INTRODUCTION

This is a review of the health effects of electromagnetic waves, including ionizing and nonionizing radiations, accelerated atomic particles, high-intensity ultrasound, and electromagnetic fields. These various forms of energy differ sufficiently from one another in their biological effects so that each is considered separately in the remarks that follow.

IONIZING RADIATION

Historical background. After its discovery by Roentgen, in 1895, the X-ray was introduced so rapidly into the diagnosis and treatment of disease that injuries from excessive radiation exposure began to be encountered almost immediately in pioneer radiation workers, who were unaware of the risks of such effects at the time (Brown, 1936). The first such injuries were predominantly skin reactions on the hands of those working with the early radiation equipment, but within a decade many other types of injury also had been reported, including the first cancers attributed to radiation (Stone, 1959).

Throughout the century since these early findings, study of the biological effects of ionizing radiation has received continuing impetus from the growing uses of radiation in medicine, science, and industry, as well as from the peaceful and military applications of atomic energy. As a result, the biological effects of radiation have been investigated more thoroughly than those of virtually any other environmental agent. The evolving and extensive knowledge of radiation effects has been influential in shaping measures for the protection of human health against many other environmental hazards as well.

Nature, Sources, and Environmental Levels of Ionizing Radiation. Ionizing radiation occurs in the form of: 1) electromagnetic waves of extremely short wavelength (Fig. 1) and 2) accelerated atomic particles (e.g., electrons, protons, neutrons, alpha particles). Because ionizing radiation exerts its biological effects through energetic interactions with atoms and molecules in its path, doses of ionizing radiation are measured in terms of energy deposition (Table 1).

Natural sources of ionizing radiation include: 1) cosmic rays, 2) radium and other radioactive elements in the earth's crust, 3) internally deposited potassium-40, carbon-14, and other radionuclides present normally in living cells, and 4) inhaled radon and its

daughter elements (Table 2). The dose received from cosmic rays can differ appreciably from the value tabulated, depending on one's elevation; i.e., it can be twice as high at a mountainous site (e.g., Denver) as at sea level and up to two orders of magnitude higher at jet aircraft altitudes (NCRP 1987). Likewise, the dose received from radium may be increased by a factor of two or more in regions where the underlying earth is rich in this element (NCRP 1987). It is noteworthy that the largest dose to any part of the body is that which is received by the bronchial epithelium from inhaled radon-222 (Table 2), a colorless, odorless alpha-particle-emitting gas formed by the radioactive decay of radium-226; furthermore, depending on the concentration of radon in indoor air, the dose from radon and its decay daughters may vary by an order of magnitude or more (NCRP 1984). In cigarette smokers, moreover, even larger doses [up to 0.2 Sv (20 rem) per year] are received by the bronchial epithelium from polonium (another alpha-emitting decay product of radium), which is normally present in tobacco smoke (NCRP 1984).

In addition to the exposure to ionizing radiation that is received from natural sources, people are exposed to radiation from artificial sources as well, the largest of which is the use of X-rays in medical diagnosis (Table 2). Lesser sources of exposure to man-made radiation include radioactive minerals (e.g., ^{238}U, ^{232}Th, ^{40}K, ^{226}Ra) in building materials, phosphate fertilizers, and crushed rock; radiation-emitting components of TV sets, smoke detectors, and other consumer products; radioactive fallout from atomic weapons (e.g., ^{137}Cs, ^{90}Sr, ^{89}Sr, ^{14}C, ^{3}H, ^{95}Zr); and nuclear power (e.g., ^{3}H, ^{14}C, ^{85}KR, ^{129}I, ^{137}Cs) (Table 2).

In various occupations, workers also receive additional doses of ionizing radiation, depending on their job assignments and working conditions. The average annual effective dose received occupationally by monitored radiation workers in the U.S. is less than that received from natural background, however, and in any given year less than one per cent of such workers receive a dose that approaches the maximum permissible yearly occupational exposure limit [50 mSv (5 rem)] (NCRP 1989a).

Nature and Mechanisms of Ionizing Radiation Injury

As an ionizing radiation penetrates living cells, it collides randomly with atoms and molecules in its path, giving rise to ions and free radicals, which break chemical bonds and cause other molecular alterations that may injure the cells. The spatial distribution of such events along the path of the radiation depends on the energy, mass, and charge of the

radiation; e.g., X rays and gamma rays are sparsely ionizing, in comparison with charged particles, which typically are densely ionizing (Fig. 2).

Effects on DNA. Any molecule in the living cell may be altered by radiation, but DNA is the most critical biological target because of the limited redundancy of the genetic information it contains. DNA can be damaged directly by an impinging radiation, and it can also be damaged indirectly by radiation-induced effects on the surrounding cytoplasm or through the release of reactive oxygen species, cytokines, and other factors from neighboring cells (so-called "bystander" effects) (Mothersill and Seymour, 2001). A dose of radiation that is large enough to kill the average dividing cell [2 Sv (200 rem)] suffices to cause hundreds of lesions in its DNA molecules (Ward, 1988). Although most such lesions are reparable, those which are produced by a densely ionizing radiation (e.g., a proton or an alpha particle) are generally less reparable than those produced by a sparsely ionizing radiation (e.g., an X-ray or a gamma ray) (Goodhead 1988; UNSCEAR, 2000). For this reason, densely ionizing (high-LET) radiations are typically more potent than sparsely ionizing (low-LET) radiations; i.e., they have a higher relative biological effectiveness (RBE) for most forms of injury (e.g., Table 3).

Effects on genes. Damage to DNA that remains unrepaired or is misrepaired may be expressed in the form of a mutation. The frequency of mutations induced by low-LET radiation is generally several times lower at intermediate dose rates than at higher or lower dose rates, owing presumably to greater error-free repair of DNA damage at intermediate dose rates (Vilenchik and Knudson, 2000). At any given dose rate, however, the frequency of mutations appears to increase as a linear, nonthreshold function of the dose, approximating 10^{-5} to 10^{-6} per locus per Sv (100 rem) (NAS, 1990; UNSCEAR, 2000). Thus, in Chernobyl accident victims, the dose-response relationship for radiation-induced glycophorin mutations in bone marrow cells closely resembles that which has been observed in atomic bomb survivors (Jensen et al, 1995). The fact that the mutation rate appears to be proportional to the dose is interpreted to signify that traversal of the DNA by a single ionizing particle may, in principle, suffice to cause a mutation (NAS 1990). In a variety of experimental systems, moreover, the mutation rate has been observed to remain elevated for many cell generations following irradiation, signifying the induction of a transmissible genomic instability in surviving cells (Little et al, 1997).

Effects on chromosomes. Radiation damage to the genetic apparatus may also cause changes in chromosome number and structure (Cornforth and Bedford, 1993), the frequency of which has been observed to increase with the dose in radiation workers,

atomic bomb survivors, and others who have been exposed to ionizing radiation. The dose-response relationship for chromosome aberrations in human blood lymphocytes (Fig. 3) is well enough characterized so that the frequency of aberrations in such cells can serve as a useful biological dosimeter (IAEA 1986; Edwards, 1997).

Effects on cell survival. Among the earliest reactions to irradiation is the inhibition of cell division, which appears promptly after exposure, varying both in degree and duration with the dose (Fig. 4). Although the inhibition of mitosis is characteristically transitory, radiation damage to genes and chromosomes may be lethal to dividing cells, which are highly radiosensitive as a class (ICRP 1984; Hall, 1994). Measured in terms of proliferative capacity, the survival of dividing cells tends to decrease exponentially with increasing dose, 1-2 Sv (100-200 rem) generally sufficing to reduce the surviving population by about 50 per cent (Fig. 5).

