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 (Aβ1–40)-induced neuronal cell death in primary cultured cerebellar granule neurons (CGNs). The effects of TQ against Aβ1–40-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 μM) and subsequent exposure to 10 μM Aβ1–40 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 Aβ1–40 alone. TQ pretreatment inhibited Aβ1–40-induced apoptosis of CGNs via both extrinsic and intrinsic caspase pathways. Thus, the findings of this study suggest that TQ may prevent neurotoxicity and Aβ1–40-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