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RADIATION

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RADIATION

1.0 ADMINISTRATION

SYNONYMS

GENERAL TOXICITY HAZARD RATING

REPRODUCTIVE HAZARD RATING

1.1 SYNONYMS

A)

IONIZING **RADIATION**
RADIATION, IONIZING
GAMMA RAYS
BETA PARTICLES
ALPHA PARTICLES
POSITRONS
X-RAYS
NEUTRONS
RADIOACTIVE MATERIAL
RADIOACTIVE MATERIAL, ARTICLES MANUFACTURED from NATURAL OR DEPLETED URANIUM AND NATURAL THORIUM
RADIOACTIVE MATERIAL, EMPTY PACKAGE-ARTICLES MANUFACTURED from NATURAL OR DEPLETED URANIUM OR THORIUM (NATURAL)
RADIOACTIVE MATERIAL, EMPTY PACKAGES
RADIOACTIVE MATERIAL, EXCEPTED PACKAGE-EMPTY PACKAGING
RADIOACTIVE MATERIAL, EXCEPTED PACKAGE-LIMITED QUANTITY OF MATERIAL
RADIOACTIVE MATERIAL, EXCEPTED PACKAGES
RADIOACTIVE MATERIAL, INSTRUMENTS AND ARTICLES
RADIOACTIVE MATERIAL, LIMITED QUANTITY, N.O.S.
RADIOACTIVITY
RADIOISOTOPES
RADIONUCLIDES
URANIUM METAL, PYROPHORIC
YELLOW CAKE (SLANG FOR URANIUM OXIDE)

1.4 GENERAL TOXICITY HAZARD RATING

A) 3

1.5 REPRODUCTIVE HAZARD RATING

A) A

2.0 INTRODUCTION

A) FORMS

1) ALPHA ionizing **radiation** is:

1) 2 neutrons and 2 protons

2) Highly ionizing

3) Travels several centimeters in air and a few microns in tissue
4) Stopped by a thin paper or clothing
5) Threat is inhalation or absorption of alpha emitter in wounds.

2) BETA ionizing **radiation** is:
1) High energy "electron" emitted from nucleus
2) Can have wide range of energies depending upon particular radionuclide
3) Moderately penetrating
4) Up to a few meters in airMillimeters in tissue
5) Some protection by PPE

3) GAMMA or X-Ray photons are:
1) High energy rays
2) Very penetrating
3) Difficult to shield (need lead or other dense material)
4) PPE will not protect against photon **radiation**

4) NEUTRONS ionizing **radiation** are:
1) Neutral particles emitted from the nucleus
2) Can be very penetrating
3) Requires special consideration for shielding

5) Units of measure of **radiation** include (1) gray (Gy) - basic unit for measuring **radiation** dose; (2) rem - quantifies the amount of damage that is suspected from a particular type of **radiation** (Jarrett, 1999).

6) **Radiation** half-life is the time required for a radioactive substance to lose half of its radioactivity. Each radionuclide has a unique half-life, with half-lives ranging from extremely short (fraction of a second) to millions of years (Jarrett, 1999).

B) SOURCES

1) **Radiation** exposure may occur in medical, industrial, and laboratory accidents in which individuals are exposed to unacceptably high doses of **radiation** (ICRP, 1977; Gains, 1989; Wagner et al, 1994). With increased use of radioactive materials, there is also an increased need for transport of such materials with the attendant risk of transport accidents and releases. In addition, there remains the possibility of exposure of large masses of people through detonation of nuclear weapons (Conklin et al, 1983).

2) Ionizing **radiation**, resulting from decay of unstable nuclei, consists of three classes of ionizing **radiation**: alpha, beta, and gamma **radiation** (NATO , 1995).

a) Alpha **radiation** (alpha particles) are helium nuclei which are mainly emitted from heavy nuclei such as uranium-235 or plutonium-239. The range of these particles is a few centimeters in air, and a few tenths of a millimeter in body tissue. These particles generally can not penetrate human skin, but may cause damage when ingested or inhaled.

b) Beta **radiation** (beta particles) consists of electrons and positrons which can be stopped by 1 cm of water or 10 m of air. In body tissue beta **radiation** has a range of up to 10 mm. Strontium-90, a beta-emitting substance, can penetrate skin and can also be systemically absorbed through ingestion and inhalation.

c) Gamma **radiation** (gamma rays) consists of photons with a large range as compared with alpha and beta **radiation**. A human body may only partly stop gamma **radiation** and it is only minimally stopped by air. Shielding by lead or other dense material is required to stop gamma **radiation**.

3) SOURCES OF LARGE RADIOACTIVE DISCHARGES

a) REACTOR ACCIDENTS - Radioactive substances such as I-131 may be released from nuclear power reactors (Becker, 1987) Jarrett. 1999). Nuclear energy production carries the extremely small risk of **radiation** accidents and **radiation** exposure of the general population worldwide (Champlin et al, 1988).

b) WINDSCALE (Sellafield, England, 1957): 30,000 Ci of iodine-131, 12,000 Ci of tellurium-132, and 600 Ci of cesium-137 were released (Diffre, 1990). A curie (Ci) is a unit of activity equal to 3.61 x 10(10) disintegration per second.

c) KYSCHTYMSK (Urals, 1957): 1 x 10(6) Ci of strontium-90 were estimated to have contaminated an area of 100 to 1,000 square kilometers (Diffre, 1990).

d) THREE MILE ISLAND (USA, 1979): Approximately 6 x 10(11) Bq of iodine-131 was released (Diffre, 1990). A becquerel (Bq) is a unit of activity, with 1 Bq equal to 1 disintegration/second. 1 curie equals 3.61 x 10(10) Bq.

e) CHERNOBYL (Byelorussia/Ukraine, 1986): 10 x 10(7) Ci of strontium-90 was reported to have been released, causing 2000 to 3000 square kilometers of soil to be unfit for agriculture (Diffre, 1990).

4) OTHER SOURCES

a) MILK: Iodine-131 is concentrated in the milk of herbivorous animals.

b) RADIUM-226: Is absorbed by plants and animals and its concentration in the human body results from

ingestion of food (Diffre, 1990).

c) RAINWATER: Usually contains beryllium, carbon-14, tritium, strontium-90, and cesium-137; total normal beta radioactivity is around 1 Bq/L (Diffre, 1990).

d) UNDERGROUND WATER: Contains uranium-238 and related products and radium-226. It also contains radon-222 which can be highly concentrated at the source of thermal springs (Diffre, 1990).

e) SEAWATER: Beta radioactivity, primarily from potassium-40, is around 10 Bq/L (Diffre, 1990).

f) BULLETS: Depleted uranium, "DU", used in armor-piercing shells contains 99.75 percent U-238; soldiers handling or shot with these bullets may be exposed (Christensen, 1993).

g) Common fission products from nuclear tests that have fallen onto the surface of the globe include: strontium-90, cesium-137, iodine-129, and iodine-131 (Diffre, 1990).

1) Strontium-90 has chemical properties similar to calcium and is deposited in bone.

2) Cesium-137 behaves similarly to potassium, but it remains in the human body 2 to 5 times longer.

3) Iodine-129 has an atmospheric half-life of 16 x 10(6) years. Radioactive iodine is stored by mammals in the thyroid gland; there its concentration is 3 to 4 times that of exposed grass.

C) USES

1) AMERICIUM-241 is a decay daughter of plutonium and is an alpha emitter, readily detectable with a standard radiac instrument due to emission of a 60-kEv gamma ray. Its use includes smoke detectors and other instruments, and it is found in fallout from a nuclear weapon detonation. It is considered a heavy metal poison, but in large radioactive doses, can cause whole-body irradiation. 75% of an initial lung burden is absorbed with 10% of the particles retained in the lung. GI absorption is minimal, but absorption through skin wounds may be rapid. Elimination occurs via urinary and hepatic excretion (Jarrett, 1999).

2) CESIUM-137 may be found in medical radiotherapy devices. It was reported to be used in the Chechen RDD threat against Moscow. Both gamma rays and beta **radiation** are emitted and can be readily detected by gamma instruments. Absorption is complete via the lungs, GI tract and skin wounds. It is soluble in most forms and is treated by metabolism as a potassium analog. Excretion is via the urine. Whole-body irradiation is the primary toxicity, with deaths due to acute **radiation** syndrome reported (Jarrett, 1999).

3) COBALT-60 is commonly used in medical radiotherapy devices and commercial food irradiators. Most commonly, contamination is discovered after improper disposal, or after destruction of a hospital or commercial facility. Generation of high-energy gamma rays and 0.31-MeV beta rays are produced. A gamma detector provides easy detection. Uses of cobalt include as a contaminant in an improvised nuclear device to make fallout more radioactive. Rapid absorption occurs from the lungs, but less than 5% will be absorbed via the GI tract. Wound absorption is not known. Whole-body irradiation and acute **radiation** syndrome are its primary toxicities (Jarrett, 1999).

4) DEPLETED URANIUM (DU) emits limited alpha, beta, and some gamma **radiation**, but poses no significant **radiation** threat. It is found in armor-piercing munitions, armor, and aircraft counterweights and is readily detectable with a typical end-window G-M (Geiger-Mueller) counter. Inhaled uranium compounds can be metabolized and result in urinary excretion. DU oxides may be inhaled during tank fires or by entering destroyed armored vehicles without a protective mask. Absorption is determined by the chemical state of the uranium, with soluble salts being readily absorbed and the metal not being absorbed. If DU metal fragments become encapsulated in wounds, they are gradually metabolized, resulting in whole-body distribution, especially to bones and kidney. No renal toxicity has been documented to date. DU does cross the placenta (Jarrett, 1999).

