RESEARCH REVIEW JOURNALS
Volume-04
Issue-05
May-2019

ISSN: 2455-3085 (Online)
RESEARCH REVIEW International Journal of Multidisciplinary
www.rrjournals.com[UGC Listed Journal]

Study on Schematic Structure of Radionuclide Transfer from Industries and Power Plants to Atmosphere; in Air, Soil and Water

Mr. S.N. Patil

Assitant Prof. Rani Laxmibai College, Parola, Jalgaon (India)

ARTICLE DETAILS

Article History
Published Online: 15 May 2019

Keywords
radionuclide, power plants.

ABSTRACT

Human exposure to ionizing radiation is not a new phenomenon. We have facing consequences of its side-effects since its discovery. Studies on survivors of nuclear explosion and accidental exposure of occupational workers provided a picture of long-term effects of ionizing radiation.

1. Introduction

Human exposure to ionizing radiation is not a new phenomenon. We have facing consequences of its side-effects since its discovery. Studies on survivors of nuclear explosion and accidental exposure of occupational workers provided a picture of long-term effects of ionizing radiation. However, biological aspects of radiation effects are poorly understood. Post-irradiation damage to DNA initiates the repair process.

A cell may either survive or die. Cells that survive with mutations in DNA may lead to cancer. Long-term exposure to low doses up to 0.1 Gy of ionizing radiation is reported to modify genetic sequences (UNNRC, 2001). However exposure to ionizing radiation has dose-dependent acute and long-term effects on health. Exposure to doses more than 0.5 Gy may be carcinogenic, and after some period of time it can lead to various types of human cancers (Koukourakis 2012).

On the other hand exposure to high dose of ionizing radiation can kill numerous cells, which leads to organ damage and ultimately mortality. The latter effect, involving a rapid response, is referred to as acute radiation syndrome (ARS) (Casarett 1968). Damage due to ionizing radiation exposure depends upon several parameters, such as the distance from the radiation source, the duration of the exposure and the dose rate with which the radiation is emitted.

It is not always not necessary that the effects of ionizing radiation exposure to be visible as an injury in a short time period; instead effects/symptoms may appear later. Exposure to ionizing radiation can have deterministic or stochastic effects. The effects of exposure to ionizing radiation depend on the kind of radiation and the dose received.

Deterministic effects such as cell death are immediate effects following radiation exposure. Deterministic effects have thresholds, above which severity increases with dose. The responses and thresholds vary from individual to individual and even from one tissue to another (Armao and Smith 2014, Holmes et al., 2015).

Acute deterministic effects such as sterility, cataracts etc. require high doses of 1 to 2 Gy. On the other hand stochastic radiation effects are long-term effects, such as mutations. Mutations can lead to hereditable changes and cancer as well. Stochastic effects have a long latent period and sometimes can be seen only in F1 or F2 generations. Oxidative modification in DNA bases initially may not be expressed at cellular or tissue levels. However, this modification can lead to mutations in upcoming replication cycles. The protein product of such mutated genes will malfunction, and cells may lose important biological functions.

2. Literature Review

Exposure during clinical practices such as diagnostic procedures like X-ray imaging and exposure to X-rays during security checks are direct modes of exposure. The International Commission for Radiation Protection (ICRP) has recommended exposure dose limits for different occupations, which are divided into three categories: workers, patients and physicians. The annual average dose for the world population is approximately 2.8 mSv, and in the USA it is 3 mSv. Approximately 85% exposure in humans is due to background radiation and the remaining 15% is from artificial sources. Artificial sources include medical exposure, representing 14% of the total exposure limit, and the remaining 1% represents nuclear testing fallout, including Chernobyl accident fallout and nuclear fuel (Holmes et al., 2015). The stochastic effects have a long latent period, and the risk of cancer induction due to medical uses of radiation is difficult to interpret, as extrapolating effects of low dose radiation is complicated (Holmes et al., 2015).

Studies on Biological Effects of Ionizing Radiation (BEIR) suggested that the chance of developing cancer among individuals exposed during medical procedures is 1 out of 3 individuals. Though the imaging based risks of cancer formation appear small from an individual perspective, this risk factor multiplies with a large and growing population of patients getting exposed to medical radiation (Armao and Smith 2014). According to an estimate in the USA, 80 million computed tomography (CT) scans were performed in the year 2010, whereas this figure was 3 million in 1980 (Armao and Smith 2014). The per capita exposure to natural ionizing radiation was 3 mSv per year, and medical radiation exposure accounted for an average per capita effective dose of 0.53

mSv per year. Over a period of 30 years the natural ionizing radiation remained nearly same. However, medical ionizing radiation exposure has increased 600% to 3mSv per capita per year over 30 years (Mettler et al., 2009, Einstein 2012). Some diagnostic techniques involve ingestion of radioactive materials in to the body. During thyroid diagnosis, iodinated water is given to the patient, and patients are scanned for accumulation of radioactive material (Casarett 1968).

