Thermal impulse response and the temperature preference of *Escherichia coli*

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Short Abstract — Swimming *Escherichia coli* responds to changes in temperature by modifying its motor behavior. Previous studies using populations of cells have shown that *E. coli* accumulate in spatial thermal gradients, but these experiments did not cleanly separate thermal responses from chemotactic responses. Here we have isolated the thermal response by studying the behavior of single, tethered cells. The motor output of cells was measured in response to small, impulsive increases in temperature, delivered by an infrared laser, over a range of ambient temperature (23° to 43°C). The thermal impulse response at temperatures < 31°C is similar to the chemotactic impulse response: Both follow a similar time course, share the same directionality, and show biphasic characteristics. At temperatures > 31°C, some cells show an inverted response, switching from warm- to cold-seeking behavior. The fraction of inverted responses increases nonlinearly with temperature, switching steeply at the preferred temperature of 37°C.

***Keywords* — Sensory behavior, Thermotaxis, *E. coli*, Locomotion**

Like chemotaxis, thermotaxis is widespread among organisms, both prokaryotic and eukaryotic. Previous studies of bacterial thermotaxis have been difficult to interpret because the experiments were done with populations of cells [1-2]. The problem is that the cells themselves produce chemical and oxygen gradients and this complicates the study of the thermal response. Here we have used the tethered-cell assay to measure the thermal impulse response of bacteria [3]. Since our experiments stimulate and measure responses from single cells, we have cleanly decoupled the thermal response from the chemotactic response.

Our main finding is that the thermal preference of bacteria will steeply switch from warm-seeking to cold-seeking at 37 deg. C. We also have shown that the thermosensory system is robust to temperature changes yet still remains sensitive to thermal stimuli throughout its physiological temperature range. And finally, we have discovered that the normal and inverted impulse response have different time courses, indicating that the computations the cell is making in these temperature regimes are fundamentally different.

From a broad perspective, exposure to environmental temperature changes is a universal condition of living organisms, and in principle every step of a biochemical signaling system is temperature dependent. Enzymatic rates are generally exponential functions of temperature and typical Q10 values (increase in enzymatic rate per 10°C) are from 1.2 – 2.5, implying rate increases from 2 – 16 fold over 30°C. This wide variation of reaction rates presents an obvious challenge for robust design, particularly for systems that need to transmit quantitative information, e.g. signaling systems. Our results show that the thermotactic network is buffered against temperature changes, but still is able to respond to thermal stimuli. Understanding how the bacterial thermotactic network remains robust and yet responsive to changes in temperature will likely lead to a broader understanding of how temperature compensation is achieved in general.

References

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