1P133 極性タンパク質 PAR-2 の非対称局在は、細胞質における拡散によって安定化される

Stable maintenance of the cortical PAR-2 asymmetry by the cytoplasmic diffusion in the one-cell *C. elegans* embryo

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Asymmetric localizations of polarity proteins are maintained by feedback controls, but how the dynamics are balanced *in vivo* remains unexplored. Using single-molecule detection technologies, we measured dynamics of *C. elegans* polarity protein PAR-2 comprehensively. We identified the novel cortical fractions that dissociated in a fast rate and were laterally immobile on the cortex. Single-molecule imaging revealed that the novel fractions contain less-oligomerized and highly-phosphorylated PAR-2. Our mathematical model showed that dynamics measured *in vivo* measurements were sufficient for the cortical asymmetry by bi-stability, but unexpectedly emphasized a role of the components for the stability of asymmetry. A similar mechanism may function in other polarity systems.

1P134 組織応力の異方性が細胞の六角格子化を促進する
Anisotropic tissue stress promotes ordering in hexagonal cell packing

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Many epithelial tissues pack cells into a honeycomb pattern to support their structural and functional integrity. Developmental changes in cell packing geometry have been shown to be regulated by mechanical and biochemical interactions between cells; however, it is largely unknown how molecular and cell-level dynamics and tissue mechanics are orchestrated to realize the correct and robust development of hexagonal cell packing. By combining mechanical and genetic perturbations along with live imaging and Bayesian force inference, we identified the mechanism through which the mechanical anisotropy in a tissue promotes ordering in cell packing geometry. We are currently investigating a regulatory mechanism of force sensation and generation of F-actin during this process.

1P135 細胞間に非対称性を生み出すしくみの再構成
Reconstitution of an intercellular symmetry breaking mechanism

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Cells diversify into different cell-types during development. We have recently reconstituted a mechanism for creating a stable difference among cells, which is mediated by Delta-Notch signaling. In our synthetic gene circuit, production of a membrane protein, Delta, in a cell inhibits production of Delta in the adjacent cells. Such intercellular mutual inhibition functions as a (double-negative) positive feedback loop, which amplifies a small initial difference between adjacent cells. In fact, mammalian cultured cells engineered with our gene circuit spontaneously split into two distinct cell-types: Delta-positive and -negative populations. Interestingly, the ratio of two cell-types was robust against perturbation and also adjustable by the architecture of gene circuit.

1P136 低温電子顕微鏡による骨格筋の細いフィラメントの構造解析
Structural analysis of Ca²⁺ regulated thin filament from skeletal muscle by electron cryomicroscopy

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Muscle contraction is caused by sliding between the thick and thin filaments. The thin filament is composed of actin filament, tropomyosin (Tm) and troponin (TnC, TnI, TnT). It is thought that Ca²⁺ released from sarcoplasmic reticulum binds to TnC, leading to a conformational change of Tm on the actin filament to allow actin-myosin interaction. To understand this regulatory mechanism by electron cryomicroscopy, we developed a method to isolate skeletal muscle thin filament from a crab, *Portunus trituberculatus*. We treated the thin filament by glutaraldehyde to crosslink Tm and Tn with actin filament and obtained a density map of the filament fully decorated with Tm. Interestingly, the cross-linked thin filament retained the ability of Ca²⁺ regulation.

1P137 中性子非弾性散乱による F-アクチン及びミオシン S1 のダイナミクス解析
Difference in dynamics between F-actin and myosin S1 measured by neutron scattering

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The dynamics of F-actin and myosin S1 were studied by quasi-elastic neutron scattering (QENS). The QENS measurements were conducted on D₂O solution samples of F-actin and S1 at 300 K using the cold-neutron disk chopper spectrometer AMATERAS in J-PARC. In the current analysis, it was found that while the correlation time (τ) of atomic motions of S1 was similar to that of other proteins such as hemoglobin, the τ value of F-actin was shorter than S1. Furthermore, F-actin had a population of the atoms undergoing diffusive motions with larger amplitudes than S1. These results suggest that F-actin is more flexible than other proteins including S1. The results of a more detailed analysis will be given in the presentation.

1P138 アクチン疎水性ヘリックスに導入した変異は細胞性粘菌の細胞運動と細胞分裂を阻害する
Mutations introduced into the hydrophobic helix of the *Dictyostelium* actin impaired cell motility and cytokinesis

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The hydrophobic helix of actin is a part of myosin-binding site(s). *Dictyostelium* cells carrying plasmids expressing the mutant actin (Tyr337Cys/Leu349Cys) showed dominant negative phenotypes. The size of starvation-induced mounds was smaller. The number of mounds was increased 10 times. When co-cultured with *E. coli* B/r, they migrated slower than wild-type cells. When cultured in suspension, the number of nuclei per cell increased by a factor of two, indicating impaired cytokinesis. The results are consistent with the reports showing the hydrophobic helix is important for the interaction with myosin. Surprisingly, ultracentrifugation analysis of the purified mutant actin showed the significantly higher polymerizability than the expressed wild-type actin.