東京大学環境安全本部
EHS Environment, Health and Safety, UTokyo

RADIATION RE-EDUCATION MATERIALS THE UNIV. OF TOKYO DOC No. 41 (2023)

Edited by Division for Environment, Health and Safety

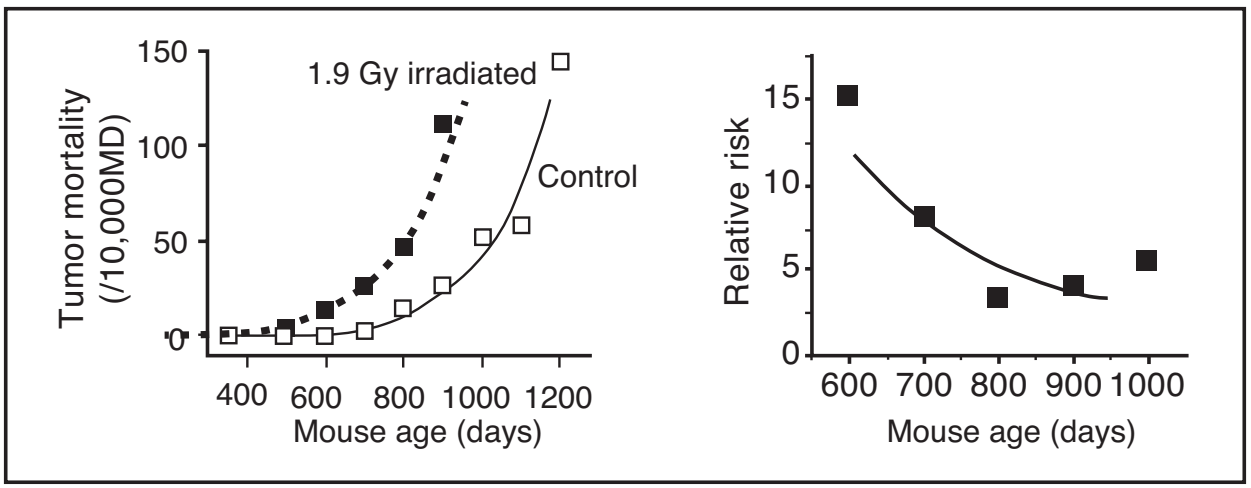
1 Involvement of Tissue Inflammation in Radiation Carcinogenesis

Why are we afraid of exposure to low levels of radiation? This is because we worry about the possibility of developing cancer in somatic cells and the possibility of mutations occurring in germ cells which can affect the next generation. I personally felt this way for nearly 40 years as I worked as a radiation scientist. However, recently I began to feel that my fear was an overreaction, which indicates that it is important to base our fears on real risks which are described by current research findings.

In this short narrative, I will touch on the first issue: radiation carcinogenesis. I imagine that many of us believe that radiation exposures can cause cancer and I thought so until recently. However, I recently came to the conclusion that radiation does not induce cancers, but instead, causes alterations in the cellular microenvironment which are favorable for an earlier development of naturally occurring cancers. Although this new concept is not yet widely appreciated, I believe it will be widely accepted soon.

Before explaining the reasons which support this concept, some background information should be addressed. First, the relative risk for cancer is defined as the ratio of cancer mortality (or incidence) observed in radiation-exposed and control groups. Here, mortality is defined as the ratio of the number of individuals who have died from cancer during an observation period (e.g., one year) to the number of people who were alive on the first day of the observation period. As the cancer mortality increases rapidly after 60 years of age (with the fifth power of age), one may think that the increased risk can be a result of not only an increased mortality (upward shift in the mortality curve) but also a shortened latency (leftward shift). This may occur through the induction of tissue inflammation which leads to stimulation of naturally occurring tumor cells.

