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Progress in pharmacological mechanisms of terandrine and its therapeutic use in digestive diseases

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Abstract

AIM: To review the progress in pharmacological mechanisms of terandrine (Tet) and its therapeutic use in digestive diseases.

METHODS: We reviewed almost all the papers related to Tet from various magazines published in English and Chinese in recent years.

RESULTS: It has been demonstrated that Tet had multiple bioactivities: (1) Tet could act as a Ca^{2+} antagonist *via* blocking cellular plasma membrane voltage or receptor operating Ca^{2+} channels, inhibiting extracellular Ca^{2+} entry into the cell and intracellular Ca^{2+} mobilization to the cytosol, so as to prevent hepatocytes, cardiomyocytes, pancreas cells and neurocytes from toxic or ischemia reperfusion injuries. However, in HL-60 and leukemic T cells, Tet promoted Ca^{2+} releasing from mitochondria and microsomes, increased the concentration of intracellular Ca^{2+}, and induced cell death; (2) Tet inhibited phobol 12-myristat 13-acetate (PMA) plus ionomycin-induced T cell proliferation, interleukin-2 secretion and expression of the T cell activation antigen, CD71. It could also interrupt the integrity of macrophages, and reduced respiratory burst of neutrophils and macrophages and proinflammatory cytokines secretion through minimizing nuclear transcriptional factor kappa B DNA binding activity; (3) Tet could induce tumor cell apoptosis, and down regulate P-glucoprotein activity; and (4) Tet has the therapeutic effects on hepatic fibrogenesis, portal hypertension, immunomodulation, *etc*.

CONCLUSION: Tet can act as a Ca^{2+} channel blocker, inhibit proinflammatory factors releasing, modulate immunoreaction, and induce tumor cell apoptosis. It can be used to prevent hepatocyte injury induced by toxins and virus, inhibit hepatic fibrogenesis, reduce portal venous pressure, and can be used as an anti-tumor drug as well.

Key words: Terandrine; Pharmacology, clinical; Digestive system diseases/drug therapy; Antineoplastic agents, phytogenic; Calcium channels; Apoptosis

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