A mechanism for precision-sensing via a gradient-sensing pathway: A model of *E. coli* thermotaxis

Lili Jiang[1,2], Qi Ouyang[1], and Yuhai Tu[2]

***Short Abstract* — Thermotaxis is the phenomenon where an organism directs its movement towards its preferred temperature. So far, the molecular origin for this precision-sensing behavior remains a puzzle. Here we propose a model of *E. coli* thermotaxis and show that the precision-sensing behavior in *E. coli* can be carried out by the gradient-sensing chemotaxis pathway, that is, *E. coli* thermotaxis is achieved by the cryophilic and thermophilic responses for temperature above and below a critical temperature T_c, which is encoded by internal pathway parameters. Our model results are supported by both experiments with adaptation-disabled mutants and the recent temperature impulse response measurements for wild type cells.**

***Keywords* — thermotaxis, precision sensing, gradient sensing, modeling, adaptation.**

I. INTRODUCTION

One of the main challenges in modern biology is to understand the molecular mechanisms for multiple cellular processes. For *E. coli* thermotaxis, Imae et al. [1,2] found that the thermotactic behavior is governed by the same signaling pathway responsible for *E. coli* chemotaxis (see recent review [3]).

Imae et al. [2,4] shown that one major chemoreceptor Tar switches from warm sensing to cold sensing as receptor methylation level increases pass a critical level, which behaviorally cause cells to migrate to a particular temperature [5]. This is just precision-sensing, different from gradient-sensing in chemotaxis. Recently several quantitative measurements on thermotaxis [6,7] found that response to a temperature impulse is inverted as the base temperature increases pass certain critical value and *E. coli* thermotaxis behaviors depends strongly on the chemical background and growth condition.

In this paper we develop a general mathematical description of *E. coli* themotaxis by extending a model for chemotaxis to include temperature effects. Our model can not only explain the recent impulse response, the observed overshoot, but also can be used to study integration and interference between chemical and thermal stimuli. Finally we suggest the general mechanism for precision sensing may provide insights in studying other systems, such as thermotaxis in *C. elegans*.

[1]Center for Theoretical Biology and School of Physics, Peking University, Beijing, China. E-mail: bnujianglili@163.com

[2]IBM T.J. Watson Research Center, Yorktown Heights, New York, USA. E-mail: yuhai@us.ibm.com

II. RESULTS

Using the temperature dependent methylation energy in our model, we reproduce the inverted response at high methylation levels for cheRcheB mutants, in agreement with previous experiments [2].

For thermotaxis in wt *E. coli* cells, we apply a temperature dependent adaptation kinetics and know that the critical temperature is encoded in the adaptation process using steady state analysis. We use the same temperature profile as experiment [6] to show the inverted thermoresponse. Also our model explains the presence and absence of overshoot in temperature impulse response for base temperature below and above the critical temperature T_c respectively found in the experiment.

About chemical and thermal interference and integration, we predict that the critical temperature decreases with increasing attractant ligand concentration, as verified by direct simulation of our model. Also an increase of the ligand/receptor dissociation constant with temperature can further decrease the critical temperature.

III. CONCLUSION

In this paper we have developed a quantitative model for *E. coli* thermotaxis by introducing temperature effects in the chemotaxis pathway. Their adaptation processes differ significantly, perfect adaptation in chemotaxis and imperfect adaptation in thermotaxis, allowing the system to carry out both precision sensing (of temperature) and gradient sensing (of ligand concentration).

REFERENCES

[1] Maeda K, Imae Y (1979) Thermosensory transduction in *Escherichia coli*: inhibition of the thermoresponse by L-Serine. PNAS 76, 91-95

[2] Mizuno T, Imae Y (1984) Conditional inversion of the thermoresponse in *Escherichia coli*. J Bacteriol 159, 360-367

[3] Baker MD, Wolanin PM, Stock JB (2006) Signal transduction in bacterial chemotaxis. Bioessays 28, 9-22

[4] Nishiyama SI, Umemura T, Nara T, Homma M, Kawagishi I (1999) Conversion of a bacterial warm sensor to a cold sensor by methylation of a single residue in the presence of an attractant. Mol Microbiol 32, 357-365

[5] Maeda K, Imae Y, Shioi JI, Oosawa F (1976) Effects of temperature on motility and chemotaxis of *Escherichia coli*. J Bacteriol 127, 1039-1046

[6] Paster E, Ryu WS (2008) The thermal impulse response of *Escherichia coli*. PNAS 105, 5373-5377

[7] Salman H, Libchaber A (2007) A concentration-dependent switch in the bacteria response to temperature. Nat Cell Biol 9, 1098-110