第 27 回　生化学セミナー

日時：2023 年 11 月 17 日（金曜日）17:00 - 18:30
場所：東邦大学医学部 2 号館　M2 階　第２講義室

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Cell death, inflammation, wounds and hypoxia

TNF is a master inflammatory cytokine and blocking it clinically with TNF antagonists has transformed the treatment of inflammatory diseases such as psoriasis, rheumatoid arthritis and inflammatory bowel disease. TNF can induce inflammation by upregulating transcription of inflammatory mediators, including other cytokines or by inducing apoptotic or necroptotic cell death, but the relative importance of each of these outcomes in the inflammatory process is still debated. I will discuss our published and unpublished data demonstrating the importance of TNF induced cell death in inflammatory disease models and also the importance of the context of that cell death including the amount of cell death and the contribution of the microbiota. It has been appreciated that inflammatory diseases such as rheumatoid arthritis correlate with hypoxia and I explore the consequence of a hypoxic environment on TNF signalling and cell death.

References:

1. Anderton H, et al. Langerhans cells are an essential cellular intermediary in chronic dermatitis. ***Cell Rep*** 39, 110922 (2022).
2. Liu Z, et al. Oligomerization-driven MLKL ubiquitylation antagonizes necroptosis. ***EMBO J*** 40, e103718 (2021).
3. Lalaoui N, et al. Mutations that prevent caspase cleavage of RIPK1 cause autoinflammatory disease. ***Nature*** 577, 103-108 (2020).

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