UPPSALA UNIVERSITET

**Project in Computational Science 2019**


Fredrik Gustafsson
fredrik.gustafsson.8585@student.uu.se

Fredrik Jonasson
fredrik.jonasson.3188@student.uu.se

Oscar Larsson
oscar.larsson.4858@student.uu.se


**Supervisors**

Stefan Engblom
Robin Eriksson

# Data driven modeling of avascular tumors

## Introduction

The project aim was to implement and compare three different cancer growth models and then to combine the models into one new model based on which parameters of each model was the most important.

## Implementation

The three models were implemented using FEniCS, which is a library that provides methods and routines for formulating and solving finite element method problems.

## Models

The first model was presented by Pietro Mascheroni and his team in 2017. The main difference when compared to the other two models is that the governing equations are expressed in polar coordinates.

$$\frac{\partial \varepsilon^t}{\partial t} - \frac{1}{r^2}\frac{\partial}{\partial r}\left(r^2 \varepsilon^t \frac{k}{\mu_l}\Sigma' \frac{\partial \varepsilon^t}{\partial r}\right) + \frac{1}{\rho}\left(\underset{growth}{M^{l\to t}} - \underset{lysis}{M^{t\to l}}\right) = 0$$

*Figure 1. One of the three governing equations of the first model*

Due to spherical symmetry the problem is reduced to 1D. The equation above is then solved for $\varepsilon^t$, the volume fraction of tumor cells.

The second model was proposed by Mark A. J. Chaplain et al. In 2014. Their model consists of three equations which tries to model the growth of tumors as the increase in concentrations

$$\frac{\partial}{\partial t}c = \nabla[D_1 \nabla c - cA(t,x,\mathbf{u}(t,\cdot))] + \mu_1(t)c(1-\rho(\mathbf{u}))$$

*Figure 2. Equation from second model governing the change in concentration of tumour cells.*

The third model was presented by a team led by Giuseppe Sciumè in 2015. The model includes the area between the cells in a new way, which relaxes the limitations of the model.

$$\begin{aligned}
&\left[\frac{\epsilon S^t}{K_T} + \frac{S^t(1-\epsilon)}{K_S}\left(S^t + u(1)S^t_{der}\right) + \epsilon S^t_{der}\right]\frac{\partial u(1)}{\partial t} \\
&+\left[\frac{\epsilon S^t}{K_T} + \frac{S^t(1-\epsilon)}{K_S}\left(1 - S^t - u(2)S^t_{der})\right)\right]\frac{\partial u(2)}{\partial t} \\
&+\left[\frac{\epsilon S^t}{K_T} + \frac{S^t(1-\epsilon)}{K_S}\right]\frac{\partial u(3)}{\partial t} \\
&=\nabla\cdot\left[\frac{k^t_{rel}\boldsymbol{k}}{\mu^t}\cdot\nabla\left(u(1)+u(2)+u(3)\right)\right] \\
&-S^t\left(\mathbf{1}:\boldsymbol{d}^s\right) - \nabla S^t\cdot\left(\epsilon \boldsymbol{v}^s\right) + \frac{1}{\rho^t}M^{l\to t}
\end{aligned}$$

*Figure 3. The first governing equation for the third model, which shows the mass balance for the tumor cell*

## Results

Due to troubles with implementing the models in FeniCS, none of the three models are correctly implemented entirely and as such the results presented below are partial results where some term or expression is missing from the original models.

Simulating the equation in figure 2 with *A* = 0, r, from t = 0 to t = 60 yields the results shown in figure 4.

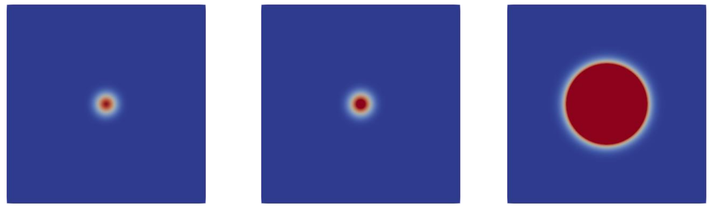

*Figure 4. Results from simulating the model presented by Chaplain at t=0, t=30 and t=60.*

## Conclusion

From figure 4 one can see that when the haptotaxic term *A*, in Chaplains model, is set to zero the tumor grows like a circle with increasing radius.