1P272 大自由度遺伝子発現制御モデルにおける適応応答の協同的進化
Cooperative Adaptive Responses in Gene Regulatory Networks with Many Degrees of Freedom

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Cells generally adapt to environmental changes to achieve homeostasis. Although simple mechanisms for adaptation through network motifs consisting of a few genes are well understood, how regulatory networks involving many genes that activate or repress each other can generate adaptive behaviors is unclear. Here, by numerically evolving gene regulatory networks, we obtained a class of genes whose expression dynamics showed adaptation over almost all genes, albeit sometimes partial adaptation, from which we revealed the general logic underlying such adaptive dynamics with many degrees of freedom, which was not reducible to common motifs with a few genes. These results have implications in the significance to the biological homeostasis of systems with many components.

1P273 細胞の集団的意思決定の設計原理
A Design Principle of Group-level Decision Making in Cell Populations

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Although the gene circuits that underlie the switches are well understood at the level of single cells, the ways in which such circuits work in concert among many cells to support group-level switches are not fully explored. Here, we theoretically show two distinct forms of bistability are uniquely determined by a dimensionless parameter, which compares the synthesis and the transport of the inducing molecules. The parameter in bacterial quorum-sensing circuits appears to be tuned so that the cells can use either the cell-level or the group-level switch. Furthermore, in negative feedback circuits, the same parameter determines the group-level transitions. The design principle identified here serves as the basis for the control of cellular collective decision making.

1P274 Large deviation properties of population averages: An indicator of gene expression dynamics in a single cell

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Population of genetically identical cells are heterogenous in their phenotypes. Consider a model of gene expression network where activated genes produce mRNA and protein in a sequential manner. The output data of a collection of cells give us the mean signal which contains only partial information. Instead, we calculate the sample mean for a set of sub-population of cells, obtaining a distribution whose variance and large deviation properties are characterised by the underlying gene expression network. To perform this calculation, we obtain the probability generating function for chemical master equations for three simple regulatory networks and also exactly calculate the first and second moments in the steady state.

1P275 細胞システムの内因的・外因的ゆらぎに対するロバスト性に関する理論的基礎
Theoretical basis for robustness of intracellular systems against intrinsic and extrinsic fluctuation

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Intracellular systems, even with intrinsic stochasticity in intracellular reactions, can robustly and dynamically sustain their homeostatic state by flexibly adapting to unpredictably changing environment. The underlying mechanisms how this robustness and flexibility emerge from stochastic components have yet to be revealed. In this work, I demonstrate that Bayesian and information theories can be applied for unveiling the mechanisms by reframing the problem of the robustness as efficient information transfer in noise and uncertainty. I also clarify that the network that transfer information effectively behaves as if it utilizes noise and fluctuation. This result may serve as a theoretical basis for the origin of spontaneous activity in living cells.

1P276 イノシトールリン脂質代謝系が細胞の自発運動への効果の理論と実験による検証
Theoretical and Experimental Analysis for the Effect of Phosphatidyl Inositol System on Spontaneous Cell Movement

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We have analyzed the effect of phosphatidyl inositol system on spontaneous cell movement from single molecule to a cell level, experimentally and theoretically. In our experiment, PTEN mutants were used to modulate phosphatidyl inositol system. Then, the on and off rate of PTEN mutants were measured by using single molecule imaging to understand the mutation effect on membrane localization. Next, the localization of PTEN mutants, and spontaneous cell movement were measured. Finally, we have constructed a mathematical model, and we compared the model and experimental data (on and off rate, localization, and cell movement). As a result, our model explained experimental results. We believe our model becomes fundamental model to connect molecule and cell level.

1P277 間葉－アメーバ型遊走に関する理論モデル
A Theoretical Model for Mesenchymal-Amoeboid Modes for Migration

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Tumor cells exhibit two distinct modes for migration, mesenchymal and amoeboid modes, in three-dimensional environment. The Mesenchymal tumor type is similar to fibroblasts that synthesize the extracellular matrix. In contrast, the amoeboid type is rather similar to leukocytes that eliminate external microbes. Certain types of tumor cells can transform one mode to the other. To understand why the tumor cells exhibit two distinct modes, a theoretical model is introduced. The simulated cell on a flat substrate forms two distinct, amoeboid and keratocyte-like shapes by changing model parameters. In three-dimensional substrates, the amoeboid type cell again forms three-dimensional amoeboid shape but the keratocyte type transforms its body into fibroblast-like shape.