A reaction-diffusion system to model symmetry-breaking in the C. elegans worm

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The main aim of a signaling pathways in cells is the transduction of a signal. These pathways regulate the cellular response to external cues. Such a situation occurs for example during cell polarization, where the cell undergoes a symmetry-breaking process in response to a weak external cue. Most cells have polarity and consequently have two different sides. A case where this is evident is for the cells that form the skin, which have an inside and outside. The study of the mechanisms by which cells become polarized is very important. These mechanisms are actually very similar for all animals[1].

A standard model organism where cell polarization has been studied extensively[1] is the nematode worm *Caenorhabditis elegans*. A group of genes (called the PAR genes) are in charge of the regulation of cell polarity in *C. elegans* and many other organisms. Prior to the first unequal cell division in the *C. elegans* embryo, the PAR proteins become distributed asymmetrically in distinct anterior and posterior domains[2,3].

A possible way of describing mathematically this problem is with a two-variable reaction-diffusion system. In this model each variable describes the concentration of a group of PAR proteins. The model accounts for the segregation of the PAR proteins and this separation can be triggered either convectivelly by cortical flows or spontaneously by random perturbations. In a recent publication appeared in *Science*[4], we have shown that the spontaneous symmetry-breaking leading to the polarization of the cell is induced by a mechanism similar to a Turing instability. However, in our model the fastest growing spatial perturbation's wavelength is equal to the system size. Many features of this model are in agreement with experimental observations in *C. elegans*. In Ref.[4], we have been also able to experimentally measure many of the model's parameters.

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