5) IODINE-131, 132, 134, and 135 are found after reactor accidents and following the destruction of a nuclear reactor by hostile forces. Radioactive iodine (RAI), a normal fission product found in reactor fuel rods, is released by rupturing the reactor core and its containment vessel. Wind patterns at the time of destruction determine the fallout pattern. Most of its **radiation** is beta rays, with some gamma. Toxicity is primarily to the thyroid gland. RAI concentrates in the thyroid due to uptake by this gland, and allows local irradiation similar to therapeutic thyroid ablation. Following the Chernobyl disaster, a high incidence of childhood thyroid carcinoma was reported (Jarrett, 1999).

6) PHOSPHORUS-32 is usually found in research laboratories and in medical facilities with use as a tracer. It emits strong beta rays and can be detected with the beta shield open on a beta-gamma detector. It is completely absorbed from all sites and is deposited in the bone marrow and other rapidly replicating cells. Local irradiation results in cell damage (Jarrett, 1999).

7) PLUTONIUM-239, -238 is produced from uranium in reactors and is the primary fissionable material in nuclear weapons and is the predominant radioactive contaminant in nuclear weapons accidents. Its primary **radiation** the form of alpha particles, thus not presenting an external irradiation hazard. It is ALWAYS contaminated with americium, which is a fairly readily detectable x-ray by use of thin-walled gamma probe. Primary toxicity is via the inhalation route, with 5-micron or smaller particles remaining in the lung and metabolized based on its salt solubility. Local irradiation damage is caused by remaining particles in the lungs. The chemical state of plutonium

determines its GI absorption, with the metal not being absorbed. After 24 hours, stool specimens are positive and after 2 weeks urine specimens are positive. Wound absorption is variable. Plutonium is able to be washed from intact skin (Jarrett, 1999).

8) RADIUM-226 has no military use, but may be found in FSU equipment as instrument illumination, in industrial applications, and in older medical equipment. Its primary **radiation** is due to alpha particles, but daughter products emit beta and gamma rays, which in quantity may present a serious external irradiation hazard. Exposures are usually by ingestion, with 30% absorption. Wound absorption is not known, but radium will follow calcium to bone deposition. Leukemia, aplastic anemia, and sarcomas are associated with chronic exposures (Jarrett, 1999).

9) STRONTIUM-90 is a direct fission product (daughter) of uranium, with it and its daughters emitting both beta and gamma rays which can be an external irradiation hazard if present in quantity. Strontium follows calcium and is readily absorbed via both respiratory and GI routes. Up to 50% of a **radiation** dose will be deposited in bone (Jarrett, 1999).

10) TRITIUM (hydrogen-3) is hydrogen with a nucleus composed of two neutrons and one proton. It has found use in nuclear weapons and in the U.S. (and other Western countries) in luminescent gun sights and muzzle-velocity detectors. It is NOT likely to be a hazard except within a confined space. Tritium gas is rapidly diffused into the atmosphere. Since tritium is a beta emitter, it is NOT a significant irradiation hazard. Water formed from tritium (HTO) is completely absorbed and equilibrates with body water. Excretion is via the urine, with urine samples positive within an hour of significant exposure. A single acute exposure has NOT been reported to result in any significant health effects (Jarrett, 1999).

11) URANIUM-238, -235, -239 can be found, in increasing order of radioactivity, in depleted uranium (DU), natural uranium, fuel rods, and weapons-grade material. Alpha, beta, and gamma **radiation** are emitted from uranium and its daughters. Neither DU nor natural uranium present any serious irradiation threats. Significant levels of gamma particles are emitted from used fuel rods and weapons-grade (enriched) uranium containing fission products. Following placement of enough enriched uranium together, a critical mass may form and emit lethal levels of **radiation**. This scenario could occur in a fuel-reprocessing plant or melted reactor core. Following inhalation, uranium compounds may be metabolized and excreted in the urine. Following an acute exposure, uranium urine levels of 100 mcg/dL may cause renal failure. Absorption is determined by the chemical state of the uranium, with soluble salts readily absorbed and the metal not absorbed (Jarrett, 1999).

3.0 EFFECTS OF ACUTE EXPOSURE

A) The biological effects of exposure to ionizing **radiation** in humans are well known. Acute exposures greater than 1 Gray (100 rads) may result in **radiation** sickness, with the primary targets being blood-forming cells, the intestinal mucosa, and the brain. With whole-body **radiation**, less than 200 RADs is not lethal, 200 to 1000 RADs may be lethal, and exposure to greater than 1,000 rads is supralethal (Conklin et al, 1983).

B) ACUTE **RADIATION** SYNDROME may vary in nature and severity, depending on the total dose, dose rate, dose distribution, and individual susceptibility (NAS, 1963) and is divided into four distinct clinical phases: PRODROME, LATENT, MANIFEST ILLNESS, and RECOVERY.

C) The PRODROME is the initial phase, which generally appears within minutes to hours after exposure. It is characterized by nausea, vomiting, diarrhea, excessive salivation, intestinal cramps, dehydration, fatigue, weakness, apathy, fever, and hypotension (Conklin et al, 1983).

D) The LATENT period is an intervening time between exposure and the development of obvious symptoms; duration may be from several days to weeks.

E) MANIFEST ILLNESS occurs when effects on the blood-forming system become apparent. It is characterized by reduction in the counts of all types of blood cells (pancytopenia, thromobytopenia), resulting in infections, increased risk of bleeding, and anemia. The chronological order of the cellular loss is lymphocytes, granulocytes, and platelets, followed by red blood cells (Conklin et al, 1983).

F) The central nervous system (CNS) syndrome associated with low-level exposure can be manifested as subtle neuropsychological symptoms such as mental cloudiness, listlessness, drowsiness, and lethargy. With higher-level exposure, CNS signs are more obvious: ataxia, psychological and behavioral disturbances, nystagmus, and tremor. Seizures may occur rarely.

G) Other consequences of acute exposure to ionizing **radiation** are dermal erythema (NAS, 1983) and burns (Andrews & Cloutier, 1965). Epilation (hair loss) generally occurs about two weeks after acute exposure when the skin dose exceeds 300 RADs (Andrews & Cloutier, 1965).

H) Partial body exposure to high doses of beta- and gamma-**radiation** can produce delayed skin lesions (bruises, ulcers, and scars) as occurred in victims of the Chernobyl incident (Peter et al, 1994).

I) Brains of mice irradiated with either mid-brain or whole-body doses of at least 7 Gy produced dose-dependent increases of tumor necrosis factor-alpha, interleucin-1 beta, intercellular adhesion molecule-1, alpha

1-antichymotrypsin, interleukin-1 alpha and glial fibrillary acidic protein. These responses were suppressed by dexamethasone or pentoxifylline (Hong et al, 1995).

4.0 EFFECTS OF CHRONIC EXPOSURE

A) The major difference between survivors of atomic bomb explosions and persons occupationally exposed to ionizing **radiation** is that atomic bomb survivors received a large acute **radiation** dose, while occupational exposures generally involve small, chronic **radiation** doses. Chronic exposure to ionizing **radiation** results in an increased cancer risk without the risk of acute **radiation** syndrome.

B) An increased risk of developing lung cancer and other lung diseases has been confirmed in uranium miners whose main exposure to ionizing **radiation** is radon daughters (Brandom, 1978). Workers in the Florida phosphate industry have also been followed in retrospective mortality studies. In one study conducted by NIOSH of virtually all workers at a phosphate fertilizer plant in Polk County, Florida, an excess of lung cancers in nonwhite males was seen upon stratification into subgroups by duration of employment (p 25). The investigators concluded that these results were only suggestive.

C) A retrospective mortality study was carried out amongst all employees of the Florida phosphate industry (Checkoway, 1985). This study, which followed 22,323 workers from 1949 to 1978, found a small excess standard mortality ratio (SMR) for lung cancer when compared to the national average, but not when compared to the higher average for Florida. No trends were seen with duration of employment.

D) Occupational exposure to low doses of ionizing **radiation** has been associated with damage to the microcirculation, as revealed by capillary microscopy of the fingernail-fold (Tomei et al, 1996).

5.0 CARCINOGENIC EFFECTS

HUMAN, SUMMARY

HUMAN, STUDIES

5.2 HUMAN, SUMMARY

A) Ionizing **radiation** has carcinogenic effects in many tissues.

B) Acute ionizing **radiation** exposure survivors have increased long-term cancer risks. A dose-response relationship exists between exposure to ionizing **radiation** and the risk for the subsequent development of cancer.

5.3 HUMAN, STUDIES

A) ATOMIC BOMB SURVIVORS

1) GENERAL

a) Survivors of acute exposures have long-term risks, as seen in Japanese atomic bomb survivors. There is a dose-response relationship between exposure to ionizing **radiation** and risk for cancer. **Radiation**-induced tumors among atomic bomb detonation survivors include acute leukemia, thyroid cancer, breast cancer, lung cancer, gastric cancer, colon cancer and skin cancer (Shintani et al, 1997). The major toxicity of low- and moderate-dose ionizing **radiation** is cancer induction (Schneider & Burkart, 1998; Broerse & Dennis, 1990; Chau, 1987). There is a significantly reduced risk of developing cancer with increasing age at the time of exposure (Little et al, 1998).

b) All atomic bomb-related cancers have had a **radiation** dose dependence, but the shape of the dose-response curves differs with different cancers, suggesting that the mechanism(s) of **radiation** induced cancers is complex. Although dosimetry is still being refined, in general the risk of developing cancer was significant if the dose was 100 rads or greater. The first 30 years of follow-up of the Japanese atomic bomb survivors are published in Okada et al (1975).

c) LEUKEMIA

1) LEUKEMIAS were the first evident cancers in atomic bomb survivors, with incidence peaking 7 to 8 years after exposure. The incidence of both acute and chronic leukemias was elevated, except for the notable absence of chronic lymphocytic leukemia (Little, 1993). The peak onset for acute leukemias was age-dependent, with incidence reaching a maximum at an age corresponding to approximately 1.5 times the age of the subject at the time of exposure. The time of onset for chronic leukemias did not have such a striking age effect; the difference in time of onset for different age groups was only about 3 years. The incidence of all leukemias has subsided with time, but it is not

clear if the risk for leukemia has declined to background values. With the decline of leukemias, the onset of other types of cancer has become apparent in atomic bomb survivors. To date there is a clearly increased risk for cancers of the THYROID, BREAST (female), and LUNG. Stomach cancer and cancers of the salivary gland are suspected but not yet confirmed. In contrast to the leukemias, the time of onset for breast cancer has not been earlier than expected. Rather, breast cancer has appeared at a higher, dose-related frequency at the ages when it usually occurs.
All these cancers have had a **radiation** dose dependence, but the shape of the dose-response curves differs for different cancers, suggesting that the mechanism(s) of **radiation**-induced cancers is complex and perhaps different for different cancers. Although the dosimetry is still being refined, in general the risk for cancer was significant if the dose was 1 Gy or greater. The first 30 years of follow-up of this population are published in Okada et al (1975).

