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Kaempferol attenuates COX-2 expression in IL-6-induced macrophages and carrageenan-induced mouse paw edema by targeting STAT3 and NF-kB

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Abstract

Dietary polyphenols are reported to possess varied pharmacological activities, viz. antioxidant, anti-inflammatory, anti-cancer, anti-allergic actions. Here, we report the efficacy of Kaempferol (kae) to attenuate expression of IL-6 induced cycloxygenase-2 (COX-2), an inducible isoform of cycloxygenase enzyme family that catalyzes synthesis of inflammatory mediators, prostanoids and prostaglandins. IL-6 is a pleiotropic cytokine involved in both acute and chronic inflammation. Our results showed that kae attenuated COX-2 expression at both mRNA and protein level in IL-6-induced THP1 macrophages. This attenuation of COX-2 expression by kae involved dose-dependent inhibition of phosphorylation of STAT3 (Tyr 705) and NF-kB p65 (Ser 536) leading to their deactivation and reduced nuclear localization in THP-1 macrophages. Moreover, kae modulates COX-2 expression as well as STAT3 and NF-kB activation in carrageenan-induced mouse paw edema model. RT-PCR and western blot analysis from paw tissues were harvested after kae injection (i.p) followed by carrageenan-treatment in sub-plantar region of right hind paw. Results showed that kae attenuated COX-2 expression and STAT3 and NF-kB activation in carrageenan-induced mouse paw edema, suggesting that inhibition of both IL-6-STAT3-COX-2 and IL-6-NFkB-COX-2 axes by kae might be stimulus-independent. To understand binding affinity of kae with NF-kB and STAT3, docking analysis was performed using Patchdock server. From our findings, we observed strong binding affinity and transient interaction in both NF-kB/kae and STAT3/kae complexes. We noticed negative atomic contact energy and greater interface area for both the complexes. Selected complexes obtained from Patchdock were refined using Firedock online server which also suggested similar negative binding energy profile. It is plausible that kae attenuates COX-2 expression by directly binding to both STAT3 and NF-kB proteins and inhibiting their activation and nuclear translocation.

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