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The Attenuating Effects of 1,2,3,4,6 Penta-O-Galloyl-β-D-Glucose (PGG) on the Expression of Proteins Involved in Alzheimer's Disease in LPS/IFNγ Activated BV-2 Microglial Cells.

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Alzheimer's disease (AD) is a chronic and progressive neurodegenerative disorder associated with memory and cognition impairment. The main pathological hallmarks are abnormal intraneuronal cytoskeletal changes, known as neurofibrillary tangles, and the extracellular protein deposits of amyloid beta plaques (Aβ). Although the currently available drugs for AD do not prevent or reverse the formation of Aβ deposits, medicinal plants have been reported for possible anti-AD activity and are promising therapeutic agents for neurodegenerative disorders. 1,2,3,4,6-Penta-O-galloyl-β-D-glucose (PGG), which is a naturally occurring polyphenolic compound, has shown to exhibit anti-aggregation activity on Alzheimer's Aβ proteins. Its oral intake reduced Aβ plaque burden and Aβ peptide content in brain tissue of transgenic mice. In this study, we investigated the effect of PGG on the expression of proteins involved in neurodegenerative diseases in LPS/IFNγ activated BV-2 microglial cells. Proteomic results identified 17 proteins whose expression level was significantly downregulated by PGG, including septin-7, ataxin-2, and adenylosuccinate synthetase isozyme 2 (ADSS). These proteins were previously described as being expressed in neurodegenerative diseases and/or involved in the Wnt signaling pathway, which is associated with the development of neuronal circuits and control of AD pathogenesis. PGG inhibitory effect on ataxin-2, septin-7, and ADSS was confirmed at the protein and transcription level, and the proteins were classified according to biological processes and molecular functions using NCBI and PANTHER databases. Since these selected proteins are involved in synapse impairment induced by Aβ protein, they are interesting candidates for possible involvement in the underlying mechanisms of synaptic dysfunction in neurodegenerative diseases. Therefore, these results suggest that PGG may have a role in halting neuropathologic effects of AD by targeting ataxin-2, septin-7, and ADSS.

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