2) Deaths from cancer in relation to **radiation** exposure have been analyzed in the UK National Registry for **Radiation** Workers (Kendall et al, 1992). Significant elevation was seen for leukemias (excluding chronic lymphatic). Resulting lifetime risk was 10.0 percent per Sv for all cancers, and 0.76 percent per Sv for leukemia (excluding chronic lymphatic). These risks are somewhat higher than those proposed by the International Commission on Radiological Protection. There was no association seen with prostate cancer.
3) Draper et al (1997) concluded that the hypothesis of paternal preconception irradiation being a cause of childhood leukemia and non-Hodgkins lymphoma was NOT supported based on a case control study of 35,949 children diagnosed with cancer together with matched controls. Busby & Cato (1998) refute the above findings, stating that exposure to internal radioisotopes may be responsible for some cancers, which was not taken into consideration. Alexander (1998) also refutes the above findings, stating that effects of population mixing may be diluted in the workers described in the study.

d) MENINGIOMA

1) MENINGIOMA - Shintani et al (1997) reported a dose-response effect in atomic bomb detonation survivors with meningioma. Incidence of meningioma increased between 1975 and 1994 in survivors of the Hiroshima atomic bomb detonation (Shintani et al, 1997).

e) MUCOEPIDERMOID TUMORS

1) In the Life Span Study cohort of atomic bomb detonation survivors followed by the **Radiation** Effects Research Foundation, 145 tumors of the salivary glands were identified. Frequency of mucoepidermoid tumors was disproportionately high at higher **radiation** doses (p=0.04). Frequency of Warthin's tumor increased with increasing **radiation** dose (p=0.06). A causal role is suggested for ionizing **radiation** in salivary gland tumorigenesis (Saku et al, 1997).

B) OCCUPATIONAL EXPOSURES

1) Doody et al (1995) conducted a case-control study of breast cancer and employment practices among female radiologic technologists (over 105,000 female medical **radiation** workers). Breast cancer cases (n=528) were matched to approximately 5 control subjects each (n=2628). No significant increase in breast cancer with occupational ionizing **radiation** exposure was found when compared to controls.
2) A study of 95,673 nuclear industry workers in the USA, UK, and Canada found no association between **radiation** dose and all causes of cancer deaths (Cardis et al, 1995).

a) Less than 15 percent of this cohort were women and their mean cumulative **radiation** dose was 7-1/2 times less than that of men.

b) Mortality from multiple myeloma and leukemias (except chronic lymphocytic leukemia) was significantly related to **radiation** doses. Of 119 leukemia deaths in this cohort, 6 were in workers with cumulative exposures in the 400 millisievert range.

c) In these workers, there was a significantly increased risk of developing leukemia at relatively low ionizing **radiation** doses and a dose-related increased multiple myeloma mortality (Cardis et al, 1995).

3) In a mortality study of 15,727 white male workers at the US Los Alamos National Laboratory hired between 1943 and 1977, statistically positive dose-related trends were found for development of Hodgkin's disease, malignant brain tumors, and esophageal cancers (Wiggs et al, 1994). The brain tumors reported as the cause of death may have been metastatic rather than primary tumors (Wiggs et al, 1995).
4) In a cancer mortality study of 8997 male employees of Atomic Energy of Canada, workers exposed to external low-linear-energy transfer ionizing **radiation** had a positive dose-related association between exposure and death from leukemia (although this was based on only 4 deaths) (Gribbin et al, 1993).
5) No significant association was found between development of lung cancer and ionizing **radiation**

in a cohort of 5657 workers of the former Spanish Nuclear Energy Board (Junta de Energia Nuclear) in a retrospective cohort study carried out between 1954 and 1992 (Rodriquez Artalejo et al, 1997).

a) There was excess mortality due to malignant brain tumors in this cohort (6 observed cases).

6) In a nested case-control study of US Airforce personnel, there was no association between development of brain tumors and ionizing **radiation** exposure (Grayson, 1996).

7) In a health survey of 79,016 female certified radiologic technologists, employment in this profession was not found to increase the risk of developing breast cancer (Boice et al, 1995).

8) A cohort analysis was conducted to evaluate the incidence of mortality following occupational exposure to uranium and vanadium, and involved 1484 uranium mill workers employed in one of seven uranium mills for at least one year on or after January 1, 1940. The analysis showed that mortality from all malignant neoplasms was less than expected, although there were non-significant increases in mortality from trachea, bronchus, and lung cancer, and lymphatic and hematopoietic malignancies (primarily lymphosarcoma, reticulosarcoma, and Hodgkin's disease). Overall, mortality from all cancers was highest among those workers with the shortest duration of employment and lowest among those with the longest duration of employment. However, firm conclusions regarding the association between occupational exposure from uranium mills and the incidence of mortality from various cancers cannot be established due to limitations of the analysis including the small cohort size, limited power to detect a moderately increased risk for some outcomes of interest, the inability to estimate individual exposures, and the lack of smoking data (Pinkerton et al, 2004)

C) COMMUNITY BASED EXPOSURES

1) Increased occurrence of childhood and adult thyroid cancer has been documented with a 4 to 5 year latency in Belarus, the Ukraine, and the USA following releases of I-131 from the Chernobyl disaster, distant US nuclear weapons plants, US atmospheric atomic weapons detonations, and a release from the Millstone nuclear power plant in the USA (Mangano, 1996; Hamilton et al, 1987).

a) In a cohort of 2473 persons potentially exposed to fallout from US nuclear weapons testing, a statistically significant excess of thyroid neoplasms (both benign and malignant) was found, although only 19 persons developed these tumors (Kerber et al, 1993).

2) A community-based health survey, from 1944 to 1995 and involving 801 individuals who had lived downwind of a U.S. plutonium production facility located in Hanford, Washington, was conducted in order to determine the type and incidence of cancers that occurred in the community during that time period. Of the 801 residents downwind from the plutonium plant ("downwinders"), 294 residents (36.7%) reported at least one type of cancer as compared to 43 of 423 individuals (10.2%) in a control group of patients from a medical practice in Portland, Oregon. The most commonly occurring cancers among the downwinders included breast cancer (n=53, 10.2%), thyroid cancer (n=33, 4.1%), colon cancer (n=30, 3.7%), and CNS neoplasms (n=20, 2.5%). Comparison of the incidence of thyroid cancer within this population with other populations exposed to radioactive fallout showed the incidence rates from other populations were considerably lower than those for the downwinders. The crude incidence rate (cancers per 100,000 persons per year) for the downwinders study population was 82.4% (n= 33, total study population = 801) as compared to 8% for the Chernobyl population of 4 Russian regions (n=3,004, total study population = 3,113,000). It is speculated that the increased incidence rates for the downwinders may be associated with continuous environmental contamination of radioactive iodine as well as a longer follow-up period (50 years) as compared to the population involved with the Chernobyl accident (12 years) (Grossman et al, 2003).

3) Possible excesses of childhood cancers have been reported in populations living near nuclear installations in Britain, particularly in Sellafield, Seascale, Dounreay, Aldermaston, Burghfield, and Harwell (Gardner, 1991; Wakeford, 1995). These associations have been reviewed from historical and analytical perspectives and an association between paternal preconception exposure and childhood leukemia was only found at Seascale (Wakeford, 1995). Some in-depth reviews conclude that childhood and adult cancer rates are NOT increased in populations living near normally-operating nuclear plants (Boice & Lubin, 1997; Wakeford & Berry, 1996).

a) Paternal preconception exposure to internal or external ionizing **radiation** was NOT an important risk factor for childhood cancers in children whose fathers were employed as radiologists, surgeons, veterinarians, dental surgeons, or industrial radiographers (Sorahan & Roberts, 1993; Sorahan et al, 1995; Wakeford & Berry, 1996).

D) RISK TO OFFSPRING

1) Paternal exposure to ionizing **radiation** may be associated with an increased risk of cancer in the offspring. Development of leukemia in British children in the Sellafield area has been associated with paternal exposure to whole-body penetrating ionizing **radiation** (Gardner, 1991).

2) Besides being associated with an increased risk for mental defects, pre-natal exposure to ionizing **radiation** also increases the risk of childhood cancer, primarily in the first 10 years of life (Mole, 1987).

E) PANCREATIC CANCER

1) Ionizing **radiation** was identified as a risk factor for pancreatic cancer in a nationwide case control study in Finland (Kauppinen et al, 1995).

2) In a retrospective study, an increased risk of basal cell carcinoma (but not squamous cell carcinoma) was associated with prior therapeutic **radiation** (Karagas et al, 1996).

F) LEUKEMIA

1) In a cohort study of over 46,000 children of nuclear industry employees, fewer than 3 leukemias could potentially be attributed to offspring of male employees who had accumulated a preconceptual dose of > 100 mSv. No significant trends were discovered between increasing **radiation** dose and leukemia. Findings suggested that the incidence of cancer and leukemia among children of nuclear industry workers is similar to that in the general population (Roman et al, 1999).

