Review Paper:

The genomic effects of radiation exposure: induction of cancers and genetic aberrations in mammalian cells

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Abstract

Radiation exposure can be a two-edged sword. On one hand, the deployment of radiotherapy is one of the most acceptable modalities for malignancy treatment, for the reason of its ability to kill cancer cells. Equally, one is scared of the exposure to man-made emissions in the form of nuclear disasters or related to any form of hilarious power generation during the war. It is rational to facilitate the body enduring small intensity of emissions without any marked side effects. However, there are countless detrimental side effects of being exposed to elevated levels of radiation. These harmful side effects form an underlying fear in the general public in understanding the actual mechanism and concrete remediation of radiation exposure.

The present study investigates in detail the principles of radiation safety and how different types of radiation emissions affect the human body when exposed at different levels of their intensity. Furthermore, it is also conferred how cellular mutations are originated in different organs and body systems due to different levels of radiation exposure and also the potential of organisms to develop resistance (threshold limit) to counteract harmful effects of radiation.

Keywords: Cellular mutagenesis, DNA lesions, Radiation emissions, Chromosomal aberrations, Cancer induction.

Introduction

The biological damage that results from ionizing radiations has been a point of concern for several years. The most important radiation-related human damage was reported hardly a few months after the exploration of X-rays by W.C. Rontgen in 1985 and the primary cause of emission-stimulated malignancy was identified just seven years after it[11,76]. The shred of evidence for the harmful effects of radiation exposure can collectively be made available from the health experiences of radiation miners working in mines, uncovered to airborne radioactivity and the workers operating in the radium industry[17,50]. The major apprehension of knowledge in this regard has been assembled from past experiences: health studies of Japanese nuclear bomb survivors of Hiroshima and Nagasaki, victims of accidents at nuclear installations including the Chernobyl incident etc. (Fig. 1). The pattern of such events evidenced the ill-effects of total body radiation exposure because of ionizing radiations and documented in the reported literature[55,70].

The mechanistic approach behind the radiation-induced injuries

DNA lesions of significant biological importance: The high-intensity ionizing radiations like X-rays, gamma rays, electrons, protons and alpha particles in the biological system result from different types of physical, chemical and biological products and chemical lesions formed inside the irradiated living cells that result in the evolution of these lesions from one to another with time. The physical processes of excitation and ionization are momentary and proceed only for 10-15 sec. Since 80% part of a living cell is composed of water, therefore the physical processes generate H_2O radicals -OH radicals, -RH radicals and other chemical disorders[14,58,74].

These emissions are competent for inducing a broad range of DNA abrasions which include damages not only to the nucleotide bases and their cross-linking but also confer to the single and double-strand breaking of DNA molecules and are significantly important for inducing the mutagenesis (both chromosomal deformity and gene transmutation). These radicals and chemical scratches disseminate and act in response to the other biomolecules present within the cell such as DNA genetic molecule, consequently creating DNA free radicals and other bimolecular lesions in the biotic stuff present inside the cell[38,73].

DNA radicals can be formed either by an undeviating chemical interaction of radiations on the targeted DNA atom, thus damaging it directly, or by an indirect radiation effect that refers to the targeting of DNA molecules indirectly by chemical reactions (Fig. 2)**.** It has been confirmed that certain types of DNA base pair damage (such as thymine glycol and hydroxyldeoxyguanosine) play an important role in radiation stimulating mutagenesis. The biomolecular abrasions are comparatively vulnerable and can wind up in minutes, days, weeks, months, or even sometimes in years in a cell, thus making the entire process extremely complex[25,44].

Radiation-induced mutagenesis: It has been pragmatic from the experimental studies that radiation induced both point mutations and deletions. However, there is no direct evidence for the site-specific transformations induced by radiations. The structural adaptations connected to direct radiation are supposed to be different from that of impulsive radiation. It is apparent that radiation induces several stable

chromosomal aberrations that include both the deletion and shared translocations and plays an important role in radiation-stimulated carcinogenesis[40,43].

The cancer cells being aneuploid include several chromosomal alterations that are linked to some specific types of tumors including leukemia etc. The radiation exaggerates cancer, endorses several discrepancies in the chromosomal restructuring and is associated with the concern of certain oncogenes or tumor suppressor genes in the body cells[27,34].

Effect of radiation exposure on specific organs: The deleterious effect of radiation on the whole body and specific organs results in the generation of mutations that are transmitted from generation to generation. The types of mutations induced on individual organs and their mutagenic effect due to high energy radiation exposure have been summarized in fig. 3.

