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Activation of olfactory receptor 1A1 suppresses PPAR- γ expression by induction of HES-1 in cultured hepatocytes

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The olfactory receptors (ORs) are the largest G-protein coupled receptor gene superfamily. Recent studies demonstrated that ORs are also ectopically expressed in non-olfactory organs including metabolically active tissues, however, their biological functions in non-olfactory tissues are largely unknown. In this study, we investigated the metabolic role of OR1A1 in cultured hepatocytes. OR1A1 was moderately but significantly expressed in HepG2 cells assessed with RT-PCR, immunoblotting analysis, and fluorescence-activated cell sorter analysis. Activation of OR1A1 in cells stimulated by (-)-carvone, a known OR1A1 ligand, increased cAMP but not intracellular calcium concentrations, thus induced PKA activity and subsequently phosphorylated CREB. The gene and protein expressions of PPAR- γ and the expression of its target gene and key enzyme in triglyceride synthesis, mitochondrial glycerol-3-phosphate acyltransferase, were downregulated. Suppression of PPAR- γ was mediated by upregulation of HES-1, a CREB responsive PPAR- γ co-repressor. Interestingly, the intracellular triglyceride levels and lipid accumulation were significantly reduced in cells stimulated with (-)-carvone and OR1A1 knockdown with siRNA diminished these effects of (-)-carvone. These results indicate that OR1A1 is expressed in hepatocytes, may play function as a non-redundant receptor to regulate cAMP-PKA signaling axis, thus modulates hepatic triglyceride metabolism.

Biography

Chunyan Wu is now studying for her PhD degree in the Laboratory of Food Biomedical Science in Korea University. Her research is mainly focused on effects of aroma compounds in cellular lipid metabolism and the regulation of natural compounds on PPAR activity.

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