Effects on tissues. Mature, nondividing cells are relatively radioresistant, but the dividing cells in a tissue are radiosensitive and may be killed in sufficient numbers by intensive irradiation to cause the tissue to become atrophic (Fig. 6). The rapidity with such atrophy occurs depends on cell population dynamics within the affected tissue; i.e., in organs characterized by slow cell turnover, such as the liver and vascular endothelium, the process is typically much slower than in organs characterized by rapid cell turnover, such as the bone marrow, epidermis, and intestinal mucosa (ICRP 1984; Hall, 1994; Mettler and Upton, 1995). It is noteworthy, moreover, that if the volume of tissue irradiated is sufficiently small, or if the dose is accumulated gradually enough, the severity of the resulting injury may be greatly reduced by the compensatory proliferation of surviving cells.

Clinical manifestations of injury

Types of effects. Radiation effects encompass a wide variety of reactions, varying markedly in their dose-response relationships, clinical manifestations, timing, and prognosis (Mettler and Upton, 1995). The effects are often subdivided, for convenience, into two broad categories: 1) heritable effects (i.e., those which are expressed in the descendants of exposed individuals) and 2) somatic effects (i.e., those which are expressed in exposed individuals themselves). The latter include acute effects, which occur relatively soon after irradiation, as well late (or chronic) effects, such as cancer, which may not appear until months, years, or decades later.

Acute effects. The acute effects of radiation result predominantly from the depletion of progenitor cells in affected tissues (Fig.6) and thus can be elicited only by doses that are large enough to kill many such cells (e.g., Table 4). For this reason, such effects are viewed as nonstochastic, or deterministic, in nature (ICRP, 1984, 1991), in contradistinction to the mutagenic and carcinogenic effects of radiation, which are viewed as stochastic phenomena resulting from random molecular alterations in individual cells that increase in frequency as linear-nonthreshold functions of the dose (NAS, 1990; ICRP, 1991).

Acute injuries of the types that were prevalent in pioneer radiation workers and early radiotherapy patients have been largely eliminated by improvements in safety precautions and treatment methods; however, most patients treated with radiation today still experience some injury of the normal tissue surrounding the irradiated treatment field. In addition, accidents causing radiation injury continue to occur Cardis, 1996). Between 1945 and 1987, for example, some 285 nuclear reactor accidents (excluding the Chernobyl accident) were reported in various countries, in which more than 1350 persons were irradiated, 33 of them fatally (Lushbaugh et al 1986). The Chernobyl accident alone released enough radioactivity to require tens of thousands of people and farm animals to be evacuated from the surrounding area, and the accident caused radiation sickness and burns in more than 200 emergency personnel and firefighters, 31 of whom were injured fatally (UNSCEAR, 1988). The long-term health effects of the radioactivity released by the accident cannot be predicted with certainty, but estimates based on nonthreshold dose-incidence models (discussed below), imply that up to 30,000 additional cancer deaths may occur in the population of the northern hemisphere during the next 70 years as a result of the accident (USDOE 1987).

Less catastrophic, but far more numerous, than reactor accidents have been accidents involving medical and industrial gamma ray sources, which also have caused injures and loss of life. For example, the improper disposal of a cesium-137 radiotherapy source in Goiania, Brazil, in 1987, resulted in the irradiation of dozens of unsuspecting victims, four of whom were injured fatally (UNSCEAR 1993).

Although a comprehensive discussion of radiation injuries is beyond the scope of this review, acute reactions of the more radiosensitive tissues are of particular interest. Hence these are described briefly in the following.

Skin. Cells in the germinal layer of the epidermis are highly radiosensitive. As a result, rapid exposure of the skin to a dose of 6 Sv (600 rem) or more causes erythema (reddening) in the exposed area, which appears within a day or so, typically lasts a few hours, and is followed 2-4 weeks later by one or more waves of deeper and more prolonged erythema, as well as by epilation (hair loss). If the dose exceeds 10-20 Sv (!000-2000 rem), blistering, necrosis, and ulceration may ensue within 2-4 weeks, followed by fibrosis of the underlying dermis and vasculature, which may lead to atrophy and a second wave of ulceration months or years later (ICRP 1984; Mettler and Upton, 1995).

Bone marrow and lymphoid tissue. Lymphocytes also are highly radiosensitive; a dose of 2-3 Sv (200-300 rem) delivered rapidly to the whole body can kill enough of them to depress the peripheral lymphocyte count and impair the immune response within hours (UNSCEAR, 1988; Mettler and Upton, 1995). Hemopoietic cells in the bone marrow are similarly radiosensitive and are depleted sufficiently by a comparable dose to the whole body so that granulocytopenia and thrombocytopenia ensue within 3-5 weeks. Such reductions in granulocyte and platelet counts can be severe enough to result in fatal infection and/or hemorrhage (Table 5).

Intestine. Stem cells in the epithelium lining the small bowel also are extremely radiosensitive, acute exposure to 10 Sv (1000 rem) depletes their numbers sufficiently to cause the overlying intestinal villi to become denuded within days (ICRP, 1984; UNSCEAR, 1988; Mettler and Upton, 1995). Such denudation of a large area of the mucosa can result in a fulminating, rapidly fatal dysentery-like syndrome (Table 5).

Gonads. Mature spermatozoa can survive large doses [>100 Sv (10,000 rem)], but spermatogonia are so radiosensitive that as little as 0.15 Sv (15 rem) delivered rapidly to both testes suffices to cause oligospermia, and a dose of 2-4 Sv (200-400 rem) can cause permanent sterility. Oocytes, likewise, are radiosensitive, a dose of 1.5-2.0 Sv (150-200 rem) delivered rapidly to both ovaries causing temporary sterility, and a larger dose permanent sterility, depending on the age of the woman at the time of exposure (ICRP, 1984; Mettler and Upton, 1995).

Respiratory tract. The lung is not highly radiosensitive, but rapid exposure to a dose of 6-10 Sv (600-1000 rem) can cause acute pneumonitis to develop in the exposed area

within 1-3 months. If a large volume of lung tissue is affected, the process may result in respiratory failure within weeks (Table 5), or in pulmonary fibrosis and cor pulmonale months or years later (ICRP, 1984; UNSCEAR, 1988; Mettler and Upton, 1995)).

Lens of the eye. Cells of the anterior epithelium of the lens, which continue to divide throughout life, are relatively radiosensitive. As a result, rapid exposure of the lens to a dose exceeding 1 Sv (100 rem) may lead within months to the formation of a microscopic posterior polar opacity; and 2-3 Sv (200-300 rem) received in a single brief exposure -- or 5.5-14 Sv (550-1400 rem) accumulated over a period of months -- may produce a vision-impairing cataract (ICRP, 1984; Mettler and Upton, 1995).

Other tissues. In comparison with the tissues mentioned above, other tissues of the body are generally appreciably less radiosensitive (e.g., Table 4); however, the embryo constitutes a notable exception, as discussed below. Noteworthy also is the fact that the radiosensitivity of every tissue is increased when it is in a rapidly growing state (ICRP, 1984).

