Review Article

Harnessing nuclear energy: health risks

Amit Bhasin*, Aparna Ahuja**

Abstract

With increasing use of nuclear energy for various purposes all over the world, there is a growing debate over its ill-effects. Biological effects of radiation are dependent on the dose of radiation to which a person is exposed, dose rate of exposure, distance from radiation source, shielding and type of radiation and also type of cells and tissues being exposed with the most critically affected tissues in adults being the spermatocytes in the testis, haematopoietic precursor cells in the bone marrow and crypt cells in the intestines.

Radiation injury can manifest early after the exposure, known as Acute Radiation Syndrome (ARS) or after a latent period of many months to years. Classic clinical syndromes associated with ARS include the Haematopoietic, Gastrointestinal, and Cerebrovascular (formerly known as cardiovascular and central nervous) syndromes with local radiation injury presenting as Cutaneous Syndrome (CS). Four phases may be identified in ARS- Prodromal Phase, Latent Phase, Stage of Manifest Illness and Death or Recovery Phase. Long term effects of exposure include cancers, genetic damage and cataracts.

As controlled experiments regarding radiation exposure are neither feasible nor ethical, sources of information are the various disasters as the atomic bomb detonations in Japan (in the second world war), Chernobyl reactor explosion, Fukushima disaster (post-tsunami & following earthquake) in Japan and Mayapuri (Delhi, India) cobalt leak being some of them.

Key words: Radiation injuries; health hazards; nuclear energy; radiation protection.

Introduction

With increasing use of nuclear energy for generating power, therapeutic medical purposes and also for nuclear terrorism, there has been also an increase in radiation accidents and hence there is need for adequate medical knowledge and response. The nuclear debate is growing in importance as governments everywhere are looking for ways to maintain economic growth and reduce the effects of global warming. But it now seems like the disadvantages are just too great. Nuclear power provides about 6% of the world's energy and 13–14% of the world's electricity, with the U.S., France and Japan together accounting for about 50% of nuclear generated electricity [1,2]. Nuclear power is controversial and there is an ongoing debate about the use of nuclear energy. Proponents, such as the World Nuclear Association and International Atomic Energy Agency (IAEA), contend that nuclear power is a sustainable energy source that reduces carbon emissions. Opponents, such as Greenpeace International and NIRS, believe that nuclear power poses many threats to people and the environment [3,4]. These threats include the problems of processing, transport and storage of radioactive nuclear waste, the risk of nuclear weapons proliferation and terrorism, as well as health risks and environmental damage from uranium mining. They also contend that reactors themselves are enormously complex machines where many things can and do go wrong, and there have been serious nuclear accidents [5,6]

Determinants of radiation injury- The *amount of radiation* (i.e. radiation dose) absorbed by the patient's tissues is highly predictive of its biological effects. Such doses are defined as the amount of

energy of ionising radiation deposited per unit of tissue mass at a specific point. Some amount of exposure naturally occurs during certain medical imaging such as: a standard chest x-ray delivers a dose of 6 to 11 mrem (0.06 to 0.11 mSv, 0.06 to 0.11 mGy). Interventional cardiologists working in a high-volume catheterisation laboratory may have collar badge exposures exceeding 600 mrem (6 mSv) per year. A barium enema with 10 spot images delivers a dose of approximately 0.7 rem (700 mrem, 7 mSv, 7 mGy). Similar doses (7-8 mSv) are delivered from a CT scan of the chest or a PET scan, while a combined PET/CT scan is estimated to deliver a dose of 25 mSv [7-9]. But these exposures are not hazardous. The lowest radiation dose resulting in an observable effect in man on bone marrow depression, with a resultant decrease in blood cell counts, is in the range of 10-50 rem (100 to 500 mSv, 0.1 to 0.5 Gy). The lowest total body dose at which the first deaths may be seen following exposure to ionising radiation is in the range of 1.0 to 2.0 Gy. Depending upon the type of support given, 50 percent of people exposed to a dose of 3 to 4 Gy will be expected to die of radiation-induced injury. There is virtually no chance of survival following a total body exposure in excess of 10 to 12 Gy. The term "Lethal dose of radiation" has thus been defined — which is the dose associated with death in 50 percent of those similarly exposed (i.e., the LD 50). Depending on the incident, estimates for the LD 50 have ranged from 1.4 Gy among atomic bomb survivors in Japan to 4.5 Gy following uniform total-body exposure to external photons [10,11].

