Effect of Radiation on Physical and Mental Health

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Abstract: We reviewed available evidence in medical literature concerning experimental models of exposure to ionizing radiations (IR) and their mechanisms of producing damages on living organisms. The traditional model is based on the theory of “stochastic breakage” of one or both strands of the DNA double helix. According to this model, high doses may cause the breaks, potentially lethal to the cell by damaging both DNA strands, while low doses of IR would cause essentially single strands breaks, easily repairable, resulting in no permanent damages. The available evidence makes this classical model increasingly less acceptable, because the exposure to low doses of IR seems to have carcinogenic effects, even after years or decades, both in the exposed individuals and in subsequent generations. In addition, the cells that survived the exposure to low doses, despite being apparently normal, accumulate damages that become evident in their progeny, such as non clonal chromosomal aberrations, which can be found even in cells not directly irradiated due to the exchange of molecular signals and complex tissue reactions involving neighboring or distant cells. For all these reasons, a paradigm shift is needed, based on evidence and epigenetics.

Keywords: Ionizing radiations, cellular damage, carcinogenic and epigenetic mechanisms

I.INTRODUCTION

The risk of ionizing radiation (IR) on human health has been widely known since the last century. There's a general agreement that excessive doses of IR represent a main threat to human health. At the opposite end of the spectrum, many scientists have expressed developing doubts and proposed different models regarding the dangers connected to chronic exposures to small doses of ionizing radiations, which can be much more common than accidental exposure to excessive doses. Those capacity risks could recognize new organic mechanisms of harm, such as epigenetic, procarcinogenic pathways and Tran’s generational transmission. The adoption of the patterns of exposure, danger assessment, and damage (especially carcinogenicity) in environmental health (specifically IR) are inevitably affected by the manner in which history determined and conditioned the research. It's miles for this reason that, to better apprehend the necessity of a paradigm shift, we need to start from a brief historical evaluation of radiobiology, a discipline ruled by using physicists who described for many years the interactions among radiations and living rely mainly in terms of energy transfers and DNA harm. In reality, radio biologists consciousness on a passive, mechanistic model of DNA damage, even if rising evidence in the area of molecular biology indicates that the interactions between IR and dwelling organisms, starting from the arguable issue of carcinogenesis, must be studied in a systemic way, deliberating the complexity of tissues, cell signaling and genetic reactions involved.

The so-called linear and no-threshold model (LNT) has been identified for 1/2 a century as the methodological foundation for predicting long-term organic harm caused by IR. This version is still accepted by the most relevant global businesses and researchers. The second pillar of classical radiobiology arose from a more unique definition of the number one damage to DNA, which followed the description (in 1961) of stochastic breakage of 1 or both strands of the double helix (single-strand breaks (SSBs); double-strand breaks (DSBs), interpreted as the number one lesions in DNA uncovered to IR. On this foundation, in 1973, the linear quadratic equation (LQ-Linear Quadratic equation) became formulated, based at the concept that low doses of ionizing radiation need to basically purpose SSBs, without difficulty repairable, while excessive doses would motivate the breaks, doubtlessly lethal to the cell, of both strands of the double helix of DNA (for low doses we mean, alongside the textual content, doses underneath zero. Five gray). In keeping with this model, simplest a big exposure to IR (of the order of one–2 gray or extra) could decide large damages to tissues or human health, and the results need to be distinguished by deterministic (caused by direct mobile damages) and stochastic consequences. The deterministic effects are nearly immediately: the quick-time period publicity to large doses of IR on proliferating tissues (bone marrow, blood, and epithelial cells in growing organisms; many one-of-a-kind cell kinds in growing organisms) might cause the loss of life of thousands and thousands of at once affected cells. The effects should be directly proportional to the extent of the harm and the period of the publicity: bone marrow aplasia, bleedings, blood poisoning, coma, and demise may want to arise inside min/hours from massive exposures to IR; anemia, ageing, diarrhea can be induced by way of greater diluted big exposures, consistent with this model, additionally the stochastic results could rely from the entire dose of IR, and could cause—through the free radicals and reactive oxygen species (ROS) produced by using the radiolysis of water—“stochastic” damages on DNA ensuing in procarcinogenic results if the affected genes are involved in mobile cycle manipulate or in programmed cell demise (apoptosis) and DNA restore (proto-oncogenes and tumor suppressor genes).

