Phase transitions in embryo morphogenesis

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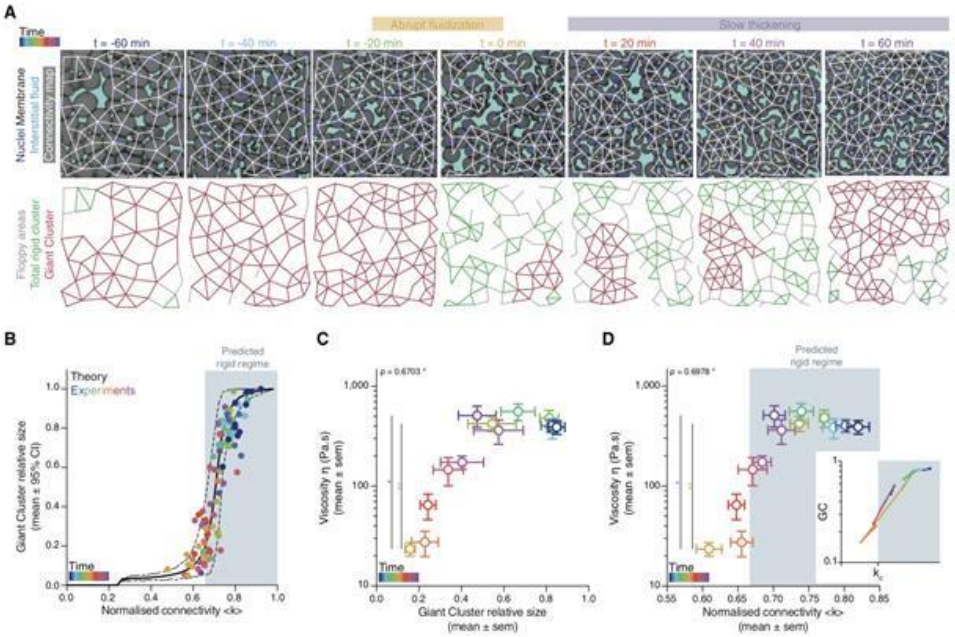
Embryo morphogenesis is impacted by dynamic changes in tissue material properties, which have been proposed to occur via processes akin to phase transitions. In this talk I will show that rigidity percolation provides a simple and robust theoretical framework to predict material/structural phase transitions of embryonic tissues from local cell connectivity. Rigidity percolation is the point in a network where the topological structure absorbs all the potential degrees of freedom of the nodes, thereby preventing the possibility of independent movements. This critical point has a dramatic impact in the material properties of the system. Combined with directly monitoring dynamic changes in tissue rheology and cell contact mechanics in real (zebrafish) embryos, I will show how the embryonic tissue undergoes a genuine phase transition, brought about by a small reduction in adhesion-dependent cell connectivity below a critical value. I will show how we quantitatively predicted and experimentally verified hallmarks of phase transitions, including traces of power-law exponents and associated discontinuities of macroscopic observables. The massive deformation occurs right after the "melting" of the tissue, which enhances such change of deformation at minimal energy cost. Consistently, when the deformation is triggered, the tissue tends to run backwards in the phase space, thereby solidifying again and fixing the deformation. Interestingly, the success of the deformation occurring right after crossing the critical point of the phase transition depends on embryonic (stem) cells undergoing asynchronous divisions causing random and, consequently, uniform changes in cell connectivity. Collectively, the theoretical and experimental findings presented here reveal the structural basis of material phase transitions, for the first time, in the context of a living organism.



(A) Exemplary 2D confocal sections at the 1st–2nd deep-cell layer of the central blastoderm with overlaid connectivity maps (top) and their rigidity profile (bottom) at consecutive time points during the fluidization/thickening process (color coded). Floppy areas are illustrated in gray, rigid areas in green, and the rigid GC in red. Shaded yellow and purple areas indicate the time period of tissue fluidization and thickening, respectively. (B) Plot of the fraction of the network occupied by the Giant Rigid Cluster (GC) as a function of normalized connectivity <k> at different time points during the fluidization/thickening process agreeing with the theoretical expectation. We observe the collapse of the GC at t=0, when the rigid-to-floppy/fluid phase transition occurs. (C) Plot of tissue viscosity as a function of the GC relative size for experimental networks of the central blastoderm at different time points during the fluidization/thickening process. (D) Plot of tissue viscosity as a function of normalized connectivity <k> for the samples described in (C) (for viscosity n = 129 embryos, N = 12 embryo batches; for normalized connectivity <k> n = 103 blastoderms, N = 11 embryo batches). The integrated plot illustrates the time trajectory (color coded) of the central blastoderm material phase state (relative size of GC) as a function of its connectivity (kc). The gray-shaded region in (B) and (D) indicates the rigid regime above the critical point of rigidity percolation kc.