2) In a population-based cohort study of 3877 commercial jet cockpit crew, crew members flying over 5000 hours were reported to have significantly increased frequency of acute myeloid leukemia (5.1 times that expected). Increased risk of melanoma (2.4 times that expected) was also found among crew flying more than 5000 hours (Gundestrup & Storm, 1999).

3) A study of US radiology technologists found the relative risks for non-chronic lymphocytic leukemia was increased 6.6-fold for those working 5 or more years before 1950 and 2.6-fold for those holding patients 50 or more times for x-ray examinations. Working as a radiology technologist was not associated with the risk of non-Hodgkin's lymphoma, Hodgkin's lymphoma, chronic lymphocytic leukemia, or multiple myeloma (Linet et al, 2005).

4) In a study population of 2,558 uranium miners, using a stratified case-cohort sampling design, a relative risk of 1.75 (95% CI 1.10 to 2.78) was reported for all leukemias in workers with high radon exposure (110 working level months (WLM); 80th percentile) compared with low radon exposure (3 WLM; 20th percentile) and 1.98 (95% CI 1.10 to 3.59) for chronic lymphocytic leukemia. The relative risks for myeloid leukemia and Hodgkin lymphoma were elevated but not statistically significant; multiple myeloma and non-Hodgkin lymphoma were not associated with radon (Rericha et al, 2006).

G) BREAST CANCER

1) A dose-dependent increased risk for breast cancer was seen in relation to exposure to low-linear energy transfer ionizing **radiation** in a large cohort of 31,917 Canadian women exposed to fluoroscopy during treatment for tuberculosis between 1930 and 1952. The results were consistent with those of the Japanese atomic bomb survivors (Howe & McLaughlin, 1996).

2) MALE BREAST CANCER - The incidence of breast cancer was studied in 45,880 male atomic bomb survivors diagnosed between January 1, 1958 and December 31, 1998. Nine exposed patients were diagnosed with male breast cancer as compared with 3 non-exposed individuals diagnosed with male breast cancer, indicating a statistically significant dose-response relationship reported between exposure to ionizing **radiation** and the development of male breast cancer (Ron et al, 2005).

H) CANCER RISK

1) In a multinational retrospective study of 407,391 workers monitored for external **radiation** relative risk per sievert for all cancers excluding leukemia was 0.97 (95% CI 0.14 to 1.97). Relative risk for leukemia, excluding chronic lymphocytic leukemia was 1.93 (95% CI <0 to 8.47) and for solid cancers was 0.87 (95% CI 0.03 to 1.88) (Cardis et al, 2005).

I) AGE AT EXPOSURE AND MORTALITY

1) A study of age at exposure and cancer mortality was conducted in workers at the United States Department of Energy Hanford Site. There was little association between mortality and cumulative doses of ionizing **radiation** accrued at ages 15 to 34, 35 to 44, and 45 to 54. For cumulative doses accrued at 55 years and older (10 year lag), the estimated excess relative risk per sievert was 9.05 (90% CI 2.96 to 17.92) for lung cancer and 3.24 (90% CI 0.80 to 6.17) for all cancers (Wing & Richardson, 2005).

J) PLUTONIUM-RELATED MORTALITY

1) At the United States Department of Energy Hanford Site, the relationship between length of exposure to plutonium and death rates from cancer was studied. For workers 50 years and older, death rate increase per year was 2.6 +/- 2.0% for all cancer, 4.9 +/- 3.3% for cancers of tissues where plutonium deposits, 7.1 +/- 3.4% for lung cancer, and 5.9 +/- 4.8% for digestive cancer (Wing et al, 2004).

6.0 GENETIC EFFECTS

SUMMARY

MUTAGENICITY

CHROMOSOME ABBERATIONS

6.1 SUMMARY

A) Ionizing **radiation** is genotoxic and causes breaks in the structure of DNA, resulting in mutations or chromosomal structural aberrations. Double strand breaks in the mutagenic and carcinogenic effects of have been reported. Incorrectly rejoined break leads to DNA misrepair which in turn leads to DNA deletions and rearrangements. Large scale changes in DNA structure appear to be typical of most **radiation**

B) Chromosomal translocations in persons who lived in houses (up to 16 years) in Taiwan contaminated with cobalt-60 has been reported. Compared to controls (no exposure to cobalt-60), the overall translocation yield in the residents was 5 times higher. Chromosomes 2, 4 and 12 were affected in 500 metaphases per person. The FISH method for reciprocal chromosomal translocations was used (Chen et al, 2000).

6.3 MUTAGENICITY

A) Japanese atomic bomb survivors have been followed for possible heritable effects from acute ionizing **radiation** exposure. Even in this population, no clearly demonstrable induced heritable defects have been found (Otake & Schull, 1984). No significant differences in mutation rates in DNA repetitive sequences were found in children of atomic bomb survivors whose parents received a mean gonadal dose of 1.9 Sv, in comparison with unexposed controls (Satoh et al, 1996).

B) Workers exposed to low levels of ionizing **radiation** had increased frequencies of hprt-mutated lymphocytes and changed CD4/CD8 lymphocyte subset ratios (Siefert et al, 1993). A 4.6-fold increase in hprt mutations in blood cells was seen in Brazilian children exposed to 15 to 70 cGy during a radiological accident (Saddi et al, 1996). A doubling dose of 173 (+/- 47) cGy was seen for inducing hprt mutation and micronuclei in victims of a Cs-137 radiological accident in Goiania, Brazil (Dacruz et al, 1997).

C) Persons living near a uranium processing site did not have increased frequencies of mutated somatic cells, as measured by hprt mutations, loss of glycophorin A alleles, or micronuclei (Wones et al, 1995).

D) Increased glycophorin A mutations were seen in former Australian uranium miners 30 years after last exposure (Shanahan et al, 1996).

E) Human cells containing mutant p53 proteins did not have delayed cell replication after irradiation; this is consistent with the occurrence of mutated p53 proteins in some cancers (Zolzer et al, 1995). In related studies, cells from patients with ataxia telangiectasia (AT) had a reduced or delayed increase in p53 protein after gamma-irradiation (Birrell & Ramsay, 1995). Cells from persons heterozygous for AT had an intermediate response. Cells from most breast cancer patients were essentially normal in their response, but 18 percent of the patients responded in the range of AT heterozygotes. This test of p53 induction may be useful in identifying persons at increased risk of DNA-damaging effects of ionizing **radiation** (Birrell & Ramsay, 1995). AT is a heritable disease characterized by increased **radiation** sensitivity and risk for cancer.

F) In limited studies, the serum of persons exposed to ionizing **radiation** contains clastogenic factors, which have persisted for over 30 years in A-bomb survivors. Such factors have been found in the serum of 33 of 47 recovery workers from the Chernobyl incident, in dose-related levels (Emerit et al, 1995).

6.4 CHROMOSOME ABBERATIONS

A) Hospital workers exposed to low levels of ionizing **radiation** had 13 and 11 times greater frequencies of chromosomal aberrations in peripheral lymphocytes as compared to unexposed controls (Paz-y-Mino et al, 1995).

1) Workers were exposed to mean x-ray doses of 1.84 millisieverts/year and 1.67 millisieverts/year for 3 to 20 years.

2) These workers had a higher frequency of chromosomal gaps and breaks, endoreduplications, hyperdiploidies, and chromosomal loss.

B) Nuclear medicine and radiology hospital workers had a mean group frequency of chromosomal aberrations (chromosomal gaps and breaks) in peripheral lymphocytes significantly higher than that of unexposed controls (Hagelstrom et al, 1995).

C) The frequency of chromosomal aberrations in the peripheral lymphocytes of hospital radiodiagnostic, radiotherapy, and nuclear medicine employees was greater than in controls (Barquinero et al, 1993). There were no significant differences between exposed and control groups in the frequency of chromatid gaps and breaks, while significant differences were noted for acentric fragments with or without chromosomal gaps and breaks and

total structural aberrations (Barquinero et al, 1993).
D) The was a statistically significant increased total aberration frequency in peripheral lymphocytes in a small group of civilian aircrew members as compared to controls (Romano et al, 1997). Aircrew members are presumed to have increased exposure to cosmic **radiation** than the general public because of more time spent at high altitudes during flight (Zwingmann et al, 1998) Okansen, 1998; (Friedberg et al, 1989).
E) Two years after total-body or total-body plus partial-body exposure to gamma **radiation**
Estonia, 5 persons had a stable level of translocations present in peripheral blood lymphocytes (Lindholm et al, 1998).
F) In 100 medical workers exposed to x-rays, there was no time-dependent recovery of chromosomal aberrations in peripheral blood lymphocytes (Kasuba et al, 1998).
G) Children exposed to low doses of ionizing **radiation** from the Chernobyl disaster had more acentric fragments in peripheral blood lymphocytes than did control subjects, but there were no significant differences in chromosome or chromatid breaks (Grollino et al, 1998).
H) Chromosome aberrations in Norwegian reindeer following the Chernobyl accident (radiocesium exposure) appeared to affect mainly calves during the immediate post-accident period in the highest
(Roed & Jacobsen, 1995).
I) Increased chromosomal aberrations, especially acentric fragments, were found in lymphocytes from hospital workers exposed to low doses of ionizing **radiation** (1.6 to 42.71 mSv). No dose-effect relationship was seen (Barquinero et al, 1993). In a group of 47 children exposed to **radiation** in the Chernobyl incident, low frequencies of chromosome aberrations were evident several years later (Padovani et al, 1993).

7.0 REPRODUCTIVE EFFECTS

7.1 SUMMARY

A) In addition to an increased risk of cancer, exposure to ionizing **radiation** is known to affect human reproduction.
B) Prenatal ionizing **radiation** exposure may cause congenital anomalies, mental retardation, and an increased incidence of seizures.