There are many supporters of the mutagenesis theory of radiation carcinogenesis, including myself. Historically, in the 1960s to 1970s when animal studies were actively conducted, there were reports which suggested that an exposure to radiation might shorten the latency of cancers (the earlier onset hypothesis), but no further studies to explore this concept were conducted. In addition, starting in the 1970s, there was a major wave of discoveries of cancer-related genes such as *Ras*, *P53* etc., which made the mutagenesis hypothesis appear to be the most likely model, and the earlier onset hypothesis started to be forgotten. However, just a few years ago I began to realize that the earlier onset hypothesis could be a more reasonable hypothesis after examining epidemiologic studies of atomic-bomb survivors and experimental studies in mice.



The figures show solid cancer mortality (left panel) and the relative risk of cancer death (right panel) in mice irradiated with 1.9 Gy of gamma rays (1). The open and closed symbols indicate control and irradiated groups, respectively. The dotted line indicates the mortality curve for the control group, which was shifted toward younger ages by 200 days, and it appears as though it is a curve fitted to the observed data points for the irradiated group (the closed squares). The relative risk (RR: right panel) value initially exceeded 10 but decreased rapidly with an increase in age. If one takes the decreasing trend of the RR at its face value, it appears necessary to think that mice which developed cancers at younger ages were more sensitive to radiation exposures while those which developed cancers later in life were less sensitive, which contradicts biological intuition. The mice that were used in these experiments are F1 hybrids of two inbred strains and are therefore genetically identical, and therefore any genetic heterogeneity present among individuals would not explain the decreasing trend in the RR. In humans, the same decreasing trend in the RR has been reported but no biological explanations have ever been offered.

These considerations led me think that the radiation-related increase in cancer risks could be better understood (or less contradictory) if one considers that the mortality shift is not in an upward direction, but rather in a leftward direction following an exposure to radiation. In fact, shifting the mortality curve leftward was found to faithfully reproduce past observations of the RR in atomic-bomb survivors (1). In the survivor study, exposure to 1 Gy at age 30 caused RR of 1.4 for cancer death at age 70, and these same exposure conditions may also be interpreted as a loss of life of 6 years if one died with cancer. Assuming a linear-non-threshold model, the RR is then 1.04 at 100 mGy, while life shortening is 0.6 years or 220 days. At 10 mGy the RR is 1.004 or 22 days of life lost. In other words, it is clearly understandable that the RR of 1.004 is quite small. However, if you imagine that you developed a cancer after an exposure to 10 mGy, you might think that you have fallen into the group with the excess 0.004 RR, and feel that you had experienced a major run of bad luck in your life. On the other hand, it is curious to find that the same risk is expressed as 22 days of life lost through cancer, and this does not seem to be a really major run of bad luck.

From the viewpoint of cancer risk from radiation, many researchers consider that an exposure below 100 mGy is a low dose. This is mainly because statistically significant increases in cancer risk are observed only above 100 mGy exposures in epidemiologic studies of atomic-bomb survivors. This does not mean that low doses are safe because a statistically significant increase is detected above 100 mGy, and so the question here is how to connect the two points between 100 mGy and zero mGy. The International Commission for Radiological Protection (ICRP) proposes a linear non-threshold model (LNT model) for the purpose of radiation protection, but at least one country (France) has adopted a threshold model.

With the mutation induction model, because the RR decreases with an increase in attained age, no single value can represent an optimal value for the risk through one's life. In contrast, if the risk is expressed as a shortening of life, there is no such problem, and a single value can be applied throughout one's life (and is much easier to explain to the public). It should be noted that there may be exceptions to the early onset hypothesis such as childhood leukemia in humans and thymic lymphomas in mice, both of which occur rarely spontaneously, but with a short latency period following a radiation exposure. These tumors appear to better fit the hypothesis of radiation induction.

