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with the severity of these diseases. The endogenous Ang II, generated by the catalytic action of the ACE, binds to a unique receptor designated the AT1 receptor and stimulates several cell signaling cascades that contribute to neuronal degeneration in the CNS. One mechanism by which Ang II induces neuronal damage is by increasing the release of pro-inflammatory cytokines and chemokines. Ang II increases the expression of genes that encode pro-inflammatory cytokines such as IL-1, IL-6, and TNF-alpha, and this effect may contribute to neuronal cell death. The AT1 receptor is also coupled to several G proteins, among which, Gq has been implicated in mediating many of the actions of Ang II. Activation of the AT1 receptor and Gq proteins is likely to play an important role in the pathophysiology of acute and chronic neurodegenerative diseases.{ "compilerOptions": { f988f36e3a