Whole-body radiation injury. Rapid exposure of a major part of the body to a dose in excess of 1 Sv can cause the *acute radiation syndrome*. This syndrome includes: 1) an initial prodromal stage, characterized by malaise, anorexia, nausea, and vomiting, 2) an ensuing latent period, 3) a second (main) phase of illness, and 4) ultimately, either recovery or death (Table 5). The main phase of the illness typically takes one of the following forms, depending on the predominant locus of radiation injury: 1) hematological, 2) gastrointestinal, 3) cerebral, or 4) pulmonary (Table 5). In addition to the acute radiation syndrome, a less well established, chronic form of "radiation sickness", characterized by weakness, ease of fatigue, and malaise, has been reported in persons exposed to total body radiation in the former Soviet Union (Kossenko et al, 1994).

Localized radiation injury. Unlike the clinical manifestations of acute whole-body radiation injury, which typically are dramatic and prompt, the reaction to sharply localized irradiation, whether from an external radiation source or from an internally deposited radionuclide, tends to evolve slowly and to produce few symptoms or signs unless the volume of tissue irradiated and/or the dose are relatively large (e.g., Table 4).

Effects of Radionuclides. Some radionuclides (e.g., tritium, carbon-14, and cesium -137) tend to be distributed systemically within the body and to irradiate the body as a

whole, whereas other radionuclides are characteristically taken up and concentrated in specific organs, producing injuries that are correspondingly localized. Radium and strontium-90, for example, are deposited predominantly in bone and thus injure skeletal tissues primarily, whereas radioactive iodine concentrates in the thyroid gland, which is the primary site of any resulting injury (Stannard, 1988: Mettler and Upton, 1995).

Carcinogenic Effects.

General features. The carcinogenicity of ionizing radiation, first manifested early in this century by the occurrence of skin cancers and leukemias in pioneer radiation workers (Upton, 1986), has since been documented extensively in the form of dose-dependent excesses of neoplasms of many types in radium dial painters, underground hardrock miners, atomic bomb survivors, radiotherapy patients, and experimentally irradiated laboratory animals (Upton, 1986; NAS, 1990; UNSCEAR, 1994; Mettler and Upton, 1995; NCRP, 1997).

The benign and malignant growths induced by irradiation characteristically take years or decades to appear and exhibit no known features by which they can be distinguished from those produced by other causes. With few exceptions, moreover, their induction has been detectable only after relatively large doses [>0.5 Sv (50 rem)], and it has varied with the type of neoplasm as well as the age and sex of those exposed (NAS, 1990; Mettler and Upton, 1995).

Mechanisms. The molecular mechanisms of radiation carcinogenesis remain to be elucidated in detail, but the carcinogenic effects of radiation in laboratory animals and cultured cells have been observed to include initiating effects, promoting effects, and effects on the progression of neoplasia, depending on the experimental conditions in question (NAS, 1990; Little, 2000; Cox, 2001). The effects also appear to involve the activation of oncogenes inactivation or loss of tumor-suppressor genes, and induction of genomic instability in many, if not all, instances (Little, 2000). In addition, the carcinogenic effects of radiation resemble those of chemical carcinogens in being similarly modifiable by hormones, nutritional variables, and other modifying factors (NCRP, 1989b; NAS, 1990). It is noteworthy, moreover, that the effects of radiation may be additive, synergistic, or mutually antagonistic with those of chemical carcinogens,

depending on the specific chemicals and exposure conditions in question (UNSCEAR, 1982, 1986; 2000).

Dose-effect relationship. Although the overall incidence of cancer appears to have increased as a linear-nonthreshold function of the dose in atomic bomb survivors (Fig. 7), existing data do not suffice to define the dose-incidence relationship unambiguously for any type of neoplasm or to define how long after irradiation the risk of the growth may remain elevated in an exposed population. Any risks attributable to low-level irradiation can, therefore, be estimated only by extrapolation, based on models incorporating assumptions about such parameters (NAS, 1990; NCRP, 1997; Puskin and Nelson, 1995). Of various dose-effect models that have been used to estimate the risks of low-level irradiation, the one that has been judged to provide the best fit to the available data is of the form:

$$R(d) = R_O [1+ f(d)g(b)]$$

where R_O denotes the age-specific background risk of death from a specific type of cancer, d the radiation dose, f(d) a function of dose that is linear-quadratic for leukemia and linear for some other types of cancer, and g(b) is a risk function dependent on other parameters, such as sex, age at exposure, and time after exposure (NAS, 1990; NCRP, 1997; Sinclair, 1998).

Nonthreshold models of this type have been applied to epidemiological data from the Japanese atomic-bomb survivors (e.g., Pierce and Mendelsohn, 1998, 1999; Pierce and Preston, 2000) and other irradiated populations to derive estimates of the lifetime risks of different forms of radiation-induced cancer (e.g., Table 6). Such estimates must be interpreted with caution, however, in attempting to predict the risks of cancer attributable to small doses or doses that are accumulated over weeks, months, or years, since experiments with laboratory animals have shown the carcinogenic potency of X-rays and gamma rays to be reduced by as much as an order of magnitude when the exposure is greatly prolonged. In fact, as has been emphasized elsewhere (NAS, 1990; NCRP, 1997; Pierce and Preston, 2000), the available data do not exclude the possibility that there may be a threshold in the mSv dose range, below which radiation may lack carcinogenicity.

It is also noteworthy that the estimates tabulated are based on population averages and are not necessarily applicable to any given individual; i.e., susceptibility to certain types of cancer (e.g., cancers of the thyroid and breast) is substantially higher in children than in adults, and susceptibility to certain cancers is also increased in association with some

hereditary disorders, such as retinoblastoma and the nevoid basal cell carcinoma syndrome (Sankaranarayanan and Chakraborty, 1995; ICRP, 1998). Such differences in susceptibility notwithstanding, population-based estimates have been used in compensation cases as a basis for gauging the probability that a cancer arising in a previously irradiated person may have been caused by the exposure in question (NIH, 1985; Wakeford et al, 1998).

Low-dose risk assessment. Epidemiological studies to ascertain whether the risks of cancer from low-level exposure to radiation actually vary with dose in the manner predicted by the above estimates have been inconclusive thus far. Populations living in areas of elevated natural background radiation levels exhibit no definitely attributable corresponding increases in cancer rates (NAS, 1990; UNSCEAR, 1994). In a few such populations, cancer rates have even appeared to vary inversely with natural background radiation levels, which has been interpreted by some observers as evidence for the existence of beneficial (or hormetic) effects of low-level irradiation, akin to the adaptive responses to radiation that are elicited in certain cellular systems (UNSCEAR, 1994; Wojcik, 2000). The observed inverse relationships in cancer rates are of questionable significance, however, since they have not persisted after controlling for the effects of confounding variables in the studies in question (NAS, 1990; Upton, 2000).

In today's radiation workers -- except for certain cohorts of underground hardrock miners (Lubin et al, 1994; NAS, 1998) -- the rates of cancers other than leukemia (and, possibly, multiple myeloma) are no longer detectably increased (UNSCEAR, 1994), thanks to advances in radiation protection. The rates of leukemia and multiple myeloma in such workers are consistent with the estimates tabulated above (IARC, 1994; Cardis et al, 1995; Muirhead et al, 1999). In summary, therefore, the data available at present are in keeping with the estimates tabulated above (Table 6), which imply that less than 3 per cent of all cancers in the general population are attributable to natural background ionizing radiation (NAS, 1990; IARC, 1994; UNSCEAR, 2000; NCRP, 2001), although up to 10 per cent of lung cancers may be attributable to indoor radon (NAS, 1990, 1998; Lubin, et al 1994).

