Two cellular events for inversion in *Volvox carteri*, cell shape changes and migration: which drives the morphogenetic movement?

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Abstract:

Volvox and their close relatives serve as a good model for investigating molecular and cellular bases of morphogenetic processes and how such processes arise during the evolution of multicellular organisms from their unicellular ancestors. The key morphogenetic process in *Volvox* development is called inversion, in which the spherical embryo turns inside out. The pre-inversion embryo is a cellular monolayer in which neighboring cells are linked to one another at the mid-level of their cell bodies by cytoplasmic bridges. The phialopore where such linkages are missing is the opening where the inversion movement will start. First, the cells become flask shaped by extending long, thin stalks from their outermost ends. Next, cells near the phialopore migrate relative to their cytoplasmic bridges until they are linked to their neighbors only at the outermost tips of their stalks, which causes the cell sheet to turn outward. In the previous work, a series of morphological mutants in *V. carteri* that failed inversion was isolated and it has been shown that these two cellular events, cell elongation and cell migration, are genetically independent. In a mutant called *InvA*, cells in the inversion stage change their shape to be elongated normally but they cannot migrate relative to the cytoplasmic bridges and the inversion arrests halfway. A similar defect of embryo morphology is also seen for another type of mutant strains, in which cell migration seems to occur normally but cells cannot elongate. Thus, it is assumed that both cellular events are complement and working together for the *V. carteri* embryo to complete the inversion process. When we examined the two cellular processes in smaller colonial species such as *Eudorina* and *Pandorina*, we noticed that the cells elongated much shorter than that in *Volvox* and inhibiting cell migration using an antisense construct of *P. morum InvA* gene was enough to prevent *P. morum* from getting its spherical shape and kept it to be a flat colony, as somehow similar to a *Gonium* colony. These results proposed that reconsideration of the inhibition mechanism in the *V. carteri* mutants without cell shape changes might be required. Video microscopy indicated that when inversion stopped for such mutants, the embryo contacted with the vesicles surrounding itself and TEM also suggested that the vesicle might interfere with embryo inversion. I would like to discuss about why cell shape changes are required for the embryo of large *Volvox* to solve such issue.