Testis and Ovaries: The testis and ovaries are considered to be the vital reproductive organs that should be protected from radiation-induced mutations[20]. The cells of the testis are highly sensitive to damage from external radiation exposure. It has been demonstrated through experimental studies that radiation exposure in the range < 0.2Gy enhances the level of reactive oxygen species (ROS) which in turn induces ER stress and apoptosis in testicular cells[45,49].

In addition, it has also been observed from experimental studies that a longer duration of radiation exposure led to changes in the expression of miRNA which induces apoptosis due to oxidative stress and generates aberrant gene expressions[56]. Similar results were also reported in the case of ovaries where a radiation exposure of the range of 0.36Gy damaged almost 50% of the germ cells in ovaries with subsequent reduction in the number of follicles, showing severe hormetic effects in the ovary[6,22].

Thyroid: The thyroid is supposed to be one of the sensitive organs (glands) vulnerable to radiation exposure. The radiation-induced effect can be through CT-scan, occupational exposure, nuclear accidents etc. It has been demonstrated in various experimental studies that radiation exposure to a range of 0.5Gy can induce a considerable risk of generating ROS and DNA damage in response[5,47]. The radiation exposure effect on the thyroid can also be pragmatic physically by a considerable expansion in the gland size, colloid density, hemorrhage enhancement, diminution in thyroid hormone secretion etc.[41,53] In similar related studies, it has been reported that radiation dose exposure of 0.5Gy range results in the initiation of disease (*thyroiditis)* during enhanced auto-immunity disorders. In conclusion, radiation exposure to the thyroid can easily harm the gland through significant DNA damage[57,63].

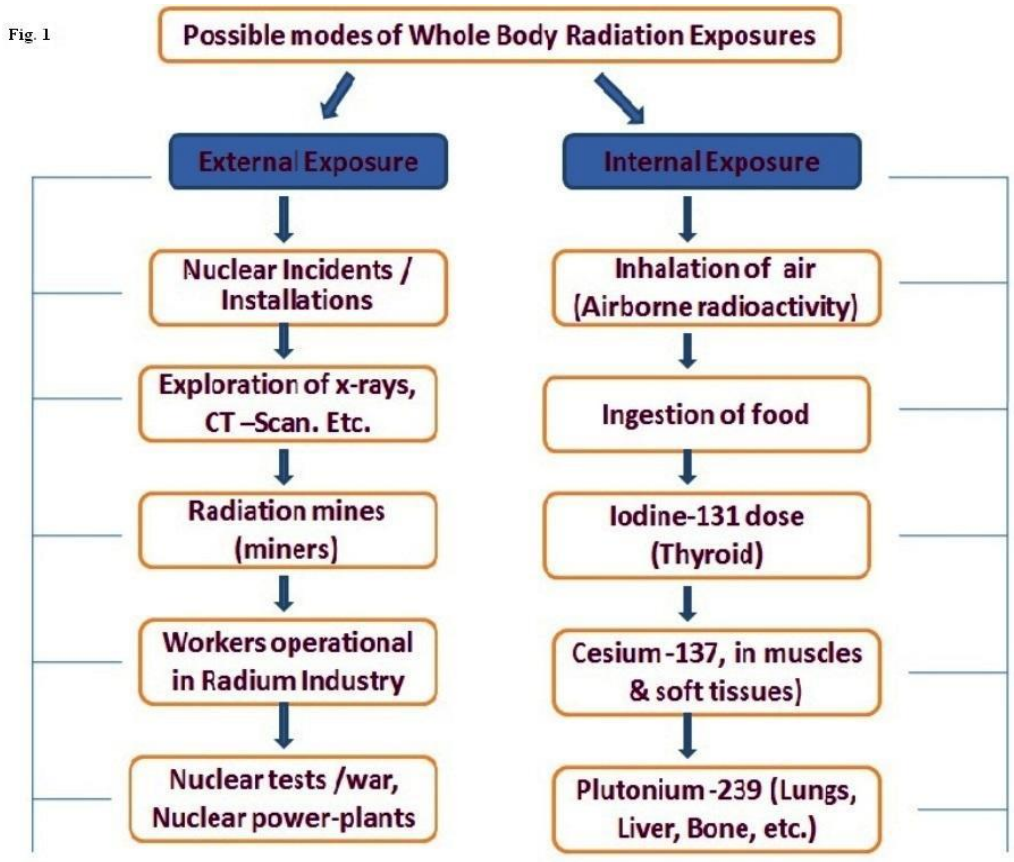

Figure 1: Different transmission modes of radiation exposure in the body include both external sources or may generate internally in different organs of the body.

