Clarification about model and future possible changes

In our current model (see "report myxo.pdf") the speed and quantity of RomR binding to the poles is regulated by the number of "active receptors" (AR) at that specific location. This number affects the binding rate s1=n*sigma1, where n is the number of active receptor (at that location and time) and sigma1 is the "basic" binding rate.
At the moment based on the data from the movies analyzed by Cameron we have established a number of 1100 AR at the lagging pole and 600 AR at the leading pole. Also based on the same movies we have determined that there seems to be none or very limited binding away from the poles. Therefore, the number of AR is decreased linearly until reaching 1 AR in a small zone adjacent to the poles.
Also during division RomR gradually starts binding to the central location. To incorporate this effect the number of AR around the center in our model increases from 1 AR to 400 AR during the division process with a certain velocity that can be adjusted to match the experimental movies.

The reversal in RomR concentration is then achieved through an inversion of the leading and lagging poles identity with consequent change in number of AR. In our model this happens based on times sampled from a gamma distribution whose parameters were determined experimentally.
However we would like to be able to make the reversal a natural occurrence emerging from the nature of the model itself instead of a prescribed occurrence.

My idea is that Myxo may have a system of proteins that through iteration with each other creates peaks of concentration moving from one pole to the other and that one of these protein could "deactivate" or "occupy" a certain quantity of active receptors while present reducing their number from 1100 AR in the lagging pole to 600 AR in the leading pole and limiting their number at the center during the division process.
This concept was inspired by my previous experience in modeling the MinCDE system of proteinsin Escherichia Coli. This system exhibits exactly the behavior previously described and in E. Coli helps to determine the localization of the division site (for more details on MinCDE see "Final report.pdf" from a class in modeling biological problems I took last Fall).

To check if this hypothesis may explain what we have seen in the experiments I will try to incorporate a MinCDE-like system of equations to our model and include an algebraic equation for the number of AR based on the concentration of either MinC or MinD.
This system will be modeled as described in the first model of "Final report.pdf" (1D model).
In a first moment I will modify the parameters of the model to match the average reversal time. However, being able to determine, either from the literature or from experiments, the diffusion rates of both RomR and other proteins known to be involved in its reaction cascade would be extremely helpful, since the simplicity of the model allows to obtain the same oscillation period with more than a single parameter set.

To better determine the RomR mechanisms would be also useful to be able to observe "hypo-reversing" and/or non-reversing RomR labeled bacteria to observe difference with the "normal" ones, as was previously done with the "hyper-reversing" batch where a third stable local concentration of RomR was observed closed to the center also when the cell was not dividing (that would produce a fourth one). Also being able to label other protein connected to RomR at the same time could be enlightening.

The following pictures I hope will clarify the AR profiles used in different phases of the model.

