INTRODUCTION

About a century ago, radiotherapy advanced with multibeam techniques and rotational therapy sets, by which radiation dose received by normal tissues can be reduced and the dose distributed in tumors can be conformal. Given that the fractionated dose used in radiotherapy is higher than the thresholds of many critical organs, the treatment plans of radiotherapy are inevitably made in accord with the physical characteristics of an individual for sparing normal tissues and lowering the risks of secondary cancers. The practice of personalized regimens, however, has been less applied by radiographers.

While patient radiation dose in the scale of several rems to several millisieverts (mSv) often results in cellular adaptive defense upon mild damage of DNA and other cellular constituents, long-term repetitive exposure to low-dose radiation (<100 mSv) still leads to increased cancer mortality. Studies have shown low dose and dose rate radiation exerted radioprotective effects by manifesting declines in cell transformation frequency and increases of cancer latency on animals [1,2]; however, marked survival reduction was observed in relation with repetitive CT scans [3]. Patients with cystic fibrosis undergoing five-times of annual CT scans with higher yearly dose (5.0 mSv vs. 0.2 mSv) were more prone to carcinogenesis (solid cancer: 5.94% higher in males and 5.66% higher in females; hematologic cancer: 1.91% higher in males and 0.93% higher in females) and short-lived (reduction in males: 6.67 months; reduction in females: 5.11 months) [3].

It is speculated that bystander effects, elicited on unirradiated surrounding cells through cell-cell and cell-matrix communication, overturn the beneficial effects of radiation-induced hormesis and promote cell malignant transformation. Delayed cell death, genetic instability and mutagenesis have been attributed to prosurvival cytokines, hazardous oxygen species and growth factors released by low-dose irradiated cells [4]. In addition, radiation-induced hormesis was found absent in flies that were susceptible to low-dose radiation [5]. Correspondingly, humans with p53 and ATM loss are subject to radiation-induced carcinoma as a result of insufficient DNA repair [6,7]. In observance of increased incidence of cataracts and breast cancers on subjects with ATM heterozygosity [6,8], the genotype profiles of patients should be evaluated prior to radiologic examinations for preventing the exposure of every individual's head and chest to sublethal but carcinogenic radiation.

To reduce potential stochastic effects arising from cumulative bystander or non-bystander effects, healthcare providers should well-estimate the excess radiation risk of each patient for making personalized radiotheranostic plans. Approaches for patient radiation protection include but not limited to understanding low-dose radiation epidemiology, application of internal and
external personal protective equipment/materials, and administration of antioxidants as well as radiation dose surveillance and follow-ups and review of cancer incidence [9-13].

REFERENCES