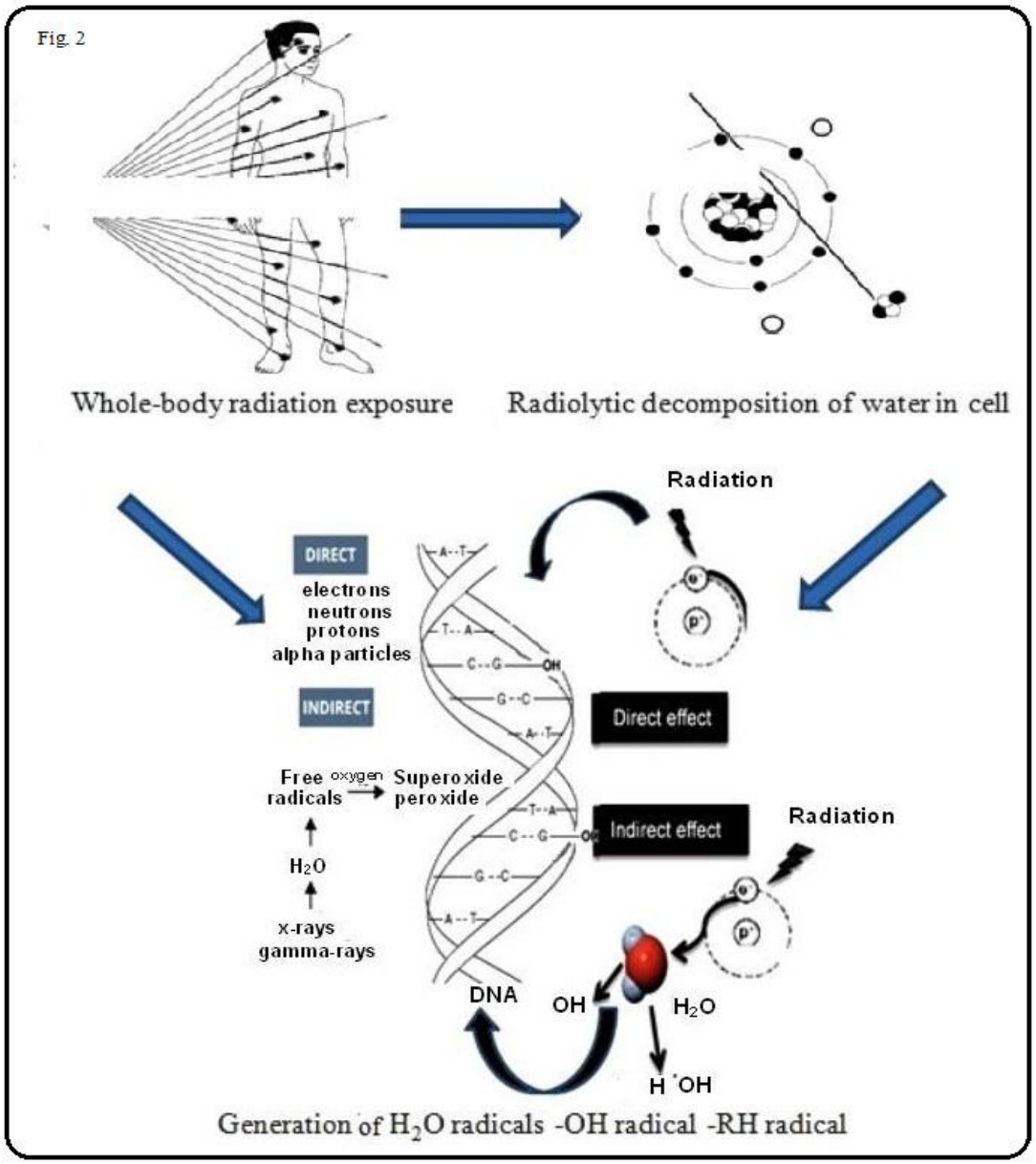

Figure 2: Effect of radiation exposure on the body: Generation of free radicals and initiation of DNA abrasions through single and double-strand breaking, inducing both chromosomal and gene deformities

Skin: Skin is another organ that is primarily exposed to radiation and gets affected by it. Even at a low radiation dose (LDR) exposure of 0.1Gy molecular alterations in the skin takes place. In HaCaT cells of the skin, low radiation of 0.1Gy causes induction of P-21 expression resulting in the differentiation in the keratinocytes[30,31]. A similar related study highlights the effect of radiation on the skin through dysregulating the articulation of TP53, BAX and BCL-2 genes, resulting in a cell-cycle clutch with stimulation of pro-apoptotic gestures[32]. Similarly, the manifestation of collagen type-1 and alpha-1 in dermal fibroblast was reported to be enhanced due to the radiation exposure in the 0.5Gy range.

