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LPS 유도된 HCT116 인간 대장암세포에서 cordycepin의 prostaglandin E2-EP4 receptor 감소 조절을 통한 세포의 이동과 전이 억제 효과

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Cordycepin Inhibits LPS-induced Cell Migration and Invasion in Human Colorectal Carcinoma HCT116 cells through Down-regulation of Prostaglandin E2-EP4 Receptor

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Prostaglandin E2(PGE2), a major product of cyclooxygenase-2 (COX-2), plays an important role in the carcinogenesis of many solid tumors, including colorectal cancer. Because PGE2 functions by signaling through PGE2 receptors (Eps), which regulate tumor cell growth, invasion, and migration, there has been a growing amount of interest in the therapeutic potential of targeting Eps. In the present study, we investigated the role of EP4 on the effectiveness of cordycepin in inhibititing the migration and invasion of HCT116 human colorectal carcinoma cells. Our data indicate that cordycepin suppressed lipopolysaccharide (LPS)-enhanced cell migration and invasion through the inactivation of matrix metalloproteinases (MMP)-9 as well as the down-regulation of COX-2 expression and PGE2 production. These events were shown to be associated with the inactivation of EP4 and activation of AMP-activated protein kinase (AMPK). Moreover, the AMPK inhibitor, compound C, as well as AMPK knockdown via siRNA, attenuated the cordycepin-induced inhibition of EP4 expression. Cordycepin treatment also reduced the activation of CREB. These findings indicate that cordycepin suppresses the migration and invasion of HCT116 cells. Through modulating EP4 expression and the AMPK-CREB signaling pathway. Therefore, cordycepin has the potential to serve as a potent anti-cancer agent in therapeutic strategies against colorectal cancer metastasis.

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