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Galangin and its Anti Inflammatory Properties

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Editorial

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Editorial

Galangin (4H-1-benzopyran-4-one-3,5,7-trihydroxy-2-phenyl and 3,5,7-trihydroxyflavone), a flavonoid derived from medicinal herbs including Alpiniaofficinarum Hance, Alnuspendula Matsumand Plantagomajor L..Propolis, a resinous substance from honeybees, has beenused as an antiseptic in folk medicine and its biologicactivity depends on the presence of galangin [1]. Galangin has antimutagenic, enzyme regulatory and antioxidant effects because of its chemical structure [2].

It is claimed that the antii flammatory effects of galanginarises from the supression of eicosanid synthesis by inhibition of cyclooxygenase (COX)-2 enzyme [3]. Insomestudiesit is shown that galangin inhibits phospholipase A2 enzyme and reduce anti-inflammatory effects by decreasing adhesion molecule expression [4].

Choi et al. (2014) investigated the effects of galangin on atopicdermatitis (AD)-like skin lesions and underlying mechanisms of action. They have estabilished an atopic dermatitis model in BALB/c mice. In this study, topical application of galangin reduced AD symptoms based on ear thickness and histopathological analysis, in addition to serum IgE and IgG2a levels. Galangin inhibited mastcellin filtration into the ear and serum histaminelevel; suppressed DFE/DNCB-inducedexpression of interleukin (IL)-4, IL-5, IL-13, IL-31, IL-32, and interferon (IFN)-γ in the ear tissue and significantly inhibited the expression of cytokines and chemokine by the down-regulation of nuclearfactor-κBandmitogen-activated protein kinases in Ha CaT cells [5].

In another study, Kim et al. (2012) investigated galangin-induced apoptosis of human gastric cancer SNU-484 cells. In this study, galangin (50-200 μMfor 24 h and 48 h)inhibited proliferation of SNU-484 cells in a dose- and time-dependent manner.Galangin-induced cell death was characterized with the changes in cellmorphology, DNA fragmentation, cellcycle, activation of caspase-3/-9, poly (ADP-ribose) polymerase (PARP) cleavage, andexpression of MAP kinasesuch as ERK1/2 and JNK [4].

Similarly, Zha et al. (2013) investigated that galangin can abrogateovalbumin- (OVA) induced airway inflammation by negative regulation of NF-B. In this study, BALB/c mice sensitized and challenged with OVA developed air way hyper responsiveness (AHR) and inflammation. Galangin dose dependently inhibited OVA-induced increases in total cellcounts, eosinophilcounts, and interleukin-(IL-) 4, IL-5, and IL-13 levels in bronch oalveolar lavage fluid, and reduced serum level of OVA-specific IgE [6].

In conclusion, we can say that the antiinflammatory effects of galangin can be seen in different inflammatory conditions in a dose dependent manner.

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