There are many reports which have described the induction of inflammation in irradiated normal tissues. I cannot present many reports here, but an example is the observation that transplanted tumor cells in the hind legs of mice can grow better if the hind legs are pre-irradiated with doses below 5 Gy (2). Tumor growth was less vigorous when higher doses of radiation were given, which was probably caused by a decreased ability to form blood

vessels. The results clearly show that that exposure to radiation in cancer radiotherapy affects not only tumor cells but also surrounding tissues (tumor bed). Another example is that when tumor cells are transplanted into host animals, they do not form tumors when the number of transplanted cells is lower than a critical number. Under such conditions, however, tumors can grow if a wound was induced near the transplant site on the skin (3). This seems likely to occur because various factors are released from inflammatory cells gathered near the wound to lead to tissue repair, and this type of condition aids tumor cell growth as well.

In the past, radiation-related inflammation was thought to occur after an exposure to large doses of radiation such as radiotherapy for cancer. However, recent studies have broadened the concept of inflammation because many inflammatory factors have been identified, and detailed analysis has become possible (see ref. 4 for an example). Reports show that 1 Gy of radiation can induce unstable-type chromosome aberrations in as many as 17% of the cells (5) which is enormous, but the damaged cells do not die immediately. Problems occur later when the first post-irradiation cell division occurs at some time after the exposure. And when cells die in our body, the cellular debris needs to be cleared by macrophages, and these macrophages release various factors to attract supporting cells to the target or inflamed site. In other words, these damaged cells may function as a biological time bomb, which may explain at least partly why radiogenic inflammation persists for a long time (6). Because radiation-related inflammation does not accompany general signs of senescence, this long-lasting inflammation is different from accelerated aging (7).

If we understand that radiation carcinogenesis requires inflammatory processes to develop following an exposure to radiation, it would seem that anti-inflammatory treatments could interfere with the carcinogenic processes inflicted by irradiation. The mutation hypothesis does not provide us with any idea or model which can prevent carcinogenesis because it is impossible to reverse mutations. However, we know that there are multiple ways to fight inflammation using non-steroidal anti-inflammatory drugs (NSAIDs). 7,12-dimethylbenzanthracene (DMBA) is one of the potent chemical mutagens (mainly AT to TA base-change mutation is induced) and induces mammary tumors within 100 days after its administration in rats. However, feeding rats with NSAID-containing food resulted in a decreased frequency of mammary tumors by more than 50% (8). In contrast, radiation effects are relatively weak since the appearance of tumors requires more than 400 days (Figure), and hence is a much weaker carcinogen than DMBA. Thus, it seems very likely that NSAIDs may also be effective in preventing radiation carcinogenesis.

Because multiple pathways are involved in radiation-induced inflammation, it seems unlikely that any single NSAID can accomplish this task, and thus a combination of them would be necessary. It also seems logical to expect that such an approach could contribute to the suppression of naturally occurring carcinogenesis. In this regard, it has been indicated that people taking low doses of aspirin for many years show a lower risk for colon cancer (9).

We wish you join radiation sciences in the 21st century!

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Commentary: Mechanisms of Carcinogenesis

Cancer is a general term for a disease in which cells grow and invade surrounding normal tissues (malignant transformation), rendering them incapable of supporting life. For many years, the classical “multi-step carcinogenesis” theory explained that cancer is a group of highly malignant and homogeneous cells formed by the accumulation of mutations in a single normal somatic cell. However, cancer cells are found to be a diverse population resulting from the accumulation of mutations in genes that regulate normal cellular activities such as proliferation, repair, energy production, cellular senescence and cell death. They diversify spatially further and adapt to different environments within the body by building robust ecosystems with surrounding non-cancerous cells.

Inflammation is one of the initial immune defenses against infection and tissue damage, but chronic inflammation is detrimental and strongly associated with tumor progression. During this process, cancer cells possess ‘non-self’ characteristics that set them apart from normal cells. These cells are monitored and eliminated by the cancer immune systems, and cancer cells are more likely to survive when the ability of the immune systems are impaired.

