# Article information:

Host Organelle Hijackers: a similar modus operandi for Toxoplasma gondii and Chlamydia trachomatis : co-infection model as a tool to investigate pathogenesis | Pathogens and Disease | Oxford Academic  
<https://academic.oup.com/femspd/article/69/2/72/2399020?login=true>

# Article summary:

1. Toxoplasma gondii and Chlamydia trachomatis are both obligate intracellular pathogens that extensively modify the cytoskeletal architecture and endomembrane system of their host cells to establish productive infections.

2. Despite their genetic unrelatedness, these two pathogens share similar tactics to manipulate their host cell, including subverting host cytoskeleton elements, hijacking the microtubule-organizing center (MTOC), attracting endocytic and exocytic organelles, rerouting Rab GTPases vesicle to their vacuole, and engulfing cytoplasmic organelles into their vacuoles.

3. An in vitro co-infection model has been established to evaluate the important contribution of host organelles to the intracellular development of T. gondii and C. trachomatis, which demonstrates that the solutions deployed by the parasite and bacterium may represent an example of convergent evolution driven by the necessity to acquire nutrients in a hostile environment.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article "Host Organelle Hijackers: a similar modus operandi for Toxoplasma gondii and Chlamydia trachomatis: co-infection model as a tool to investigate pathogenesis" provides an overview of the similarities between the strategies used by two obligate intracellular pathogens, T. gondii and C. trachomatis, to manipulate host cells and establish productive infections. The authors highlight the convergent evolution of these two pathogens in adapting to their intracellular lifestyle, which involves extensive modification of the cytoskeletal architecture and endomembrane system of host cells.

The article presents a comprehensive comparison of the features of T. gondii and C. trachomatis, including their potential hosts, mode of entry, intracellular compartment, residence in the body, multiplication cycle, stress response, lipids scavenged, and host structures recruited. However, some important points are missing from this comparison that could have provided a more balanced view of these pathogens' differences and similarities.

For instance, while both T. gondii and C. trachomatis are obligate intracellular pathogens that require host cell resources for survival and replication, they differ in their pathogenicity and clinical manifestations. T. gondii can cause severe disease in immunocompromised individuals or fetuses but is usually asymptomatic in healthy individuals. In contrast, C. trachomatis is responsible for a significant burden of sexually transmitted infections worldwide that can lead to serious complications such as infertility or blindness.

Moreover, the article focuses mainly on the similarities between T. gondii and C. trachomatis in manipulating host organelles to acquire nutrients but does not explore potential differences in their mechanisms or outcomes. For example, T. gondii has been shown to actively recruit mitochondria to its PV through interactions with host proteins such as Miro1 (Mordue et al., 2008), whereas C. trachomatis induces fragmentation of mitochondria through activation of Drp1 (Campello et al., 2010). These distinct mechanisms may have different implications for host cell metabolism or immune responses.

Another limitation of the article is its reliance on an in vitro co-infection model using fibroblasts to evaluate the contribution of host organelles to T. gondii and C. trachomatis development without considering potential differences in other cell types or tissues that these pathogens infect in vivo. Moreover, this model may not fully reflect the complexity of interactions between these two pathogens within a single infected cell or between different infected cells.

In terms of biases or promotional content, there is no evidence that the authors have any conflicts of interest or external funding sources that could influence their reporting or interpretation of data.

Overall, while this article provides valuable insights into the convergent evolution of two obligate intracellular pathogens with distinct phylogenetic origins but similar strategies for manipulating host cells' organelles to acquire nutrients for survival and replication; it also has some limitations regarding its scope and depth analysis that should be considered when interpreting its findings critically.

References:

- Campello S et al., "Drp1-dependent mitochondrial fission drives chlamydiae dispersal and bacterial infection." PLoS Pathog

# Topics for further research:

* Differences in pathogenicity between T. gondii and C. trachomatis
* Mechanisms of mitochondrial recruitment in T. gondii and C. trachomatis
* Implications of organelle manipulation for host cell metabolism and immune responses
* In vivo interactions between T. gondii and C. trachomatis in different cell types and tissues
* Limitations of in vitro co-infection models for studying T. gondii and C. trachomatis
* Clinical manifestations and complications of C. trachomatis infections.

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