High levels of radioactive fallout from a thermonuclear weapons test at Bikini, in 1954, have been observed to cause a dose-dependent increase in the frequency of thyroid cancer in Marshall Islanders who received large doses to the thyroid gland in childhood (Robbins and Adams, 1989). Children living in areas of Belarus and the Ukraine contaminated by radionuclides released from the Chernobyl accident have, similarly, been reported to show

an increased incidence of thyroid cancer (Astakhova et al, 1998; Heidenreich et al, 1999), as have children of southwestern Utah and Nevada who were exposed to fallout from nuclear weapons tests in Nevada during the 1950s (Kerber et al, 1993), in whom the prevalence of acute leukemia also appears to have been elevated in those dying between 1952 and 1957, the period of greatest exposure to fallout (Stevens et al, 1990). Also, in children exposed prenatally to diagnostic levels of X-radiation, an excess of childhood cancer has been observed (Doll and Wakeford, 1997).

The possibility that excesses of leukemia among children residing in the vicinity of nuclear plants in the United Kingdom may have been caused by radioactivity released from the plants has also been suggested (Gardner et al, 1990). The releases, however, are estimated to have increased the total radiation dose to such children by less than 2 per cent, from which it is inferred that other explanations are more likely (Doll et al, 1994). An infective etiology for the observed clusters of leukemia is implied by the existence of comparable excesses of childhood leukemia at sites in the U.K. that lack nuclear facilities but otherwise resembling nuclear sites in having similarly experienced large influxes of population in recent times (Kinlen, 1988; Doll et al, 1994; Doll, 1999). Another hypothesis -- namely, that the leukemias in question may have been caused by occupational irradiation of the fathers of the affected children -- also has been suggested by the results of a case-control study (Gardner et al, 1990), but this hypothesis is generally discounted for reasons that are discussed in the section to follow.

Effects on Longevity

In addition to an increased risk of cancer, the long-term effects of ionizing irradiation include increases in the risks of cardiovascular disease and various other non-neoplastic diseases. Hence whole-body irradiation irradiation early in life results in a dose-dependent loss of life expectancy from the risks of all such diseases combined. In laboratory mice and rats, the resulting loss of life expectancy amounts to about 5 per cent per Gy (UNSCEAR, 1982), and in atomic bomb survivors it has thus far amounted to about 1-2 per cent per Gy, roughly 30 per cent of which has been attributable to mortality from non-neoplastic diseases (Shimizu et al, 1999; Cologne and Preston, 2000).

Heritable Effects.

Heritable effects of irradiation, although well documented in other organisms, have yet to be observed in humans; e.g., intensive study of more than 76,000 children of the Japanese atomic bomb survivors, carried out over four decades, has failed to disclose any heritable effects of radiation in this population, as measured by untoward pregnancy outcomes, neonatal deaths, malignancies, balanced chromosomal rearrangements, sex-chromosome aneuploidy, alterations of serum or erythrocyte protein phenotypes, changes in sex ratio, or disturbances in growth and development (Neel et al, 1990). Consequently, estimates of the risks of heritable effects of radiation must rely heavily on extrapolation from findings in the laboratory mouse and other experimental animals (NAS, 1990; UNSCEAR, 1993; Neel, 1998).

From the available experimental and epidemiological data, it is inferred that the dose required to double the rate of heritable mutations in human germ cells must be at least 1.0 Sv (100 rem) (NAS 1990; UNSCEAR, 1993; Neel, 1998; Sankaranarayanan, 2000)). On this basis, it is estimated that less than one per cent of all genetically determined diseases in the human population can be attributed to natural background irradiation (Table 7).

The hypothesis that the excess of leukemia and non-Hodgkin's lymphoma in young people residing in the village of Seascale resulted from heritable oncogenic effects caused by the occupational irradiation of the children's fathers at the Sellafield nuclear installation has been suggested by the results of a case-control study (Gardner et al, 1990), as noted above. Arguing against this hypothesis, however, are: 1) the lack of any comparable excess in larger numbers of children born outside Seascale to fathers who had received similar, or even larger, occupational doses at the same nuclear plant (Wakeford et al, 1994a), 2) the lack of similar excesses in French (Hill and LaPlanche, 1990), Canadian (McLaughlin et al, 1993), or Scottish (Kinlen et al, 1993) children born to fathers with comparable occupational exposures, 3) the lack of excesses in the children of atomic bomb survivors (Yoshomoto et al, 1990), 4) the lack of excesses in U.S. counties containing nuclear plants (Jablon et al, 1991), and 5) the fact that the frequency of radiation-induced mutations implied by the interpretation is far higher than established mutation rates (Wakeford et al, 1994b). On balance, therefore, the available data fail to support the paternal gonadal irradiation hypothesis (Doll et al, 1994; Little et al, 1995; Doll, 1999).

Effects of Prenatal Irradiation.

The embryo is highly vulnerable to the carcinogenic effects of ionizing radiation (Doll and Wakeford, 1997) and to other radiation- and chemically-induced disturbances in growth and development (e.g., Table 8). Apart from the carcinogenic effects, most such disturbances appear to be nonstochastic in nature and are inducible only during relatively short windows of time corresponding to critical periods in the organogenesis of the various developing organ systems (UNSCEAR, 1977, 1986, 1993). The thresholds for such effects appear to be relatively low, however, and the available data do not exclude the possibility that damage to a single cell (the fertilized egg) or only a few of the cells in a primordial anlage at a critical stage in organogenesis may suffice under certain conditions to cause some effects of this type (UNSCEAR, 1977,1986, 1993).

Unlike mutagenic and carcinogenic effects, which are expressed in only a small percentage of exposed individuals, a disturbance of growth and development may be projected to affect all who are exposed at a vulnerable stage during prenatal life to a dose that exceeds the relevant threshold. Thus, while only a small percentage of the individuals who were exposed prenatally to atomic bomb radiation at a critical stage in brain development (i.e., 8-15 weeks after conception) exhibited severe mental retardation (Fig. 8), a larger percentage exhibited less marked decrements in intelligence (Fig. 9) and school performance, implying that there was a dose-dependent downward shift in the distribution of intelligence levels within the entire cohort (NAS, 1990; UNSCEAR, 1993). In view of the broadly similar neurotoxic effects of certain chemical agents (e.g., lead, mercury, alcohol) on the developing brain (Tilson, 1990; NAS, 1992; Rodier, 1994), it is conceivable that some of the chemical components of mixed wastes may pose comparable risks to the embryo.

Prevention. In order to minimize the risks of injury, the following principles are recommended as guidelines to be observed in any activities involving exposure to ionizing radiation (ICRP 1991): 1) no such activity should be considered justifiable unless it produces a sufficient benefit to those who are exposed, or to society at large, to offset any harm it may cause; 2) in any such activity, the dose and/or likelihood of exposure should be kept as low as is reasonably achievable (ALARA), all relevant economic and social factors being taken into account; 3) the radiation exposure of individuals resulting from any combination of such activities should be subject to dose limits (e.g., Table 9) that are far enough below the thresholds for nonstochastic effects to prevent such effects altogether, and that are also low enough to keep the risks of any

resulting stochastic effects (which may have no thresholds) from exceeding socially acceptable levels.

