Rupture dynamics and chromatin loss in deformed nuclei

Dan Deviri

Weizmann institute of science, Rehovot, Israel

During migration of cells in vivo, in both pathological processes such as cancer metastasis or physiological events such as immune cell migration through tissue, the cells must move through narrow interstitial spaces which can be smaller than the nucleus. This can induce deformation of the nucleus which, according to recent experiments, may results in rupture of the nuclear envelope that can lead to cell death, if not prevented or healed within an appropriate time. The nuclear envelope, which can be modeled as a viscoelastic gel whose elasticity and viscosity primarily depend on the lamin composition, may utilize mechanically induced, self-healing mechanisms that allow the hole to be closed after the deformation-induced strains are reduced by leakage of the internal fluid. Here, we present a viscoelastic model of the evolution of holes nucleated by deformations of the nuclear envelope and estimate the loss of chromatin through the rupture and its relation to the lamin A/C to B ratio in the nuclear envelope.