Psoralidin, a natural furanocoumarin, is isolated from Psoralea corylifolia L. possessing anti-cancer properties.

IC50 value:
Target: Anticancer natural compound
in vitro: PSO dramatically decreased the cell viabilities in dose- and time-dependent manner. Autophagy inhibitor 3-MA blocked the production of LC3-II and reduced the cytotoxicity in response to PSO. Furthermore, PSO increased intracellular ROS level which was correlated to the elevation of LC3-II [1]. Psoralidin at 10 μM was able to induce the maximum reporter gene expression corresponding to that of E2-treated cells and such activation of the ERE-reporter gene by psoralidin was completely abolished by the cotreatment of a pure ER antagonist, implying that the biological activities of psoralidin are mediated by ER [2]. Psoralidin enhanced TRAIL-induced apoptosis in HeLa cells through increased expression of TRAIL-R2 death receptor and depolarization of mitochondrial membrane potential [3]. Psoralidin inhibited the IR-induced COX-2 expression and PGE(2) production through regulation of PI3K/Akt and NF-κB pathway. Also, psoralidin blocked IR-induced LTB(4) production, and it was due to direct interaction of psoralidin and 5-lipoxygenase activating protein (FLAP) in 5-LOX pathway. IR-induced fibroblast migration was notably attenuated in the presence of psoralidin [4].

in vivo: Moreover, in vivo results from mouse lung indicate that psoralidin suppresses IR-induced expression of pro-inflammatory cytokines (TNF-α, TGF-β, IL-6 and IL-1 α/β) and ICAM-1 [4].

References: