令和 5 年 04 月 10 日 Apr. 10, 2023

大学院学生各位 To All Graduate Students

基盤医学特論 開講通知

Information on Special Lecture Tokuron 2023.4-2024.3

Title: **Targeting pancreatic cancer through the pancreatic stellate cell-pancreatic cancer cell bidirectional signaling axis via up-regulating the metastasis suppressor, NDRG1.**

Teaching Staff: **Des R. Richardson; Professor and Director, Centre for Cancer Cell Biology and Drug Discovery, Griffith Institute for Drug Discovery, Griffith University, Australia**

日時: 令和 5 年 5 月 19 日(金) 17:00-18:30
Time and Date: **17:00-18:30, May 19th (Fri.), 2023**

場所: 基礎棟一階 会議室 2(生協印刷部隣)
Room: **Conference Room 2, Building for Basic Medical Research 1F**

Language: **English**

Abstract: Pancreatic cancer (PaCa) is characterized by dense stroma that hinders treatment efficacy, with pancreatic stellate cells (PSCs) being a major contributor to this stromal barrier and PaCa progression. Activated PSCs release hepatocyte growth factor (HGF) and insulin-like growth factor (IGF-1) that induce PaCa proliferation, metastasis and resistance to chemotherapy. This presentation demonstrates that the metastasis suppressor, N-myc downstream regulated gene 1 (NDRG1), is a potent inhibitor of the PaCa-PSC cross-talk, leading to inhibition of HGF and IGF-1 signaling. NDRG1 also potently reduced the key driver of PaCa metastasis, namely GLI1, leading to reduced PSC-mediated cell migration. The novel clinically trialed anticancer agent, di-2-pyridylketone 4-cyclohexyl-4-methyl-3-thiosemicarbazone (DpC), which upregulates NDRG1, potently de-sensitized PaCa cells to ligands secreted by activated PSCs. DpC and NDRG1 also inhibited the PaCa-mediated activation of PSCs via inhibition of sonic hedgehog (SHH) signaling. In vivo, DpC markedly reduced PaCa tumor growth and metastasis more avidly than the standard chemotherapy for this disease, gemcitabine. Uniquely, DpC was selectively cytotoxic against PaCa cells, while "re-programming" PSCs to an inactive state, decreasing collagen deposition and desmoplasia. Thus, targeting NDRG1 can effectively break the oncogenic cycle of PaCa-PSC bi-directional cross-talk to overcome PaCa desmoplasia and improve therapeutic outcomes.

Information about the speaker:

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[注意] 事前連絡は不要です。Notice: No registration required.

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