

New ICRP Publication 131: Stem Cell Biology with Respect to Carcinogenesis Aspects of Radiological Protection

O. Niwa^a, M.H. Barcellos-Hoff^b, R.K. Globus^c, J. Harrison^d, J.H. Hendry^e, P. Jacob^f, M.T. Martin^g, T.M. Seed^h, J.W. Shayⁱ, M.D. Story^j, K. Suzuki^k, S. Yamashita^l

^aICRP Main Commission, Fukushima Medical University, Fukushima, Japan

^bRadiation Oncology and Cell Biology, New York University School of Medicine, New York, USA

^cBone and Signalling Laboratory, Space Biosciences Research Branch, NASA Ames Research Center, CA, USA

^dICRP Committee 2 Chair, Centre for Radiation, Chemical and Environmental Hazards, Health Protection Directorate, Public Health England, Chilton, Oxon, UK

^eChristie Medical Physics and Bioengineering, Christie Hospital NHS Foundation Trust and University of Manchester, UK

^fHelmholtz Zentrum Munchen, German Research Center for Environmental Health, Neuherberg, Germany

^gLaboratoire de Genomique et Radiobiologie de la Kertinopoiese, CEA, Evry, France

^hMaple Avenue, Bethesda, USA

ⁱRadiation Oncology, Simmons Cancer Center, University of Texas, Southwestern Medical Center, Dallas, Texas, USA

^jMolecular Medicine, Graduate School of Biomedical Sciences, Nagasaki University, Japan

Current knowledge of stem cell characteristics, maintenance and renewal, evolution with age, location in stem cell “niches”, and radiosensitivity to acute and protracted exposures, is reviewed regarding haematopoietic tissue, mammary gland, thyroid, digestive tract, lung, skin, and bone. This foundation is used to provide a biological insight to protection issues such as the linear-no-threshold and relative risk models, cancer risk differences among tissues, dose-rate effects, and changes in the risk of radiation carcinogenesis by age at exposure and attained age. The identity of the target cells for carcinogenesis continues to point to the more-primitive and mostly-quiescent stem-cell population, able to accumulate the protracted sequence of mutations necessary to result in malignancy. Several biological processes could contribute in protecting stem cells from mutation accumulation: (1) accurate DNA repair, (2) rapid induced death of injured stem cells, (3) retention of the intact parental DNA-template strand during divisions in some tissues, so that mutations are passed to the daughter differentiating cells, and (4) stem cell competition, whereby undamaged stem cells outcompete damaged stem cells for residence in the vital niche. DNA repair operates mainly within a few days after irradiation, while stem cell competition requires weeks or many months depending on the tissue type.