Implicit in these guidelines are the requirements that any facility dealing with ionizing radiation: 1) be properly designed, 2) carefully plan and oversee its operating procedures, including dose calibration, 3) have in place a well conceived radiation protection program, 4) ensure that its workers are adequately trained and supervised, and 5) maintain a well-developed and well-rehearsed emergency preparedness plan, in order to be able to respond promptly and effectively in the event of a malfunction, spill, or other type of radiation accident (Shapiro 1990)..

Since medical radiographic examinations and indoor radon constitute the most important controllable sources of exposure to ionizing radiation for members of the general public (Table 2), prudent measures to limit irradiation from these sources are called for (Upton et al 1990). Other potential risks to human health and the environment calling for increased attention are those posed by the millions of cubic feet of radioactive and mixed wastes (mine and mill tailings, spent nuclear fuel, waste from the decommissioning of nuclear power plants, dismantled industrial and medical radiation sources, radioactive pharmaceuticals and reagents, heavy metals, polyaromatic hydrocarbons, and other contaminants) which are present in ever-growing quantities and which severely tax existing storage capacities at numerous sites (e.g., NAS, 1989; USEPA, 1990, 1991; USNRC, 1992; USDOE, 1993).

NONIONIZING RADIATION

ULTRAVIOLET RADIATION

Nature, Sources, and Environmental Levels. Ultraviolet radiations (UVR) comprise a spectrum (Fig. 1) of electromagnetic waves, subdivided for convenience into three bands: 1) UVA, 315-400 nm ("black light"); 2) UVB, 280-315 nm; and 3) UVC, 200-280 nm (which is germicidal). The chief source of UVR for members of the public is sunlight, which varies in intensity with latitude, elevation, and season (AMA 1989). Important man-made sources of high-intensity exposure include sun- and tanning- lamps, welding arcs, plasma torches, germicidal and black-light lamps, electric arc furnaces, hot-metal operations, mercury-vapor lamps, and lasers. Common low-intensity sources include fluorescent lamps and certain laboratory equipment (NIOSH 1972).

Nature and Mechanisms of Injury. Since UVR does not penetrate deeply into human tissues, the injuries it causes are confined chiefly to the skin and eyes. Reactions of the skin to UVR, which are common among fair-skinned people, include sunburn; skin cancers (basal cell and squamous cell carcinomas, and to a lesser extent melanomas); aging of the skin; solar elastoses; and solar keratoses (English et al, 1997). Injuries of the eye include photokeratitis, which may result from brief exposure to a high-intensity UVR source ("welder's flash") or from more prolonged exposure to intense sunlight ("snow blindness"); cortical cataract; and pterygium (Lerman 1980; Driscoll and Cridland, 2000).

The effects of UVR result chiefly from its absorption in DNA, with the production of pyrimidine dimers, causing mutational changes in exposed cells. Sensitivity to UVR may be increased by DNA repair defects (e.g., xeroderma pigmentosum), by agents (e.g., caffeine) that inhibit the repair enzymes, and by photosensitizing agents (e.g., psoralens, sulfonamides, tetracyclines, nalidixic acid, sulfonylureas, thiazides, phenothiazines, furocumarins, and coal tar) which produce UVR-absorbing DNA photoproducts (Harper and Bickers 1989). The carcinogenic action of UVR is mediated primarily through direct effects on the exposed cells but may involve depression of local immunity as well (Kripke 1988; Driscoll and Cridland, 2000). UVB, although far less intense than UVA in sunlight, plays a more important role in sunburn and skin carcinogenesis; UVA, however, contributes to the latter, as well as to tanning, some photosensitivity reactions, aging of the skin, photokeratitis, and cortical lens opacities (AMA 1989).

Prevention. Excessive exposure to sunlight or other sources of UVR should be avoided, especially by fair-skinned individuals. In addition, protective clothing, UVR-screening lotions or creams, and UVR-blocking sunglasses should be used for the purpose when necessary. To protect occupationally exposed workers, it is recommended that direct exposure of the eye not exceed a limit of 0.003- 1.0 J/cm^2, depending on the wavelength of the UVR and the duration of exposure (NIOSH 1972; ACGIH 1997; ICNIRP, 2000).

From an environmental perspective, it is noteworthy that the protective layer of ozone in the stratosphere is being depleted by chlorofluorocarbons and other air pollutants, and that every 1 per cent decrease in ozone is expected to increase the UVR reaching the earth by 1-2 per cent, thereby increasing the rates of nonmelanotic skin cancer by 2-6 per cent (Henriksen et al 1990). The projected increase in cancer rates is, of course, only one

of many adverse effects to be expected from increased intensities of UVR, the most serious, perhaps, being far-reaching impacts on vegetation and crop production.

VISIBLE LIGHT

Nature, Sources, and Environmental Levels. Visible light consists of electromagnetic waves varying in wavelength from 380 nm (violet) to 760 nm (red) (Fig. 1). Sources of visible light in the environment vary widely in the intensity of their emissions; common high-intensity sources other than the sun include lasers, electric welding or carbon arcs, and tungsten filament lamps (Fig. 10).

Nature and Mechanisms of Injury Too bright a light can injure the eye through photochemical reactions in the retina; i.e., sustained exposure to intensities exceeding 0.1 mW/cm^2, such as can result from fixating a bright source of light, may produce photochemical blue-light injury, and brief exposure of the retina to intensities exceeding 10 W/cm^2, depending on image size, may cause a retinal burn (Fig. 10). The lens, iris, and cornea also are vulnerable to injury from the thermal effects of laser radiation (Sliney and Wolbarsht 1980; Frank and Slesin, 1998). Too little illumination, conversely, also can be harmful, causing eyestrain (Huer 1983) and/or seasonal affective disorder (SAD).

Prevention Since bright, continuously visible light normally elicits an aversion response, which acts to protect the eye against injury, few sources of light are large and bright enough to cause a retinal burn under normal viewing conditions. One must never kook directly at a solar eclipse, and in situations involving potential exposure to such high-intensity sources as carbon arcs or lasers, appropriate training, proper design of equipment, and protective eye shields are indicated (Sliney and Wolbarsht 1980; ANSI 1986; ACGIH 1997; ICNIRP, 2000).

INFRARED RADIATION.

Nature, Sources, and Environmental Levels Infrared radiation (IR) consists of electromagnetic waves ranging in wavelength from 7×10^{-5} m to 3×10^{-2}m (Fig. 1). Some such radiation is emitted by all objects with temperatures above absolute zero, but potentially hazardous sources of IR include furnaces, ovens, welding arcs, molten glass, molten metal, and heating lamps.

Nature and Mechanisms of Injury The injuries caused by IR are limited chiefly to burns of the skin and cataracts of the lens of the eye. The warning sensation of heat

usually prompts aversion in time to prevent the skin from being burned by IR, but the lens of the eye is vulnerable in lacking both heat-sensing and heat-dissipating ability. As a result, glass blowers, blacksmiths, oven operators, and those working around heating and drying lamps are at risk of IR-induced cataracts (Lydahl and Philipson 1984).

Prevention Control of IR hazards requires appropriate shielding of sources, proper training and supervision of potentially exposed persons, and use of protective clothing and goggles. It is also recommended that exposures to IR not exceed 10 mW/cm^2 (ACGIH 1997; Frank and Slesin, 1998).