Soon after the discovery of X-rays by Dr, Roentgen (1895), it was confirmed that many doctors and technicians who worked with X-rays suffered from skin cancer from chronic ulcers. In 1911, radiation-induced leukemia was reported. Since then, long-term follow-up studies of atomic bomb survivors in Hiroshima and Nagasaki and large-scale animal experiments have been conducted, and a large amount of data on radiation carcinogenesis has been accumulated. Since radiation induces mutations efficiently, studies on the initial processes of carcinogenesis, such as DNA damage generation and repair, cell death, and mutation induction, are in progress. However, to truly understand the data on radiation-induced cancer epidemiology, it is necessary to understand the late-stage carcinogenesis mechanism that is formed by the cross-talk between cancer cells and non-cancer cells. “Stem Cell Biology with Respect to Carcinogenesis Aspects of Radiological Protection (ICRP Publ. 131)” recommended by the International Commission on Radiological Protection discusses various aspects from this perspective. This article by Dr. Nori Nakamura also focuses on the possibility that radiation exposure may activate inflammatory factors such as stromal fibroblasts and macrophages and cause microenvironmental changes that promote spontaneous tumor cell growth.

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2 Radiation Protection and Dose Assessment for Internal Exposure

1. Laws and regulations related to dose assessment for internal exposure

Regarding dose assessment for radiation exposure, “the Act on the Regulation of Radioisotopes, etc. (ARR) [1]" stipulates that licensed and notified users must measure the quantity of radiation received by any person who entered usage facilities, etc. (Article 20, Paragraph 2 of ARR). The Regulation for Enforcement of ARR (REARR) [2] also stipulates that “the quantity of radiation” here is the radiation dose due to both external and internal exposure (Article 20, Paragraph 2 of REARR). In particular, the following standards for implementing dose assessment for internal exposure were specified (Article 20, Paragraph 2, Item 2 of REARR):

- When radioisotopes were incorporated into the body via inhalation or ingestion
- Within every period not exceeding 3 months for those who entered an area where one may inhale or ingest radioisotopes (however, within every period not exceeding 1 month for pregnant women)
- It is not necessary to implement dose assessment for non-radiation workers who temporarily entered a radiation-controlled area if the level of internal exposure is unlikely to exceed the dose specified by the Nuclear Regulation Authority (NRA)

Here “the dose specified by the NRA” is prescribed to be 100 μSv in effective dose by the Notification No.6 of the NRA (Article 18, Paragraph 2) [3].

The effective dose due to internal exposure, E (mSv), should be assessed by summing the dose calculated by the following formula for each radioisotope listed in Column 1 of Appended Table 2 in the Notification No. 6 of the NRA (Article 19) [3]:

$$E = e \times I$$

where e is the effective dose coefficient (mSv/Bq) and I is the intake of the radioisotope (Bq). The effective dose coefficients are provided in Column 2 (inhalation) and 3 (ingestion) of Appended Table 2 in the Notification No. 6 of the NRA [3].

2. Methods for assessing intake of radioisotopes

The intake of the radioisotope can be calculated from information such as the air concentration of radioactivity in the work environment, working hours, and breathing rate [4]. However, it should be noted that the uncertainty associated with the assessment may be large since the radioactivity actually incorporated into the body is not measured in this method.

Rather, a more detailed assessment based on *in vivo* measurements and bioassay may be performed if unexpected internal exposure occurs. The former method can detect radiations emitted inside of the body, and γ emitting radionuclides are the main target of this method; the latter method can detect radioisotopes contained in biological samples (such as feces and urine), and α- and β-emitting radionuclides are also included to be the target of this method, but it will take about 1 week for organics decomposition, nuclide separation, electrodeposition for sample preparation, and radioactivity measurement. As these values measured in *in vivo* measurement or bioassay are the radioactivity remaining in the body (namely, residual amount) or the radioactivity excreted in biological samples (namely, excreted amount), the intake amount, I (Bq), can be calculated by dividing the

[1] Ratio of the residual/excreted amount to 1 Bq intake. Since the amount of radionuclides incorporated into the body decreases according to their physical and biological half-lives, the retention and excretion rates depend on the elapsed time after intake, t.

residual/excreted amount, m (Bq), by the retention/excretion rate, $r(t)$ [1], as shown in the following formula:

$$I = m / r(t)$$

The retention and excretion rates were calculated from biokinetic models developed by International Commission on Radiological Protection (ICRP), and those data for "the reference worker" are provided in ICRP Publ. 78 [5]. Because the retention and excretion rates as well as effective dose coefficients depend on the chemical form of the radionuclide handled at the time when internal exposure occurred, collection of available information regarding the characteristics of radionuclide incorporated into the body is also important for dose assessment.

