



# CDB SEMINAR

## Eiji Hara

Division of Protein Information, Institute for Genome Research,  
The University of Tokushima

Thursday, June 21, 2007

16:00~17:00 A7F CDB Conference Room

## The role of cellular senescence in cancer and aging

### Summary

Most, if not all, normal human somatic cells permanently stop dividing after a finite number of cell divisions in culture and enter a state termed "cellular senescence". These cells are irreversibly arrested in the G1 phase of cell cycle and are no longer able to divide despite remaining viable and metabolically active for a long period of time. Most tumors, however, contain cells that appeared to have bypassed this pathway and evaded senescence. Moreover, the genes that either enforce or bypass cellular senescence have been proven to be involved in tumorigenesis. Therefore, cellular senescence is considered as a barrier to cancer and plays an important role in tumor suppression. An important development during the past years has been the realization that cellular senescence is a complex and heterogeneous process. Cellular senescence occurs naturally when cultured human cells reach the end of their replicative lifespan, triggered by the inexorable loss of telomeric DNA, but a similar phenotype can be induced when cells are challenged by a variety of potentially oncogenic stimuli, such as DNA damage or certain oncogene expression. Our major focus is the understanding of the molecular mechanisms that induce cellular senescence in vivo and to elucidate how these mechanisms are perturbed in cancer. I will introduce you current research from our laboratory.

### Host:

#### Toru Kondo

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