Preface

Nagasaki University has been the "Global Strategic Center for Radiation Health Risk Control", which was selected as a Global Center Of Excellence (GCOE) Program by the Japanese Ministry of Education, Culture, Sports and Technology in 2007. One of the important purposes of our GCOE program is to educate young scientists for leaders of radiation research in the next generation and to promote academic collaboration with leading universities and institutes around the world. For the purpose, the "Nagasaki Global COE Program Young Investigators' International Symposium" was held on February 7-8, 2009 at Ryojun Hall, Nagasaki University School of Medicine, Nagasaki, Japan. It is our great pleasure and honor to have an opportunity to publish the symposium proceedings as a supplement of Acta Medica Nagasakiensia.

This symposium, entitled "Perspective of Radiation Research", aimed at getting deeper insight into molecular and cellular response to ionizing radiation. Ionizing radiation is presumably the oldest environmental stress to which all life on earth has been exposed until now, and thus, the mechanisms of cellular and molecular radiation response are well conserved among the species. Classical radiation research established the "central dogma of radiation biology", which is: radiation produces DNA double-strand breaks, and cells survive if the DNA damage is repaired, otherwise cells die. Furthermore, recent studies unraveled the molecular mechanisms of DNA damage repair, checkpoint, and apoptosis, which made us realize how precise and sensitive radiation response is. However, the recent findings simultaneously have told us that we must take into account what we've never considered. For instance, we now must consider the response to DNA double-strand breaks not only on DNA level but also on chromatin level, which is best exemplified by phosphorylation of histone H2AX and its focus formation. Moreover, recent studies revealed that chromatin remodeling, histone post-translational modification, and nucleosome assembly/disassembly, which are once thought as processes of gene transcription and/or DNA replication, are also involved in DNA double-strand break repair. And there is accumulating evidence that interaction between irradiated-, and non-irradiated cells can cause DNA double-strand breaks or can affect DNA repair efficiency. To discuss and exchange ideas, 10 young distinguished scientists in radiation biology/DNA damage response field were invited from all over the world. Their papers, which are collected in the present volume, really inspired us all and gave us deep insight. I am confident that the symposium ended up being fruitful.

Finally, I would like to express my sincere appreciation to all contributors for these interesting and valuable papers and, especially, to Professor Masataka Uetani for his great help in editing this publication.

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