# 鈴木啓司准教授が日本放射線影響学会「Highly Cited Review Article Award (2021-2025)」を受賞

2025 年 10 月 24 日～26 日に開催された日本放射線影響学会第 68 回大会／第 6 回アジア放射線研究会議（JRRS/ACRR2025）合同大会において、鈴木啓司准教授（原研医療）は、Highly Cited Review Article Award(2021-2025)を受賞しました。

本賞は、2021～2025 年の Journal of Radiation Research に掲載された総説論文の中で、特に引用回数が多かったものの筆頭著者に授与される賞です。

Associate Professor Keiji Suzuki, Department of Molecular Oncology, was awarded the Highly Cited Review Article Award (2021–2025) at the Joint Meeting of the 68th Annual Meeting of the Japanese Radiation Research Society and the 6th Asian Congress of Radiation Research (JRRS/ACRR 2025).

# Highly Cited Review Article Award (2021-2025)

## 鈴木 啓司 殿

**Molecular and cellular basis of the dose-rate-dependent adverse effects of radiation exposure in animal models. Part II: Hematopoietic system, lung and liver**

Keiji Suzuki*, Tatsuhiko Imaoka, Masanori Tomita, Megumi Sasatani, Kazutaka Doi, Satoshi Tanaka, Michiaki Kai, Yutaka Yamada, Shizuko Kakinuma

***Journal of Radiation Research*, 64(2): Pages 228-249, 2023**

上記の貴殿の論文は、2021～2025年に *Journal of Radiation Research* に掲載されたReview Articleの中で、引用回数が特に高いと判断されましたので、ここに表彰いたします

令和7年10月24日

一般社団法人 日本放射線影響学会

理事長 田代 聡

# Highly Cited Review Articles (2021-2025)

(from the category of "Fundamental Radiation Science")

## The role of mitochondrial oxidative stress and the tumor microenvironment in radiation-related cancer

Tsutomu Shimura*

*Journal of Radiation Research,* Volume 62, Issue Supplement_1, April 2021, Pages i36–i43,
https://doi.org/10.1093/jrr/rraa090
Published: 05 May 2021

### Abstract

The health risks associated with low-dose radiation, which are a major concern after the Fukushima Daiichi nuclear power plant accident (the Fukushima accident), have been extensively investigated, and the cancer risks from low-dose radiation exposure (below ~ 100 mSv) are thought to be negligible. According to World Health Organization and the United Nations Scientific Committee on the Effects of Atomic Radiation reports, the level of radiation exposure from the Fukushima accident is limited, estimating no significant increased risk from the accident. Radiation-induced cell injury is mainly caused by oxidative damage to biomolecules, including DNA, lipids and proteins. Radiation stimulates metabolic activation within the mitochondria to provide energy for the DNA damage response. Mitochondrial respiratory chain complexes I and III are the most important intracellular source of reactive oxygen species (ROS) during oxidative phosphorylation in eukaryotic cells. Manganese superoxide dismutase and glutathione are key players in redox control within cells. However, perturbation of the antioxidant response leads to chronic oxidative stress in irradiated cells. Excess ROS of mitochondrial origin is reported in cancer-associated fibroblast and promotes carcinogenesis. The aim of this review paper is to discuss critical roles of mitochondria in radiation-related cancer by introducing our recent studies. In particular, elevated mitochondrial ROS in stromal fibroblasts potentiate transforming growth factor-beta (TGF-β) signaling, which triggers smooth muscle actin (α-SMA) expression to stimulate myofibroblast differentiation. Radiation-induced myofibroblasts promote tumor growth by enhancing angiogenesis. Thus, radiation affects both malignant cancer cells and neighboring stromal cells through secretion of soluble factors.

## Molecular and cellular basis of the dose-rate-dependent adverse effects of radiation exposure in animal models. Part II: Hematopoietic system, lung and liver

Keiji Suzuki*, Tatsuhiko Imaoka, Masanori Tomita, Megumi Sasatani, Kazutaka Doi, Satoshi Tanaka, Michiaki Kai, Yutaka Yamada, Shizuko Kakinuma

*Journal of Radiation Research,* Volume 64, Issue 2, March 2023, Pages 228–249,
https://doi.org/10.1093/jrr/rrad003
Published: 11 February 2023

### Abstract

While epidemiological data have greatly contributed to the estimation of the dose and dose-rate effectiveness factor (DDREF) for human populations, studies using animal models have made significant contributions to provide quantitative data with mechanistic insights. The current article aims at compiling the animal studies, specific to rodents, with reference to the dose-rate effects of cancer development. This review focuses specifically on the results that explain the biological mechanisms underlying dose-rate effects and their potential involvement in radiation-induced carcinogenic processes. Since the adverse outcome pathway (AOP) concept together with the key events holds promise for improving the estimation of radiation risk at low doses and low dose-rates, the review intends to scrutinize dose-rate dependency of the key events in animal models and to consider novel key events involved in the dose-rate effects, which enables identification of important underlying mechanisms for linking animal experimental and human epidemiological studies in a unified manner.

Author Notes: K. Suzuki, T. Imaoka, M. Tomita and M. Sasatani contributed equally.