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How eukaryotic phagocytes locate and interact with microorganisms: Lessons from the social amoeba *Dictyostelium discoideum*

Tow eukaryotic cells find and interact with bacteria is a fundamental question in biology. Eukaryotic phagocytes and their interactions with bacteria began when single-celled life forms, protozoans, appeared about 2.5 billion years ago. Since then, multicellular organisms endowed with increasingly complex genomes gradually formed and phagocytic cells from these organisms, such as invertebrates and vertebrates, patrol in a host body to detect, recognize and eliminate invading pathogenic bacteria for host immunity. The current dogma is that phagocytic cells use at least two types of receptors for defense against invading pathogens: One for detecting and chasing pathogens via chemotaxis and another for recognizing and eliminating them via phagocytosis. Detection and chasing is facilitated by G-protein-coupled receptors which sense diffusible chemo-attractants derived from bacteria. Recognition and elimination employs Pattern-Recognition Receptors (PRRs), such as Toll-like receptors, for recognizing Microbial-Associated Molecular Pattern (MAMPs) and/or phagocytic receptors for bacterial surface-bound complements or immunoglobulins. However, the social amoeba Dictyostelium discoideum does not encode orthologs of any known PRRs or phagocytic receptors; yet, they are highly evolved as professional phagocytes that chase bacteria via chemotaxis and consume them as food through phagocytosis. We find that this stereotypical phagocyte, breaking the dogma, assembles a simple and elegant molecular machinery to detect a diffusible chemoattractant



Figure-1: A model shows that a chemoattractant GPCR recognizes diffusible chemical for cell migration and binds immobilized ligands on the bacterial surface for engulfment.

and recognize an immobile component on the bacterial coat for both chasing and engulfing bacteria. Our studies on the social amoeba *Dictyostelium discoideum* sheds new light on the origin of bacterial recognition by eukaryotic phagocytes, the path through which PRRs evolved and the unexpectedly close mechanistic connection between chemotaxis and phagocytosis.

Recent Publications

- 1. Gera N, Swanson K D and T Jin (2017) β -Arrestin 1-dependent regulation of Rap2 is required for fMLP-stimulated chemotaxis in neutrophil-like HL-60 cells. *J Leukoc Biol*; 101(1): 239-251.
- 2. Xu X, Wen X, Veltman, Keizer-Gunnink I, Pots H, D M, Kortholt A and T Jin (2017) GPCR-controlled membrane recruitment of negative regulator C2GAP1 locally inhibits Ras for adaptation and long-range chemotaxis. *PNAS*; 114 (47):

Biography

Tian Jin is a Senior Investigator and Chief of the chemotaxis signal section in Laboratory of Immunegenetics, NIAID, NIH. His work focuses on molecular mechanisms underlying chemotaxis and phagocytosis.

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