7.2 TERATOGENICITY

A) CONGENITAL ANOMALY

1) Four major effects of ionizing **radiation** on the fetus include: growth retardation; severe congenital malformations (including errors of metabolism); embryonic, fetal, or neonatal death; and carcinogenesis. The most pronounced permanent growth retardation occurs following irradiation in the fetal period (Jarrett, 1999).

a) When the fetus is irradiated during organogenesis, the peak incidence of teratogenesis occurs. In humans, **radiation**-induced malformations of bodily structures other than the CNS are uncommon. Reports on atomic bomb survivors indicate that microcephaly may result from a free-in-air dose of 100 to 190 milliGrays.

2) Prenatal exposure to ionizing **radiation** is well known to induce birth defects in humans, as documented in the children of pregnant atomic bomb detonation survivors (Otake & Schull, 1998; Brent, 1989). However, nuclear power industry workers exposed to low-levels of ionizing **radiation** do not appear to have an increased risk of having a liveborn child with a congenital anomaly (Green et al, 1997).

a) Exposures greater than 2 Gy can cause microcephaly and severe mental retardation. The critical dose and period of exposure for microcephaly is at least 0.10 to 0.19 Gy at 4 to 17 weeks, and for mental retardation is at least 0.2 to 0.4 Gy at 8 to 15 weeks (Miller, 1990). Other kinds of effects described in the

literature are retarded growth, pre- and post-natal death, structural malformations, and functional impairment.

b) The developing fetus is most vulnerable to ionizing **radiation** at 8 to 15 weeks postconception (Ikenoue et al, 1993; Otake & Schull, 1998). There is a second period of somewhat reduced vulnerability at 18 to 27 weeks of gestation (Ikenoue et al, 1993).

3) Two children conceived while their mothers were undergoing I-131 therapy for thyroid cancer were born with fatal birth defects (Smith et al, 1994).

4) An increased prevalence of Down's syndrome (trisomy 21) has been suggested but not confirmed to be associated with periods of increased environmental ionizing **radiation** (Bound et al, 1995; Verger, 1997).

a) A cluster of DOWN'S SYNDROME (trisomy 21) cases was seen in the Lothian region of Scotland in 1987, temporally associated with the Chernobyl incident in April, 1986. This was unlikely to have been due to chance, but could not be readily explained by the documented low **radiation** region (Ramsay et al, 1991). A significant increase in cases of Down's syndrome was noted in Germany after the Chernobyl disaster, with the highest rates in the most contaminated regions (Sperling et al, 1991).

5) Maternal mediated neonatal and developmental toxicity resulted in mouse pups after maternal intake of cesium in drinking water (Messiha, 1994).

6) LOW-LEVEL IONIZING **RADIATION** - Offspring of men and women occupationally exposed to low-level ionizing **radiation** were studied to determine any increased risk of congenital malformations. No evidence of a link between exposure before conception and increased risk of adverse reproductive outcome was noted in men (n=11697) or women (n=1903) (Doyle et al, 2000).

7) The mechanism of neurological defects may involve **radiation**-induced cell death.

B) MENTAL DEFICIENCY

1) Exposures in the range of 0.01 to 0.1 Gy may produce mental retardation and increase the risk for childhood cancers. No gross malformations seem to occur at exposures less than about 0.05 Gy (REPROTOX , 1999). The developing central nervous system may be the most sensitive target for the effects of ionizing **radiation**, with the critical period being between the 10th and 17th weeks of pregnancy (Mole, 1985; Cockerham & Prell, 1989).

2) Studies of the offspring of pregnant atomic bomb survivors have found mental retardation at exposures of less than 0.05 Gy, with NO APPARENT THRESHOLD (Otake & Schull, 1998). The probability of mental retardation in this population was 40 percent per gray of fetal tissue dose (Otake & Schull, 1984). Expressed another way, there was a reduction of 21 to 29 IQ points per Gy of exposure (Miller, 1990). The frequency of mental retardation increased from a background of 0.8 to 46 percent with prenatal exposure to 1 Gray (Gy) or greater in children of atom bomb detonation survivors (Ikenoue et al, 1993). Special note should be made of the fact that these were acute exposures, which in general have more severe biological effects than equivalent doses delivered over a longer period of time.

a) In a cohort of 888 children whose mothers were exposed to ionizing **radiation** detonations, those who were exposed at 8 to 15 weeks postovulation had significantly worse scores on repetitive action tests. Those exposed at 0 to 7 weeks postovulation had decreased IQ's (Yoshimaru et al, 1995). These effects were not seen in children whose mothers were exposed at weeks 16 to 25 postovulation.

b) Severe mental retardation has been described in children exposed in utero to ionizing atomic bomb detonations at gestation ages 8 to 15 weeks (Mole, 1990).

C) ANIMAL STUDIES

1) Many similar effects in the offspring have been produced in laboratory animals, such as low birth weight and behavioral changes including hyperactivity (Norton, 1986). In experimental animal studies, induction of structural malformations has been seen, but these are lacking in exposed humans (Mole, 1987).

2) Fetal Swiss albino mice had different effects from a single 0.5 Gy dose of gamma different times of gestation. Dosing during the preimplantation period increased prenatal mortality. Exposure between days 2 and 4 produced increased resorptions. Dosing between days 9 and 13 resulted in small heads, low brain weight, and microphthalmia (Devi & Baskar, 1996).

3) Prenatal exposure to ionizing **radiation** produced an increased risk of cancer and reproductive defects in mice. Female mice exposed to 1.0 and 2.7 Gy of Cf(252) and Co(60) in utero had an increased incidence of tumors of the pituitary gland, mammary gland, liver, and lung for up to two years; as well as dysfunctional ovaries (Nitta et al, 1992).

7.3 PREGNANCY EFFECTS

A) SPONTANEOUS ABORTION

1) Increased incidences of spontaneous abortions and toxicosis of pregnancy have been seen following maternal **radiation** exposure, especially in women affected by the Chernobyl incident (Lieberman et al, 1990).

B) OTHER

1) Commercial and military flightcrew members have exposure to cosmic **radiation** general public, which may be of concern during pregnancy (Geeze, 1998).

C) LACK OF EFFECT

1) Use of video display terminals by pregnant women does not appear to represent a significant source of **radiation** to the fetus. Available data do not support a teratogenic risk. As field strength decreases significantly with distance, it may be prudent for pregnant women to sit at least 50 cm from the screen (Paul, 1993).

2) The reproductive risk from therapeutic doses of I-131 are low: in 3 studies there were no excess malformations, stillbirths, or early deaths (Dottorini, 1996).

7.4 BREAST FEEDING

A) BREAST MILK

1) Cesium has been shown to penetrate the human placenta and breast milk in mothers following exposures (Messiha, 1994).

7.5 FERTILITY EFFECTS

SUMMARY

FEMALE

MALE

7.5.1 SUMMARY

A) STILLBIRTH

1) Parker et al (1999) reported stillbirths among offspring of male **radiation** preconceptional irradiation) at a nuclear reprocessing plant. A point estimate range of 0 to 31.9 (95% confidence limits) stillbirths (n=130) was calculated. These estimates are stated to be qualitatively consistent with animal models. However, Selby (2000) quantitatively compares animal studies to the **radiation** workers at the nuclear reprocessing plant and suggests a point estimate of 0.1 stillbirth caused by paternal preconceptional irradiation.

2) Most occupational standards for exposure to ionizing **radiation** preceded publication of findings linking low-level exposure to more subtle effects in the unborn. The National Commission for Protection (NCRP) has recommended a cumulative fetal dose not to exceed 0.5 rad (NCRP, 1977). Using a weighting factor of 0.25 for the gonads (ILO, 1983), this would correspond to an annual whole-body dose of 2.67 rems of gamma-**radiation**.

7.5.2 FEMALE

A) HUMANS

1) Female reproduction can be affected by ionizing **radiation**, which alters the viability of ova and can disrupt the function of the endocrine system which produces female sex hormones. Human oocytes may be more resistant to ionizing **radiation** than those of laboratory animals.

7.5.3 MALE

A) HUMANS

1) At exposures less than 5 to 10 rads, effects on male reproduction are not well understood. X-rays at doses as low as 15 rads can temporarily depress sperm production. At doses of 50 rads, there is temporary elimination of sperm production, and at doses of 236 to 365 rads, damage to sperm production can persist for months. Exposure to greater than 400 rads can cause complete and permanent damage to sperm production.

2) Of twelve men with chronic **radiation** dermatitis after the Chernobyl incident, all had impaired sexual function including impotence, aspermia, azoospermia, abnormal sperm shapes, decreased plasma testosterone levels, increased follicle stimulating hormone (FSH) levels, and decreased luteinizing hormone (LH) levels (Birioukov et al, 1993).

8.0 PREDISPOSING CONDITIONS

A) GENETIC

1) Eight complementation groups of human DNA repair genes which define sensitivity to ionizing been identified on chromosomes 2, 5, 7, 8, 14, 19, and 22 (Thompson & Jeggo, 1995).

2) **Radiation** sensitivity in 20 human carcinoma cell lines correlated with allelic status of the p53 tumor suppressor gene, with the wild-type allele conferring **radiation** resistance (Servomaa et al, 1996).

9.0 BIOMONITORING

9.2 MEDICAL SURVEILLANCE

A) Cytogenetic monitoring may be a sensitive means of monitoring groups of persons exposed to ionizing **radiation**. Certain chromosomal aberrations, such as dicentrics, exchanges, and rings, are sensitive markers for exposure to ionizing **radiation**. However, there seems to be no relationship between the frequency of abnormal chromosomes and risk for cancer in a given individual (Okada, 1975).

B) Chromosome painting (fluorescence hybridization with DNA probes specific for certain chromosomes) was shown to be valid for detecting chromosome exchanges in human lymphocytes exposed to ionizing vitro; chromosomes containing exchanged material appear bi-colored (Tucker et al, 1993).

C) Of established methods, induction of HPRT (hypoxanthine-guanine phosphoribosyl transferase) mutations in lymphocytes correlates best with recent ionizing **radiation** exposure, and loss of glycophorin A variants from red blood cell membranes appears to indicate cumulative **radiation** exposure (Mendelsohn, 1990).

