

Role of mitochondria-associated membrane (MAM) in *Legionella* infection



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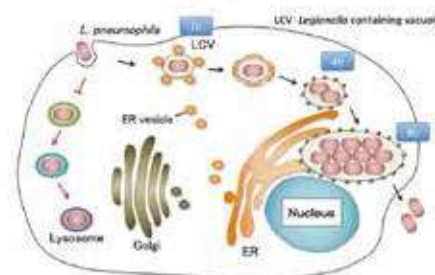
Research summary

Many microbes and viruses create intracellular environments advantageous for their survival and growth by hijacking host physiological machinery. Recent studies have revealed that some microbes and viruses manipulate function of organelle contact sites such as the ER (endoplasmic reticulum)-mitochondria contact site. In this project, I will try to understand the role of organelle contact sites, in particular, the ER-mitochondria contact site in intracellular pathogenesis of *Legionella pneumophila* that is known to cause severe pneumonia.

Figure

Intracellular pathogenesis of *Legionella pneumophila*.

After uptake into the host via phagocytosis, *Legionella pneumophila* prevents its degradation by inhibiting the delivery to lysosome. Simultaneously, Legionella recruits host ER-derived vesicles to the Legionella-containing vacuole to convert it into ER-Golgi intermediate compartment like structures, and then the pathogen-occupied membrane fuse with the ER and *Legionella* start to replicate.



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