

Theoretical Modelling of Dynamic Phenotypes of Human HSC Affected by Clinical Agent

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The deformation of cells involves active, energy-consuming processes such as the membrane protrusions and the remodeling of cytoskeletons. To numerically describe the effect of clinical agents on dynamic phenotypes of cells, we study the dynamic deformation and motion of human hematopoietic stem cells (hHSC) on supported lipid bilayers functionalized with the cell adhesion molecule N-cadherin as an *in vitro* model of bone marrow surfaces. The deformation and migration of HSC is investigated by live cell imaging in the absence and presence of a clinical agent ADH-1, which is a cyclic penta-peptide selectively blocking the homophilic interaction between N-cadherin molecules. To account for the experimental observations, we utilize a simple and general theoretical model that describes “crawling” cells as deformable, self-propelled particles. Our experimental data shows that ADH-1 significantly reduces the adhesion and active deformation of hHSCs on the surface displaying N-cadherin. The combination of the simple theoretical model and the label-free, quantitative *in vitro* experiments of primary hHSCs opens a large potential to numerically identify the differential effects of clinical drugs on dynamic phenotypes of primary cells.