D) Increased frequencies of HPRT-mutated lymphocytes and changed CD4/CD8 lymphocyte subset ratios were correlated with dosimeter readings of occupational exposure to low levels of ionizing 1993).

E) HPLC-coupled 32P-postlabeling analysis has been used to quantitate levels of 8-hydroxydeoxyguanosine in leukocyte DNA from persons exposed to therapeutic doses of ionizing **radiation** the level of such adducts, as compared with unexposed individuals (Wilson et al, 1993).

11.0 SUMMARY AND CONCLUSIONS

A) Because of its well-documented effects in increasing cancer risk and in causing acute IONIZING **RADIATION** is in Class 3 (may cause irreversible effects which can be life-threatening) for general toxicity.

B) Because of its known effects on male and female reproduction and on the unborn, ionizing (human reproductive hazard with known no-effect dose) for reproductive hazard.

C) Unnecessary ionizing **radiation** exposure should be avoided.

12.0 REFERENCES

12.2 SPECIFIC REFERENCES

1) Adelstein SJ: Uncertainty and relative risks of **radiation** exposure. JAMA 1987; 258:655-657.

2) American Heart Association: 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2005; 112(24 Suppl):IV 1-203. Available from URL: http://circ.ahajournals.org/content/vol112/24_suppl/. As accessed 12/14/2005.

3) Andrews GA & Cloutier RJ: Accidental acute **radiation** injury. Arch Environ Health 1965; 10:498-507.

4) Anon : Army Field Manual FM 8-10-7. Chapter 2. Nuclear, Biological, and Chemical Weapons Effects. U.S. Army. Washington, DC, USA. 2000. Available from URL: http://www.nbc-med.org/SiteContent/ MedRef/OnlineRef /FieldManuals/fm8_107/chapter2.htm. As accessed Accessed July 12, 2000.

5) Anon: Managing **Radiation** Emergencies. Oak Ridge Associated Universities. Oak Ridge, TN, USA. 2002.

Available from URL: www.orau.gov/reacts/internal/htm. As accessed 01/26/04.
6) Barquinero JF, Barrios L, & Caballin MR: Cytogenetic analysis of lymphocytes from hospital workers occupationally exposed to low levels of ionizing **radiation**. Mutat Res 1993; 286:275-279.
7) Becker DV: Reactor accidents: Public health strategies and their medical implications. JAMA 1987; 258:649-654.
8) Behar A, Cohen-Boulakia F, & Othmani S: Chernobyl three years later: radiobiologic evaluation of a radioactive contamination. J Environ Pathol Toxicol Oncol 1990; 10:281-285.
9) Benitz WE & Tatro DS: The Pediatric Drug Handbook, 3rd ed, Mosby-Year Book Inc, Chicago, IL, 1995.
10) Birioukov A, Meurer M, & Peter RU: Male reproductive system in patients exposed to ionizing irradiation in the chernobyl accident. Arch Androl 1993; 30:99-104.
11) Birrell GW & Ramsay JR: Induction of p53 protein by gamma **radiation** in lymphocyte lines from breast cancer and ataxia tenangiectasia patients. Br J Cancer 1995; 72:1096-1101.
12) Boice JD & Lubin JH: Occupational and environmental **radiation** and cancer. Cancer Causes Control 1997; 8:309-322.
13) Boice JD, Mandel JS, & Doody MM: Breast cancer among radiologic technologists. JAMA 1995; 274:394-401.
14) Bonassi S, Forni A, & Bigatti P: Chromosome aberrations in hospital workers: evidence from surveillance studies in Italy (1963-1993). Am J Ind Med 1997; 31:353-360.
15) Bound JP, Francis BJ, & Harvey PW: Down's syndrome: prevalence and ionising west England 1957-91. J Epidemiol Community Health 1995; 49:164-170.
16) Brandom WF: Radiat Res 1978; 76:159-171.
17) Brenner DJ & Sachs RK: Chromosomal "fingerprints" of prior exposure to densely ionizing Res 1994; 140:134-142.
18) Brent RL: The effects of embryonic and fetal exposure to X-ray, microwaves, and ultrasound: Counseling the pregnant and nonpregnant patient about these risks. Seminars in Oncology 1989; 16:347-368.
19) Broerse JJ & Dennis JA: Dosimetric aspects of exposure of the population to ionizing Radiat Biol 1990; 57:633-645.
20) Broyles AA: The effect of dose rate on **radiation** injury. Health Physics 1989; 56:933-937.
21) CDC: Facts about DTPA. Centers for Disease Control. Atlanta, Ga, USA. 2003a. Available from URL: cdc.gov.**radiationemergencies**. As accessed 01/26/04.
22) CDC: Facts about neupogen. Centers for Disease Control. Atlanta, GA, USA. 2003. Available from URL: www.bt.cdc.gov/**radiation**/neupogenfacts.asp. As accessed 01/05/04.
23) Cardis E, Gilbert ES, & Carpenter L: Effects of low doses and low dose rates of external ionizing Cancer mortality among nuclear industry workers in three countries. Radiat Res 1995; 142:117-132.
24) Cardis E, Vrijheid M, Blettner M, et al: Risk of cancer after low doses of ionising cohort study in 15 countries. BMJ 2005; 331(7508):77-.
25) Chamberlain JM, Altieri MA, & Futterman C: A prospective, randomized study comparing intramuscular midazolam with intravenous diazepam for the treatment of seizures in children. Ped Emerg Care 1997; 13:92-94.
26) Champlin RE, Kastenberg WE, & Gale RP: **Radiation** accidents and nuclear energy: Medical consequences and therapy. Ann Intern Med 1988; 109:730-744.
27) Chau NP: **Radiation** carcinogenesis in humans: Is it necessary to revise exposure dose limits based on recent estimates of lifetime risks?. Health Physics 1987; 52:753-761.
28) Checkoway H: J Occup Med 1985; 27:885-892.
29) Chen FD, Chen KY, & Ngo FQH: Chromosomal damage in long-term residents of houses contaminated with cobalt-60 (letter). Lancet 2000; 355:726.
30) Christensen J: Two reports warn of radioactive bullets. High Country Times 1993; 25:4.
31) Cockerham LG & Prell GD: Prenatal **radiation** risk to the brain. Neurotoxicol 1989; 10:467-474.
32) Conde-Salazar L, Guimaraens D, & Romero LV: Occupational radiodermatitis from Ir(192) exposure. Contact Dermatitis 1986; 15:202-204.
33) Conklin JJ, Walker RI, & Hirsch EF: Current concepts in the management of trauma. Surg Gynecol Obstet 1983; 156:809-829.
34) Correia MP, Santos D, & Jorge M: Radiotherapy-induced pemphigus (Portugese). ACTA Med Port 1998; 11:581-583.
35) Cronkite EP & Wallace JF: **Radiation** and electrical injuries, in: Isselbacher KJ, Adams RD, Braunwald E et al (Eds), Harrison's Principles of Internal Medicine, 8th ed, McGraw Hill, New York, NY, 1977, pp 748-752.
36) Curnes JT, Laster DW, & Ball MR: Magnetic resonance imaging of **radiation** Neuroradiol 1986; 7:389-394.
37) Dacruz AD, Volpe JP, & Saddi V: **Radiation** risk estimation in human populations -- lessons from the radiological accident in Brazil. Mutat Res 1997; 373:207-214.

