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Revisit the Effects of Low Dose Radiation in Diagnosis and Therapy

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Editorial

Since Wilhelm Conrad Rontgen discovered X-rays in 1895 followed by Big-Bang breakthrough of researches in modern physics, radiation has become part of human life more than one century. For common populations, the medical radiation is probably the most well-known application of this basic physic research. As a powerful weapon in clinical, radiation has been widely used in diagnosis and therapy. Nowadays, medical radiation by external beams and nuclear medicine account for approximately 50% of annual radiation exposure in United States (National Council on Radiation Protection and Measurements, NRCP Report No. 160, 2009). General speaking, the dosage of medical radiation is believed to be ablated to avoid side effects. However, accumulated literatures concern the abused application of radiation diagnosis, especially the Computed Tomography (CT) scan that may cause unexpected side effects [1]. Additionally, the intensity-modulated radiotherapy (IMRT) originally designed for delivery of precise doses to tumor with minimized dose to surrounding tissues has been reported to increase the probability of side effects and secondary cancer induction, although such a risk may depend on tissue types and patient selection (such as age at exposure) [2]. Therefore, the effects of low dose radiation should not be overlooked for diagnosis and therapy.

The effects of low dose radiation remain falling in debate from basic science to public health. The Linear No-Threshold (LNT) model has been adopted more than 50 years for radiation safety, based on a "no safe dose" hypothesis. Some researchers believe this is wrong model because the data from radiation biology and experiments did not agree LNT model [3]. However, a recent report using the Chernobyl data showed that the heritable effects of radiation at low dose range are greatest rather than linear relationship [4]. Because CT scans are the major concerns of low dose radiation (over 10 mSv) applied in diagnosis of human diseases, the safety of CT was argued. An Epi-CT study held in nine European nations has been triggered by recruiting a million young patients, and the analytic results will be released in 2017 [1]. Indeed, as Dr. David Brenner in Columbia University pointed, the anxiety about increasingly use of radiation based instruments in clinical should base on profound basic and epidemic researches rather than prejudice to radiation itself.

For medical radiation, the risk of low dose radiation mainly correlates to the probability for inducing death or mutation of normal tissues. Although the evidence of radiotherapy induced secondary malignancy is largely based on the epidemic research, the underlying mechanisms remain to be addressed. High dose ionizing radiation is believed to induce DNA damage followed by a series of signaling pathways to trigger apoptosis, cell cycle arrest and DNA repair. Low dose radiation, on the contrary, induced more complicate biological effects that may explain the controversy of LNT model. Low dose range from 1 mGy to 2 GY can induce the γ-H2AX foci formation, representing the increase of Double Strand Breaks (DSBs) [5]. If this is the common phenomenon, different signaling pathways activated by low dose radiation may stem from the different levels of DSBs. A single signaling pathway may be not sufficient to explain the biological effects caused by low dose radiation. Therefore, it is about time to investigate how dose radiation influences the intracellular signaling pathways on different tissues and cancer types. Moreover, whether pathways of interests are only activated by different windows of low dose radiation should be examined.

Compared to high dose, low dose radiation may not induce severe cell death. It is speculated that DNA damage levels are low and easy to be repaired. The stress induced by low dose radiation remains affecting the signaling pathways for other cell responses, such as cell cycle arrest and senescence. Immediately early response genes that are usually raised by various stimuli are considered to be affected by low dose radiation. Some of these early response genes such as c-Myc and Ras are classified as oncogenes that may account for potent side effect of low dose radiation. Oncogene-Induced Senescence (OIS) has been proposed as a process of malignancy, although the underlying

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mechanisms are still obscure. Whether low dose radiation would induce OIS to cause secondary malignancy is of interest to further investigate. Our lab had found that c-myc inhibitor could suppress low dose radiation induced senescence in tumor cells, which could promote the invasion of unirradiated tumor cells (unpublished data). Induction of early response genes by low dose radiation may be an effect for induction of secondary malignancy that can be prevented by certain inhibitors to alleviate the adverse effect caused by low dose radiation.

It is believed that radiation will still largely used in clinics even new alternative approaches are developing. Reduction of the side effects and potent mutation issues by low dose radiation remains a critical topic to improve the quality and safety of patient therapy. Revisit the complex molecular biological networks re-established by low dose radiation would be one of the keys to solve this plight of radiation based diagnosis and therapy.

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