Relation between velocity and density in an advancing cell monolayer

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Cell migration plays an important role in governing various biological processes such as shape and vascularize tissues, in wound healing, tumour spreading and as a part of immune response. Although epithelia are generally assumed to be spatially constrained, it has been observed that in morphogenesis cells rearrange slightly but in a highly coordinated way in order to maintain tissue integrity. Collective cell migration is a dynamic process that requires physical (mechanical) and also chemical (signalling) interactions with the environment which are essential for cell movement.

We are focused in understanding the mechanisms of tissue repair in epidermal cell monolayers which is a model problem less complex than wound healing. The purpose of this study is to introduce a quantitative approach of the biological problem through in vitro assays and subsequent image analysis which allows to compute cellular velocities and densities for two different set of problems, normal conditions and starvation, inhibiting cell proliferation.

We found clear differences in the mean border displacement between experiments at normal conditions and those carried out in starvation that at some point they stop, although for some time both show similar velocity. We also explore the spatio-temporal evolution of velocity and density inside the monolayer obtaining that the distribution of the density within the monolayer at the end of the experiment is quite similar among all cases, whether the gap is closed or not.

In contrast, the velocity distribution at different depths shows that initially, the shallower cell layers develop the same speed in both cases but in normal conditions close to the edge the fastest cells are found. In other words, the velocity decays slowly with depth. However, in starvation the velocity is zero everywhere, consistently with the null advance velocity of the front. This is a clear evidence that the density is not driving the population of cells.

Finally, since the distribution of the density within the monolayer at the end of the experiment is quite similar between among all cases, whether the gap is closed or not. This is a clear evidence that the density is not driving the population of cells. Density might be considered given by an universal distribution or "advance formation" with respect to the density profile which is kept even when the population stops.