Similar other related studies conclude with a general apprehension that the genes regulating the extra-cellular matrix (ECM) were induced both by high and low radiation dose exposure. However, the genes relating to the cytoskeleton and intercellular signaling can be induced even by low radiation exposure without causing any significant alterations in the genomic DNA[30,13].

Immune Cells: Several experimental studies were carried out discussing the particular molecular actions and signaling pathways in response to radiation exposure to immune cells. Low radiation exposure of 0.1Gy slowed the expansion of bone marrow mesenchymal cells (BM-MSCs) and induced the proliferation of hematopoietic cells (CD34+, CD38+ cells), through enhanced expression of IL-6 and suppression of tyrosine kinase-3 ligand.

Although the results obtained from different studies are not consistent to explain the proliferation of bone-marrow stem cells, a common inference that can be drawn from all such

related studies is that low radiation exposure enhances hematopoiesis by increasing the secretion of cytokines[21,65].

Innate immunity is believed to be a key player in maintaining a defense mechanism against various antigens through phagocytosis. Any change in innate immunity cells due to radiation has a direct influence on immune responses. It has been observed that an LDR < 0.5Gy is supposed to decrease the oxidative strain by triggering superoxide dismutase (SOD) and restraining mitochondrial permeability. In one such related study, it has been demonstrated that radiation exposure in the range of 0.25 to 0.5Gy induces glutathione expression by activating protein-1 and NF-KB pathways in macrophages[16].

In addition, a reduction in the secretion of IL-6 and tumor necrosis factor-a (TNF-a) in macrophages has been reported. Taken together with a common quintessence of all associated studies, it can be concluded that radiation exposure to innate immunity cells results in inhibition of immunity through inflexible disparity in the concentration of cytokines level and oxidative stress[48,69].

Brain and Heart: Investigational learnings have provided sufficient authentication in support of brain scratch in reaction to energy exposure. It has been observed that a low dose radiation exposure of 0.1Gy brings changes in the gene expression linked to DNA damage, resulting in long-term depression.

These radiation-induced abnormalities enhanced the expression of genes associated with DNA repairs such as (PTPN-1), (PMS2), (HMGN-2) and interferon regulatory factors. All such aberrant changes lead to the establishment of signaling pathways in the brain resulting in certain neuronal defects. One of the common diseases that are related to heart exposure to external radiation, is Atherosclerosis.

It has been evidenced through experimental studies that radiation exposure induced harmful effects on cardiac tissues, thus increasing oxidative stress and decreasing protein expressions in the cardiac cells[12,37]. It has been demonstrated from the studies that even a low radiation dose led to an alteration in the metabolism of heart muscles and malfunctioning. The mechanism behind that is a consistent low-dose radiation exposure suppressed the lipid metabolism, which in turn increases the cardiac expression of miRNAs[9,29].

