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**Lipin-2 mRNA inhibition aggravates TLR ligands induced inflammation**Seung-Heon Hong, Sung-Joo Park and Dae-Seung Kim  
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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 PPAR $\gamma$  and PGC1 $\alpha$  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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