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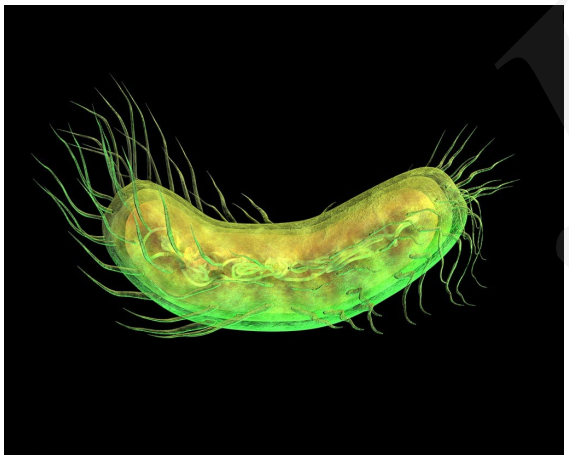
Pseudomonas aeruginosa-induced mitochondrial dysfunction inhibits proinflammatory cytokine secretion and enhances cytotoxicity in mouse macrophages in a reactive oxygen species (ROS)-dependent way

Key words: ***Pseudomonas aeruginosa***; **Mitochondria; Mitophagy; Proinflammatory cytokines**

Research Summary

This study provides insight into the role of mitochondrial dysfunction in the host regulation of *Pseudomonas aeruginosa* infection, and will provide reference for formulating targeted treatment strategies for *P. aeruginosa* infection.



- ***P. aeruginosa* infection**
- **Mitochondrial dysfunction**
- **Proinflammatory cytokine secretion**
- **A ROS-dependent way**

Innovation points

A series of comprehensive figures were generated to the molecular mechanisms of *P. aeruginosa* infection in the host

Figure 1 | *P. aeruginosa* induces mitophagy and mitochondria dysfunction in mouse peritoneal macrophages.

Figure 2 | *P. aeruginosa* clears the production of ROS in mouse peritoneal macrophages by inducing mitophagy.

Figure 3 | Mitophagy-mediated reduction of ROS inhibits *P. aeruginosa*-induced secretion of inflammatory cytokines in mouse peritoneal macrophages.

Figure 4 | Mitophagy-mediated reduction of ROS enhances *P. aeruginosa* infection-induced toxicity in mouse peritoneal macrophages.

Figure 5 | Activation of mitophagy aggravates *P. aeruginosa* infection in mice.