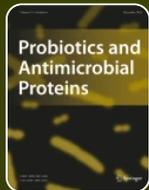


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Pediococcus pentosaceus LAB6- and *Lactiplantibacillus plantarum* LAB12-Derived Cell Free Supernatant Inhibited RhoA Activation and Reduced Amyloid-B In Vitro

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Abstract

Alzheimer's disease (AD) is characterized by aggregation of amyloid beta (A β) plaque. RhoA may serve as a potential target for prevention against AD given its role in the

amyloidogenic pathway. The recent emergence of the gut–brain axis has linked lactic acid bacteria (LAB) to neuroprotection against AD. This study assessed the importance of RhoA inhibition in mediating the neuroprotective potential of LAB. To this end, de Man, Rogosa and Sharpe (MRS) broth fermented by lactobacilli or pediococci were tested against SK–N–SH (a human neuroblastoma cell line) in the presence of RhoA activator II for 24 h after which the RhoA activity was measured using the G–LISA Kit. Fluorescence staining of f-actin stress fibres was performed to validate RhoA inhibition. SK–N–SH was transfected with plasmid expressing amyloid precursor protein (APP) gene. The A β concentration in transfected cells exposed to LAB–derived cell free supernatant (CFS) in the presence of RhoA activator II was measured using the ELISA kit. Furthermore, this study measured organic acids in LAB–derived CFS using the gas chromatography. It was found that LAB–derived CFS yielded strain–dependent inhibition of RhoA, with LAB6– and LAB12–derived CFS being the most potent *Pediococcal*– and *Lactiplantibacillus*–based RhoA inhibitor, respectively. Lesser stress fibres were formed under treatment with LAB–derived CFS. The LAB–derived CFS also significantly inhibited A β in SK–N–SH transfected with APP gene in the presence of RhoA activator II. The LAB–derived CFS was presented with increased lactic acid, acetic acid, butyric acid and propionic acid. The present findings warrant in–depth study using animal models.

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