

The Effect of *Uncaria tomentosa* (Cat's Claw) and Gut Microbiome Dysbiosis Repair With *Bacillus subtilis* in Locomotion and Alpha Synuclein Aggregation in *Caenorhabditis elegans* Transgenic Strain NL5901, an Animal Model for Parkinson's Disease

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Background: Parkinson's disease (PD) is a neurodegenerative disease caused by degeneration of dopaminergic neurons due to alpha-synuclein (AS) aggregation. Microbiome dysbiosis has been recognized as a possible mechanism leading to AS aggregation. *Caenorhabditis elegans* (CE), is an excellent PD animal model. Transgenic NL5901 CE possess an AS mutation in its muscle cells, generating AS aggregation and impairing worm's locomotion. Prior experiments have shown AS anti-aggregating effects of *Bacillus subtilis* (BS) and *Uncaria tomentosa* (UT) in CE NL5901. Hypothesis: CE NL5901 cultured with BS and treated with UT will have higher improvements in locomotion and reduction in AS aggregation compared with cohorts treated without BS and not treated with UT. Methods: 120 worms (60 WT and 60 NL5901 strain) were divided in 12 cohorts of 10. These cohorts had worms exposed to BS, EC, BS/EC and exposed vs non-exposed to UT. Worms locomotion was measured at days 0, 1, 3, and 6. AS aggregation was measured at day 6 post-treatment. Results: At day 6, the CE NL5901 cohorts treated with BS, UT or both had better locomotion and lower levels of aggregated AS compared with other cohorts. Combined treatment with UT and BS had the highest anti-AS aggregating effect. Conclusions: Treatment with BS and UT enhances locomotion and lowers AS aggregation in CE NL5901 strain. Applications: This research increases our knowledge regarding the AS anti-aggregating effect of BS (restoring microbiome dysbiosis) and UT in an animal model for PD, which may contribute to the search of neuroprotective medications in PD patients.

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