Lipin-2 mRNA inhibition aggravates TLR ligands induced inflammation

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Lipins are phosphatidic acid phosphatases involved in synthesis of phospholipids and triglycerides, although they regulate cellular levels of important signaling lipids. Lipin-1 contributes positively to macrophage stimulation through TLR4, and other TLRs, by affecting MAPKs and AP-1 activation and, as a consequence, the generation of pro-inflammatory factors. Lipin-2 reduces pro-inflammatory signaling induced by saturated fatty acids in macrophages. Here we examined whether LPIN-2 mRNA inhibition affects TLR mediated inflammatory signaling in HT29, a colon cancer cell line. The LPIN-2 siRNA pre-treatment reduced the up-regulated defensins stimulated by TLR ligands, LPS and flagellin. And the increased level of IL-8 mRNA by LPS and R848 were more increased by LPIN-2 mRNA inhibition. And LPS and R848 induced JNK and ERK phosphorylation whose expressions were more elevated by Lipin-2 inhibition. On the other hand, the lipid transcription factors like PPARγ and PGC1α did not change by LPIN-2 siRNA pre-treatment. Taken together, LPIN-2 inhibition aggravates TLR ligands induced inflammatory signaling through ERK and JNK phosphorylation.

Biography
Seung-Heon Hong works as a Professor at the Department of Oriental Pharmacy, College of Pharmacy, Wonkwang University, Iksan, Korea. Since 2005, he has been an Editor of Oriental Pharmacy and Experimental Medicine and an Editorial Board Member of Evidence-based Complementary and Alternative Medicine. His research interest is to investigate pharmacological effect of herbal medicine on cancer, allergic inflammation and obesity.

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