From a mathematical model of a unique tumour cell to a model of a dynamic population of interacting cells

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When modelling tumorigenesis, there are different levels of complexity to consider: (1) the diversity of the intracellular signalling pathways that are deregulated in cancer patients; (2) the heterogeneity between cells, i.e., not all tumour cells have the same genomic profile and thus the parameters of the mathematical model should be adapted; (3) the tumour microenvironment matters: tumour cells interact with other cell types such as immune cells, fibroblasts, etc. Depending of the biological question, the model should be able to account for all these levels of complexity in order to optimize its predictive power and, ultimately, anticipate the effect of drug treatments.
To address these modelling aspects, we have developed a series of tools that extend from the modelling of an individual tumour cell (MaBoSS) to a dynamic population of interacting cells (UPMaBoSS, PhysiBoSS), considering the heterogeneity of these populations (PROFILE, EnsembleMaBoSS). During this talk, I will introduce some of these tools, show their limitations and illustrate them with a concrete example.