# 第 380 回つくば分子生命科学セミナー

TSUKUBA MOLECULAR LIFE SCIENCE SEMINAR

**演題：**Apurinic/apyrimidinic endonuclease 1, a close encounter of the two kinds: repair of oxidative DNA damage and cellular redox status

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**日時：**2013 年 10 月 9 日（水）17:00-18:30

**会場：イノベーション棟 1 階 105 室**

**要旨：**

Reactive oxygen species (ROS) are generated continuously, not only by exogenous and environmental genotoxins and radiation, but also from mitochondria as byproducts of respiration in our body. ROS play significant roles in plethora of cellular activities and in the transformation of cells into malignant cancer. Apurinic/apyrimidinic endonuclease 1 (APE1) in mammals, a linchpin in repair of oxidative DNA damage, is also a redox sensor to activate many transcription factors. However, APE1 has not been directly studied for its effect on intracellular redox status. The difficulty to study the role of APE1 in the cells stems from the fact that no APE1 defective cell lines exist due to its essentiality. The presenter's laboratory has established mouse embryonic fibroblasts (MEFs) that express APE1 at an extremely low level compared to their wild-type control MEFs. Analyzing these unique resources have led to the conclusion that APE1 affects mitochondrial respiration and cellular redox status. Together with its role for activating c-Jun/Fos and other transcription factors, the finding has a significant implication in the etiology of cellular degeneration, early events of cancer transformation and adaptation of tumor cells toward chemo and radiation resistance.

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