

Opinion





Role of green tea as anti-oxidative stress agent in neurodegenerative diseases

Abstract

Aims and Scopes: Alzheimer's disease (AD) and Parkinson are common kind of progressive neurodegenerative diseases of the aged brain, many promising chemicals have failed because of therapeutic limitations i.e. only making symptomatic relief. Antioxidant system, is known in etiology of these. The imbalance may originate from an overproduction of free radicals or a reduction in antioxidant defenses. Green tea has been proven as anti-oxidative herbal agent in prevention of some neurodegenerative disease as parkinson, alzheimer and depression, antioxidative properties of green tea belong to polyphenol compounds as cathechins. According to these backgrounds, this mini-review has been prepared.

Keywords: alzheimer, green tea, passive avoidance, oxidative stress, cathechins

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Introduction

Neurodegenerative diseases are defined as functional loss or dysfunction of nerve cells in brain and spinal cord. The most important neurological disease in aging as Alzheimer's disease (AD) has been shown with mitochondrial dysfunctions, excitotoxicity and finally apoptosis.1 Disturbance of pro-oxidant/antioxidant homeostasis brings oxidative stress that could make further Reactive oxygen species (ROS) generation in neurons. The brain is with high rate of oxidative activity and high polyunsaturated fatty acids and low antioxidant capacity, so it is very susceptible to oxidative damage.^{2,3} It has been shown that mitochondrial damage has an important role in the pathogenesis of AD.4 Several researches demonstrated that various xenobiotic increase risk of AD by mitochondrial dysfunction or oxidative stress.^{5,6} Hence protection of mitochondria and reducing oxidative damage has been considered as a therapeutic for AD.^{1,4} Today, attentions focused on the potential of neuroprotective effects of flavonoids against the neuronal deficits associated with age-related neurodegenerative diseases.7

Green tea as anti-oxidant

Green tea constitutes some active materials rich in flavonoids as Cathechins, Epicathechin (EC) and Epigalocathechins. Cathechins are absorbed readily by oral and are stable in gastric and intestinal areas (81) Studies have shown that green tea usage can reduce risk of neurodegenerative disease as Parkinson. Also Epi-Galocathechins in green tea can protect dopaminergic neurons against toxicity by MPTP. Flavanols, such (-)-epicatechin (EC), are major class of flavonoids which are commonly in some plants as Camellia sinensis (green tea. Besides, studies in animals using EC extracts, demonstrated positive effects on decreasing of oxidative stress, and enhancing cognitive function and memory performance. In one study, we have shown the beneficial effects of EC on oxidative stress and mitochondrial damage induced by Hcy using isolated rat hippocampus mitochondria in vivo.

Also in one of our previous studies we have shown that Curcumin as another herbal anti-oxidant can prevent lipid peroxidation induced by homocysteine in rat hippocampus significantly and decreased MDA and SOD levels.¹²

Epicathechin

Epicathechin(EC) is a flavonoid, in green and black tea extract.¹³ It has been shown that the bioavailability of EC is greater than of other catechins in rat.14 Also this flavonoid can transfer the bloodbrain-barrier after oral ingestion easily. 15 According some DATA, EC could inhibit cell death induced by hydrogen proxide.¹³ Also, it had radical scavenging activity.16 It was suggested that Hcy which can accumulated during some neurodegenerative disease might generate reactive oxygen species which could attacks the poly unsaturated fatty acids of neuronal cell membranes and induces lipid peroxidation in the hippocampus.¹⁷ Moreover, with some studies, EC could cause a significant decrease in lipid peroxidation and reactive oxygen species. 1,7,18 In our previous studies, we found that administration of EC significantly decreased the rate of ROS production, induced by Hey in hippocampus isolated mitochondria of rat. Furthermore, It has been proved that EC and epigallocatechin gallate were more potent than catechin as neuroprotectants. According to its simpler structure and more efficiency for blood-brain barrier penetration, EC might be the best therapeutic which can be candidate for neurodegenerative diseases.19

Assessment of Adverse effects of human studies with various green tea preparations or EGCG which monitored safety of green tea have shown that the most prevalent adverse effects were gastrointestinal irritations and hepatotoxicity which occurred at a low rate.²⁰ Also evidences have not shown any genotoxic or carcinogenic effect based on the results of carcinogenicity and genotoxicity assays.²⁰ The results of some analyses, have shown that the composition of green tea traditional preparations is safe. Preparations of concentrated extracts,



containing high levels of constituents, as EGCG, which consumed in solid dosage form, may require health-based guidance to assure their safety especially with considering hepatotoxicity as critical effect, for adults with normal liver function.²⁰

Conclusion

According to results of many studies it can be suggested that green tea according to its' antioxidant properties, can be useful for people at risk of neurodegenerative disease and Cathechins which are in high constituent in green tea might be with much propensity of responsibility for these protective effects.