Several factors determine the lethality of ionising radiation. These include: 'Dose rate' (doses received over a shorter period of time cause more damage), 'Distance from the source' (For point sources of radiation, the dose rate decreases as the square of the distance from the source {inverse square law}), 'Shielding' (Shielding can reduce exposure, depending upon the type of radiation and the material used, 'Type of radiation' (alpha particles can be stopped by a sheet of paper or a layer of skin, beta particles by a layer of clothing or less than one inch of a substance such as plastic, and gamma rays by inches to feet of concrete or less than one inch of lead) [12].

Radiation exposure causes different degree of damage to various body structures depending upon certain inherent properties of cells. Radiosensitivity varies directly with the 'rate of cellular proliferation'. Rapidly dividing cells are more profoundly affected. Radiosensitivity varies directly with the 'number of future divisions'. Long-lived gonadal and haematopoietic stem cells fall into this category. Radiosensitivity varies indirectly with the 'degree of morphologic and functional differentiation'. As an example, cells at the growth plate in bone, which have not yet developed into bone or cartilage, are more sensitive than those of the diaphysis. Accordingly, growth arrest of bone is commonly seen after radiation exposure to the growth plate in children, as may occur in the treatment of malignancy. Variation in sensitivity to radiation is an inherited genetic trait, although candidate gene studies have been largely unsuccessful in identifying the genetic variants underlying most phenotypes [13,14]. While all tissues composed of short-lived cells are directly and indirectly affected by radiation, the most critically affected tissues in adults include the following: spermatocytes in the testis, haematopoietic precursor cells in the bone marrow and crypt cells in the intestines.

Dose-dependent effects on various organs have also been identified. They are of two types, deterministic and stochastic: A 'deterministic' effect is one in which the severity is determined by the dose (e.g. depression of blood counts). A dose threshold (i.e. a dose below which an effect is not seen) is characteristic of this effect. As an example, the threshold absorbed dose for a "deterministic effect" on bone marrow (0.5 Gy) is lower than that for all other organs, except for the testis (0.15 Gy). A 'stochastic' effect represents an outcome for which the probability of occurrence (rather than severity) is determined by the dose. An example is radiation-induced carcinogenesis, which occurs after a prolonged and variable delay (latency) after exposure. These effects do not have an apparent threshold dose. The mechanisms underlying deterministic and stochastic effects remain unknown. Studies showing the impact of radiation on gene function may shed light in this area.

Radiation injury

The damage caused by radiation exposure can be categorised according to whether the symptoms and signs develop immediately or are delayed by months or years. The ensuing damage results from the sensitivity of cells to radiation, with the most rapidly dividing cells being the most sensitive to the

acute effects of radiation. The inherent sensitivity of these cells results in a constellation of clinical syndromes that occur within a predictable range of doses after a whole-body or significant partial-body exposure. Symptoms arising from such exposures are referred to as radiation sickness or acute radiation syndrome (ARS). Classically, the threshold dose for ARS is a whole-body or significant partial-body irradiation of greater than 1 Gy delivered at a relatively high dose rate.

Acute radiation syndrome

Acute changes, which are seen within the first two months following exposure, include signs and symptoms resulting mainly from damage to the skin, central nervous system, lung, gastrointestinal tract, and haematopoietic tissues. Classic clinical syndromes associated with ARS include the haematopoietic, gastrointestinal, and cerebrovascular (formerly known as cardiovascular and central nervous) syndromes, although there is significant clinical overlap.

Local radiation injury, sometimes called the Cutaneous Syndrome (CS), is especially common and important in patients with ARS consequent to a non-uniform exposure. The CS may include changes ranging from epilation to radionecrosis. The presence of ARS complicates the management of CS, due to poor wound healing, infections and bleeding, while the converse is also true. As an example, severe CS dramatically affected the course of

victims of the Chernobyl accident, and was the main cause of death in more than half of lethal cases [15].

There are four main phases to the ARS [16]-

The Prodromal Phase usually occurs in the first 48 hours following exposure, but may develop up to six days after exposure.

The Latent Phase is a short period characterised by improvement of symptoms. However, this effect is transient, lasting for several days to a month. The duration of this phase is inversely related to the dose of radiation received, and may be absent at the highest, fatal doses.