Direct exposure to ionizing radiation also includes exposure during treatments such as radiotherapy. Almost all the cancer patients undergo radiotherapy as a part of their treatment protocol (Joiner and Kogel 2009). The main problem in radiotherapy is radiation toxicity to non-cancerous tissue/cells in the neighborhood of target (cancerous) tissue/cells. The responses of non-cancerous tissues to ionizing radiation depend on type of tissue and can be immediate or long-term. Early effects include onset of erythema, hair loss, dry or wet desquamation and also oral mucositis (Joiner and Kogel 2009). Long-term effects are largely irreversible and can be seen after months or even years of radiotherapy. Late effects depend on the kind of tissue irradiated during treatment. These include cataracts, necrosis, fibrosis, sclerosis and so on (Joiner and Kogel 2009). Despite toxic effects on non-cancerous tissues, radiotherapy remains an important modality in cancer treatment.

3. Accidental Exposure

Apart from direct exposure to ionizing radiation during medical diagnosis or treatments and security checks, exposure to ionizing radiation can occur through contamination. Accidental exposure through contamination is not restricted to a group of the population or to a few individuals wherever contamination occurs; the complete biota in that region including plants, animals and even microbes are exposed to radiation. When a radioactive substance accidentally gets released into the environment in the form of gases, liquids or solids, it leads to contamination of that environment. Radioactive substances released in the environment get deposited on the skin of animals and can enter the body through skin cuts/wounds, may get ingested into the body through food or water, or may get inhaled along with air (Yoshida and Kanda 2012).

Accidents at the Chernobyl nuclear power plant (1986) and the Fukushima Dai-ichi Nuclear Power Plant (2011) led to contamination of the environment and ultimately led to human exposure to the radiation. After the accident at Chernobyl nuclear power plant, a cocktail of radioactive material was released into the atmosphere. Over 50,000 people were evacuated following the accident, and 134 nuclear plant workers and fire fighters controlling the fire received radiation doses of 800-16,000 mSv. Consequently, they suffered from acute radiation syndrome. Two of these people died after a few days of exposure, and 28 died within three months of exposure (U.S.NRC 2011). Twenty-five years after the Chernobyl tragedy, in 2011 one more mishap happened on the coast of Japan in a nuclear power plant located in Daiichi, Fukushima. This was the second largest nuclear accident after Chernobyl.

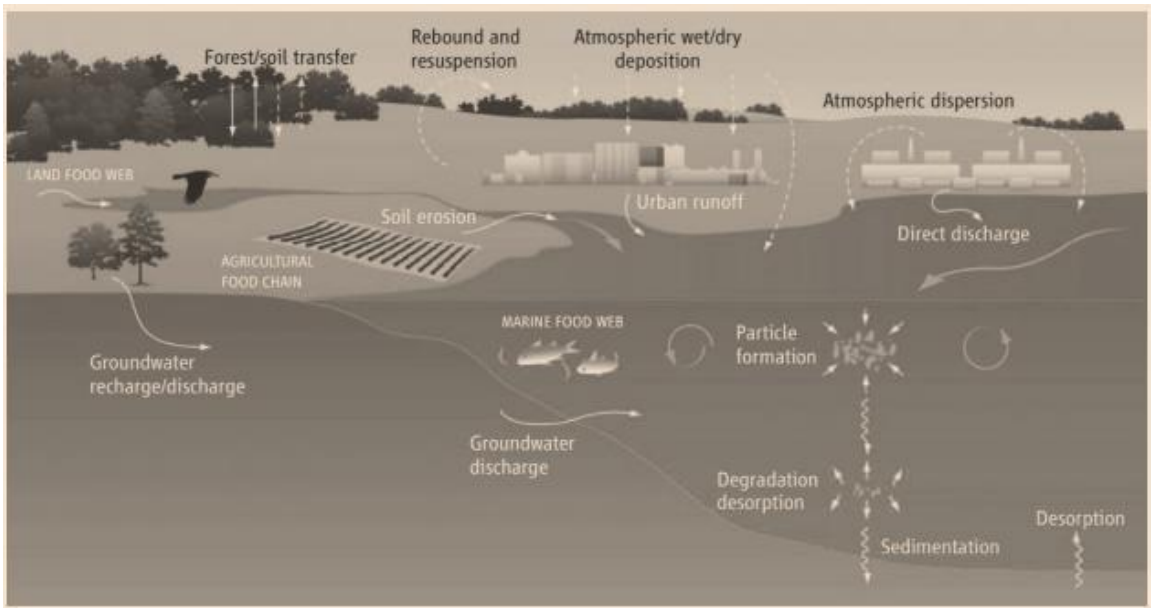

Figure: Schematic diagram of radionuclide transfer from industries and power plants to atmosphere; in air, soil and water (Yoshida and Kanda 2012).

