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Emerging Role of Curcumin in Parkinson's Disease

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Curcumin, a yellow pigment present in turmeric has been shown to exhibit numerous activities, which was first isolated in 1815 and obtained in crystalline form in 1870. In 1910, the feruloylmethane skeleton of curcumin was confirmed and synthesized by **Goel** et al. [1]. What is more, extensive research over the last half century has revealed several important functions of curcumin. Such effects include antioxidant, anti-inflammatory, antimicrobial, hepatoprotective, anticarcinogenic, thrombosuppressive and cardiovascular [2].

Among them, the most key biological activity of curcumin related to neuroprotection is its anti-oxidant function that can protects substantia nigra neurons, increases striatal dopamine level and chelates Fe^{2+} in the 6-hydroxydopamine rat models of Parkinsons disease (PD) [3]. Furthermore, consequent to its anti-oxidant activity, curcumin offers mitochondrial protection in peroxynitrite-mediated nitrosative stress and damage of brain mitochondria *in vitro* with direct therapy [4]. Meanwhile, the most compelling and pivotal rationale for the continuing use of curcumin is its extremely good safety profile.

To date, no researches in either animals or humans have showed any toxicity associated with the use of curcumin, and it is report that curcumin is not toxic even at high doses [5,6]. Moreover, several clinical studies recommend that a special attention must to be paid to the systematic documentation of potential toxicities in long-term administration of curcumin in healthy volunteers [7]. Furthermore, there are no experimental or clinical studies focusing on curcumin toxicities in healthy subjects per se without a concomitant morbidity or a disease model. Thus, future studies designed for research need to select suitable subjects for explicit curcumin toxicity.

Several studies in different experimental models of PD strongly support the clinical application of curcumin in PD. Song et al. provided evidence that curcumin may be considered as a promising candidate for PD management, whose underlying molecular mechanism was implicated in regulating intracellular SOD/GSH/NGF/Hsp70 expressions, thereby improving neurofunctions in the substantia nigra neurons [8].

Bharathi et al. demonstrated that curcumin binds toa-synuclein oligomeric form and prevents further fibrillization of a-synuclein, which might aid the development of disease modifying agents in preventing or treating PD [9]. Moreover, Limamanen et al. showed that curcumin treatment could replenish depleted brain dopamine levels in early Parkinson disease, clearly suggesting its limitation as a therapeutic agent in late-onset stage [10].

Overall, *in vivo* and *in vitro* studies have revealed many important bioactivities of curcumin in PD. However, to date, curcumin only has been used in clinical trials on various inflammatory diseases and cancers. In the future, it will be necessary to focus attention on the clinical application of curcumin in neurodegenerative diseases, such as PD, because many experiments have clarified the potential value of curcumin in these areas.

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