The Stage of Manifest Illness may last for weeks, and is characterized by intense immunosuppression. It is the most difficult to manage. If the person survives this stage, recovery is likely.

Death or Recovery Phase — Those patients who recover will require close follow-up for the first year, owing to the risk for unusual infections, as aberrant immune reconstitution is probable in those with significant exposure. Survivors will require life-long follow-up to monitor for long-term complications, such as organ dysfunction and carcinogenesis.

The onset, duration, and dominant pattern of the acute radiation syndrome depend upon the dosage of radiation received (Table 1) [17,18]. As examples, the prodromal syndrome is often minimal in those

Table 1- Phases of radiation injury

Dose (Gy)	Symptoms				Prognosis
	Prodrome	*Haematologic*	*GI*	*Neurologic*	
0.5-1	+	+	0	0	Survival almost certain
1-2	+/++	+	0	0	Survival >90 percent
2-3.5	++	++	0	0	Probable survival
3.5-5.5	+++	+++	+	0	Death in 50% at 3.5 to 6 wks
5.5-7.5	+++	+++	++	0	Death probable in 2-3 wks
7.5-10	+++	+++	+++	0*	Death probable in 1-2.5 wks
10-20	+++	+++	+++	+++	Death certain in 5-12 days
>20	+++	+++	+++	+++·	Death certain in 2-5 days

Gy: dose in Grey; GI: gastrointestinal effects; 0: no effects; +: mild; ++: moderate; +++: severe or marked.
* Hypotension. Also cardiovascular collapse, fever, shock.

exposed to doses of ≤ 1 Gy, while those exposed to doses of 10 to 20 Gy may have a rapid compression of phases and proceed from the prodromal phase to death in two days or less.

The Prodromal Syndrome is generally mild or absent at total body doses of 1 Gy or less. Patients whose symptoms begin more than two hours after exposure were probably exposed to doses <2 Gy. They can be expected to fully recover within one month, although long-term sequelae may develop. Onset of symptoms within the first two hours usually indicates significant and potentially lethal exposures exceeding 2 Gy. At these doses, sloughing of the gastrointestinal epithelium also occurs (i.e. the gastrointestinal syndrome), adding to the symptomatology. At doses between 2 to 10 Gy, it is difficult to establish a prognosis based solely on the existence and/or severity of the prodromal syndrome. At high doses (e.g, 10 to >20 Gy), prodromal symptoms occur in virtually all patients within minutes of exposure [19-21]. These gradually merge into loss of consciousness and hypotension, components of the cerebrovascular syndrome. Death often occurs within a few days to weeks after such exposures. Accordingly, a rapid and severe prodromal response is the harbinger of a poor clinical outcome.

The Cerebrovascular Syndrome, also called the neurovascular syndrome or CNS syndrome, results from localised changes in the central nervous system. These include impaired capillary circulation with damage to the blood-brain barrier, interstitial oedema, acute inflammation, petechial haemorrhages, inflammation of the meninges, and hypertrophy of perivascular astrocytes. Paroxysmal spike and wave discharges may be evident on the EEG, and the presence of swelling and oedema may be documented by CT scan and MRI of head [22].

There may be a latent period of a few hours in which there is apparent improvement, but within five to six hours watery diarrhoea, secondary to severe gastrointestinal syndrome, respiratory distress, fever, and cardiovascular collapse ensue. The final picture, which may mimic that of sepsis, includes hypotension, cerebral oedema, increased intracranial pressure, and cerebral anoxia, with death in about two days time.

The Gastrointestinal Syndrome typically develops within five days of the initial exposure (Table 2) [18]. At doses <1.5 Gy, only the prodromal phase of nausea, vomiting, and gastric atony are observed [23]. More severe symptoms develop at doses between 5 and 12 Gy, secondary to loss of intestinal crypt cells and breakdown of the mucosal barrier, with sloughing of the epithelial cell layer and denudation of the bowel wall [24]. These changes result in crampy abdominal pain, diarrhoea, nausea and vomiting, gastrointestinal bleeding with resultant anaemia, and abnormalities of fluid and electrolyte balance. This early phase is often followed by a latent phase lasting five to seven days, during which symptoms abate. Vomiting and severe diarrhoea accompanied by high fever make up the manifest illness. Systemic effects at this time may include malnutrition from malabsorption.

