Neuroprotective effect of fermented spent coffee grounds extract using SH-SY5Y human neuronal cell model

王建薇 (5120)

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Outline

1. Introduction
2. Fermented SCG hydrolysate by lactic acid bacteria
3. SCG ferments extracted with different ethanol concentrations
4. Neuroprotective effects on SH-SY5Y human neuronal cells
5. Conclusion

Abstract

Parkinson's disease (PD) is a neurodegenerative movement disorder caused by dopamine deficiency, α-synuclein misfolding, mitochondria dysfunction, and oxidative stress. Hydroxydopamine (6-OHDA) is a dopamine neurotoxin that induces the production of reactive oxygen species (ROS), leading to oxidative stress and cell damage or death, and it's often used to induce PD cell patterns. Spent coffee grounds (SCG) contain chlorogenic acid, which protects neuronal cells against oxidative damage caused by H_2O_2. This study investigated the protection of fermented SCG against oxidative stress caused by H_2O_2 and 6-OHDA. Seven strains of lactic acid bacteria fermented SCG hydrolysate using xylanase, and FKR2526 and FLC2528 showed the highest growth counts of 9.11 and 9.16 log CFU/mL, respectively. FLC2528 had the highest total phenol and total flavonoid content, with a 46% and 17.38% increase, respectively, compared to unfermented SCG hydrolysate. The fermented SCG freeze-dried powders of FKR2526 and FLC2528 were extracted using microwave-assisted ethanol extraction at different concentrations. The highest concentration of total phenols and total flavonoids was observed in the 20% ethanol extraction. The fermented SCG extracts significantly increased the viability of H_2O_2- or 6-OHDA-treated SH-SY5Y cells. They also increased intracellular superoxide dismutase (SOD) activity (63.72-129.02%), glutathione peroxidase (GPx) activity (13.10-23.93%), and catalase (CAT) activity (36.57-98.55%). Additionally, they significantly reduced intracellular acetylcholinesterase (AChE) activity (14.21-34.28%). In summary, SCG xylanase enzyme hydrolysate fermented by lactic acid bacteria FKR2526 and FLC2528 could increase total phenol and total flavonoid content and protect against H_2O_2- and 6-OHDA-induced oxidation by increasing intracellular SOD and CAT activity and inhibiting AChE activity, with the potential for neuroprotective effects and prevention of PD.

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