This accident caused the release of radioactive material into the environment; however, the quantity of radioactive contaminants in the Fukushima tragedy was far less than at Chernobyl. Following the Fukushima accident, there was lower worker group and public exposure to radiation compared to Chernobyl. Thirty of the plant workers received about 100 mSv, and this dose is significantly below the dose require to cause radiation sickness, but this dose was sufficient to increase the risk of cancer. Two workers who waded through contaminated water received a dose of 600 mSv (Boice Jr 2012). Studies on butterflies from the contaminated areas of Fukushima and nearby cities revealed mosaic mutations in some species. Lepidoptera are comparatively radio-tolerant. There was an improvement in radiation tolerance of Pseudozizeeriamaha butterflies (Taira et al., 2014).

4. Radiation Protection

Free radicals are generated during metabolic activity through mitochondrial respiration. ROS generated post-radiation exposure are the product of radiolysis of water, and they react with molecules in their vicinity. ROS transfer their reactive electrons to other bio-molecules that, in turn, undergo oxidative modifications. To counter ROS cells have a pool of antioxidant molecules, which are of two kinds. The first are enzymatic antioxidants, specialized protein molecules involved in neutralizing ROS generated. These include superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx) and glutathione S-transferase (GST).The second kind are non-enzymatic molecules, which are low molecular weight non-enzyme antioxidants such as glutathione, trehalose ,uric acid, flavonoids, ascorbic acid, thiols, bilirubin, carotenoids, α-tocopherol and amino acids like cysteine (Sies 2007).

Superoxide dismutase (SOD) is a family of enzymes that are ubiquitous metalloproteins. These are considered to be true antioxidants because of their involvement in scavenging superoxide radicals generated in mitochondria through the respiratory electron transport chain (Ramasarma 2007). SOD is present in the cytoplasm as well as in mitochondria. SOD1 and SOD2 are present in the cytoplasm and mitochondria, respectively (Kirby et al., 2002). The role of SOD in scavenging oxidative free radicals generated due to ionizing radiation exposure has been reported earlier (Datkhile et al., 2009a). The reaction carried out by SOD is,

$$2O2 + 2H2O \rightarrow 2H2O2 + O2,$$

If H2O2 not neutralized; H2O2 thus by itself generates hydroxyl radicals, but compared to superoxide radicals H2O2 is less potent. SOD utilizes two superoxide radicals and converts them into less harmful H2O2 and an oxygen molecule.

5. Conclusion

Catalase is present in almost all aerobic organisms. Catalases are a class of hydroperoxidases that has been studied extensively. There are various types of catalases, and they are engaged in variety of functions. Catalase was reported to be involved in folic acid oxidation in rats and some non-primate species.

References

1. United Nation's Nuclear Regulatory Commission (UNNRC) (2001),United Nation's Nuclear Regulatory Commission, protecting people and environment, United States Environmental Protection Agency, office of air and radiation, EPA-402-K-10-008
2. Koukourakis, M. I. (2012). Radiation damage and radioprotectants: New concepts in the era of molecular medicine. British Journal of Radiology, 85(1012), 313–330.
3. Casarett, A. P. (1968). Radiation biology. Prentice-Hall, Englewood Cliffs, New Jersy.
4. Armao, D., & Smith, J. K. (2014). The health risks of ionizing radiation from computed tomography. N C Med J, 75(2), 126–131.
5. Holmes, E. B. (2015). Ionizing radiation exposure with medical imaging. Medscape.
6. Mettler, F. A., Bhargavan, M., Faulkner, K., Gilley, D. B., Gray, J. E., Ibbott, G. S., &Yoshizumi, T. T. (2009). Radiologic and nuclear medicine studies in the united states and worldwide: Frequency, Radiation dose, and comparison with other radiation sources- 1950– 2007. Radiology, 253(2), 520–531.
7. Joiner, M., &Kogel, A. V. D. (2009). Basic clinical radiobilogy. Hodder Arnold, UK,4th edi .
8. Einstein, A. J. (2012). Effects of radiation exposure from cardiac imaging: How good are the data? Journal of the American College of Cardiology, 59(6), 553–565.
9. Yoshida, N., & Kanda, J. (2012). Tracking the Fukushima radionuclides. Science, 336(6085), 1115–1116.
10. Boice, J. D. (2012). Radiation epidemiology: A perspective on Fukushima. Journal of Radiological Protection, 32(1), 33-40.
11. Sies, H., (2007). Total antioxidant capacity :Appraisal of a concept. The Journal of Nutrition, (February), 1493–1495.
12. Ramasarma, T. (2007). Many faces of superoxide dismutase, originally known as erythrocuprein. Current Science, 92(2), 184–191.
13. Datkhile, K., Mukhopadhyaya, R., Dongre, T., &Nath, B. (2009a). Increased level of superoxide dismutase (SOD) activity in larvae of Chironomusramosus (Diptera: Chironomidae) subjected to ionizing radiation. Comparative Biochemistry and Physiology - C Toxicology and Pharmacology, 149(4), 500–506.

