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CURCUMIN SUPPRESSES ACROLEIN-INDUCED COX-2 EXPRESSION AND PROSTAGLANDIN PRODUCTION IN ENDOTHELIAL CELLS

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Abstract

Inflammation is crucial to limiting vascular disease. Previously we reported that acrolein, a known toxin in tobacco smoke, might play an important role in the progression of atherosclerosis via an inflammatory response involving cyclooxygenase-2 (COX-2) and prostaglandin (PG) production in human umbilical vein endothelial cells (HUVECs). Curcumin has been known to improve vascular function and have anti-inflammatory properties. In this study, we investigated whether curcumin prevents the induction of inflammatory response caused by acrolein. We show that curcumin suppresses acrolein-induced COX-2 expression at the protein level, and this inhibition is involved in the abolition of oxidative stress and ER stress in HUVECs. We also found that curcumin treatment blocked acrolein-induced activation of PKC , p38 MAPK and CREB pathway. Curcumin attenuates inflammatory response via inhibition of COX-2 expression and prostaglandin production in acrolein-induced human endothelial cells. This inhibition by curcumin results in the abolition of phosphorylation of PKC, p38 MAPK and CREB. Furthermore, curcumin suppresses the production of ROS and ER stress caused by acrolein. These results suggest that curcumin might be a useful agent against endothelial dysfunction caused by acrolein-induced inflammatory response.

Keywords

inflammation; acrolein; curcumin; cyclooxygenase-2; HUVECs