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Review

Biophoton signaling in mediation of cell-to-cell communication and radiation-induced bystander effects

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ABSTRACT

This paper presents a comprehensive overview of the historical trajectory and development in biophoton studies over the past 100 years, with a particular focus on the recent progress regarding the pivotal role of biophoton in mediating radiation-induced bystander effects (RIBE). The exploration of biophoton mystery starts from the initial observation of mitogenetic radiation and continues to develop to the contemporary science of biophotonics. The properties and underlying mechanisms of biophoton emission are described with illustrative examples from diverse biological systems such as plants, animals and humans. The conclusive evidence of cell-to-cell communication facilitated by biophoton signaling is presented, followed by an elaborate interpretation of potential mechanisms through which biophoton mediates RIBE. The engagement of mitochondria and exosomes in this process is extensively clarified, by highlighting their significant roles in biophoton-mediated RIBE. The advances in biophoton research in respect of bystander response to ionizing radiation may offer profound insights into radiobiology and provide for possible future applications as well in radiation medicine and protection.

1. Introduction

Low-dose ionizing radiation (LDIR) responses refer to the reactions or effects observed in biological systems when exposed to ionizing radiation (IR) at low doses (<100 mGy) and low dose rates (<6 mGy/h), which are considered to bring about biological responses involving changes in biological functions, cellular processes, or potential health risks with low-level radiation exposure.[1,2] The classical and contemporary studies on low-dose radiation effects have been illuminated and thoroughly examined, particularly for hormesis[3–6] and adaptive radiation response,[7] along with bystander effects and other non-targeted effects (NTE).[7,8] Notably, these effects were observed to exhibit nonlinearity within the low-dose range of IR. Mechanistic investigations have revealed that crucial cellular processes, such as gene induction,[9,10] gene expression and epigenetic control,[11] protein expression,[12] DNA repair,[13] and intracellular signaling related to cell cycle arrest and apoptosis,[14] all manifest nonlinearity at low doses. Additionally, interconnected intracellular and extracellular signaling processes have also been explored accordingly.[15,16]

A comprehensive series of reviews have delved into and commented on the significance of the bystander effect induced by IR. Initially, these studies focused on the potentially adverse biological effects of LDIR, including cytotoxicity,[15,17–19] mutagenicity,[20–22] genomic instability,[23–25] carcinogenic effects,[26] and inflammatory responses.[27] These radiation-induced deleterious effects to the organism were later defined as bionegative effects.[7] However, subsequent research provided deeper insights, revealing that the bystander effects could also bring about positive biological outcomes[7,28–30] in extended dose ranges. These biopositive effects, defined as radiation-induced beneficial effects to the organism, were mediated by systemic and long-distance abscopal reactions, hormesis and adaptive responses, leading to favorable consequences in the organism.[31–35] Nevertheless, in some cases, these effects can also be bionegative.[36]

Hormesis serves as an example of beneficial low-dose IR effects,[37–39] specifically in terms of enhancing mitochondrial functions.[40] In fact, low dose IR can effectively prompt cell proliferation and survival, while elevating antioxidant and immune responses. This intricate mechanism acts to strengthen cellular defense systems, thereby enabling cells to endure subsequent stress. Hormesis may be induced via a direct stimulation or by over compensation to a disruption of homeostasis.[41]

Protective adaptive radiation processes are evolutionarily conserved.[42] The adaptive radiation response has been aptly characterized by the observations[43] that when human lymphocytes are first exposed to a low "priming" radiation dose from low concentrations of tritiated thymidine, followed by a subsequent, higher challenging dose of 1.5 Gy of X-rays, they exhibit fewer chromosomal aberrations than when

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