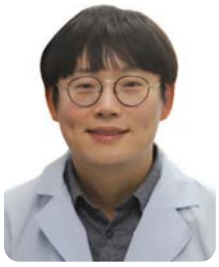
CURRICULUM VITAE

이은우



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[관심분야]

Ferroptosis, Lipid Metabolism, ROS, Senescence, Athersclerosis

[논문]

1. Oh M. et al., Darapladib, an inhibitor of Lp-PLA2, sensitizes cancer cells to ferroptosis by remodeling lipid metabolism. Nat Commun. 14(1):5728 (2023).
2. Kim J. et al., FSP1 confers ferroptosis resistance in KEAP1 mutant non-small cell lung carcinoma in NRF2-dependent and -independent manner. Cell Death Dis, 14(8):567 (2023).
3. Kim J. et al., An integrated view of lipid metabolism in ferroptosis revisited via lipidomic analysis. Exp Mol Med. 55(8):1620-1631 (2023).
4. Lee JY. et al., Polyunsaturated fatty acid biosynthesis pathway determines ferroptosis sensitivity in gastric cancer. PNAS, 117(51): 32433-32442 (2020).
5. Park TJ. et al., Quantitative proteomic analyses reveal that GPX4 downregulation during myocardial infarction contributes to ferroptosis in cardiomyocytes. Cell Death Dis. 10, 835 (2019).

Potential roles of ferroptosis in atherosclerosis

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Ferroptosis is a form of regulated cell death that relies on iron and is triggered by lipid peroxidation of polyunsaturated fatty acids (PUFAs) in membrane phospholipids. Extensive in vitro and in vivo studies have underscored the crucial roles of ferroptosis in various human diseases, including neurological disorders, ischemia-reperfusion injury, kidney damage, and blood disorders. Notably, cancer cells, particularly those resistant to chemotherapy, are highly susceptible to ferroptosis inducers, presenting a promising avenue for anti-cancer therapy. We previously reported that arachidonic acid (AA; C20:4) and adrenic acid (C22:4) biosynthesis from linoleic acid (C18:2), the predominant fatty acid in the blood, by fatty acid desaturases (FADSs) and elongases (ELOVLs) is critical for ferroptosis sensitivity. I will introduce that the cell-autonomous recycling of AA facilitated by lipoprotein-associated phospholipase A2 (Lp-PLA2) is a rapid mechanism influencing the abundance of AA-containing phosphatidylethanolamine (PE) species, contributing to ferroptosis resistance. Inhibition of Lp-PLA2 with darapladib, which failed in phase III clinical trials for acute and chronic coronary diseases, can sensitize cells to ferroptosis both in vitro and in vivo, offering a novel therapeutic strategy for enhancing ferroptosis in cancer treatment. Additionally, I will explore the initiation and propagation of lipid peroxidation to the plasma membrane, causing membrane damage and ferroptosis. Our recent findings indicate that a specific protein translocates to the plasma membrane within 15 minutes of ferroptotic stimuli. Cells lacking this protein exhibit lipid peroxidation signals solely in the cytoplasm or subcellular organelles, not at the plasma membrane, resulting in ferroptosis resistance. Finally, I will propose that inhibiting ferroptosis by targeting this protein or using a lipophilic antioxidant could represent a potential therapeutic approach for atherosclerosis.