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Form-Function Relationship in E. coli Chemotaxis[1]

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Cell-to-cell variations in protein abundance in clonal cell populations are ubiquitous in living systems. Because protein composition determines responses in individual cells, it stands to reason that the variations themselves are subject to selective pressures. However, the functional role of these cell-to-cell differences is not well understood. One way to tackle questions regarding relationships between form and function is to perturb the form (e.g., change the protein abundances) and observe the resulting changes in some function. We take on the form-function relationship from the inverse perspective, asking instead what specific constraints on cell-to-cell variations in protein abundance are imposed by a given functional phenotype [1]. We develop a maximum entropy based approach to posing questions of this type and illustrate the method by application to the well-characterized chemotactic response in *Escherichia coli*. We find that full determination of observed cell-to-cell variations in protein abundances is not inherent in chemotaxis itself but, in fact, appears to be jointly imposed by the chemotaxis program in conjunction with other factors (e.g., the protein synthesis machinery and/or additional non-chemotactic cell functions, such as cell metabolism). These results illustrate the power of maximum entropy as a tool for the investigation of relationships between biological form and function.

[1] Sayak Mukherjee, Sang-Cheol Seok, Veronica J. Vieland, and Jayajit Das, Proceedings of National Academy of Sciences **110** 18531 (2013).

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