**Cholesterol assembles armies of Motor proteins on Phagosomes**

Cells of our immune system engulf pathogens by enclosing them in a compartment called the phagosome. The phagosome undergoes programmed maturation, where the pathogen is degraded by acidification. Intimately linked to this degradation is active transport of the phagosome inside cells by motor proteins. Early phagosomes move in back-and-forth manner near the cell periphery, and mature by fusing with other compartments during this period. In contrast, late phagosomes move in almost unidirectional manner towards the cell centre. This early-to-late phagosome switch is important for the degradation of pathogens. Mycobacterium Tuberculosis and Salmonella can abort this switch.

We have investigated the mechanism of this switch using fluorescence imaging, protein biochemistry, lipid
chromatography, detergent resistant membrane isolation, drug-induced interference and optical trap based force measurement. Our results suggest that the "switch" in a phagosome's fate is because of the formation  of cholesterol-rich domains called lipid rafts on the phagosome membrane. Motors cluster into these raft-like domains, and by doing so are able to work cooperatively in large teams. This cholesterol induced activation of motors transports the phagosome towards acidic lysosomes for degradation. I will discuss the biophysical evidence for this clustering, the biochemical mechanisms by which it may be effected and what it implies for phagosome biogenesis.