Physiologically structured PDE models of proliferation in cell populations to optimise anticancer treatments

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Abstract:

The main two pitfalls of therapeutics in clinical oncology, that limit increasing drug doses, are unwanted toxic side effects on healthy cell populations and occurrence of resistance to drugs in cancer cell populations.

Depending on the constraint considered in the control problem at stake, toxicity or drug resistance, we present two different ways to model the evolution of proliferating cell populations, healthy and cancer, under the control of anti-cancer drugs.

In the first case, we use a McKendrick-like age-structured model of the cell cycle, whereas in the second case, we use a model of evolutionary dynamics, physiologically structured according to a continuous phenotype standing for drug resistance.

In both cases, we mention how drug targets may be chosen so as to accurately represent the effects of cytotoxic and of cytostatic drugs, separately, and how one may consider the problem of optimisation of combined therapies.