3. Management of internal exposure based on concentration limit

Daily radiation management for "areas" in addition to "individuals" is required to be taken in usage and storage of radioisotopes to prevent radiation hazards (Article 15, Item 1 and Article 16, Item 1 of ARR). In particular, areas where people enter must not exceed the air concentration limit for the purpose of managing internal exposure (Article 14, Item 4 and Article 17, Item 4 of REARR). The air concentration limit, L (Bq/cm^3), corresponding to 1 mSv/week can be basically calculated using the effective dose coefficient, e (mSv/Bq), the breathing rate, R (cm^3/h), and the working hours, T (h/week), as shown in the following formula [2]:

$$L = \frac{1}{e \cdot R \cdot T}$$

The air concentration limit for each radioisotope calculated by substituting 1.2×10^6 (cm^3/h) for R and 40 (h/week) for T are provided in Column 4 of Appended Table 2 in the Notification No. 6 of the NRA [3]. The average air concentration during a week is compared with the limit in actual management, and in case that two or more types of radioisotopes are present in the air, the sum of the ratios of the air concentration to the limit for them should not exceed 1 (Article 7 of the Notification No. 6 of the NRA).

It is also necessary to take measures to prevent radiation hazards in case of waste management of radioisotopes, etc. (Article 19 of ARR), and the concentration of radioisotopes in exhaust gas and drainage must be below the limit prescribed by NRA (Article 19 of REARR). The concentration limit for exhaust gas/drainage, L (Bq/cm^3), corresponding to 1 mSv/year in average from 0 to 69 years old (70 mSv in total of 70 years) can be basically calculated using the effective dose coefficient, e_i (mSv/Bq), and the breathing/water intake rate, R_i (cm^3/year), by age i, as shown in the following formula [3]:

$$L = \frac{70}{\Sigma_i^{69} (e_i \cdot R_i)}$$

The concentration limit for exhaust gas/drainage calculated by substituting the breathing/water intake rate shown in Table 1 are provided in Column 4 (exhaust gas) or 5 (drainage) of Appended Table 2 in the Notification No. 6 of the NRA [3]. The average concentration during three months is compared with the limit in actual management, and in case that two or more types of radioisotopes are present in the exhaust gas/drainage, the sum of the ratios of the concentration to the limit for them should not exceed 1 (Article 14 of the Notification No. 6 of the NRA).

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[2] See reference [6] for derivation of air concentration limits for inert gases and radon.

[3] See reference [6] for derivation of air concentration limits for tritium, inert gases, and radon.

Table 1 Breathing and water intake rates by age [6]

age i	Breathing rate R_i (cm^3/year)	Water intake rate R_i (cm^3/year)
0	1.0×10^9	5.1×10^5
1-2	1.9×10^9	5.1×10^5
3-7	3.2×10^9	5.8×10^5
8-12	5.6×10^9	6.6×10^5
13-17	7.3×10^9	8.8×10^5
18-69	8.1×10^9	1.3×10^6

4. Protection principles for handling unsealed radioisotopes

Protection based on the five (3D and 2C) principles, i.e., “Dilute”, “Disperse”, “Decontaminate”, “Contain”, and “Concentrate”, is effective when handling unsealed radioisotopes which may cause internal exposure. Specifically, the following countermeasures can be considered.