38) Devi PU & Baskar R: Influence of gestational age at exposure on the prenatal effects of gamma-
Internat J Radiat Biol 1996; 70:45-52.
39) Diffre P: Radionuclides in the environment. J Environ Pathol Toxicol Oncol 1990; 10:276-280.
40) Dottorini ME: Genetic risk assessment after iodine-131 exposure: an opportunity and obligation for nuclear medicine. J Nucl Med 1996; 37:612-615.
41) Doyle P, Maconochie N, & Roman E: Fetal death and congenital malformation in babies born to nuclear industry employees: report from the nuclear industry family study. Lancet 2000; 356:1293-1299.
42) Drum DE & Jankowski CB: **Radiation** injury, in: May HL (Ed), Emergency Medicine, John Wiley & Sons, New York, NY, 1984, pp 491-498.
43) Dunn K, Yoshimaru H, & Otake M: Prenatal exposure to ionizing **radiation**
seizures. Am J Epidemiol 1990; 131:114-123.
44) Edsall K: **Radiation** exposure, North American Congress of Clinical Toxicology, Orlando, FL, 1999.
45) Emerit I, Oganesian N, & Sarkisian T: Clastogenic factors in the plasma of Chernobyl accident recovery workers -- anticlastogenic effect of Ginkgo biloba extract. Radiat Res 1995; 144:198-205.
46) Finch SC: Acute **radiation** syndrome. JAMA 1987; 258:664-667.
47) Finkelstein E: Nifedipine for **radiation** oesophagitis (Letter). Lancet 1986; 1:1205-1206.
48) Friedberg W, Faulkner DN, & Synder L: Galactic cosmic **radiation** exposure and associated health risks for air carrier crewmembers. Aviat Space Environ Med 1989; 1104-1108.
49) Fry RJ, Grosovsky A, & Hanawalt PC: The inpact of biology on risk assessment -- workshop of the National Research Council's Board on **Radiation** Effects Research. July 21-22, 1997, National Academy of Sciences, Washington, DC. Radiat Res 1998; 150:695-705.
50) Gains M: **Radiation** exposure. Practitioner 1989; 233:1631-1634.
51) Gardner MJ: Childhood cancer and nuclear installations. Public Health 1991; 105:277-285.
52) Geeze DS: Pregnancy and in-flight cosmic **radiation**. Aviat Space Environ Med 1998; 69:1061-1064.
53) Gibson PG, Bryant DH, & Morgan GW: **Radiation**-induced lung injury: A hypersensitivity pneumonitis?. Ann Intern Med 1988; 109:288-291.
54) Goldsmith JR, Merkin L, & Quastel MR: Evaluation of the **radiation** exposures of Chernobyl "liquidators": Exploratory studies of a sample of immigrants to Israel. Internat J Occup Environ Health 1997; 3:51-59.
55) Gottlober P, Bezold G, & Weber L: The **radiation** accident in Georgia: clinical appearance and diagnosis of cutaneous **radiation** syndrome. J Am Acad Dermatol 2000; 42:453-458.
56) Grayson JK: **Radiation** exposure, socioeconomic status, and brain tumor risk in the US Air Force: a nested case-control study. Am J Epidemiol 1996; 143:480-486.
57) Green LM, Dodds L, & Miller AB: Risk of congenital anomalies in children of parents occupationally exposed to low level ionsing **radiation**. Occup Environ Med 1997; 54:629-635.
58) Gribbin MA, Weeks JL, & Howe GR: Cancer mortality (1956-1985) among male employees of Atomic Energy of Canada limited with respect to occupational exposure to external low-linear-energy-transfer ionizing
Radiat Res 1993; 133:375-380.
59) Grollino MG, Eleuteri P, & Mark HF: Children exposed to chronic contamination after the Chernobyl accident: cytogenetic and radiotoxicological analyses. Arch Environ Med 1998; 53:344-346.
60) Grossman CM, Nussbaum RH, & Nussbaum FD: Cancers among residents downwind of the Hanford, Washington, plutonium production site. Arch Environ Health 2003; 58:267-274.
61) Gundestrup M & Storm HH: **Radiation**-induced acute myeloid leukaemia and other cancers in commercial jet cockpit crew: a population-based cohort study. Lancet 1999; 354:2029-2031.
62) Hagelstrom AH, Gorla NB, & Larripa IB: Chromosomal damage in workers occupationally exposed to chronic low level ionizing **radiation**. Toxicol Lett 1995; 76:113-117.
63) Hamilton TE, van Belle G, & LoGerfo JP: Thyroid neoplasia in Marshall Islanders exposed to nuclear fallout. JAMA 1987; 258:629-636.
64) Hong JH, Chiang CS, & Campbell IL: Induction of acute phase gene expression by brain irradiation. Internat J Radiat Oncol Biol Phys 1995; 33:619-626.
65) Howe GR & McLaughlin J: Breast cancer mortality between 1950 and 1987 after exposure to fractionated moderate-dose-rate ionizing **radiation** in the Canadian fluoroscopy cohort study and a comparison with breast cancer mortality in the atomic bomb survivors study. Radiat Res 1996; 145:694-707.
66) ICRP: The principles and general procedures for handling emergency and accidental exposure of workers: A report of the Commitee 4 of the International Commission on Radiologic Protection, in
Publication 28. International Commission on Radiologic Protection, 1977.
67) ILO: Encyclopaedia of Occupational Health and Safety, 3rd ed, 1-2, International Labour Organization, Geneva, Switzerland, 1983.
68) Ikenoue T, Ikeda T, & Ibara S: Effects of environmental factors on perinatal outcome: Neurological

development in cases of intrauterine growth retardation and school performance of children perinatally exposed to ionizing **radiation**. Environ Health Perspect 1993; 101:53-57.
69) Imamura N & Kimura A: Neutropenia among survivors of atomic bomb explosion (letter). Lancet 2000; 355:117.
70) Jarrett D: Medical Management of Radiological Casualties Handbook, 1st edition, Armed Forces Radiobiology Research Institute, Bethesda, MD, 1999.
71) Joksic G & Spasojevic-Tisma V: Chromosome analysis of lymphocytes from applying industry. Internat Arch Occup Environ Health 1998; 71:213-220.
72) Karagas MR, McDonald JA, & Greenberg ER: Risk of basal cell and squamous cell skin cancers after ionizing **radiation** therapy. J Natl Cancer Inst 1996; 88:1848-1853.
73) Kasuba V, Rozgaj R, & Sentija K: Chromosomal aberrations in medical staff occupationally exposed to X-rays: a follow-up study. Arh Hig Rada Toksikol 1998; 49:1-8.
74) Katayama S, Shimaoka K, & Osman G: **Radiation**-associated thyrotoxicosis. J Surg Oncol 1986; 33:84-87.
75) Kauppinen T, Partanen T, & Degerth R: Pancreatic cancer and occupational exposures. Epidemiol 1995; 6:498-502.
76) Kendall GM, Muirhead CR, & MacGibbon BH: Mortality and occupational exposure to of the National Registry for **Radiation** Workers. Br Med J 1992; 304:220-225.
77) Kerber RA, Till JE, & Simon SL: A cohort study of thyroid disease in relation to fallout from nuclear weapons testing. JAMA 1993; 270:2076-2082.
78) Khokhryakov VF, Belyacv AP, Kudryavtseva TI, et al: Successful DTPA therapy in the case of 239Pu penetration via injured skin exposed to nitric acid. Rad Protect Dosimetry 2003; 105:499-502.
79) Lahat E, Goldman M, & Barr J: Comparison of intranasal midazolam with intravenous diazepam for treating febrile seizures in children: prospective randomized study. BMJ 2000; 321:83-86.
80) Landtblom A-M, Flodin U, & Karlsson M: Multiple sclerosis and exposure to solvents, ionizing animals. Scand J Work Environ Health 1993; 19:399-404.
81) Leonard RB & Ricks RC: Emergency department **radiation** accident protocol. Ann Emerg Med 1980; 9:462-470.
82) Lieberman AN, Randarenko IG, & Pethkurov AV: Influence of small doses of ionising and result of pregnancies (Russian), in: Polonskaja EJ (Ed), **Radiation** Scientific Research Institute for **Radiation** Hygiene, Leningrad, Russia, 1990, pp 100.
83) Lincoln TA: Importance of initial management of persons internally contaminated with radionuclides. J Am Ind Hyg Assoc 1976; 37:16-21.
84) Lindholm C, Tekkel M, & Veidebaum T: Persistence of translocations after accidental exposure to ionizing **radiation**. Internat J Radiat Biol 1998; 74:565-571.
85) Linet MS, Freedman DM, Mohan AK, et al: Incidence of haematopoietic malignancies in US radiologic technologists. Occup Environ Med 2005; 62(12):861-867.
86) Little JB: Cellular, molecular, and carcinogenic effects of **radiation** 7:337-352.
87) Little MP, De Vathaire F, & Charles MW: Variations with time and age in the risks of solid cancer incidence after **radiation** exposure in childhood. Stat Med 1998; 30:1341-1355.
88) Lloyd DC, Edwards AA, & Fitzsimmons EJ: Death of a classified worker probably caused by overexposure to **radiation**. Occup Environ Med 1994; 51:713-718.
89) Mangano JJ: A post-Chernobyl rise in thyroid cancer in Connecticut, USA. Eur J Cancer Prev 1996; 5:75-81.
90) Manno EM: New management strategies in the treatment of status epilepticus. Mayo Clin Proc 2003; 78(4):508-518.
91) Mendelsohn ML: Health Phys 1990; 59:23-28.
92) Messiha FS: Developmental toxicity of cesium in the mouse (minireview). Gen Pharmac 1994; 25:395-400.
93) Mettler FA Jr, Monsein L, & Davis M: Three-phase radionuclide bone scanning in evaluation of local injury: a case report. Clin Nucl Med 1987; 12:805-808.
94) Miller RW: Heath Phys 1990; 59:57-61.
95) Milroy WC: Management of irradiated and contaminated casualty victims. Emerg Med Clin North Am 1984; 2:667-686.
96) Mole RH: Br J Radiol 1987; 60:17-31.
97) Mole RH: Radiol Protect Bull 1985; 67:18.
98) Mole RH: The effect of prenatal **radiation** exposure on the developing human brain. Interat J Radiat Biol 1990; 57:647-663.
99) Movsas B, Raffin TA, & Epstein AH: Pulmonary **radiation** injury (review). Chest 1997; 111:1061-1076.
100 NATO : NATO/CCMSNACC Pilot Study: Cross-border environmental problems emanating from defence-

related installations and activities. Summary final report. Phase 1 1993-1995; Report No. 206. North Atlantic Treaty Organization from Estonia and NATO. Islandi väljak, Estonia. 1995. Available from URL: http://www.vm.ee /nato/ccms/s00/phase1/final/finalsum.html. As accessed (cited 7/30/98).

101 NCRP: Review of NCRP **Radiation** Dose Limit for Embryo and Fetus in Occupationally Exposed Women, National Council on **Radiation** Protection and Measurements, Washington, DC, 1977.

102 Nagataki S, Shibata Y, & Inoue S: Thyroid diseases among atomic bomb survivors in Nagasaki. JAMA 1994; 272:364-370.

103 Natl Acad Sci: The treatment of **Radiation** Injury: Report of Committee on Pathologic Effects of Atomic **Radiation**, Publication 1134, National Academy of Sciences -- National Research Council, Washington, DC, 1963.

104 Nitta Y, Kamiya K, & Yokoro K: Carcinogenic effect of in utero CF-252 and CO-60 irradiation in C57Bl/6NXC3H/HEf1 (B6C3f1) mice. J Radiat Res 1992; 33:319-333.

105 Norton S: Toxicol Appl Pharmacol 1986; 83:240-249.

106 Okada S: A Review of Thirty Years Study of Hiroshima and Nagasaki Atomic Bomb Survivors. J Radiat Res 1975; 16 (Suppl).

107 Otake & Schull J: Br J Radiol 1984; 57:409.

108 Otake M & Schull WJ: **Radiation**-related brain damage and growth retardation among the prenatally exposed atomic bomb survivors. Internat J Radiat Biol 1998; 74:159-171.

109 Pacini F, Vorontsova T, & Molinaro E: Prevalence of thyroid autoantibodies in children and adolescents from Belarus exposed to the Chernobyl radioactive fallout. Lancet 1998; 352:763-766.