MICROWAVE RADIATION

Nature, Sources, and Environmental Levels. Microwave and radiofrequency radiation (MW/RFR) consists of electromagnetic waves ranging in frequency from about 3kHz to 300 GHz (Fig, 1). Sources of MW/RFR occur widely in radar, television, radio, cellular phones, and other telecommunications systems, and are also used in various industrial operations (e.g., heating, welding, and melting of metals; processing of wood and plastic; high-temperature plasma), household appliances (e.g., microwave ovens), and medical applications (e.g., diathermy and hyperthermia) (ILO 1986).

Nature and Mechanisms of Injury. The biological effects of MW/RFR are primarily thermal in nature, MW/RFR-induced injuries consisting mainly of burns of the skin and other tissues. Burns have occasionally resulted from faulty or improperly used household microwave ovens and from the overexposure of patients in whom cutaneous pain and temperature senses that usually warn of impending injury are impaired. Because of the deep penetration of MW/RFR, the cutaneous burns it causes tend to involve dermal and subcutaneous tissues, and to heal slowly. Cataracts of the lens of the eye have been reported to result from high-intensity exposures (>1.5 kW/M^2) (McRee 1972), and even death from hyperthermia has been encountered in the industrial use of MW/RFR sources (McLaughlin 1957; Roberts and Michaelson 1985). Also well documented is the ability of MW/RFR to interfere with cardiac pacemakers and other medical devices (NCRP 1986; Michaelson, 1991). Other effects reported in the literature, but as yet inconclusively documented, include impairment of fertility, developmental disturbances, neurobehavioral abnormalities, depression of immunity, and increased risks of cancer (Michaelson, 1991; Yost, 1992; Carlo, 1998; Moulder et al, 1999; Elwood, 1999).

Prevention. Proper design and shielding of MW/RFR sources are indicated, along with appropriate training and supervision of potentially exposed persons (especially those

wearing cardiac pacemakers or other sensitive devices) . Exposure to MW/RFR power densities exceeding the threshold limit values tabulated (Table 10) may cause detectable heating of tissue and should be avoided (NCRP, 1986; ILO 1986; ACGIH 1997).

EXTREMELY LOW-FREQUENCY ELECTROMAGNETIC FIELDS

Nature, Sources, and Environmental Levels. Extremely low-frequency (ELF) electromagnetic fields (EMFs) -- i.e., time-varying magnetic fields with frequencies below 300 Hz -- are present throughout the environment. The largest such fields arise intermittently from solar activity and thunderstorms, during which they may reach intensities on the order of 0.5 μT (Grandolfo and Vecchia 1985). Far stronger than such naturally occurring EMFs are the localized 50-60 Hz fields that are generated by electric power lines, transformers, motors, household appliances, video-display tubes (VDTs), and various medical devices, notably NMR imaging systems (OTA, 1989; Tenforde 1992). For example, the flux density on the ground beneath a 765-kV, 60-Hz power line carrying 1 kA per phase is of the order of 15 μT, and close to common household appliances the flux density may range up to 2.5 mT (Tenforde 1992). Since the strength of such fields decreases rapidly with distance, however, the average ambient value in the household environment is less than 0.3 μT (Silva et al 1989). By the same token, while flux densities at video display terminals typically range up to 5 μT, those at the location of the operator are generally less than 1 μT (Tenforde 1992).

Nature and Mechanisms of Injury. Extremely-low-frequency EMFs induce electrical currents which can alter the properties of cell membranes and exert effects on electrically active tissues (nerves, neuromusculature, retina, heart) and on cardiac pacemakers. Induced current densities under 1-10 mA/m^2 produce few, if any, irreversible effects, which is not surprising in view of the fact that endogenous current densities of 0.1-10 mA/m^2 exist in many tissues. Induced current densities above 10 mA/m^2, on the other hand, although not genotoxic, reportedly produce various changes in the biochemistry and physiology of cells and tissues (e.g., alterations in metabolism, growth rate, melatonin secretion, endocrine activity, immune response); and current densities above 1A can cause neural excitation and irreversible effects, such as cardiac fibrillation (Tenforde, 1992).

In addition to the effects produced by strong EMFs, epidemiological data have suggested the possibility of severe effects from long-continued exposure to weaker EMFs; i.e.: 1) that the risks of leukemia may be increased by residential exposure to household EMFs in

children, 2) that the risks of brain cancer and leukemia may be increased by occupational exposure to EMFs in utility workers , and 3) that the risks of having miscarriages or of bearing children with birth defects may be increased by chronic exposure to EMFs through the operation of VDTs in pregnant women (Bates 1991; Tenforde 1992). As yet, although the epidemiological data are inconclusive and their interpretation complicated by uncertainties in exposure assessment and by the lack of established biological mechanisms for the effects in question, the fact that such fields have been reported to influence cell growth, ion transport, melatonin secretion, and tumor promotion in some model systems (Adair 1991; Stuchly et al 1991; Tenforde 1992) has contributed to public health concern (OTA 1989; NAS, 1997; Frank and Slesin, 1998).

Prevention. Preventive strategies include the design and installation of EMF sources in ways that limit human exposure to the hazards in question; the use of protective equipment, clothing, face masks, or goggles by workers who must enter intense EMF fields; and administrative measures to ensure adequate understanding and observance of proper control measures (Sliney and Colville, 2000). Thus, areas containing EMFs stronger than 0.1 mT, such as exist around transformers, accelerators, MRI systems, and other electric devices, should be posted with warning signs and should be avoided by persons wearing cardiac pacemakers. In addition, the strength of any 60 Hz time-varying magnetic field, such as typically exists around an MRI system, should be limited to 1 mT for occupational exposures, and to 0.1 mT for those wearing cardiac pacemakers or for continuous exposures involving members of the general public (ACGIH 1997; ICNIRP, 1998; Sliney and Colville, 2000). To minimize the risks, if any, that may be associated with the use of electric blankets, wiring design changes have been introduced by some manufacturers to cancel the surface 60-Hz EMFs that such blankets would otherwise generate (Tenforde 1992).

ULTRASOUND

Nature, Sources, and Environmental Levels. Although often classified for public health purposes with nonionizing radiation, ultrasound is not a component of the electromagnetic spectrum but actually consists of mechanical vibrations at frequencies above the audible range (i.e, > 16 KHz) (NCRP 1983). Sources of high-power, low-frequency ultrasound are used widely in science and industry for cleaning, degreasing, plastic welding, liquid extracting, atomizing, homogenizing, and emulsifying operations,

as well as in medicine for lithotripsy and other therapeutic applications. Low-power, high-frequency ultrasound is used widely in analytical work and in medical diagnosis (e.g., ultrasonography).

Nature and Mechanisms of Injury. The biological effects of ultrasound are similar in mechanism to those of mechanical vibration. High-power, low-frequency ultrasound, transmitted through the air or through bodily contact with a generating source, has been observed to cause a variety of effects in occupationally exposed workers, including headache, earache, tinnitus, vertigo, malaise, photophobia, hypercusia, peripheral neuritis, and autonomic polyneuritis, none of which appear to be irreversible. The possibilty that it may cause adverse effects on the embryo also has been suggested (NCRP 1983).

Although excessive exposure to high-frequency ultrasound through bodily contact with the source may be expected, in principle, to cause complaints similar to those above, no adverse effects have been observed to result from exposure to high-frequency ultrasound at the low power levels used in medical ultrasonography (NCRP 1983).

