ORIGINAL RESEARCH PAPER
Volume-6 | Issue-11 | November-2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CURCUMIN AS A POTENTIAL THERAPEUTIC DRUG FOR NEURODEGENERATIVE COMPLAINTS

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ABSTRACT

Neurodegenerative complaints, such as Alzheimer's disease have an increasing prevalence with increasing global life expectancy, while there is no effective measure to fight against this disease. In this review, we discussed the current evidence supporting the great potential of the curcumin to treat AD, including inhibited Aβ protein aggregation, cleared generated Aβ, chelated metal ions and antioxidation.

KEYWORDS

Alzheimer's disease, curcumin, antioxidation.

Alzheimer's disease (AD) is a kind of nervous system diseases with progressive cognitive function deterioration, daily living ability decline and behavioral ability changes, clinical manifestations of cognitive, language movements, spititual behavioral disorder. Incidence is age-related and the incidence of the elderly is higher [1]. There is no the antidote to the AD, which has become a challenge for the medical community. Epidemiology shows that the incidence of AD varies from region to region [2-3].

Curcumin is a kind of yellow phenolic compound derived from the rhizome of Curcuma longa [4-5]. Particular attention has been paid to curcumin because regular diet of curcumin is one of the reasons responsible for reducing the risk of AD among the Indian populations. Curcumin has been found recently (1) inhibited Aβ protein aggregation, (2) cleared generated Aβ, and (3) chelated metal ions [6-9]. Besides, Curcumin also has a strong antioxidant effect. Curcumin has attracted wide attention due to its extensive physiological and pharmacological activity, Curcumin can also be used for the treatment of liver disease, it is also considered a potential anti-cancer agent for a variety of cancer types. In this paper, we reviewed the current in vitro and in vivo evidence to support the therapeutic potential of curcumin to AD and to promote the use of this promising drug for AD [10-13].

Study found that polyphenols drug structure of phenol ring can interaction with aromatic residues of Amyloid protein sequence, interference gathered Aβ [14]. In vitro experiments proved that curcumin can inhibit the aggregation of Aβ40, while the specific mechanism still need further study [15].

Curcumin can inhibit the deposition of amyloid and new plaque formation, and has a strong scavenging effect on the already formed plaque, can also reverse the nerve dendritic injury caused by Aβ deposition. In the experiment of APP transgenic mice, the low dose of curcumin (160×10-6) and high dose group (5000×10-6) could significantly reduce the load of Aβ plaque in the brain clearly. The low dose group can reduce the level of soluble Aβ and insoluble Aβ, while the high dose group has no such effect [16].

Metal ions can promote the aggregation of Aβ, caused oxidative damage, increased cytotoxicity, result in neuronal necrosis and brain damage. The concentration of Cu(II) and Zn(II) in the brain of AD patients was higher [17], and the curcumin had complexation of Cu(II), Zn(II) and Al(Ⅲ). In vitro experiment, Curcumin can inhibited the formation of Aβ42 induced by Al3+, and formed the Al(Ⅲ)-Curcumin comlex [18], and through the complex effect on Aβ42, make it lost stability, continuous degradation, thereby reducing the neurotoxicity of Aβ42 [19].

The β-amyloid protein deposition is a very critical step in the pathogenesis of Alzheimer's disease [20]. It has been reported that the precipitation of β-amyloid is strongly related to free radicals [21]. Oxide can cause significant damage to neurons, damages DNA or prevent DNA repair. Curcumin has a significant antioxidant effect, in vivo and in vitro can directly remove free radicals (ROS and RNS), and faster than other polyphenols to remove ROS.

In general, previous studies have shown that curcumin as a multifunctional natural drug has important implications for AD and should be given more preclinical and clinical studies.

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