- To use fume hoods (“Disperse”, “Decontaminate”, and “Contain”)
- To wear double gloves (“Disperse” and “Decontaminate”)
- To zone work area and change slippers (“Contain” and “Concentrate”)
- To use trays and absorbent (“Contain”)
- To prevent spread of contamination by curing (“Decontaminate” and “Contain”)

As radioisotopes may be incorporated into the body through three routes via: inhalation, ingestion, or wound/percutaneous absorption, it should be paid particular attention that (1) the radioactivity concentration in the work environment has not increased, (2) experimental instruments contaminated with radioisotopes do not come into contact with the mouth, and (3) radioisotopes do not adhere to the skin, in addition to appropriate measurements with a survey meter to confirm that there is no contamination.

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Re-Education Theme: Safety Handling; Laws and Ordinances

3 Status of Nuclear Waste Management in Japan

The use of radioactive isotopes or nuclear fuel materials generates a variety of radioactive waste. The series of actions involved in the treatment, storage, and disposal of radioactive waste is called radioactive waste management. This section provides an overview of waste management in Japan and the points you should be aware of.

As with household and industrial wastes, the first step in waste management is to classify the wastes. The classification of radioactive waste varies from country to country, but generally depends on half-lives and radioactivities, properties of the wastes, and sources of the wastes. The classification by half-lives and radioactivities ranges from short-lived low-level waste to high-level waste, depending on the half-lives and radioactivity concentrations of radionuclides contained in the wastes. In Japan, there is no intermediate-level category, and waste is classified into high-level waste, which is a waste generated from vitrification of liquid waste after reprocessing spent fuels, and the other low-level wastes. Low-level waste is further divided into L3 waste with extremely low radioactivity, L2 waste with relatively low radioactivity, L1 waste with relatively high radioactivity, TRU waste generated at reprocessing plants, etc. that contains long-lived transuranic elements and fission products, and uranium waste generated in the uranium refining process. Waste with extremely low concentrations of radionuclides and little or no impact on the human body can be removed from the management as radioactive waste (i.e. clearance waste) with permission and confirmation from the government after measurement. Classification by properties includes gaseous, liquid, and solid wastes, as well as combustible and incombustible wastes. Classification by source includes vitrified waste and TRU waste generated at reprocessing plants, as well as operating waste generated during facility operation and decommissioning waste generated during decommissioning. In addition, wastes generated from research and educational activities at universities and research institutes are considered laboratory wastes.

These various classifications of radioactive waste are required for later treatment and disposal, and the classified waste streams in waste management are called waste schemes. Disposal of radioactive waste can be broadly classified into dilution/dispersion and burial. Relatively small gaseous and liquid wastes (specifically, gaseous and liquid wastes that have been diluted to below the concentration required by law specified for each nuclide) are allowed to be discharged into the environment through stacks and drains. On the other hand, liquid and gaseous wastes with relatively high radioactivity concentrations can be discharged to the environment after waiting their radioactivities being decayed, or, if this is not feasible, can be disposed of by burial after being stabilized by adsorption or precipitation and being solidified into a waste body in a container with a filler such as mortar. Solid wastes are disposed of in the same manner, but since they are usually bulky, they are compacted or melted to reduce their volume and homogenize them. For combustible liquids and solid wastes, volume reduction by incineration is effective.

For the burial of solid wastes, the depth of the repository and the artificially placed barriers (i.e. engineered barriers) that delay the release of nuclides into the environment depend on the radioactivities and half-lives of radionuclides contained in the wastes. For example, the L3 wastes described above is disposed by trench disposal (equivalent to usual landfill of industrial wastes), the L2 wastes by concrete pit disposal, the L1 wastes by intermediate depth disposal below 70 m from the ground level, and the vitrified and TRU wastes by geologic disposal below 300 m. Materials such as bentonite are used as engineered barriers because of their high impermeability against groundwater intrusion and expected retardation of radionuclide migration through adsorption. The waste material itself

also contributes to the control of radionuclide leaching as an engineered barrier. In the case of wastes containing relatively high concentrations of long-lived radionuclides, such as L1 waste, vitrified and TRU waste, it is impractical to control leaching and migration using only engineered barriers, so the ability of the surrounding host rocks and soils to retain radionuclides is used as a barrier (i.e. natural barriers). Since the safety of such a disposal facility cannot be demonstrated empirically, it is assumed that if a repository were constructed, leached radionuclides would migrate through groundwater, reach the surface, and expose human being through various pathways, and the extent of this exposure is evaluated in a predictive manner and compared to safety standards. This can be seen as a type of environmental risk assessments.

