, EMBL-EBI provides freely available [data from life science experiments](#), performs [basic research](#) in computational biology and offers an extensive [user training](#) programme, supporting researchers in academia and [industry](#).”

We quote from the home page,

“BioModels Database is a repository of computational models of biological processes. Models described from literature are manually curated and enriched with cross-references. All models are provided in the [Public Domain](#). More information about BioModels Database can be found in the [FAQ](#).”

When searching through <http://www.ebi.ac.uk/biomodels-main/> under “differential equation” one finds 65 curated and 55 uncurated resources/papers. For each there is a full citation and an abstract and for many there is quick access to the complete paper for download.

To give the reader some idea of what the possibilities are here is one of the curated citations. It should be noted that while some materials are directly downloadable through hot-linked reference others will need to be obtained through library or individual data base or journal access privileges.

Clinical Cancer Research. 2012 Sep 15; 18(18):5071-80. Epub 2012 Jul 3.

A tumor growth inhibition model for low-grade glioma treated with chemotherapy or radiotherapy. Ribba B¹, Kaloshi G, Peyre M, Ricard D, Calvez V, Tod M, Cajavec-Bernard B, Idbaih A, Psimaras D, Dainese L, Pallud J, Cartalat-Carel S, Delattre JY, Honnorat J, Grenier E, Ducray F.

Abstract

PURPOSE:

To develop a tumor growth inhibition model for adult diffuse low-grade gliomas (LGG) able to describe tumor size evolution in patients treated with chemotherapy or radiotherapy.

EXPERIMENTAL DESIGN:

Using longitudinal mean tumor diameter (MTD) data from 21 patients treated with first-line procarbazine, 1-(2-chloroethyl)-3-cyclohexyl-1-nitrosourea, and vincristine (PCV) chemotherapy, we formulated a model consisting of a system of differential equations, incorporating tumor-specific and treatment-related parameters that reflect the response of proliferative and quiescent tumor tissue to treatment. The model was then applied to the analysis of longitudinal tumor size data in 24 patients treated with first-line temozolomide (TMZ) chemotherapy and in 25 patients treated with first-line radiotherapy.

RESULTS:

The model successfully described the MTD dynamics of LGG before, during, and after PCV chemotherapy. Using the same model structure, we were also able to successfully describe the MTD dynamics in LGG patients treated with TMZ chemotherapy or radiotherapy. Tumor-specific parameters were found to be consistent across the three treatment modalities. The model is robust to sensitivity analysis, and preliminary results suggest that it can predict treatment response on the basis of pretreatment tumor size data.

CONCLUSIONS:

Using MTD data, we propose a tumor growth inhibition model able to describe LGG tumor size evolution in patients treated with chemotherapy or radiotherapy. In the future, this model might be used to predict treatment efficacy in LGG patients and could constitute a rational tool to conceive more effective chemotherapy schedules