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Response of *Caenorhabditis elegans* during *Klebsiella pneumoniae* pathogenesis

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Background

Caenorhabditis elegans, owing to its amenability and conserved innate immune system was recognized as an emerging model to dissect the molecular basis of many mammalian infections. *Klebsiella pneumoniae* causes severe infections in immunocompromised patients. Hence, the host response was studied using *C. elegans* during *K. pneumoniae* infection.

Methods

Pathogenicity of *K. pneumoniae* and its LPS were assessed by variety of physiological and biochemical assays. The qPCR was used to analyze the regulation of innate immune genes.

Results

K. pneumoniae and its LPS were lethal to *C. elegans* and required 48±5hours and 24±3hours for complete killing, respectively, with cessation of pharyngeal pumping and egg laying. Infection with *K. pneumoniae* increased the bacterial load in the intestine of host upon course of infection, which was measured as $1.5x10^3$, $2.2x10^3$, $3.6x10^4$ and $3.7x10^5$ in 4,6,12 and 24hours, respectively. This increased bacterial load subsequently disseminates oxidative stress markers in host. The level of ROS was measured to increase by 24.36597nM, 35.60517nM, 39.34052nM, and 28.24774 nM/mg of protein in 6, 12, 24, and 36 hours, respectively. Infection by *K. pneumoniae* also increased the protein carbonyls to 25.57nM, 36.14nM, 35.26nM and 38.84nM/mg of protein in 6, 12, 24, and 36 hours, respectively. *K. pneumoniae* and its LPS suppressed the expression of *pmk*-1, to l-1 and antimicrobial peptides (clec-60, clec-85, clec-87, lys-1, and lys-7) and thus succumbed the host by upregulated expression of virulent genes such as *uge*, *rmpA* and *oxyR*.

Conclusion

The interplay between *K. pneumoniae* and *C.elegans* in the present study further provides more insights into the mechanism of host defense against this pathogen.

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