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None.

Conflict interest

None.

References

- Shaki F, Shayeste Y, Karami M, et al. The effect of epicatechin on oxidative stress and mitochondrial damage induced by homocycteine using isolated rat hippocampus mitochondria. *Res Pharm Sci.* 2017;12(2):119–127.
- Massimo Collino MA, Mastrocola R, Gallicchio M, et al. Modulation of the oxidative stress and inflammatory response by PPAR-γ agonists in the hippocampus of rats exposed to cerebralischemia/reperfusion. Eur J Pharmacol. 2006;530(1):70–80.
- Du H, Guo L, Yan S, et al. Early deficits in synaptic mitochondria in an Alzheimer's disease mouse model. *Proc Natl Acad Sci USA*. 2010;107(43):18670–5.
- Agnati L, Genedani S, Rasio G, et al. Studies on homocysteine plasma levels in Alzheimer's patients. Relevance for neurodegeneration. *J Neural Transm (Vienna)*. 2005;112(1):163–9.
- 6. Cuevas E, Limón D, Pérez Severiano F, et al. Antioxidant effects of epicatechin on the hippocampal toxicity caused by amyloid-beta 25-35 in rats. *Eur J Pharmacol*. 2009;616(1):122–7.
- Elvis Cuevas DL, Perez severiano F, Diaz A, et al. Antioxidant effects of Epicatechin on the hippocampal toxicity caused by Amyloid – beta 25 – 23 in rats. Eur J Pharmacol. 2009;616(1-3):12–7.
- Ramassamy C, Poirier J .Ginkgo biloba extract (EGb 761) and apolipoprotein E isoforms on the β-amyloid fibril formation. In: Christen Y, editor. Ginkgo Biloba Extract (EGb 761) and Neurodegenerative Diseases. 2001:71–91.

- Checkoway H, Powers K, Smith Weller T. Parkinson's disease risks associated with cigarette smoking, alcohol consumption, and caffeine intake. Am J Epidemiol. 2002;155:732–38.
- Levites Y, Amit T, Mandel S. Neuroprotection and neurorescue against Abeta toxicity and PKC-dependent release of nonamyloidogenic soluble precursor protein by green tea polyphenol (–) epigallocatechin-3-gallate. FASEB J. 2003;17(8):952–54.
- Manuel Gómez Guzmána MS, María José Zarzueloa, Pilar Galindoa, et al. Epicatechin lowers blood pressure, restores endothelial function, and decreases oxidative stress and endothelin-1 and NADPH oxidase activity in DOCA-salt hypertension. Free Radic Biol Med. 2012;52(1):70–9.
- 12. Ataie A, Sabetkasaei M, Haghparast A, et al. Neuroprotective effects of the polyphenolic antioxidant agent, Curcumin, against homocysteine-induced cognitive impairment and oxidative stress in the rat. *Pharmacol Biochem Behav*. 2010;96(4):378–85.
- Spencer JP, Schroeter H, Crossthwaithe AJ, et al. Contrasting influences ofglucuronidation and O-methylation of epicatechin on hydrogen peroxide-induced cell death in neurons and fibroblasts. Free Radic Biol Med. 2001;31:1139–46.
- Baba S, Osakabe N, Natsume M, et al. In vivo comparison of the bioavailability of (+)-catechin, (-)-epicatechin and their mixture in orally administered rats. J Nutr. 2001;131(11):2885–91.
- Mohsen MA, Marks J, Kuhnle G, et al. The differential tissue distribution of the citrus flavanone naringenin following gastric instillation. Free Radic Res. 2004;38(12):1329–40.
- Longpré F, Garneau P, Christen Y, et al. Protection by EGb 761 against beta-amyloid-induced neurotoxicity: involvementof NF-kappaB, SIRT1, and MAPKs pathways and inhibition of amyloid fibril formation. Free Radic Biol Med. 2006;41(12):1781–94.
- Agnati LF, Genedani S, Rasio G. Studies on homocysteine plasma levels in Alzheimer's patients for neurodegeneration. *J Neural Transm (Vienna)*. 2005;112(1):163–9.
- 18. Ban JY, Jeon SY, Bae K, et al. Catechin and epicatechin from Smilacis chinae rhizome protect cultured rat cortical neurons against amyloid β protein (25–35)-induced neurotoxicity through inhibition of cytosolic calcium elevation. *Life Sci.* 2006;79(24):2251–9.
- Nath S, Bachani M, Harshavardhana D. Catechins protect neurons against mitochondrial toxins and HIV proteins via activation of the BDNF pathway. J Neurovirol. 2012;18(6):445–455.
- Hua J, Websterb D, Caoc J, et al. The safety of green tea and green tea extract consumption in adults – Results of a systematic review. *Regul Toxicol Pharmacol*. 2018;95:412–433.