

Thymoquinone prevents β -amyloid neurotoxicity in primary cultured cerebellar granule neurons

ABSTRACT

Thymoquinone (TQ), a bioactive constituent of *Nigella sativa* Linn (*N. sativa*) has demonstrated several neuropharmacological attributes. In the present study, the neuroprotective properties of TQ were investigated by studying its anti-apoptotic potential to diminish β -amyloid peptide 1–40 sequence (A β 1–40)-induced neuronal cell death in primary cultured cerebellar granule neurons (CGNs). The effects of TQ against A β 1–40-induced neurotoxicity, morphological damages, DNA condensation, the generation of reactive oxygen species, and caspase-3, -8, and -9 activation were investigated. Pretreatment of CGNs with TQ (0.1 and 1 μ M) and subsequent exposure to 10 μ M A β 1–40 protected the CGNs against the neurotoxic effects of the latter. In addition, the CGNs were better preserved with intact cell bodies, extensive neurite networks, a loss of condensed chromatin and less free radical generation than those exposed to A β 1–40 alone. TQ pretreatment inhibited A β 1–40-induced apoptosis of CGNs via both extrinsic and intrinsic caspase pathways. Thus, the findings of this study suggest that TQ may prevent neurotoxicity and A β 1–40-induced apoptosis. TQ is, therefore, worth studying further for its potential to reduce the risks of developing Alzheimer's disease.

Keyword: Thymoquinone; β -Amyloid; Alzheimer's disease; Neurotoxicity; Primary cultured cerebellar granule neurons