Impaired barrier function of the gastrointestinal tract results in the passage of bacteria and their toxins through the intestinal wall into the bloodstream, predisposing to infection and sepsis, which may be further compromised by immunosuppression and cytopenias (secondary to development of the haematopoietic syndrome).

Other severe complications include ulceration and necrosis of the bowel wall, leading to stenosis, ileus, and perforation. In the latter case, recovery is most unlikely, as radiosensitive stem cells in the crypts of the gastrointestinal tract are permanently damaged. Consequently, there is no replacement of cells that are lost from the surface of the villi through the sloughing process, precluding recovery

Table 2- Radiation toxicity- gastrointestinal system

Symptom	Degree 1	Degree 2	Degree 3	Degree 4
Stool (/day)	2-3	4-6	7-9	≥10
Stool consistency	Bulky	Loose	Loose	Watery
GI bleeding	Occult	Intermittent	Persistent	Persistent large amount
Abdominal cramps or pain	Minimal	Moderate	Intense	Excruciating

[25]. However, mild gastrointestinal symptoms limited to one or two episodes of diarrhoea with associated abdominal pain are accompanied by virtually certain recovery, provided that the haematopoietic syndrome which follows is reversible.

The Haematopoietic Syndrome develops at doses exceeding 1 Gy and is rarely clinically significant at doses <1 Gy [20,21,24,26,27]. Mitotically active haematopoietic precursors have limited capacity to divide after whole-body doses greater than 2 - 3 Gy.

Neutropenia and thrombocytopenia reach a nadir at two to four weeks and may persist for months. Anaemia inevitably ensues, due to the combined effects of gastrointestinal blood loss from the gastrointestinal syndrome, haemorrhage into organs and tissues secondary to thrombocytopenia, and ultimately, bone marrow aplasia. In the ensuing weeks to months after exposure, hypoplasia or aplasia of the bone marrow occurs, resulting in pancytopenia, predisposition to infection, bleeding, and poor wound healing, all of which may contribute to death in the absence of appropriate supportive care. Lymphopenia is common and occurs before depression of other cellular elements, and may develop within the first 6 to 24 hours after exposure to a moderate or high dose [25,28,7]. Based on the overall levels of lymphocyte, neutrophil, and platelet counts, as well as the presence or absence of infection and blood loss, the relative severity of toxicity to the haematopoietic system can be evaluated (Table 3) [18].

The *Cutaneous* Syndrome may develop early

following exposure (e.g. one to two days). However, it may take years before becoming fully manifest. Early lesions include erythema, oedema, and dry desquamation of the skin. Such lesions may be isolated or may appear simultaneously in several locations, depending on the amount of skin receiving direct exposure. More advanced lesions include bullae, moist desquamation, ulceration, and onycholysis. The severity of the cutaneous reaction depends upon the depth dose distribution of the radiation source.

- Blisters and bullae with or without necrosis appear one to three weeks after localised exposure to doses of >30 Gy [29,30].
- Moist desquamation and ulceration are seen with localised doses of 20 to 25 Gy [30].
- The estimated threshold for erythema is a localised exposure dose of 10 to 15 Gy.
- Epilation occurs 10 to 20 days after a single localised exposure to 3 to 4 Gy or greater.

Delayed effects

Long Term Radiation Exposure results from residing in a fallout contaminated area for an extended period (external exposure), consuming food produced in a contaminated area (internal exposure), or both. If the exposure rate is low enough, no symptoms of radiation sickness will appear even though a very large total radiation dose may be absorbed over time. Latent radiation effects (i.e. cancer, genetic damage) depend on total dosage, not dose rate, so serious effects can result.

Internal Exposure: Radioisotopes may be taken up

Table 3- Levels of haematopoietic toxicity following radiation exposure

Degree	Absolute lymphocyte count	Absolute neutrophil count	Total platelet count	Bleeding and Anaemia
0*	1400-3500/µL	4000-9000/µL	150-450,000/µL	None
1	≥1500/µL	≥2000/µL	≥100,000/µL	Petechiae, bruising Normal Hb level
2	1000-1500/µL	1000-2000/µL	50-100,000/µL	Mild blood loss <10 percent decrease in Hb
3	500-1000/µL	500-1000/µL	20-50,000/µL	Gross blood loss 10-20 percent decrease in Hb

* Degree 0 represents normal reference values.

