Curcumin in neurology

Sir,

Having read the article relating plant extracts and Alzheimer's by Obulesu *et al.*,[1] I would like to extend the discussion on curcumin and its benefits in neurology as a whole. Curcumin is touted to have protective effects against cerebral ischemia/reperfusion injury.

The administration of curcumin (100 and 300 mg/kg) 60 min after middle cerebral artery occlusion significantly diminished infarct volume, brain water content, disruption of the blood-brain barrier, and behavioral deficits and improved neurological deficits in a dose-dependent manner (demonstrated on Nissl staining).[2] Curcumin significantly reduces the elevated levels of MMP-9 and MMP-2 expression and lipid peroxidation within the cerebral cortex and the middle cerebral artery. It decreases malondialdehyde levels, cytochrome c, and caspase-3 protein (anti-apoptic effects); causes higher expression of bcl-2, higher ratio of bcl-2/bax, and lower expression of bax; and upregulates transcription factors Nrf2 and HO-1. It significantly decreased the production of NO and iNOS expression induced by LPS in activated microglia cells. In intracerebral hemorrhage, it increased hematoma resolution at 72 h probably mediated by the reduced expression of the proinflammatory mediators, tumor necrosis factor-α, interleukin-6, and interleukin-1β.[3]

Curcumin restores membrane homeostasis by counteracting all the effects of fluid percussion injury (FPI) like increased levels of 4-hydroxynonenal (HNE – an intermediary for lipid peroxidation on neurons), reduces calcium-independent phospholipase A2 (iPLA$_2$), increases fatty acid transport protein (FATP), modulates levels of the NR2B subunit of the transmembrane NMDA receptor, brain-derived neurotrophic factor, and syntaxin-3. It is shown to preserve synaptic plasticity and learning capacity after experimental traumatic brain injury (TBI). In TBI, curcumin reversed the induction of aquaporin-4 (implicated in the development of cellular edema). In cultured astrocytes, curcumin blocked IL-1β-induced aquaporin-4 expression (by the reduced activation of the p50 and p65 subunits of nuclear factor κB). It preferentially attenuated phosphorylated p65 immunoreactivity in pericontusional astrocytes and decreased the expression of glial fibrillary acidic protein.

Curcumin ameliorated HIV-1gp120 V3 peptide-induced impairment of spatial learning and memory, inhibited long-term potentiation in the CA1 region of the hippocampus, and mediated oxidative stress and neuronal injury, and has potential against HIV-associated dementia (HAD).

Curcumin has a potential antiepileptogenic effect on kindling-induced epileptogenesis and can reduce the severity of seizures induced by kainate acid (KA) probably by inhibiting calcium elevation induced by KA and affecting the histone modification of chromatin.

It also has benefits in Alzheimer's disease pathology, chronic ethanol exposure, and 6-hydroxydopamine (6-OHDA) model of Parkinson's disease.

Long-term treatment (12 months) with curcumin improved parent and patient-reported quality of life

(especially self-esteem) in a case of (a 15-year-old female) Déjérine–Sottas disease.[4]

Curcumin acted synergistically with stem cells to heal an induced spinal contusion in rats (probably by its proliferative effect and reduction of usual glial scarring and swelling). Its binding to tau aggregates has applications in diagnostic pathology as curcumin fluorescence reliably detected neuronal fibrillar tau inclusions in AD and progressive supranuclear palsy and surpassed AT8 immunolabeling in visualizing later stages of FTIs, including ghost tangles.[5]

Curcumin certainly has a hoard of beneficial effects especially in neurology.

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