NCGG SEMINAR

"Characterization of new senescence regulators and evaluation of their roles in age-related diseases"

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Time and Date: at 16:00 ~ 17:00 on 26 Oct, 2016 (Wed)

Room: The 2nd floor Conference Hall Small in the 1st Research building, NCGG

<Reference for Seminar>

Cellular senescence response is activated by numerous cellular stresses such as replicative exhaustion, radiation, genotoxic, oncogenic signals, inflammation, metabolic stress as well as oxidative stress and it results in the acquisition of a specific secretome and is characterized by a stable proliferation arrest. Timely regulated senescence is thought to be beneficial as it exerts tumor suppressive activity, it contributes to wound healing and even to embryonic development. By contrast, chronic senescence such as during normal or premature aging is deleterious as it favors most, if not all, age-related diseases. In the aging context, restricted proliferation limits the organ renewal capabilities and is thereby thought to participate to organism dysfunctions. In addition, senescent cells secrete numerous factors propagating cellular senescence to the neighborhood and altering tissue architecture, tissue functions and creating an inflammatory environment that is thought to favor tissue and organism dysfunctions. By performing functional genetic screenings in different senescence settings (type of cells and senescence inducers) we have now identified several new senescence regulators. I will focus my presentation on 2 of the main topics of my lab: the first one will be on the emerging role of ion channels on regulating cellular senescence[1, 2]. I will quickly comment our published results before presenting unpublished results focusing on the role of a sodium channel, plasma membrane potential and Rb pathway on regulating cellular senescence. The second one will be on PLA2R1 (Secretory Phospholipase A2 Receptor) and its role in regulating cellular senescence and aging-related diseases. I will quickly comment some of our past results[3, 4] before presenting our PLA2R1 unpublished results on aging and age-related diseases.

1. Wiel C, Lallet-Daher H, et al., Bernard D. Endoplasmic reticulum calcium release through ITPR2 channel leads to mitochondrial calcium accumulation and senescence. ***Nature Communications***, 2014; 5:3792.
2. Lallet-Daher H, Wiel C, et al., Bernard D. Potassium channel KCNA1 modulates oncogene-induced senescence and transformation. ***Cancer Res***, 2013; 73(16):5253-5265.
3. Augert A, Payré C, de Launoit Y, Gil J, Lambeau G, Bernard D. The M-type receptor PLA2R regulates senescence through the p53 pathway. ***EMBO Reports***, 2009;10(3):271-277
4. Griveau A, Devailly G, et al., Bernard D. The PLA2R1-JAK2 pathway upregulates ERRa and its mitochondrial program to exert tumor-suppressive action. ***Oncogene***, 2016. doi: 10.1038/onc.2016.43

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