into plants through the root system, or they may be contaminated by fallout descending on the leaves. The primary risks for internal exposure are caesium-137 and strontium-90. Strontium-89, transuranics alpha emitters, and carbon-14 are also significant sources of concern. Caesium-137 is readily absorbed by food plants, and by animal tissues and distributes itself fairly evenly through the body causing whole body exposure. Strontium is chemically similar to calcium, and is deposited in bone along with calcium. Somewhat less than 10% of the strontium is retained in the bone, but since bone marrow is among the most sensitive tissue in the body to radiation, this creates a very serious hazard. If small particles of alpha emitters are inhaled, they can take up permanent residence in the lung and form a serious source of radiation exposure to the lung tissue. Uranium and the transuranic elements are bone-seekers (with the exception of neptunium) and present a serious exposure risk to bone tissue and marrow.

Cancer: The most serious long term consequence of radiation exposure is the elevation of cancer risk. Cancer risk is more or less proportional to total radiation exposure, regardless of the quantity, rate or duration. There is no evidence of a "safe dose". Safety standards are established primarily to keep the increased incidence of cancer below detectable levels. Cancer risk to radiation exposure can be expressed as the increase in the lifetime probability of contracting fatal cancer per unit of radiation. The current estimate of overall risk is about a 0.8% chance of cancer per 10 rem for both men and women, averaged over the age distribution of the U.S. population. There is also risk coefficient for specific tissue exposures (approximately):

Female Breast	1.0%/100 rem
Bone Marrow	0.2%/100 rem (0.4% for children)
Bone Tissue	0.05%/100 rem
Lung	0.2%/100 rem

Genetic Effects: Radiation damage to germ cells of the reproductive organs can cause mutations that are passed on to subsequent generations. However, no elevated mutation rate from radiation has ever been detected even in the substantial population of atomic bomb survivors and descendants. Two factors can explain this- High acute exposures to the reproductive organs can cause permanent sterility, which prevents transmission of genetic effects and cumulative effect of chronic exposure is limited by

the fact that only exposures prior to reproduction count. Since most reproduction occurs before the age of 30, exposures after that age have little effect on the population.

Cataracts: Eye tissues exposed to radiation show an increased incidence of cataracts at dose levels below which most tissues show increased cancer rates. This makes cataract risk the most important tissue dose criterion for establishing safety standards [31].

Source of information

Our understanding of the effects of total-body radiation is derived from analysis of the clinical course of individuals exposed to radiation after the detonation of two atomic bombs over Japan in 1945, as well as radiation accidents that have occurred throughout the world since that time. In some cases, this includes a large affected population (e.g. the Marshallese exposed in 1954 and individuals in the former Soviet Union and Europe exposed during the Chernobyl nuclear power plant disaster in 1986). As examples: the Chernobyl reactor explosion in the former Soviet Union resulted in high levels of exposure, with 28 people receiving doses >6 Gy, 23 receiving 4 to 6 Gy, and 53 receiving 2 to 4 Gy [32]. There were 115 cases of acute radiation syndrome and 28 deaths. In 1987, in Goiania, Brazil, an abandoned Caesium-137 teletherapy source was breached, with hundreds of people exposed to gamma and beta radiation [33]. There were 48 hospitalisations for radiation injury and four deaths. In contrast, the reactor breach at Three Mile Island in the United States was calculated to result in no more than 50 to 70 mrem of additional exposure to any individual within range. In other cases, relatively low numbers of individuals have been exposed.

The Chernobyl nuclear power plant led to radioactive contamination of aquatic systems which became a major problem in the immediate aftermath of the accident [34]. In the most affected areas of Ukraine, levels of radioactivity (particularly radioiodine: I-131, radiocaesium: Cs-137 and radiostrontium: Sr-90) in drinking water caused concern during the weeks and months after the accident. Bio-accumulation of radioactivity in fish was seen to be significantly above guideline maximum levels for consumption [34]. Groundwater

was not badly affected by the Chernobyl accident since radionuclides with short half-lives decayed away long before they could affect groundwater supplies, and longer-lived radionuclides such as radiocaesium and radiostrontium were adsorbed to surface soils before they could transfer to groundwater [35]. However, significant transfers of radionuclides to groundwater have occurred from waste disposal sites in the 30 km exclusion zone around Chernobyl. After the disaster, four square kilometres of pine forest directly downwind of the reactor turned reddish-brown and died, earning the name of the "Red Forest". Some animals in the worst-hit areas also died or stopped reproducing. Up to the year 2005, more than 6,000 cases of thyroid cancer have been reported in children and adolescents who were exposed at the time of the accident. There is no scientific evidence of increase in overall cancer incidence or mortality rates or in rates of non-malignant disorders that could be related to radiation exposure [36].

