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Anti-inflammatory Effects of Phytochemicals by the Modulation of the Innate Immunity

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Toll-like receptors (TLRs) induce innate immune responses by recognizing invading microbial pathogens leading to the activation of the adaptive immune responses. In general, TLR activation triggers the activation of two downstream signaling pathways; MyD88- and TRIF-dependent pathways leading to the expression of pro-inflammatory cytokines and type I interferons (IFNs), respectively. Many phytochemicals are known to exert anti-inflammatory effects. However, the direct molecular targets and the mechanisms for such inhibition are not fully identified. Here, we investigated the molecular target of resveratrol, EGCG and curcumin in TLR-mediated signaling pathways. Resveratrol and EGCG specifically inhibited TRIF pathway of TLR3 and TLR4 by targeting TBK1 and RIP1 kinases in TRIF complex. In contrast, curcumin inhibited ligand-induced dimerization of TLR4. Our results reveal important molecular targets of phytochemicals that are known to possess anti-inflammatory effects. Together, the results raise the possibility that certain dietary phytochemicals can alter the susceptibility to microbial infection and the risk of development of chronic inflammatory diseases through the regulation of TLR signaling pathways and target gene expression.

Key word: Phytochemicals, Toll-like receptor, resveratrol, EGCG, curcumin

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Growth Inhibitory Effect and Apoptosis by Extract of *Strychni Semen* in Human Gastric Carcinoma AGS Cells

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Strychni Semen is a well known herb material to cope with diseases of toxic and tumorigenic inflammation effectively in traditional oriental medicine. In the present study, the effects of extract of *Strychni Semen* (ESS) was examined on the growth of human gastric carcinoma cell line AGS in order to investigate the anti-proliferative mechanism and induction of apoptosis by ESS. Treatment of AGS cells to ESS resulted in the growth inhibition and morphological change in a dose-dependent manner. The induction of apoptotic cell death by ESS was connected with down-regulation of anti-apoptotic Bcl-2 and Bcl-XS/L expression, and cleavage of Bid protein without alteration pro-apoptotic Bax expression. ESS treatment induced the proteolytic activation of caspase-3, -8 and -9, and a concomitant degradation and/or down-regulation of poly(ADP-ribose) polymerase (PARP), phospholipase (PLC)- γ 1 and β -catenin protein. Additionally, induction of apoptotic bodies by ESS was connected with the activation of CAD/DFF40 and inactivation of ICAD/DFF45 protein expression.