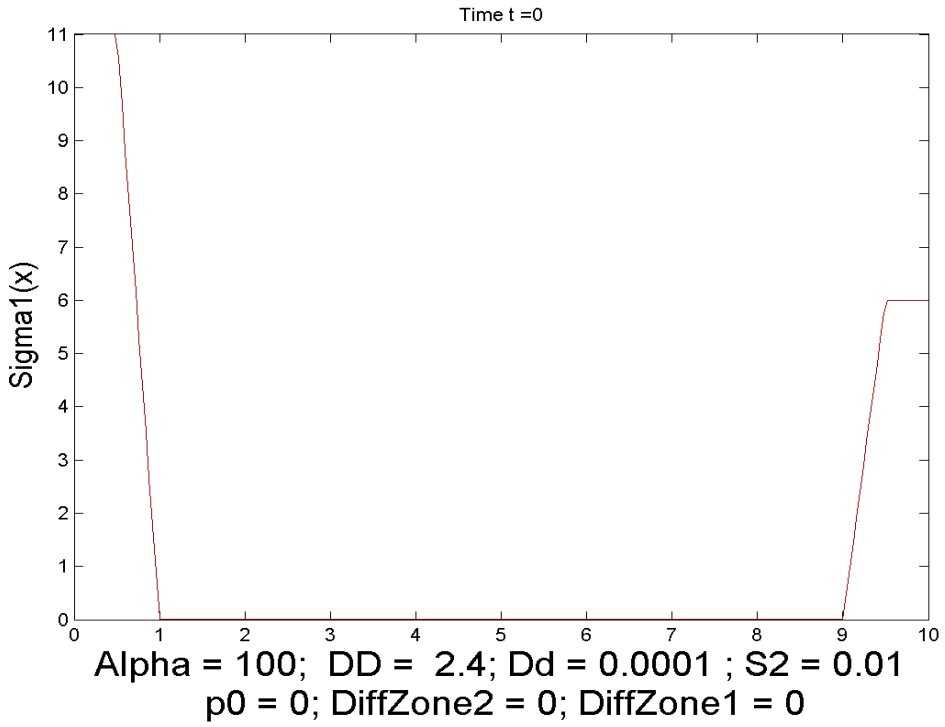

Fig 1: Initial configuration

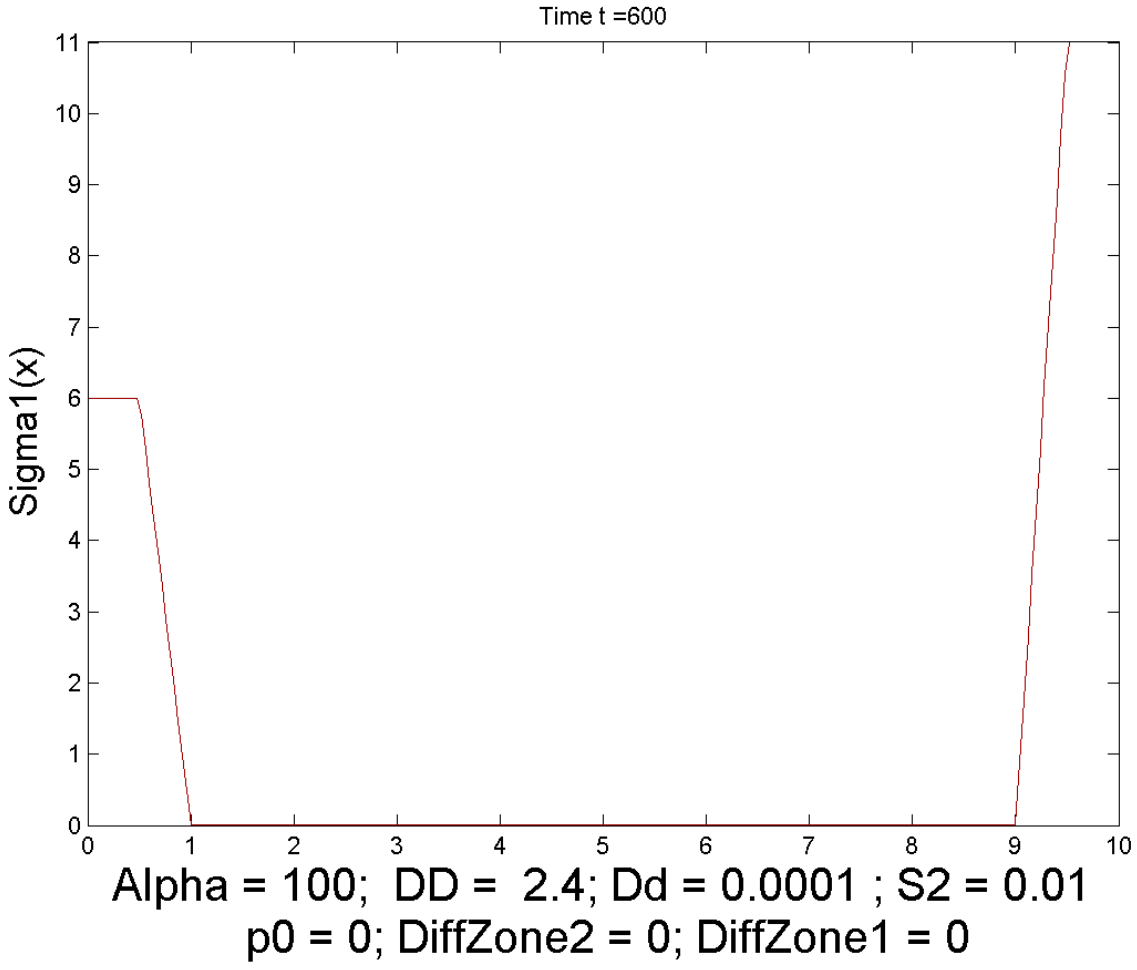

Fig 2: Reversal before division started