In April 2010, a 35 year old man died after exposure to scrap metal containing Cobalt-60 in the Mayapuri industrial area of New Delhi [37]. The man died of multiple organ failure when various treatment modalities failed to resuscitate him. Six other people from the same area were also hospitalised after being exposed to the contaminated scrap metal. Officials retrieved 11 samples of contaminated materials containing Cobalt-60, which is a radioactive material used in food irradiation and radiotherapy.

Following an earthquake, tsunami, and failure of cooling systems at Fukushima I Nuclear Power Plant and issues concerning other nuclear facilities in Japan on March 11, 2011, a nuclear emergency was declared with explosions and a fire resulting in dangerous levels of radiation. This was the first time a nuclear emergency had been declared in Japan, and 140,000 residents within 20km of the plant were evacuated [38].

Key Points

- Harmful effects of radiation have been evident, during nuclear accidents/disasters.
- Acute and delayed damage can occur following radiation exposure.

- Various factors, determine the extent and pattern of injury including the nature and dose of radiation and tissue characteristics.
- Hence, nuclear energy harnessing has to be viewed in light of all the hazards it poses to the human beings and their environment.

References

1. World Nuclear Association. Another drop in nuclear generation World Nuclear News, 05 May 2010.
2. (PDF) Key World Energy Statistics 2007. International Energy Agency. 2007. http://www.iea.org/textbase/nppdf/free/2007/key_stats_2007.pdf. Retrieved 2008-06-21.
3. Share. "Nuclear Waste Pools in North Carolina". http://www. projectcensored.org/top-stories/articles/4-nuclear-waste-pools-in-north-carolina/. Retrieved 2010-08-24.
4. Sturgis S. "Investigation: Revelations about Three Mile Island disaster raise doubts over nuclear plant safety". Southernstudies.org. http://www.southernstudies.org/2009/04/post-4.html. Retrieved 2010-08-24.
5. Sovacool BK. The costs of failure: A preliminary assessment of major energy accidents, 1907–2007. Energy Policy 36, 2008, pp. 1802-20.
6. Cooke S. In Mortal Hands: A Cautionary History of the Nuclear Age, Black Inc. 2009, p. 280.
7. Rotblat J. Acute mortality in nuclear war. In: The Medical Implications of Nuclear War, Soloman F, Marston RQ (Eds), Institute of Medicine, National Academy of Sciences, Washington, DC 1986,251-81.
8. Mole RH. The LD50 for uniform low LET irradiation of man. Br J Radiol 1984; 57:355-69.
9. Reeves GI. Radiation injuries. Crit Care Clin 1999; 15:457-73.
10. Bentzen SM. Preventing or reducing late side effects of radiation therapy: radiobiology meets molecular pathology. Nat Rev Cancer 2006; 6:702-13.
11. Barnett GC, West CM, Dunning AM, Elliott RM, Coles CE, Pharoah PD, et al. Normal tissue reactions to radiotherapy: towards tailoring treatment dose by genotype. Nat Rev Cancer 2009; 9:134-42.
12. Amundson SA, Bittner M, Meltzer P, Trent J, Fornace AJ Jr. Physiological function as regulation of large transcriptional programs: the cellular response to genotoxic stress. Comp Biochem Physiol B Biochem Mol Biol 2001; 129:703-10.

13. Amundson SA, Do, KT, Shahab S, Bittner M, Meltzer P, Trent J, et al. Identification of potential mRNA biomarkers in peripheral blood lymphocytes for human exposure to ionising radiation. Radiat Res 2000; 154:342-6.

14. Goldberg Z, Schwietert CW, Lehnert B, Stern R, Nami I. Effects of low-dose ionising radiation on gene expression in human skin biopsies. Int J Radiat Oncol Biol Phys 2004; 58:567-74.

15. Lushbaugh CC. Human radiation tolerance. In: Parker J Jr (Ed), Bioastronautics Data Book. NASA-S-30006. National Aeronotics and Space Administration Washington, DC 1973; 421.

