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Anti-tumorigenic function of the esophageal cancer related gene 4 (*Ecrg4*)

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Recent findings suggest that tissue-specific stem/precursor cells, including neural stem cells (NSCs) and oligodendrocyte precursor cells (OPCs), acquire mutations during life time and eventually either become senescent or transform into cancer cells. Cell senescence is described as an irreversible growth arrest and acts as a potent barrier to tumorigenesis. Such senescent cells are often found in the surrounding area of malignant tumors, although it is still uncertain what is the exact role of cell senescence in tumorigenesis. We recently identified a novel senescence-messaging secretome factor, the esophageal cancer related gene 4 (*Ecrg4*), which is induced in the senescent NSCs and OPCs as well as in the passaged mouse embryonic fibroblasts in culture and *in vivo*. Overexpression of *Ecrg4* induces NSCs and OPCs to become senescent, while its knockdown prevent them to do so. Using mouse glioblastoma-initiating cell (GIC) models and human glioblastoma tissues, we found a novel tumor suppressor function of *Ecrg4*. I would like to discuss about *Ecrg4* as a new tumor suppressor and a potential therapeutic target.

Biography

Toru Kondo has completed his PhD at the age of 29 years from Osaka University and postdoctoral studies from Osaka Bioscience Institute and University College London MRC Laboratory of Molecular Cell Biology. He is a Professor of Hokkaido University Institute for Genetic Medicine (IGM), the director of the IGM Center for Infection-associated Cancer, and a theme-leader & a visiting senior scientist of RIKEN. He has published more than 40 papers in reputed journals and has been serving as an Associate Editor of Stem Cells and an editorial board member of six international journals.

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