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Radiation injuries in people and animals: A lot of food for thought

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Abstract

The present Editorial deals with radiation-induced acute and chronic biological effects in humans and in animals, to which special attention should be also paid in view of the ongoing war theatres in Ukraine as well as in Near and Middle East.

Within this challenging and frightening scenario, emphasis is placed upon chronic radiation-induced effects, with particular reference to tumour development, on one hand, and to protective host cells' mechanisms like p53, on the other hand, within a One Health perspective.

Keywords: radiation-induced acute effects, chronic biological effects, war theatres in Ukraine and Middle East, p53.

Editorial

Against the backdrop of the ongoing war theatres in Europe, Near and Middle East, along with the related nuclear threats and associated (human and animal) radiation exposure risk, summarizing radiation-induced damage may turn useful.

In this respect, animals exposed to either ionizing (alfa, beta, gamma particles and X-rays) or non-ionizing (UV-rays) radiations are known to suffer from biological effects that, as a general rule, are quite similar to those commonly experienced by humans. Radiation exposure in animals can lead, in fact, to both acute and chronic injuries, with the former ones occurring relatively quickly and being characterized by skin erythema and desquamation as well as by more severe effects like "acute radiation syndrome" including nausea, vomiting and, in the worst cases, a fatal outcome. Acute effects' severity largely depends upon the duration and intensity of exposure, coupled with the distance from the radiation source (1).

Conversely, chronic radiation-induced damage, developing months if not years later, tends to manifest with organ and immune system dysfunction (immunosuppression), along with tumour development in various body sites. Notwithstanding the above, however, radiation injuries were systematically investigated in a limited number of laboratory animal species, such as mice, rats, guinea pigs, rabbits, dogs and monkeys (2). More recently, the chronic effects of ionizing radiation were experimentally predicted in 14 vertebrate species, 11 mammalian and 3 avian, with small animals (mice, sparrows) being more resistant than big ones (elephants) and with thresholds for health effects largely depending upon metabolism, longevity and individual host tissues' susceptibility (3).

Chronic radiation effects are known to originate from the biochemical damage inflicted on cell macromolecules like membrane lipids (undergoing rapidly expanding peroxidation phenomena), proteins (both enzymatic and non-enzymatic), sugars and, above all, nucleic acids (DNA and RNA), with the latter ones undergoing widespread degradation/denaturation/rupture processes, followed by genomic mutations culminating in irreversible cell alterations (1). Among the over 200 cytotypes populating terrestrial and aquatic mammals' bodies, highly variable sensitivity levels to radiation are known to exist, with "labile" - alias intensely replicating - cells (male and female gametocytes, haematopoietic stem cells, lymphoid cells, basal layer epidermis cells, etc.) exhibiting the

greatest susceptibilities, as clearly shown by the subsequently developing "aplasia/hypoplasia" and/or neoplastic processes affecting these cell populations in both natural and experimental disease models (1,4).

Up to a "given magnitude" of radiation injury to animal (and human) cells, a number of molecular players become activated in order to revert the DNA damage, thus preventing the occurrence of further genomic alterations. Within such a dynamic and challenging scenario, a key actor is represented by p53, popularly known as the "cell guardian", a transcription factor crucially involved either in DNA repair or, alternatively, in the initiation of cell apoptosis -alias "programmed cell death" -, should significant DNA alterations occur (1). Unfortunately, also the p53-encoding gene is not spared by the aforementioned radiation-induced mutations. As a consequence, rather than orchestrating the establishment of an apoptotic "cascade", mutated p53 will just do the opposite, thus leading animal (and human) cells to progressive "genetic instability" resulting in turn in tumour development (1). This is the main reason why mutated p53 is increasingly regarded as an early "biochemical signature" of neoplastic trasformation in humans and in animals, especially in dogs (1,5,6). Interestingly enough, however, a lower cancer prevalence was reported to occur in African elephants, which have at least 20 copies of wild-type p53 compared with human and animal cells, that generally harbour one single copy of the aforementioned transcription factor (7).

Additionally, I believe we should also take into adequate account the potential role of radiations as physical noxae causing mutations in the genetic make-up of microbial pathogens, with special emphasis on rna-viruses, given the apparent rise in emerging infectious diseases caused by such agents.

As a concluding remark, I believe that a multidisciplinary, holistic and "One Health"-based approach would be absolutely needed in handling, counteracting and preventing (human and animal) acute and chronic radiation-induced damage, all the more in the light of the scary war theatres in Ukraine as well as in Near and Middle East.

Historia Magistra Vitae!

Declarations

Conflict of Interest

The Author declares that there is no conflict of interest.

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