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Cyclooxygenase-2 induction by lysophosphatidylcholine in cultured rat vascular smooth muscle cells: involvement of the p38MAPK pathway

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ABSTRACT

Lysophosphatidylcholine (lysoPC) stimulates the release of prostaglandins (PGs) in various cells and tissues. Cyclooxygenase (COX)-2 has recently emerged as a key regulator of PG synthesis. We investigated whether lysoPC regulates COX-2 expression in cultured rat vascular smooth muscle cells (VSMCs). LysoPC strongly increased the expression of COX-2 mRNA in a time- and dose-dependent manner. COX-2 protein expression also was increased by lysoPC. The p38 mitogen-activated protein kinase (MAPK) inhibitor SB203580 significantly suppressed lysoPC-induced COX-2 mRNA and protein expression, but not a p42/44MAPK kinase (MEK-1) inhibitor, PD98059. LysoPC did not increased the transcription of the COX-2 gene, as assayed with a COX-2 promoter/luciferase chimeric plasmid and suppressed the decay of COX-2 mRNA. SB203580 markedly enhanced the decay of COX-2 mRNA induced by lysoPC, implying that p38MAPK activated by lysoPC helps to regulate COX-2 by stabilizing its mRNA. The COX-2 specific inhibitor NS-398 attenuated lysoPC-stimulated DNA and protein synthesis

as well as PGE_2 production by VSMCs. These results suggest that in rat VSMCs lysoPC regulates COX-2 expression and PG production and also modulates cell proliferation through p38MAPK-mediated signaling pathways.

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