In Japan, a repository for L2 waste generated from nuclear power plant operations is currently in operation in Rokkasho-mura, Aomori Prefecture. In addition, the selection of a repository for vitrified and TRU wastes is underway by the Nuclear Waste Management Organization (NUMO), and in 2020, a literature review, the first step in the site selection process, has begun in two municipalities in Hokkaido. Wastes containing RI generated from universities and research institutes and RI sources that are no longer needed can be sent to the Japan Radioisotope Association with charge for storage and future disposal. Similarly, waste containing nuclear fuel materials generated in university and research institutes is called research laboratory wastes, and the Japan Atomic Energy Agency is responsible for their disposal, although they are stored in the source facilities as there is no repository to accept them.

It is inevitable that radioactive waste will be generated in education and research activities using RI and nuclear fuel materials. Please conduct cold tests and other experiments to reduce the amounts of wastes as much as possible, and plan and prepare well before starting experiments. In addition, please be aware of the properties of generated wastes, not only the types and concentrations of radionuclides contained in the wastes, but also the coexisting materials. If the wastes contain hazardous materials other than radioactive materials, special attention should be paid to the fact that acceptance of the wastes into a repository may be restricted. Also, if there is a possibility of leakage, or if the material is unstable per se, it must be chemically stabilized. And information about the wastes should be properly recorded to ensure that there are no problems with future treatment or disposal.

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Re-Education Theme: Nuclear Materials; Application

4 Safety Control of X-ray Irradiators

The University of Tokyo has approximately 300 X-ray irradiators for researches. There are a wide variety of types, and various levels of safety functions to prevent exposure accidents. This document shows important issues when using or managing X-ray irradiators. For detailed information, please refer to the convenience book page* of Division for Environment, Health and Safety.

As a general premise, for an operator or an administrator of X-ray irradiator followings are required.

- When installing a new irradiator, do not forget predetermined procedures in advance through the safety manager of the faculty.
- Do not modify or release safety devices to prevent exposure accident of x-rays.
- If the X-ray apparatus cannot be turned off for beam adjustment and/or maintenance work, the apparatus door should be locked during the work. If the door cannot be locked for some reason, use shields and take extra care to avoid scattered X-rays when you do such work.

Especially for an operator of X-ray irradiator followings are required.

- A person who operates X-CDE irradiator (whose exposure risk is assumed to exist outside of the devise) should wear a personal dosimeter at the specified position.
- Create and maintain operation records
- Open and close the safety beam shutter after checking for the irradiation status of X-rays
- If you notice any abnormalities, immediately turn off the X-ray and contact the equipment administrator
- When sharing one X-ray irradiator with multiple laboratories or researchers, display the operator's name during the time of use. In addition, check the status inside of the device before use, and return to the basic settings after use.

Especially for an administrator of X-ray irradiator followings are required.

- Shield it based on measurement results and usage conditions if necessary
- Display the emergency contact information, results of dosimetry, feature information on the equipment, and X-ray administrator's name
- Conduct periodical inspections once a year determined by The University of Tokyo for all devices, and for X-CDE irradiators, check the working environment as defined by laws and ordinances (every six months for use at a fixed point)

Recently, most of X-ray irradiators used for researches and educations has been designed with a consideration of safety. It is important to fully understand the structure and characteristics of the safety equipment in order to operate and control it.

1.X-ray_Equipment,Electron_Microscope

https://univtokyo.sharepoint.com/sites/EHS_portal/SitePages/d/X-ray_Equipment,Electron_Microscope.aspx

2.Periodic_Inspection_of_X-rays_Device,Electron_Microscope

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Re-Education Theme: Safety Handling, Laws and ordinances