

# MODELING TUMOR CELL GROWTH INCLUDING NUTRIENT DEPENDENT AUTOPHAGY

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In *in vitro* experiments with tumor cells [1] it could be observed that lack of nutrients does not only lead to starvation in the long term, but can also enhance temporary growth if the nutrient stress is sufficient. To describe this effect, a basic system of ordinary differential equations (ODEs) is presented, which describes cell proliferation according to the available nutrients and the induction of cell necrosis and autophagy due to nutrient deprivation. Autophagy is a regulated intracellular mechanism to recycle or remove components of the cell, which are unnecessary or dysfunctional. It is a natural process i.a. to maintain proper cell function but it can also be promoted, e.g. by various stressors. In the case of nutrient deprivation, this mechanism provides the cell with energy to ensure survival and proliferation and in cancer cells this process might be upregulated [2]. In general, the balance between apoptosis and autophagy is important, when considering tumor development.

The unknown parameters of the model are estimated by using Bayesian inversion methods. The corresponding data sets for the parameter calibration are time resolved measurements of populations of liver cancer cells, which are provided with varying amounts of nutrients in an avascular environment. The presented ODE model serves as a basis for modeling and quantifying effects of mechanical properties of the extracellular matrix on the growth of tumor cells.

## References

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- [2] Livesey, K. M., et al. (2012). *p53/HMGB1 complexes regulate autophagy and apoptosis*. Cancer research 72.8, 1996-2005, <https://doi.org/10.1158/0008-5472.CAN-11-2291>