

BACTERIAL INFECTIONS and ALZHEIMER'S DISEASE: Insights from *Caenorhabditis elegans*

P3-06

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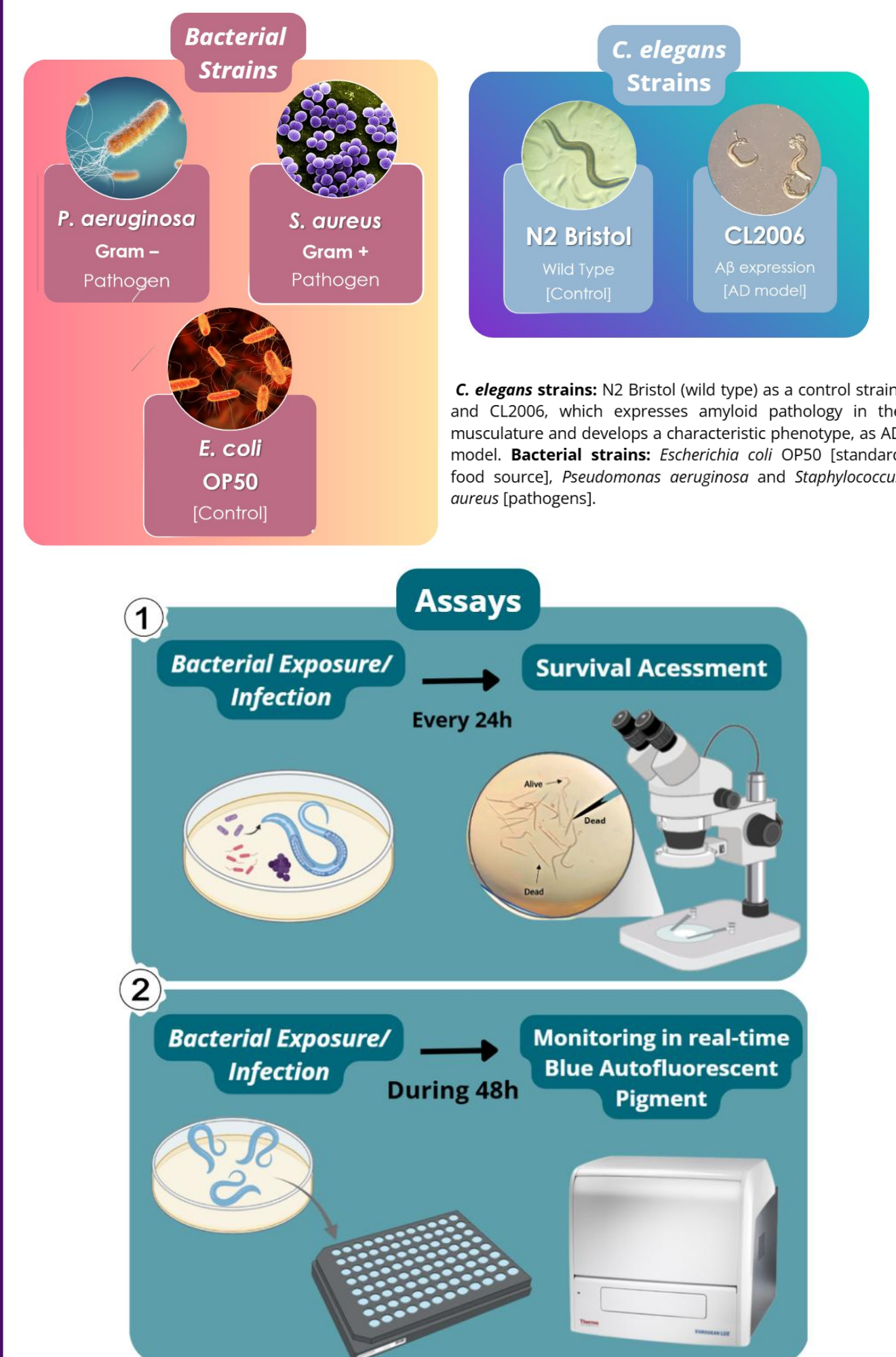
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INTRODUCTION

The microbial hypothesis has gained attention as a risk factor, proposing that infections may contribute to AD development by triggering neuroinflammation, disrupting proteostasis, and accelerating A β aggregation. The nematode *Caenorhabditis elegans* is a suitable model to investigate this hypothesis due to its genetic tractability, conserved molecular pathways, and suitability for studying host–pathogen interactions. Through *C. elegans*, it is possible to explore how pathogen exposure influences amyloid aggregation and neurodegenerative processes, providing insights into the potential role of bacteria in AD pathogenesis.

