Emerging Neuroprotective Effects of N-Palmitoylethanolamide Besides Its Significant Antinociceptive Effects

To the Editor:

I read with great interest the recent article by Gatti et al. in a recent issue of your esteemed Journal [1]. The article is highly thought provoking. N-palmitoylethanolamide (PEA) exerts a number of other neuro-protective effects besides its significant antinociceptive effects.

PEA attenuates chemotherapy-induced neuropathies by attenuating endoneurial edema in myelinated nerve fibers [2]. Similarly, PEA improves distal motor latency in individuals with carpal tunnel syndrome [3]. Similar benefits have been noticed in pudendal neuralgias [4]. PEA also exerts beneficial effects in patients with multiple sclerosis by ameliorating neuropathic pain [5]. PEA also increases the latency between tonic clonic seizures and thus exhibits anti-epileptic activity [6].

Interestingly, Yu et al. have recently demonstrated that just like fluoxetine, PEA decreases immobility in the tail suspension test in mice and thereby exerts a significant antidepressant effect [7]. Most of these neuroprotective effects of PEA are a result of direct attenuation of astrocyte infiltration [8]. Interestingly, PEA holds a potential therapeutic role in sleep disorders such as narcolepsy as it enhances waking by modulating dopamine and anandamide levels [9].

Similarly, caspase 3 activation secondary to intracerebroventricular administration of agents such as the amyloid-β 25–35 peptide is ameliorated after administration of PEA [10]. This manifests as reduced memory attenuation and illustrates a plausible role of PEA in Alzheimer’s disease and other forms of dementia. Similarly, PEA may play a plausible role in protecting neurogenic tissue from infections such as Streptococcus pneumoniae as it enhances the phagocytic ability of microglia [11].

The examples clearly illustrate the potent neuroprotective effects of PEA.

References