16. Wald N. Radiation Injury. In: Wyngaarden JB, Smith LH Jr (Eds). Cecil Textbook of Medicine, WB Saunders, Philadelphia 1982, p.2228.

17. Anno GH, Baum SJ, Withers HR, Young RW. Symptomatology of acute radiation effects in humans after exposure to doses of 0.5-30 Gy. Health Phys 1989; 56:821-38.

18. Waselenko JK, MacVittie TJ, Blakely WF, Pesik N, Wiley AL, Dickerson WE, et al. Medical management of the acute radiation syndrome: Recommendations of the strategic national stockpile radiation working group. Ann Int Med 2004; 140:1037-51.

19. Fliedner TM, Friesecke I, Beyrer, K. Medical Management of Radiation Accidents: Manual on the Acute Radiation Syndrome. British Inst Radiol, Oxford 2001;27-32.

20. Gangloff H. Hippocampal spike activity following low doses of irradiation. In: Response of the Nervous System to Ionising Radiation, Haley TJ, Snider RS (Eds), Little Brown, Boston 1964:221-42.

21. Dubois A, Walker RI. Prospects for management of gastrointestinal injury associated with the acute radiation syndrome. Gastroenterology 1988; 95:500-7.

22. Goans RE, Holloway EC, Berger ME, Ricks RC. Early dose assessment in criticality accidents. Health Phys 2001; 81:446-9.

23. Husebye E, Hauer-Jensen M, Kjorstad K, Skar V. Severe late radiation enteropathy is characterized by impaired motility of the proximal small intestine. Dig Dis Sci 1994; 39:2341-49.

24. Medical Consequences of Nuclear Warfare. Walker RI, Cerveny RJ (Eds) Office of the Surgeon General, Falls Church, VA 1989. Available at www.afrri.usuhs.mil (Accessed 8/10/04).

25. Hall EJ. Radiobiology for the Radiologist, 5th ed, Lippincott, Williams & Wilkins, Philadelphia 2000;91-111.

26. Andrews GA, Cloutier RJ. Accidental acute radiation injury. The need for recognition. Arch Environ Health 1965; 10:498-507.

27. Rezvani M, Hopewell JW, Wilkinson JH, Bray S, Morris GM, Charles MW. Time- and dose-related changes in the thickness of skin in the pig after irradiation with single doses of thulium-170 beta particles. Radiat Res 2000; 153:104-9.

28. Peter RU. Cutaneous radiation syndrome - clinical and therapeutic aspects. Rad Protect Bull 1996; 183:19-25.

29. Gale RP. Immediate medical consequences of nuclear accidents. Lessons from Chernobyl. JAMA 1987; 258:625-28.

30. Roberts L. Radiation accident grips Goiania. Science 1987; 238:1028-31.

31. Gerusky TM. Three Mile Island: assessment of radiation exposures and environmental contamination. Ann N Y Acad Sci 1981; 365:54-9.

32. Ricks RC. The radiation-accident patient in the new millennium: past history and future threats. In: The Medical Bases for Radiation-Accident Preparedness: The Clinical Care of Victims, Ricks RC, Berger ME, O'Hara FM (Eds), The Parthenon Publishing Group, Boca Raton, FL 2002. p.1.

33. Dainiak N, Ricks RC. The evolving role of haematopoietic cell transplantation in radiation injury: potentials and limitations. BJR Suppl 2005; 27:169-74.

34. Wildlife defies Chernobyl radiation, by Stefen Mulvey, BBC News. news.bbc.co.uk/2/hi/europe/4923342.stm Published April 20, 2006. (Retrieved 03/05/2011).

35. http://en.wikipedia.org/wiki/List_of_civilian_radiation_accidents#2010 (Retrieved 03/05/2011)

36. Clarens F; de Pablo J, Diez-Perez I, Casas I, Gimenez J, Rovira M (2004-12-01). "Formation of Studtite during the Oxidative Dissolution of UO2 by Hydrogen Peroxide: A SFM Study". Environmental Science & Technology 38 (24): 6656-6661.

37. Indian Man Dies after Radiation Exposure. http://www.nytimes.com/2010/04/28/world/asia/28india.html (Retrieved 03/05/2011).

38. Weisenthal Joe (11 March 2011). "Japan Declares Nuclear Emergency, As Cooling System Fails At Power Plant". Business Insider. http://www.businessinsider.com/fukushima-nuclear-plant-2011-3.(Retrieved 03/05/2011)