METHODS



Assays - Two experimental approaches were applied: **(1)** bacterial exposure/infection on solid culture medium observed every 24 hours under a stereomicroscope to assess live and dead worms. **(2)** Bacterial exposure/infection was performed in black 96-well plates containing liquid medium, and the blue autofluorescent pigment produced by the worms was monitored in real time for 48h, with measurements acquired every 5 min using a Varioskan Lux spectrophotometer (Thermo Fisher Scientific).

RESULTS

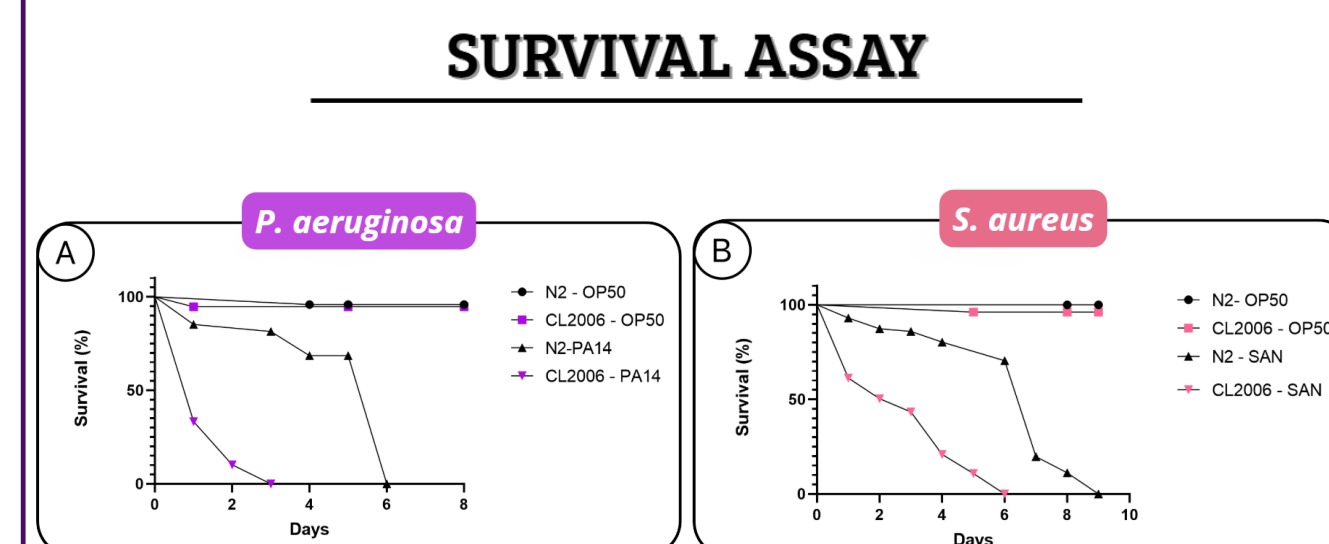
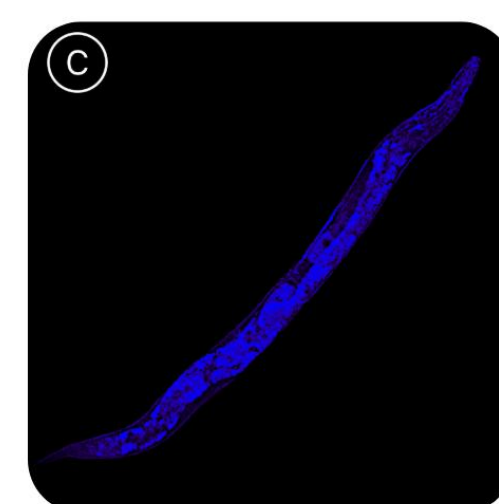
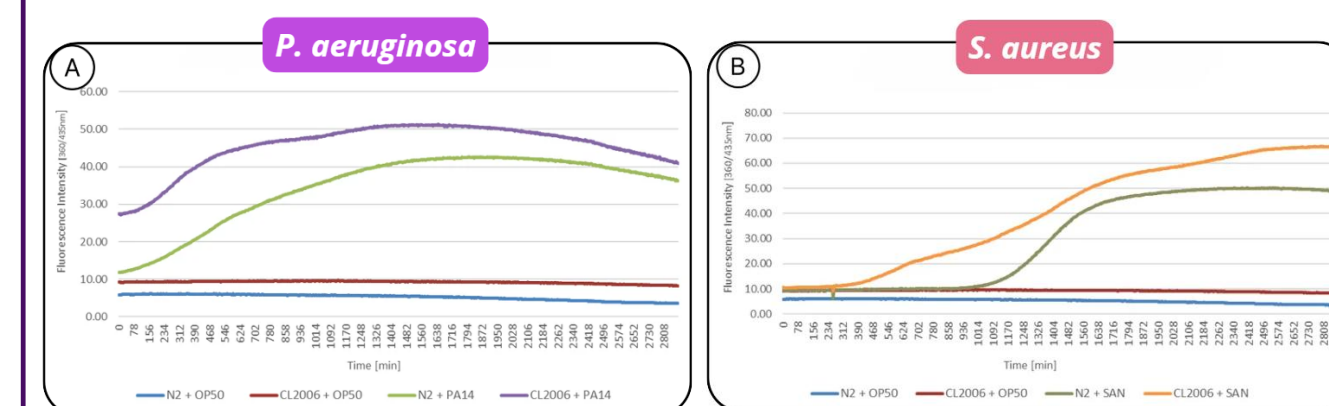


