Cell migration is a fundamentally important phenomenon underlying wound healing, tissue development, immune response and cancer metastasis. Understanding basic physics of the cell migration presented a great challenge until, in the last three decades, a combination of biological, biophysical and mathematical approaches shed light on basic mechanisms of the cell migration. I will first focus on the simplest mode of cell locomotion, lamellipodial motility. I will describe models, based on nonlinear partial differential equations and free boundary problems, which predicted that individual keratocyte cells do not linger in a symmetric stationary state, but rather spontaneously break symmetry and initiate motility. The cells can either crawl straight, or turn, depending on mechanical parameters. I will show how experimental data supported the models.

Most cells, however, migrate collectively, not individually, and in 3D. I will introduce experimental data on collective migration of two heart progenitor cells in Ciona embryo. These cells crawl cohesively squeezing between stiff ectoderm and elastic endoderm with persistent leader-trailer polarity. Most active and passive forces are concentrated in the 2D cortex of these cells, with hydrostatic pressure of the 3D cytoplasm assisting the cortex forces in generating stress balances optimizing the cell migration. I will present a computational model that sheds light on design principles of this motile system.

Wednesday, October 21st, 2020 at 2:15 pm
MPIDS, video conference at www.zoom.us
Meeting ID: 959 2774 3389
Passcode: 651129, direct link