110 Padovani L, Caporossi D, & Tedeschi B: Cytogenetic study in lymphocytes from children exposed to ionizing **radiation** after the Chernobyl accident. Mutat Res 1993; 319:55-60.

111 Paul ME: Physical agents in the workplace. Seminars in Perinatology 1993; 17:5-17.

112 Paz-y-Mino C, Leone PE, & Chavez G: Follow up study of chromosomal aberrations in lymphocytes in hospital workers occupationally exposed to low levels of ionizing **radiation**

113 Peter RU, Braunfalco O, & Birioukov A: Chronic cutaneous damage after accidental exposure to ionizing **radiation**: the Chernobyl experience. J Am Acad Dermatol 1994; 30:719-723.

114 Pinkerton LE, Bloom TF, Hein MJ, et al: Mortality among a cohort of uranium mill workers: an update. Occup Environ Med 2004; 61:57-64.

115 Product Information: Ca-DTPA, Trisodium calcium diethylenetriaminepentaacetate. Distributed by: Oak Ridge Associated Universities (www.orau.gov), Oak Ridge, TN, USA, 2003.

116 Product Information: Radiogardase(TM), Insoluble Prussian blue capsules. Haupt Pharma Berlin GmbH, Berlin, Germany, 2003.

117 Product Information: Zn-DTPA, Trisodium zinc diethylenetriaminepentaacetate. Distributed by: Oak Ridge Associated Universities (www.orau.gov), Oak Ridge , TN, USA, 2003.

118 Product Information: lorazepam injection, lorazepam injection. Bedford Laboratories, Bedford, OH, 2004.

119 REPROTOX : REPROTOX Information System, Scialli AR (Ed), (CD-ROM Version). Georgetown University Medical Center and Reproductive Toxicology Center, Columbia Hospital for Women Medical Center. Washington, DC (Internet Version). Edition expires 1999; provided by Thomson Healthcare Inc., Greenwood Village, CO.

120 Ramsay CN, Ellis PM, & Zealley H: Down's syndrome in the Lothian region of Scotland -- 1978 to 1989. Biomed Pharmacother 1991; 45:267-272.

121 Rericha V, Kulich M, Rericha R, et al: Incidence of leukemia, lymphoma, and multiple myeloma in Czech uranium miners: a case-cohort study. Environ Health Perspect 2006; 114(6):818-822.

122 Roed KH & Jacobsen M: Chromosome aberrations in Norwegian reindeer following the Chernobyl accident. Mutat Res 1995; 346:159-165.

123 Roman E, Doyle P, & Maconochie N: Cancer in children of nuclear industry employees: report on children aged under 25 years from nuclear industry family study. Br Med J 1999; 318:1443-1450.

124 Romano E, Ferrucci L, & Nicolai F: Increase of chromosomal aberrations induced by ionising peripheral blood lymphocytes of civil avaition pilots and crew members. Mutat Res 1997; 377:89-93.

125 Ron E, Ikeda T, Preston DL, et al: Male breast cancer incidence among atomic bomb survivors. J Natl Cancer Inst 2005; 97(8):603-605.

126 Saddi V, Curry J, & Nohturfft A: Increased hprt mutant frequencies in Brazilian children accidentally exposed to ionizing **radiation**. Environ Mol Mutagen 1996; 28:267-275.

127 Saenger EL: **Radiation** accidents. Am J Roentgenol 1960; 84:715-728.

128 Saku T, Hayashi Y, & Takahara O: Salivary gland tumors among atomic bomb survivors, 1950-1987. Cancer 1997; 79:1465-1475.

129 Salovsky P, Shopova V, & Dancheva V: Pneumonotoxic effects of radioactive dust (RD) from a nuclear power plant in Kozloduy, Bulgaria. Am J Ind Med 2000; 38:639-643.

130 Sasaki MS, Takatsuji T, & Ejima Y: The F value cannot be ruled out as a chromosomal fingerprint of

radiation quality. Radiat Res 1998; 150:253-258.
131 Satoh C, Takahashi N, & Asakawa J: Genetic analysis of children of atomic bomb survivors. Environ Health Perspect 1996; 104(Suppl 3):511-519.
132 Schneider G & Burkart W: Health risks of ionizing **radiation**
133 Scott R, Besag FMC, & Neville BGR: Buccal midazolam and rectal diazepam for treatment of prolonged seizures in childhood and adolescence: a randomized trial. Lancet 1999; 353:623-626.
134 Servomaa K, Kiuru A, & Grenman R: p53 mutations associated with increased sensitivity to ionizing **radiation** in human head and neck cancer cell lines. Cell Prolif 1996; 29:219-230.
135 Shanahan EM, Peterson D, & Roxby D: Mutation rates at the glycophorin A and hprt loci in uranium miners exposed to radon progeny. Occup Environ Med 1996; 53:439-444.
136 Shintani T, Hayakawa N, & Kamada N: High incidence of meningioma in survivors of Hiroshima (Letter). Lancet 1997; 349:1369.
137 Smith MB, Xue H, & Takahashi H: Iodine-131 thyroid ablation in female children and adolescents -- long-term risk of infertility and birth defects. Ann Surg Oncol 1994; 1:128-131.
138 Sorahan T & Roberts PJ: Childhood cancer and paternal exposure to ionizing
from the Oxford survey of childhood cancers. Am J Ind Med 1993; 23:343-354.
139 Sorahan T, Lancashire RJ, & Temperton DH: Childhood cancer and paternal exposure to ionizing
A second report from the Oxford survey of childhood cancers. Am J Ind Med 1995; 28:71-78.
140 Sperling K, Pelz J, & Wegner RD: Frequency of trisomy 21 in Germany before and after the Chernobyl accident. Biomed Pharmacother 1991; 45:255-262.
141 Stayner LT et al: NIOSH Report 1184B. National Institute for Occupational Safety and Health, 1983.
142 Streffer C, Muller WU, & Kryscio A: Micronuclei-biological indicator for retrospective dosimetry after exposure to ionizing **radiation**. Mutat Res 1998; 404:101-105.
143 Thompson LH & Jeggo PA: Nomenclature of human genes involved in ionizing
Res 1995; 337:131-134.
144 Tomei F, Papaleo B, & Fantini S: Vascular effects of occupational exposure to low-dose ionizing
Am J Ind Med 1996; 30:72-77.
145 Toohey RE: Internal dose assessment in **radiation** accidents. Rad Protect Dosimetry 2003; 105:329-331.
146 Tucker JD, Ramsey MJ, & Lee DA: Validation of chromosome painting as a biodosimeter in human peripheral lymphocytes following acute exposure to ionizing **radiation**
64:27-37.
147 Tucker JD, Tawn EJ, & Holdsworth D: Biological dosimetry of
facility. Radiat Res 1997; 148:216-226.
148 Vanagunas A, Jacob P, & Olinger E: **Radiation**-induced esophageal injury: A spectrum from esophagitis to cancer. Am J Gastroenterol 1990; 85:808-812.
149 Verger P: Down syndrome and ionizing **radiation**. Health Physics 1997; 73:882-893.
150 Verzijl JM, Joore JCA, & van Dijk A: In vitro binding characteristics for cesium of two qualities of prussian blue, activated charcoal and resonium-a. Clin Toxicol 1992; 30:215-222.
151 Verzijl JM, Lie RL & Savelkoul TJ et al: In-vitro binding characteristics for cesium of two qualities of Prussian blue, activated charcoal, and Resonium-A(R). Personal Communication, 1990.
152 Voelz GL: Current approaches to the management of internally contaminated persons, in: Hubner KF & Fry SA (Eds), The Medical Basis for **Radiation** Accident Preparedness, Elsevier, New York, NY, 1980, pp 311-325.
153 WHO: Radiologic Aspects, in: Guidelines for drinking-water quality, 2nd ed, World Health Organization, Geneva, Switzerland, 1993, pp 114-121.
154 Wagner RH, Boles MA, & Henkin RE: Treatment of **radiation**
1994; 14:387-396.
155 Wakeford R & Berry RJ: Nuclear facilities and cancers. Ann Acad Med Singapore 1996; 25:468-476.
156 Wakeford R: The risk of childhood cancer from intrauterine and preconceptional exposure to ionizing **radiation**. Environ Health Perspect 1995; 103:1018-1025.
157 Weinberg AD, Kripalani S, & McCarthy PL: Caring for survivors of the Chernobyl disaster. What the clinician should know. JAMA 1995; 274:408-412.
158 Wiggs LD, Johnson ER, & Cox-DeVore CA: Mortality through 1990 among white male workers at the Los Alamos National Laboratory: Considering exposures to plutonium and external ionizing
1994; 67:577-588.
159 Wilson VL, Taffe BG, & Shields PG: Detection and quantification of 8-hydroxydeoxyguanosine adducts in peripheral blood of people exposed to ionizing **radiation**. Environ Health Perspect 1993; 99261-99263.
160 Wing S & Richardson DB: Age at exposure to ionising **radiation**
workers: follow up through 1994. Occup Environ Med 2005; 62(7):465-472.

161 Wing S, Richardson D, Wolf S, et al: Plutonium-related work and cause-specific mortality at the United States Department of Energy Hanford Site. Am J Ind Med 2004; 45(2):153-164.
162 Wones R, Radack K, & Martin V: Do persons living near a uranium processing site have evidence of increased somatic cell gene mutations -- a first study. Mutat Res 1995; 335:171-184.
163 Yoshimaru H, Otake M, & Schull WJ: Further observations on abnormal brain development caused by prenatal A-bomb exposure to ionizing **radiation**. Internat J Radiat Biol 1995; 67:359-371.
164 Zolzer F, Hillebrandt S, & Streffer C: **Radiation** induced G(1)-block and p53 status in six human cell lines. Radiother Oncol 1995; 37:20-28.
165 Zwingmann IN, Welle IJ, & van Herwijnen M: Oxidative DNA damage and cytogenetic effects in flight engineers exposed to cosmic **radiation**. Environ Mol Mutagen 1998; 32:121-129.