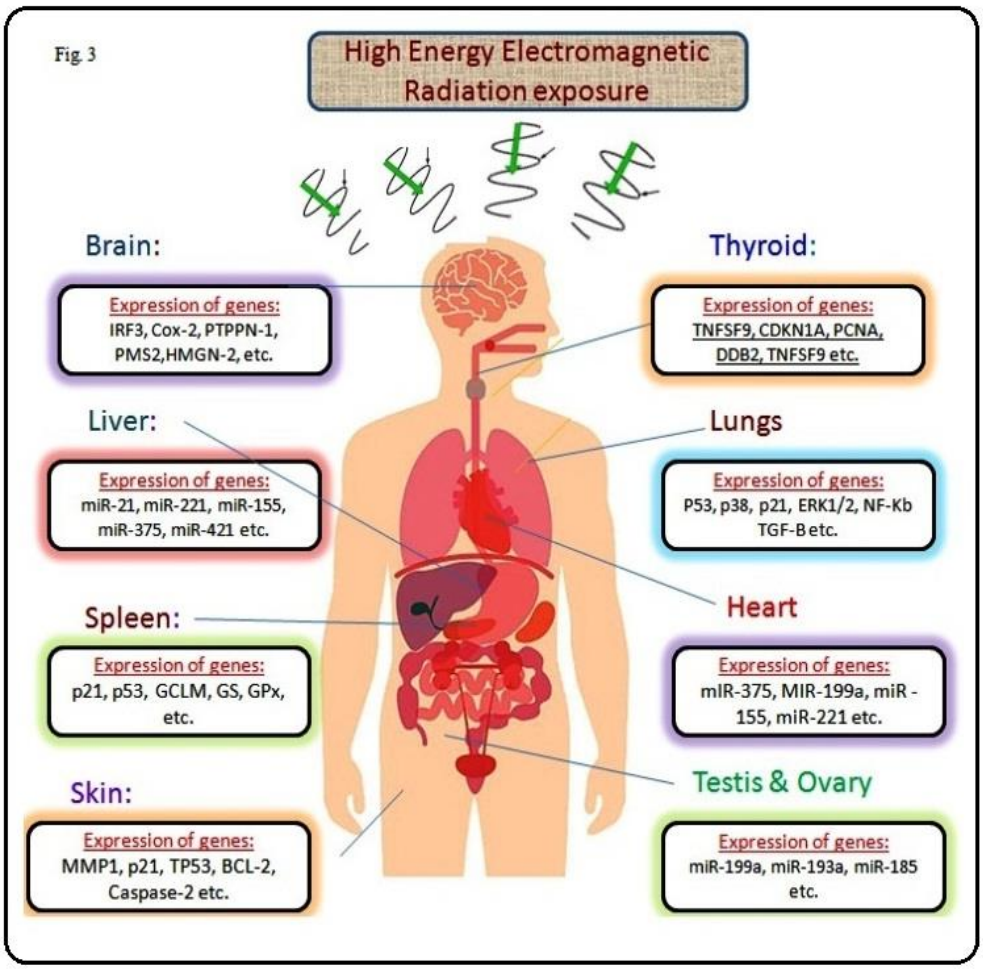

Figure 3: Expression of genes or types of mutation induced in different organs of the body due to high energy radiation emissions

Liver and Spleen: The liver is considered to be an organ regulating metabolic activities and maintaining homeostasis. Several studies have proved that systematic low-dose radiation exposure of .075Gy brings significant changes in liver dysfunction, deregulating metabolic activities. In one of the experimental studies on a mouse model, a radiation dose exposure of (0.2 - 0.5Gy) enhances the liver and urinary level expression even up to 160 hours of irradiation[32]. This rise in urine level in turn is correlated to impairments in the iron transport mechanism of the body. Another related study in support demonstrates that low x-ray radiation is responsible for interfering with maintaining iron homeostasis in a mouse model[7].

In addition, a low radiation input is also a key factor responsible for affecting glucose and lipid metabolism. Radiation exposure of range (0.1 to 0.5) Gy is supposed to curb glucose utilization and inhibit lipid metabolism. Accretion of radiation exposure (total 0.2Gy to 0.4Gy) significantly decreases gene expression concerned with fatty acid metabolism. A consistent low radiation exposure brings alteration in the miRNA (mIRr-21, mIRr-221, mIRr-421, mIRr-155 and mIRr-375) expression leading to the deregulation of iron, glucose and lipid metabolism in the liver[71].

Similar to the liver, low radiation exposure affects the damage to tissues in the spleen. A low radiation dose of range 0.02Gy to 0.2Gy shielded the tissues from oxidative damage by augmenting the production of GSH-synthase and glutathione peroxidase. However, radiation exposure of a range higher than 0.2Gy significantly induces apoptosis in the spleen by enhancing the expression of the P53 and P21 genes[8]. In response to the short-term exposure, the percentage of CD4+ T-cell, dendritic cell and macrophage counts reduced, thus maintaining a protective relationship between the spleen and the immune system. However, long-term exposure reduced the percentage of CD8+ CD28+, T-cell etc. These results demonstrate a mixed reaction of the spleen against radiation exposure and followed a hormesis model of radiation-induced responses with a threshold limit of 0.1Gy[65].

Blood cells: Several biological markers are widely used in different studies related to peripheral blood cells and to diagnose the effect of disease due to low and high-grade radiation exposure. It was demonstrated through one of the related studies that the effect of low-dose radiation exposure to whole white blood cells results in an enhancement of DNA damage-induced gene expression. It has been observed that delayed low radiation exposure is responsible for inducing the expressions of genes such as Ku70, Ku80 and histone family members (h2AX) of blood cells[33].

Another related study demonstrates an increment in DNA strand break oxidative damage and chromosomal aberration due to LDR exposure of range (0.05 Gy-0.1 Gy) in human blood cells. Low radiation exposure is supposed to enhance DNA damage with subsequent reduction in the DNA repairing capacity of peripheral blood cells.