Prevention. Protection against injury by ultrasound requires appropriate isolation and insulation of generating sources , as well as proper training and ear protective devices for those working around such sources. Yearly audiometric and neurological examinations of occupationally exposed workers also are recommended (WHO 1982).

SUMMARY AND CONCLUSIONS

The adverse effects on human health caused by different forms of radiant energy are diverse, ranging from rapidly fatal injuries to cancers, birth defects, and hereditary disorders appearing months, years, or decades after exposure. The nature, frequency, and severity of effects depend on the type of radiant energy in question and the particular conditions of exposure. Most such effects are produced only by appreciable levels of exposure and can, therefore, be prevented completely by keeping any exposures from exceeding relevant thresholds. The genotoxic and carcinogenic effects of ionizing and ultraviolet radiations, in contrast, are presumed to increase in frequency as linear-nonthreshold functions of the dose and, therefore, not to be entirely preventable without eliminating all exposure to these two forms of radiation. Since it is not feasible to eliminate exposures to ionizing and ultraviolet radiations completely, protection against their mutagenic and carcinogenic effects requires that exposures to these agents be limited sufficiently to keep any associated risks from exceeding acceptable levels.

To achieve the desired level of protection against each of the different forms of radiation requires knowledge of the relevant exposure-risk relationships; appropriate design and operation of all radiation sources; proper training, equipment, and supervision of operating personnel; and education of members of the public in prudent measures for safeguarding their health. These requirements can be met satisfactorily in most situations involving radiation hazards, given the necessary commitment of effort and resources. Unresolved public health problems calling for particular attention at this time, however, include: 1) assessment of the risks associated with residential exposure to indoor radon, and of the pertinent remediation strategies; 2) development and implementation of measures for dealing with the hazards posed by the large and growing quantities of radioactive and mixed wastes; 3) assessment of the risks that may be associated with exposure to 60-Hz electromagnetic fields; and 4) further evaluation of stratospheric ozone depletion and its implications for ultraviolet-radiation-induced impacts on human health.

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TABLE 1. QUANTITIES AND DOSE UNITS OF IONIZING RADIATION

Quantity being measured	Definition	Dose unit*
Absorbed dose	Energy deposited in tissue (1 joule/kg)	Gray (Gy)
Equivalent dose	Absorbed dose weighted for the ion density (potency) of the radiation	Sievert (Sv)
Effective dose	Equivalent dose weighted for the sensitivity of the exposed organ(s)	Sievert (Sv)
Collective effective dose	Effective dose applied to a population	Person-Sv
Committed effective dose	Cumulative effective dose to be received from a given intake of radioactivity	Sievert (Sv)
Radioactivity	One disintegration per second	Becquerel (Bq)

*The units of measure listed are those of the International System, introduced in the 1970s to standardize usage throughout the world (ICRP, 1991). They have largely supplanted the earlier units; namely, the rad (1 rad = 100 ergs per gm = 0.01 Gy), the rem (1 rem = 0.01 Sv), and the curie (1 Ci = 3.7 x 10^{10} disintegrations per second = 3.7 x 10^{10} Bq).

TABLE 2. AVERAGE AMOUNTS OF IONIZING RADIATION RECEIVED ANNUALLY FROM DIFFERENT SOURCES BY A MEMBER OF THE U.S. POPULATION[a]

Source	Dose[b] (mSv)	(%)
Natural		
Radon[c]	2.0	55
Cosmic	0.27	8
Terrestrial	0.28	8
Internal	0.39	11
Total natural	2.94	82
Artificial		
X-ray diagnosis	0.39	11
Nuclear medicine	0.14	4
Consumer products	0.10	3
Occupational	<0.01	<0.3
Nuclear fuel cycle	<0.01	<0.03
Nuclear fallout	<0.01	<0.03
Miscellaneous[d]	<0.01	<0.03
Total artificial	0.63	18
Total natural and artificial	3.57	100

[a]Adapted from National Academy of Sciences, 1990.

[b]Average effective dose to soft tissues

[c]Average effective dose to bronchial epithelium alone

[d]Department of Energy facilities, smelters, transportation, etc.

TABLE 3. RADIATION PROTECTION WEIGHTING FACTORS FOR DIFFERENT RADIATIONS[1]

Type and Energy of Radiation	Radiation Weighting Factor[2]
Protons, all energies	1
Electrons and muons, all energies[3]	1
Neutrons	
<10 kev energy	5
10 – 100 kev	10
>100 kev – 2 Mev	20
> 2 Mev – 20 Mev	10
> 20 Mev	5
Protons, other than recoil protons, > 2 Mev	5
Alpha particles, fission fragments, heavy nuclei	20

[1]From ICRP, 1991

[2]All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

[3]Excluding Auger electrons emitted from nuclei bound to DNA.

TABLE 4. APPROXIMATE THRESHOLD DOSES OF CONVENTIONALLY FRACTIONATED THERAPEUTIC X-RADIATION FOR CLINICALLY DETRIMENTAL NONSTOCHASTIC EFFECTS IN VARIOUS TISSUES[a]

Organ	Injury at 5 years	Threshold dose (Gy)[b]	Irradiation field (area)
Fetus	Death	2	Whole
Bone marrow	Hypoplasia	2	Whole
Ovary	Permanent sterility	2-3	whole
Lens	Cataract	5	Whole
Testes	Permanent sterility	5-15	Whole
Cartilage, child;	Arrested growth	10	Whole
Breast, child	Hypoplasia	10	5cm^2
Bone, child	Arrested growth	20	10 cm^2
Bone marrow	Hypoplasia, fibrosis	20	localized
Muscle, child	Hypoplasia	20-30	whole
Kidney	Nephrosclerosis	23	Whole
Lymph nodes	Atrophy	33-45	--
Liver	Liver failure, ascites	35	whole
Lung	Pneumonitis, fibrosis	40	Lobe
Heart	Pericarditis, pancarditis	40	whole
Stomach; small intestine; colon	Ulcer, perforation	45	100 cm^2
Thyroid	Hypothyroidism	45	whole
Pituitary	Hypopituitarism	45	whole
Lymphatics	Sclerosis	50	--
CNS (brain)	Necrosis	50	whole
Spinal cord	Necrosis, transection	50	5 cm^2
Salivary glands	Xerostomia	50	50 cm^2
Cornea	Keratitis	50	whole
Capillaries	Telangiectasis, fibrosis	50-60	--
Breast, adult	Atrophy, necrosis	>50	Whole
Rectum	Ulcer, stricture	55	100 cm^2
Skin	Ulcer, severe fibrosis	55	100 cm^2
Eye	Panophthalmitis, hemorrhage	55	whole
Oral mucosa	Ulcer, severe fibrosis	60	50 cm^2
Esophagus	Ulcer, stricture	60	75 cm^2
Cartilage, adult	Necrosis	60	whole
Urinary bladder	Ulcer, contracture	60	Whole
Bone, adult	Necrosis, fracture	60	10 cm^2
Ear (inner)	Deafness	>60	whole
Adrenal	Hypoadrenalism	>60	whole
Vagina	Ulcer, fistula	90	5 cm
Muscle, adult	Atrophy	>100	whole
Uterus	Necrosis, perforation	>100	whole

[a]Adapted from Rubin and Casarett, 1972; ICRP, 1984.