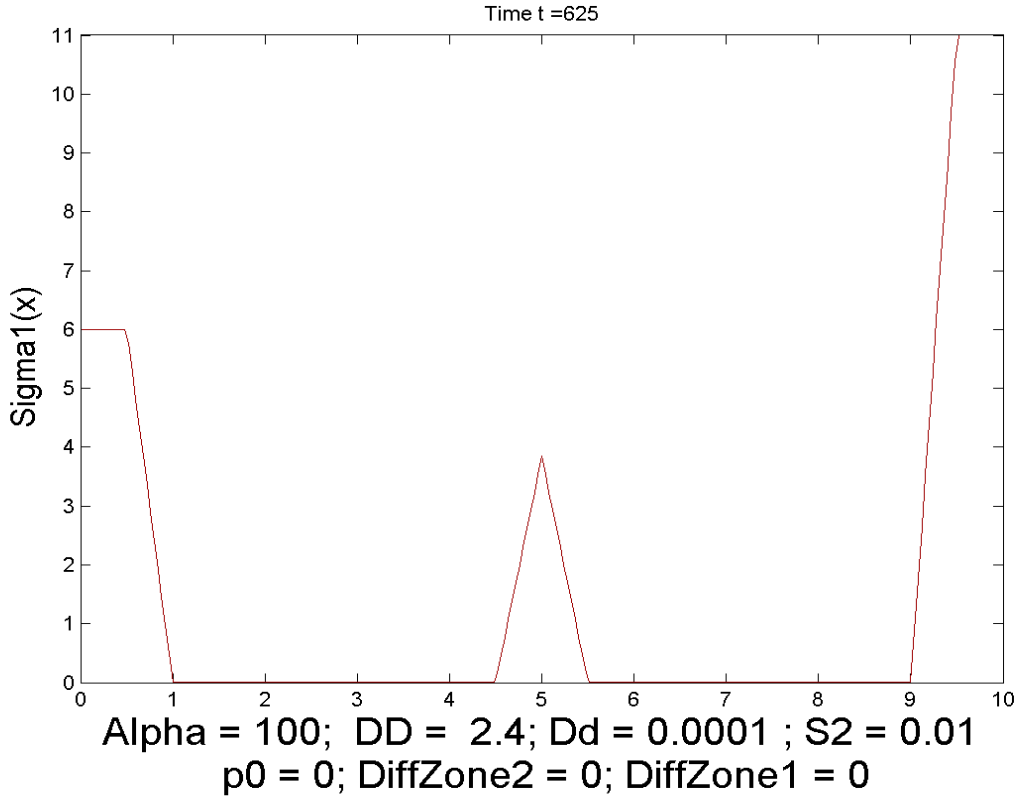

Fig 3: Division started

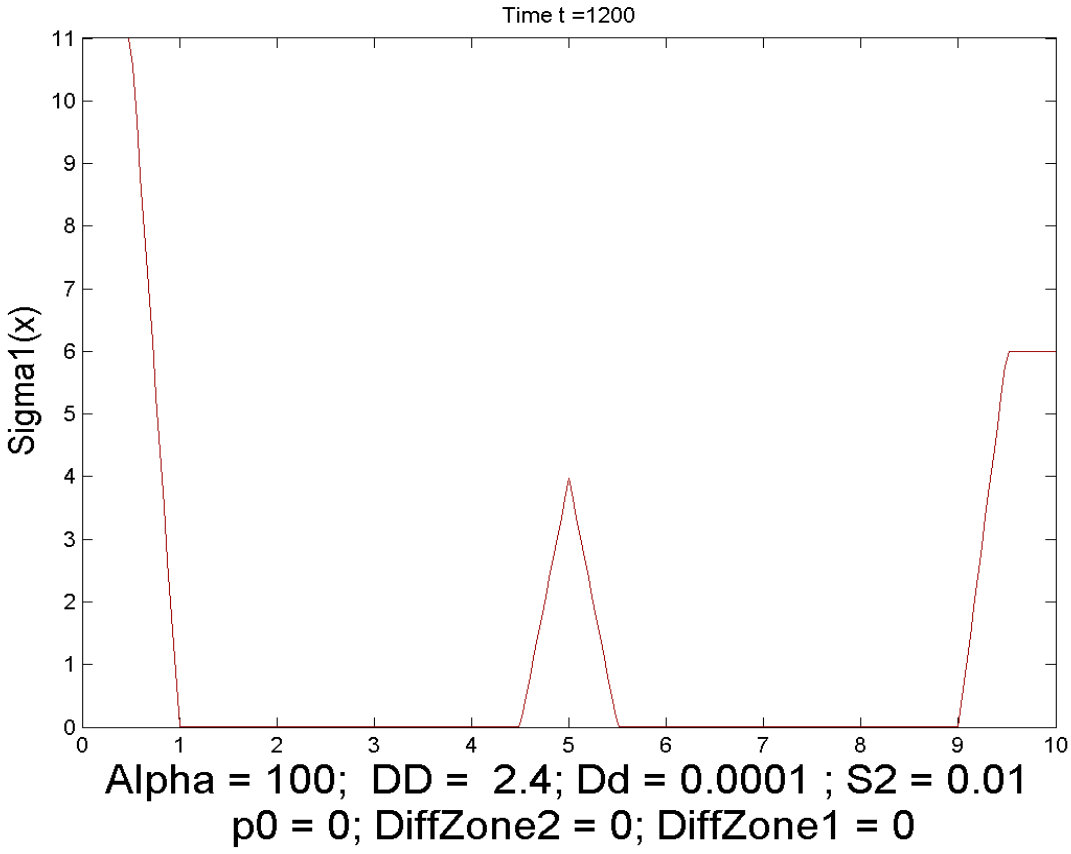

Fig 4: Reversal after division started