In addition, it was also reported that low radiation exposure decreases the WBC and platelet counts together with a drop in the quantity of T-cells, CD3+, CD4+ and CD4+/CD8+ fractions in blood cells. The low radiation dose exposure (LDR) is supposed to reduce the number of leukocyte counts which in line induces the appearance of TGF-β; Transforming Growth Factor beta and interleukin-6. Thus it can be concluded that low radiation exposure (LDR) significantly affects the class of peripheral blood cells by inducing DNA aberration, tumbling viability and sometimes restraining the immunogenicity of blood cells[67,77].

Early and Acute effect of high radiation dose exposure: The lethal effect of high-intensity radiation exposure can be measured based on the amount of radiation dose exposed, eventual survival time, death mode due to the damage of any particular organ etc. The minimum threshold radiation exposure limits that can adverse the body of a healthy adult and the possible symptomatic effects, have been reported in tables 1 and 2 respectively.

Hematopoietic syndrome: The cells of the hematopoietic and lymphoid systems are highly sensitive to radiation. These include cells of bone marrow, giving rise to all circulating cells and platelets and lymphoid tissue found in the spleen, liver, lymph nodes and thymus. It is due to the radiation intensity of 300cGy to 800cGy that mainly is the cause of radiation damage to the hematopoietic system. The decrease in blood constituents starts just after acute radiation exposure. The whole body's acute exposure to 300cGy-800cGy results in the development of symptoms like nausea and vomiting for several days followed by symptoms like manifestation and depression of blood elements in lymphocytes and granulocytes that causes infection and fever. The impairment of immune mechanism, bleeding and anemia due to continuous three-week radiation exposure has also been reported[35].

Gastro-intestinal Syndrome: Radiation exposure of more than 1000 cGy leads to gastrointestinal (GI) syndrome which includes nausea, vomiting and prolonged diarrhea that extend for seven days. The gastrointestinal tract is highly sensitive to radiation and death occurs due to the depopulation of the epithelial lining of the gastrointestinal (GI) tract. A radiation dose of the order of 1000cGy will damage a large proportion of the dividing crypt cells of intestinal villi, causing its shrinkage and shortening. The shrinkage and cell loss rate depend on the dose delivered and at the time of death, the villi turn out to be flat and get free from cells[36].

Cerebro-vascular syndrome: A whole-body radiation dose of 10,000 cGy results in death within a few hours. The gastrointestinal and hematopoietic systems both will be severely damaged and fail. At higher doses of radiation,

cerebrovascular damage brings death quickly with the consequence of the failure of other organ systems. Exposure to radiation of the above range results in the development of severe nausea and vomiting followed by coordination of muscular movement, respiratory disorders, diarrhea, convulsive seizures, coma and finally death.

The cause of the cerebrovascular syndrome is not fully understood but is assumed that the cause of death due to radiation exposure is credited to the actions taking place within the central nervous system (CNS). At higher doses, the rapid killing of the cerebellum has been reported while at lower doses of radiation, reversible changes in the nerves have been observed. Since the peripheral nerves are highly resistant to radiation, hence higher and prolonged doses of radiation exposure periods are required for the expression of such effects fully[2].

Concept of Mean Lethal Dose (LD-50/60) and its typical value for different species: The mean lethal radiation dose represented as LD50/60 can be defined as the amount of dose at which 50% of the exposed population dies within 60 days. The LD50/60 value of radiation exposure for different species has been estimated from various experimental studies and is enlisted in table 3. It has been observed from experimental studies that the main source of human LD50/60 data can be obtained from total body irradiation that can be from bone marrow transplantation, radiation accidents like Chernobyl disaster etc. from the past experiences of radiation-exposed people such as Japanese bomb survivors of Hiroshima and Nagasaki and the accidents of various nuclear installations.

From the experimental observation of different studies, it has been estimated that LD50/60 value for humans is around 3.25 Gy for young healthy adults without any medical intervention. It has been reported from some of the related studies that the LD50/60 value can go beyond up to 4Gy under conservative medical care and is expected to be around 6Gy with medical intervention. However, bone marrow transplantation is suggested to be useful within a very limited range of doses i.e. from 7.5Gy to 10Gy[19].

Late (chronic) effects of high radiation dose exposure: The prolonged and late effects of radiation exposure due to low doses and longer duration of exposure can be included as follows:

i.) Induction of radiation-induced different types of cancers
ii.) Cataract formation
iii.) Shortening of life span.