Figure 1 – **SURVIVAL ASSAY:** Infection with **(A) P. aeruginosa** showed a significant reduction on survival of CL2006 [ΔB] strain. At the first 24 hours of exposure there is less than 50% of alive animals and at the third day, 100% death. In contrast, the N2 (wild type) strain maintained survival rates above 50% until the sixth day of exposure, when the population was fully eliminated. Infection with **(B) S. aureus** showed a significant reduction on survival of CL2006 [ΔB] strain. At the first 72 hours of exposure there is less than 50% of alive animals and at the sixth day, 100% death. In turn, strain N2 (wild type) maintained survival rates above 50% until the sixth day of exposure, when a significant drop occurred, reaching 100% mortality on the ninth day. Three independent assays were performed containing 25 animals per plate, in quadruplicate (n=160/group)

BLUE AUTOFLUORESCENT PIGMENT ASSAY



BLUE AUTOFLUORESCENT PIGMENT ASSAY: (A) *P. aeruginosa* showed a marked increase in pigment levels from the early stages of exposure, compared to non-infected groups. CL2006 [Aβ] strain had the highest pigment levels, peaking around 24 hours of exposure. (B) *S. aureus* showed a progressive increase of fluorescence intensity in CL2006 [Aβ] strain, with higher levels at 48h of exposure. The N2 strain showed levels similar to those of the non-infected groups in the early stages of exposure, with a marked increase after 24 hours of exposure. Three independent assays were performed containing 100 animals per well at least in four replicates ($n=1200/\text{group}$). (C) Representative fluorescence microscopy image illustrating the blue autofluorescent pigment produced by the worms, captured using the EVOS Imaging System MS000.

CONCLUSIONS

These initial findings indicate heightened susceptibility and a potential acceleration of A β aggregation in response to bacterial infections. The ongoing work focuses on real-time monitoring and quantification of reactive oxygen species (ROS) production during pathogen-induced cellular stress. In addition, A β aggregation and plaque deposition will be evaluated using specific dyes and antibodies for microscopy and western blotting techniques, allowing direct investigation of how pathogenic bacteria influence A β production and aggregation.

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CONTACT



Contact the author for collaborations, data, and additional information.