The first-rate majority of epidemiological data regarding the effects of exposures to IR came from the cohorts of the survivors of the bombing of Hiroshima and Nagasaki and the fallout of radionuclides after the Chernobyl coincidence and, to a lesser extent, from the studies on the outcomes of extended X-ray occupational exposures (i.e., in medical diagnostics); very specific conditions, characterized by using completely assorted modes and types of publicity although, the modalities of publicity assessment, damage (specially carcinogenicity), and radioprotection were heavily promoted through these theoretical models.

Within the case of Hiroshima and Nagasaki survivors, a hundred thousand humans have been subject to a massive overall body brief-time period of publicity to huge doses of radiation at low and excessive let (linear electricity transfer). The outcomes of
such publicity have been the almost instantaneous loss of life of thousands of women, men and children; the slow agony of numerous thousand sufferers whose tissues were irreversibly damaged by way of debris and neutrons produced by the explosion; an amazing range of diseases (neoplastic or not) discovered after years or many years each among survivors and their offspring. As for most cancers the first applicable data turned into a significant boom of leukemia (excluding chronic lymphatic leukemia), recorded inside the years right now following the bombing. After a few years, the epidemiologists detected a huge boom of stable tumors in diverse tissues and organs, especially thyroid, breast, and lung cancer. but, important limitations concerning those studies were reported: the depend of the survivors became not correct, and the records started to be reliable simplest many years after the exposure but are still incomplete? Another critical word issues the hundreds of dramatic occasions (abortions and deaths of youngsters), that took place in the first weeks/months after the tragedy that have never been recorded. yet, the maximum difficult fact to give an explanation for concerned the offspring of the survivors, as most research did not file in the children born from the extra exposed dad and mom, mainly inside the early many years, neither an growth of mutations, nor an extra of congenital malformations nor a massive growth in tumors. Only a few years later, primarily based on more state-of-the-art investigations, become it viable to make clear the importance of these apparently reassuring and paradoxical information. Indeed, a quick-term exposure to massive doses of IR, ensuing in mobile irreversible damage, decided a twofold impact of selection: first on cells and tissues of the survivors, after which on the general population (because the survivors are, by means of definition, a selected factor of the whole exposed population)
The limited general count number of harmful effects in a population exposed to high total doses of IR and the supposed absence of major variations between more or much less exposed subjects lead most people of researchers and worldwide companies to underestimate for decades the risks of radiations, especially regarding the outcomes of extended exposure to low doses, which are the most common and maximum risky for human being. These serious mistakes of judgment had its roots within the manner in which the studies were carried out based on the LNT model and thinking about the whole absorbed doses on the basis of the distance from the epicenter of the explosion, via extremely complex calculations, revised numerous times

II. CONCLUSION

Large-scale projects of genome sequencing allowed significant progress in identifying the mutational profile of cancers. some mutational signatures had been related to the action of endogenous techniques or environmental exposures; e.g. tobacco smoke, ultraviolet light, aflatoxins, aristolochic acid, and IR recent research documented the lifestyles and endurance of cancer epigenetic signatures related to environmental exposures, which include cigarette smoking; but, until now this has been limited to one epigenetic mark (i.e., DNA methylation), it is in all likelihood that in the destiny the analysis of mutational and epigenetic signatures in experimental research as well as in epidemiological studies may want to constitute a completely promising area of studies on publicity to IR considering such emerging proof, there is the need for a changing paradigm inside the interpretation of the mechanisms leading to mobile damage after publicity to IR, moving from a traditional paradigm based at the “stochastic breakage” of one or both strands of the DNA double helix caused only by way of high dose exposures toward an epigenetic model that could also explain the trans generational effects of slight and chronic irradiation

REFERENCES