Table 1
Minimum threshold radiation exposure level (Gy) for screening of adverse body effects in healthy human adults

S.N.	Radiation exposure effects	Minimum threshold acute dose level (Gy)
1.	Blood Change effects.	0.50
2.	Vomiting (Threshold)	1.00
3.	Mortality (Threshold)	1.50
4.	Minimum care	3.20-3.60
5.	Supportive care	4.80-5.40
6.	Bone marrow or stem cell transplant	>7.50-10.00

Table 2
Symptomatic body effects due to the whole body radiation exposure of 4-6 Gy (Threshold limit)

The period after exposure	Symptoms and recovery stages
0-45 Hours	Loss of appetite, nausea, vomiting, fatigue and lethargy.
2 days to 2 weeks.	Recovery from the above symptoms and patient appears to be well after 2-3 weeks.
2-3 weeks to 6-8 weeks	Fever, hemorrhages, diarrhea, loss of hair, severe lethargy and death in some cases.
6-8 weeks to several months	Recovery stage, surviving patients start showing improvement and severe symptoms disappear.

Table 3
Mean Lethal Dose (LD-50/60) values for different average body weight species

S. N.	Species	LD-50/60 (Gy)
1.	Dogs	3.70
2.	Monkeys	5.25
3.	Rat	6.75
4.	Mouse	7.00
5.	Rabbits	8.00
6.	Fish (Goldfish)	20.00

Cancer Induction due to radiation exposure

Induction of Leukemia: The assessment of radiation-induced leukemia has been estimated from the reported study of two population groups: (i) Bomb survivors of Hiroshima and Nagasaki (Japan) and (ii) The patients undergoing treatment for *Ankylosing Spondylitis*. It has been concluded from the reported studies that chronic lymphocytic leukemia does not have any significant correlation with radiation exposure.

However; acute or chronic myeloid leukemia has a positive correlation with radiation exposure. It has been confirmed that the susceptibility toward severe lymphatic or stem-cell leukemia is at the utmost in childhood and declines sharply with maturation[1,28].

Induction of Lung Cancer: Radio-emission exposure is one of the detrimental carcinogens responsible for inducing lung cancer, besides cigarette smoking, asbestos, chromium salts, mustard gas, hematite etc. The assessment of lung cancer has been carried out from the case studies of Japanese bomb survivors, patients with ankylosing spondylitis and underground miners exposed to radon radiation in mines. The workers working in uranium mines have shown clear excess of lung cancer. There is a positive correlation between lung cancer and radiation exposure; however, it is difficult to segregate with contributory effects of radon and cigarette[4,24].

Induction of Bone Cancer: The incidence of radiation-induced bone cancer is reported higher in children, who underwent treatment for tinea capitis and in patients treated for ankylosing spondylitis by external radiation. The radiation-induced bone cancer is generally observed in women who work as dial painters in watch companies, ingested radium during the process of dial painting and sharpened the points of their brushes used for coating radium. These women workers were reported to be suffering from bone sarcomas and carcinomas of epithelial cell linings of their paranasal sinuses and nasopharyngeal part. The threshold dose limit of 5Gy was observed for the induction of bone cancer below which no incidence has been reported.

While above the threshold limit, there is a sharp increase in bone sarcoma symptoms. There are also some reported cases of bone sarcoma in patients injecting Radium-224 for the treatment of ankylosing spondylitis[39].

Induction of Thyroid Cancer: Children and young people are more susceptible to developing thyroid cancer than adults. The induction of radiation-induced thyroid cancer is an age-dependent phenomenon. The conclusion has been drawn from the case studies of atomic blast survivors of Hiroshima and Nagasaki, uncovered to external irradiation and ingested I-131, victims of the Chernobyl accident etc. Children treated by external beam radiotherapy for an enlarged thymus, tonsils, nasopharynx, tinea capitis etc. can be more susceptible to thyroid cancer[31,68].

Induction of Breast Cancer: The incidence of radiation-induced breast cancer was studied from the case studies of Japanese bomb female survivors of Hiroshima and Nagasaki. Female patients, subjected to more than 100 serial fluoroscopic examinations during artificial pneumothorax for pulmonary tuberculosis, or those who were treated for postpartum mastitis and other benign conditions and have received a dose from 1 to 6 Gy, were susceptible to breast cancer[52]. In these groups of patients, it has been observed that there is an extra susceptibility for the prevalence of breast malignancies. It can be concluded that the frequency of breast cancer is enhanced with an augment in the radiation dose and also the prevalence of breast cancer has a linear correlation with the radiation dose magnitude[10,59].

Induction of Skin Cancer: The radiation-induced skin cancers are diagnosed and treated at an early stage of their appearance and therefore have less mortality than other cancers. The initial case of emission-stimulated skin cancer was accounted for in 1902 by a radiologist and after that several hundred such cases were reported among general practitioners, X-ray technicians, dentists and dosimeter investigators. Such cases have a long latent period and their onset follows chronic radio-dermatitis. The frequencies of squamous and basal cell carcinomas were higher than sarcomas in the case of radiation-induced skin damage[18,48].

