Therapeutic potential of resolvin D1 in the treatment of liver sterile inflammatory diseases

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A novel genus of specialized pro-resolving mediators (SPMs) has been discovered to be involved in the active clearance and regulation of inflammatory exudates to restore tissue homeostasis. One of the lipid mediators, resolvins (Rvs), are formed via specific transcellular biosynthetic pathways at strict temporal intervals during the inflammatory response. While accumulating evidences suggest RvD1 counteracts proinflammatory signaling and promotes resolution, the specific cellular targets and modes of action for RvD1 remains largely unknown. Ischemia/reperfusion (IR) injury is an unavoidable sequela of major liver surgery and is characterized by a sterile inflammatory response jeopardizing the organ. We have recently reported that RvD1 facilitates M2 macrophage polarization of Kupffer cells and efferocytosis via ALX/FPR2 signaling in the animal model of liver IR. Moreover, our most recent *in vivo* and *in vitro* findings have implied that a crosstalk between mitochondrial oxidative stress and RvD1 is pivotal in the protection of IR-induced hepatocellular damage (*unpublished data*), which give us a clue for solving the conundrum of cellular and molecular mechanisms of liver IR injury. Our study justifies that RvD1 might be a useful pharmacological maneuver for attenuating liver sterile inflammation such as IR injury.

Speaker Biography

Jung-Woo Kang has completed his Ph.D in 2015 from School of Pharmacy, Sungkyunkwan University. Dr. Kang has currently been a postdoctoral associate in Department of Internal Medicine, Section of Digestive Diseases, Yale University. Dr. Kang have performed a number of studies investigating signaling pathways in clinically-relevant hepatotoxicity models and applying novel pharmacological strategies. Dr. Kang has published more than 20 papers in the journals of pharmacology and biochemistry.

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