

第 486 回 難研セミナー

第 59 回 難治疾患共同研究拠点セミナー

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記

日 時 : 2012 年 10 月 31 日 (水) 11 :00 ~ 12: 00

場 所 : M&D タワー21 階 研究部・教育部会議室

演 者 : Prof. Dr. Peter ten Dijke

(Leids Universitair Medisch Centrum)

演 題 : TGF- β signaling in cancer

要 旨 : The transforming growth factor- β (TGF- β) signalling pathway plays a critical and dual role in the progression of human cancer. During the early phase of tumour progression, TGF- β acts as a tumour suppressor, exemplified by deletions or mutations in the core components of the TGF- β signalling pathway. On the contrary, TGF- β also promotes processes that support tumour progression such as tumour cell invasion, dissemination, and immune evasion. Consequently, the functional outcome of the TGF- β response is strongly context-dependent including cell, tissue, and cancer type. In my talk, I will describe the molecular signalling pathways employed by TGF- β in cancer and how these, when perturbed, may lead to the development of breast cancer. Current TGF- β signalling inhibitors that are being evaluated in clinical trials for treatment of cancer patients target TGF- β or TGF- β receptors. These therapeutic agents are used systemically, and inhibit all TGF- β responses (bad and good). Therefore, while effective in inhibiting metastasis, they have a high chance of unwanted on-target side effects. With the aim to specifically target the oncogenic arm of the TGF- β and to leave the tumor suppressor arm intact, we have performed gain and loss of function genetic screens, combined with expression profiling studies of breast cancer tissues, to identify novel druggable regulators of pro-oncogenic TGF- β pathways in breast cancer. I will provide an update on our results on an E3 ubiquitin ligases TRAF4 and a deubiquitinase USP4 that regulate the stability of specific TGF- β signaling components and thereby promote the TGF- β -induced breast cancer invasion and metastasis.

連絡先 : 分子細胞生物学分野 澁谷浩司 (内線 : 4901)

共催 : 発生再生生物学分野