Effects of radiation on embryo and fetus: The developing fetus within her pregnant mother is uncovered to different radiation threats, distressing the security of both. These risk factors may consider deliberate or unintentional, irrational or inevitable on the part of the mother. These incorporate the drinking of liquor, chewing tobacco, cigarette smoking, ingestion of prescribed and non-prescribed drugs and exposure to ionizing emissions from non-remedial sources. *In uteri* radiation revelation of the fetus for the woman working or visiting areas where there is a chance of radioactive exposure, needs to understand the biological radiation risks at hand to the unborn child.

Rapidly proliferating and differentiating cells are most susceptible to radiation damage and as a result, radiation exposure can produce certain developmental abrasions, particularly in the developing brain when a developing embryo is radio-exposed prenatally. There are relatively three phases that the fetus has to surpass during its development and the magnitude of the effect of radiation which will be different in each stage[51].

Pre-implantation: This is the period that begins from the stage of insemination and ends with the establishment of a fetus in the uterus and is completed in ten days. During this phase, the exposures to radiation of animals materialize to direct 'all or none effects'. This can be explained as such that an X-ray dose of 2Gy in mice results in a high frequency of embryonic casualties; however, those that stay alive appear to be normal. It has been reported that the possibility of impulsive abortions increases to some extent during this

period. The typical prevalence of impulsive abortion in humans may be elevated up to 30-50%[3,76].

Organogenesis: This period of organogenesis is divided into early and late stages. The early organogenesis period is 15-28 days after conception whereas late organogenesis refers to the time 29-50 days after conception. In early organogenesis, the embryo becomes sensitive to lethal, growth retarding emission effects due to the high proportion of radiosensitive cells. Irradiation at this time leads to several developmental defects or abrasions. However, the case studies of human embryo victims of Hiroshima and Nagasaki did not exhibit any substantial increase in the frequency of developmental defects in developing fetuses at this stage[72].

Fetal stage: Experimental studies during this stage of development suggest that the effect of exposed irradiation is less likely to develop any noteworthy abnormalities after the first two months of gestation. The data reported available is drawn from the pregnant survivors of Hiroshima and Nagasaki. Reduced head size and mental retardation were the developmental abnormalities observed after the whole-body exposure. There is also an increased risk of childhood cancer including leukemia up to the age of ten years from exposure during pregnancy. The risk associated with prenatal radiation exposure suggests that the overall risk lies in the range of 0-1 cases per 1000 irradiated in utero, which is at least 30 times slower than the natural level of occurrence of serious handicaps in average pregnancies[42].

Cataract formation: Any detectable change that affects the normal transparency of an eye lens is described as a cataract. The lens of the eye is considered to be a self-renewal system because its cells divide continuously throughout life. There is no such mechanism reported for the damaged cells if the dividing cells are damaged by radiation. Different types of radiation have shown different response rates. The eye lens is more susceptible to beta and neutron radiation. The degree of opacity of the lens is the function of the dose also. At a low dose of radiation exposure, the opacity may become stationary with very little or no impairment of the vision. However, with high doses of radiation, the opacity becomes noteworthy with loss of vision[54].

Conclusion

The present review highlights the detrimental effects of both low and high-grade radiation exposure on different body parts both somatic and genetic. These ionizing radiations are supposed to generate free radicals which interact with fragile biological tissues such as DNA causing free radicals and chemical lesions within the body cells. Short and long-term radiation exposure brings certain deterministic effects to the body, which induces the induction of different types of cancers and other related problems and may manifest in bringing out genetic mutations and malignancies in the body. In this review, we have incorporated in detail the lethal effect of both acute and chronic radiation exposure on different body parts and systems and the minimum threshold limit of the radiation exposure that is effectively a perimeter for knowing the damage[23].

The use of ionizing radiation for medicinal purposes offers tremendous benefits to patients, but it also poses serious harm when exposed outside of a limit. For the proper use of ionizing radiation, there is a need for more detailed reporting and enforcement systems[61]. Any by-product molecule that seems to be a source of radiation exposure either medically or commercially should be restricted for not even a marginal risk to health providers, patients, or workers. Equal treatment of all ionizing radiation in medicine could be a reasonable national policy, as the risks evoked by reactor-generated by-products and other forms of radiation are equally at par. The International Commission on Radiological Protection (ICRP) regularly reviews the biological evidence of the detrimental effects of ionizing radiation and publishes appropriate recommendations regarding acceptable self-practices for the exposure of occupational workers and patients undergoing treatment / diagnosis[15,62].

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