第 10 回若手セミナー

筑波大学　テニュアトラック普及・定着事業

演題：　**Prolonged Mitotic Arrest Induces a Telomere-dependent DNA Damage Checkpoint**

演者：林　眞理　(Makoto Hayashi)
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日時：2013 年 3 月 28 日（木）　15:00-16:30

会場：医学系棟 4 階 482 室

要旨：　Telomeres protect natural chromosome ends from activating DNA-damage response pathways. Replication dependent telomere shortening and experimental disruption of telomeric components are well characterized pathways that induce telomeric DNA-damage response signaling and cell growth arrest. The former has been shown to induce replicative senescence, irreversible cell cycle arrest that supprresses tumorigenesis. I will discuss our discovery of a novel pathway, where prolonged arrest in M phase of the cell cycle induces a DNA-damage signal at mammalian telomeres. Cells that are arrested in mitosis potentially escape from mitotic arrest and become aneuploid cells, which carry abnormal number of chromosome and are implicated in tumor generation. We propose that prolonged mitotic arrest destabilizes telomeric structure, initiating a DNA damage response and growth arrest, thereby serving as a mitotic duration checkpoint to ensure cells that fail to progress through mitosis are eliminated from the cycling population. This pathway may underlie the mechanism of action of some cancer drugs, including vinblastine and taxol, that cause prolonged mitotic arrest.

REFERENCE:

1. Hayashi MT, et al., (2012) *Nat Struct Mol Biol*, 19: 387-394.
2. Hayashi MT and Karlseder J, (2013) *Oncogene*, in print
3. 林眞理 (2012) *医学の歩み*、241(11)： 823-828.

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