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• 中文摘要	查無中文摘要		
• 英文摘要	Heme oxygenase-1 (HO-1) is induced as a beneficial and adaptive response in cells and tissues exposed to oxidative stress. Herein we examined how various eicosanoids affect the induction of HO-1, and the possible mechanism underlying 15-deoxy-D12,14-prostaglandin J2 (15d-PGJ2)-induced HO-1 expression. PGH2, PGD2 and its metabolites of the PGJ2 series, and PGA1 markedly induced the protein expression of HO-1. Arachidonic acid (AA), docosahexaenoic acid (DHA), PGE2, PGF2a, and thromboxane B2 (TXB2) were shown to have no effect on the induction of HO-1. 15d-PGJ2 was the most potent activator achieving significance at 5 AM. Although 15d-PGJ2 significantly activated the MAPKs of JNK and ERK, the activation of JNK and ERK did not contribute to the induction of HO-1 as determined using transfection of dominant-negative plasmids and MAPKs inhibitors. Additional experiment indicated that 15d-PGJ2 induced HO-1 expression through peroxisome proliferator-activated receptor (PPAR)-independent pathway. 15d-PGJ2 significantly decreased the intracellular level of reduced glutathione; and the thiol antioxidant, N-acetyl-L-cysteine (NAC), and the thiol-reducing agent, dithiothreitol (DTT), inhibited the induction of HO-1 by 15d-PGJ2. Finally, NAC and DTT exhibited significant inhibition of HO-1 mRNA and HO-1 promoter reporter activity induced by 15d-PGJ2. These results suggest that thiol antioxidant and reducing agents attenuate the expression of HO-1 induced by 15d-PGJ2, and that the cellular thiol-disulfide redox status may be linked to HO-1 activation.		