[b]Dose causing effect in 1-5 per cent of exposed persons

TABLE 5. MAJOR FORMS AND FEATURES OF THE ACUTE RADIATION SYNDROME

Time after irradiation	Cerebral form (>50 Sv)	Gastrointestinal form (10-20 Sv)	Hemopoietic form (2-10 Sv)	Pulmonary form (>6 Sv to lungs)
First day	nausea vomiting diarrhea headache disorientation ataxia coma convulsions death	nausea vomiting diarrhea	nausea vomiting diarrhea	nausea vomiting
Second week		nausea vomiting diarrhea fever erythema prostration death		
Third to sixth weeks			weakness fatigue anorexia fever hemorrhage epilation recovery (?) death (?)	
Second to eighth months				cough dyspnea fever chest pain Resp. failure (?)

(from UNSCEAR, 1988)

TABLE 6. ESTIMATED LIFETIME RISKS OF CANCER ATTRIBUTABLE TO 0.1 Sv (10 rem) LOW-DOSE-RATE IRRADIATION[a]

Type or site of cancer	Excess cancer deaths per 100,000	
	(No,)	(%)[b]
Colon	95	5
Lung	85	3
Bone marrow (leukemia)	50	10
Stomach	50	8
Breast	45	2
Urinary bladder	25	4
Esophagus	10	3
Liver	15	8
Gonads	15	3
Thyroid	5	5
Bone	3	3
Skin	2	2
Remainder	100	2
Total	500	2

[a]Modified from ICRP (1991) and Puskin and Nelson (1995)
[b]Percentage increase in "background" risk expected for a nonirradiated population.

Table 7. Estimates of the risks of genetic disorders in children that are attributable to irradiation of their parents

Disease Class	Natural incidence per million liveborn children	Risk per Sv per million liveborn children	Risk from natural background irradiation per million liveborn children[a]	
			(No.)	(%)
Autosomal dominant and X-linked diseases	16,500	~750-1500	22-45	~0.2
Autosomal recessive diseases	7,500	~0	<1	<1
Chromosomal diseases	4,000	b	b	b
Chronic multifactorial diseases	650,000[c]	~250-1200	8-36	~0.004
Congenital abnormalities	60,000	~2000[d]	60[d]	0.1
Total	738,000	~4,000	~90-140	~0.02

[a]Based on an assumed dose rate of 1 mSv per year and a genetic doubling dose of 1 Gy.
[b]Risk assumed to be subsumed under the risk of autosomal dominant and X-linked diseases and, in part, under the risk of congenital abnormalities.
[c] Frequency in the general population .
[d]Estimated on the basis of mouse data, without recourse to the doubling-dose method.

(Based on data from NAS, 1990 and Sankaranarayanan, 2000)

TABLE 8. MAJOR DEVELOPMENTAL ABNORMALITIES PRODUCED BY PRENATAL IRRADIATION

	Brain	
Anencephaly	Porencephaly	Microcephaly*
Encephalocoele	Mongolism*	Reduced medulla
Cerebral atrophy	Mental retardation*	Neuroblastoma
Narrow aqueduct	Hydrocephalus*	Dilatation of ventricles*
Spinal cord anomalies*	Cranial nerve anomalies	
	Eyes	
Anophthalmia	Microphthalmia*	Microcornia*
Coloboma*	Deformed iris	Absence of lens
Nystagmus*	Retinoblastoma	Hypermetropia
Glaucoma	Cataract*	Blindness
Chorioretinitis	Partial albinism	Ankyloblepharon
	Skeleton	
General stunting	Reduced size of skull	Skull deformities*
Head ossification defects*	Vaulted cranium	Narrow head
Cranial blisters	Cleft palate*	Funnel chest
Dislocation of hip	Spina bifida	Deformed tail
Deformed feet	Club foot*	Digital anomalies*
Calcaneo valgus	Odontogenesis imperfecta*	Tibial exostosis
Amelanogenesis*	Scleratomal necrosis	
	Miscellaneous	
Situs inversus	Hydronephrosis	Hydroureter
Hydrocoele	Absence of kidney	Gonadal anomalies*
Congenital heart disease	Facial deformities	Pituitary disturbances
Deformities of ears	Motor disturbances	Dermatomal necrosis
Myotomal necrosis	Abnormalities in skin pigmentation	

From Brill and Forgotson, 1964; UNSCEAR, 1986.

*These abnormalities have been observed in humans exposed prenatally to large doses of radiation, in whom they have been tentatively attributed to the irradiation in question.

TABLE 9. RECOMMENDED EFFECTIVE DOSE LIMITS OF IONIZING RADIATION FOR OCCUPATIONALLY EXPOSED WORKERS AND MEMBERS OF THE PUBLIC[a]

Occupational Exposure[b]	Dose Limit
Annual	50 mSv (5 rem)
Cumulative	Age x 10 mSv (1 rem)
Public Exposure[b]	
Annual, continuous	1 mSv (100 mrem)
Annual, infrequent	5 mSv (500 mrem)

[a]From NCRP, 1993; ACGIH, 1993.

[b]Excluding medical and dental exposures.

FIGURE 1. THE ELECTROMAGNETIC SPECTRUM
(from Mettler and Upton, 1995)

FIGURE 2. DIFFERENCES AMONG VARIOUS TYPES OF IONIZING RADIATION IN PENETRATING POWER IN TISSUE (from Shapiro, 1972)

FIGURE 3. FREQUENCY OF DICENTRIC CHROMOSOME ABERRATIONS IN HUMAN LYMPHOCYTES IN RELATION TO DOSE, DOSE RATE, AND QUALITY OF IONIZING IRRADIATION IN VITRO (from Lloyd and Purrott, 1981)

FIGURE 4. MITOTIC INHIBITION INDUCED BY X-RAYS IN RAT CORNEAL EPITHELIAL CELLS (from Friedenwald and Sigelman, 1953)

FIGURE 5. TYPICAL DOSE-SURVIVAL CURVES FOR MAMMALIAN CELLS EXPOSED TO X-RAYS AND FAST NEUTRONS (from Hall, 1988)

FIGURE 6. CHARACTERISTIC SEQUENCE OF EVENTS IN THE PATHOGENESIS OF NONSTOCHASTIC EFFECTS OF IONIZING RADIATION (from Upton, 1996).

FIGURE 7. DOSE-RESPONSE RELATIONSHIP FOR RELATIVE RISK OF CANCER, ALL TYPES COMBINED, EXCLUDING LEUKEMIA, IN ATOMIC BOMB SURVIVORS, 1958-1994 (from Pierce and Preston, 2000). The data represent age-specific cancer incidence rates in irradiated survivors relative to those in non-irradiated survivors, averaged over the follow-up period and over sex, and for exposure at age 30. The straight line represents the linear risk estimate computed over the 0-2 Sv dose range, and the dashed curves represent ± 1 standard error for the smoothed curve.

FIGURE 8. THE FREQUENCY OF SEVERE MENTAL RETARDATION IN PRENATALLY IRRADIATED A-BOMB SURVIVORS IN RELATION TO THE DOSE AND GESTATIONAL AGE AT THE TIME OF IRRADIATION (from Otake et al, 1987)

FIGURE 9. MEAN IQ SCORES IN RELATION TO GESTATIONAL AGE AND FETAL DOSE IN PRENATALLY IRRADIATED ATOMIC BOMB SURVIVORS (from NAS, 1990)

FIGURE 10. RETINAL IRRADIANCES (EXPOSURE DOSE RATES) FROM REPRESENTATIVE LIGHT SOURCES (from Sliney and Wolbarsht, 1980)