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RADIATION, ENVIRONMENT AND CANCER – A CHALLENGE FOR BIOPHYSICAL RESEARCH

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Cancer and the environment

Increasing environmental pollution is accompanied by a growing concern over the threat of cancer incidence reaching endemic proportions. fig. 1 illustrates the atmospheric pollution due to emission from traffic, industry and domestic heating in a preferential tourist area, i. e. Province of Salzburg, Austria, renowned for its hitherto high quality of environmental conditions (GALLER, 1985). For comparison, in Fig. 2 the development of cancer mortality figures for Austria in the twentieth century is shown for countries with similar socio-economic characteristics, such as Switzerland, Sweden and the Federal Republic of Germany (UICC, 1966). It can be seen that even comparing the period of the 1960's – when environmental conditions were favourable compared to those in the 1980's – with the beginning of the century or the 1930's there is a significant overall increase in each country, with Austria mostly taking the lead. A comparison of the 1960 with 1980 shows a further significant in cancer mortality increase of cancer deaths, which causes at present 10 Austrians to die of lung cancer daily (KLIMA et al., 1985); for comparison: traffic accidents cause about three deaths daily. In the past 80 years the mean life expectancy has increased thereby increasing the chance for cancer expression; also diagnostic methods for cancer detection have improved significantly. Both factors contributing to an overall increase of cancer incidence figures.

However, there is still a high portion of the remaining cancer deaths, that may be associated with the increased deterioration of environmental conditions. Due to the long latency period many of the cancer tumors of today were actually induced by production of industrial carcinogens in the early sixties (DAVIS et al., 1978).

Over the past 40 years there has been the appearance of a new environmental agent, ionizing radiation, which causes great concern in the public about its overall contributions to the observed increasing cancer incidence. In the following the risk for cancer induction due to exposure to ionizing radiation is assessed and compared to other non-radiation risks.

Radiation sources and doses

The main sources of exposure of man to ionizing radiation and the resulting mean effective dose equivalent for a typical Austrian citizen are given in Fig. 3. The largest component (about 60–70%) is represented by the natural radiation environment (NRE), of which the gaseous radon (Rn 222) and its solid decay products cause the largest single radiation burden in parts of the respiratory tract. The second largest contributor to the total dose is due to medical application of ionizing radiation for diagnostic or therapeutical purposes (about 25–30% of total).

All other sources, e. g. fallout contribution from nuclear weapon tests, nuclear power production or occupational radiation exposure contributed between 1% and 10% of the total dose for the period before the Chernobyl-reactor accident.

Radiation–induced biological effects

At the cellular level there are several theoretical experimental indications that a radiation–exposed cell is capabel of reacting in multiple ways to potential damage caused by the subcellular energy deposition (PFALLER et al., 1979; STEINHÄUSLER et al., 1980; STEINHÄUSLER 1979; ECKL et al., 1981; STEINHÄUSLER et al., 1981; STEINHÄUSLER et al., 1983). Fig. 4 shows schematically the various substages during the three main phases (transformation, promotion, progression) of the development of a radiation-exposed normal cell into a tumor cell. As can be seen there is the possibility at several stages for the elimination of a damaged cell before it can turn into the final progression stage of a tumor cell. As long as the internal cellular repair capacity is not damaged itself, cell recovery remains a realistic possibility.

However, in applied radiation protection cellular repair is not taken into consideration. In a conservative approach it is assumed that linear extrapolation for biological effects observed at high dose levels can be applied to low dose levels, i. e. only at zero dose also the induced risk equals zero.

Theoretical risk assessment

Any hazardous activity can result in an undersirable situation, which ranges from damage to property to loss of life. The probability of these events to actually occur is assessed in risk calculation. The objective of any risk assessment is to provide a quantitative numerical base for a future prediction, using data from past experiences.

The public perception of risk can differ significantly from its actual magnitude (Fig. 5; LICHTENSTEIN et al., 1978). A comparison of several risks of every day life shows that risks considered as "normal" (e. g. cancer, stroke) tend to be underestimated by the public (i. e. below the "true" line) as compared to the actual risks, whilst "abnormal" risks, such as "tornado" or "flood" are regarded as more dangerous than they actually are from disaster statistics (i. e. above the "true" line).

In order to carry out a scientifically sound risk assessment for radiation-induced cancer a multitude of dada is needed from several different categories, such as radia-

tion physics, radiobiology, as well as demoscopical and medical characteristics of the population investigated (Fig. 6). Each data set is associated with a varying degree of uncertainties.

Taking all uncertainties into consideration Fig. 7 compares the results of reviews of international groups of experts (US-Academy of Sciences, BEIR Committee, and United Nations-UNSCEAR Committee) for the resulting lethal cancer risk due to exposure to low LET (Linear Energy Transfer)-radiation. Independent of the underlying mathematical concept of risk calculation based on the absolute or relative risk concept, the risk values for additional lethal cancer cases range from 0.7 to $1.8/10^6$ person-Sievert (Sv). Assuming a population of one million persons exposed to 1 mSv, this means, that theoretically about 12 additional lethal cancer cases will be due to the absorbed radiation dose, but at the same time more than 100.000 "normal" cancer cases will be observed, which are not related to the past radiation exposure at all.

Similar magnitudes of risk are valid for radiation-induced genetic effects. Amongst one million living births with a past radiation exposure of 10 mSv/generation will be about 1.100 cases of births with genetic defects in addition to an accepted "spontaneous" rate of genetic anomalies of 107.000 cases in the same population (Fig. 8).

Applied risk assessment

Fig. 9 compares the radiation burden to the general public from the two main sources natural radiation environment (NRE), and medical practices vs. nuclear power production. As can be seen the median dose equivalent (per caput value) from nuclear power production is a marginal fraction of the gonad NRE-dose value. The high value for the skin dose from chest X-ray is of less biological significance since skin is known to be very radiation insensitive. In order to compare the biological significance of the dose contributions of the various sources in Fig. 10 radiation-induced risks have been converted into reduction of mean life expectancy (in number of days lost) due to fatal malignancies; the same procedure has been applied to non-radiation risks (INHABER, 1979; NRC, 1975; POCHIN, 1980; HILL, 1981; COHEN, 1981). Generally risks, related to radiation exposure cause a reduction of the mean life expectancy significantly lower than 50 days; in the case of a theoretical accident in a nuclear power plant even known opponents of this form of energy production ("Union of Concerned Scientists", UCS) calculate less than five days lost. For comparison, "accepted" risks, like accidents in the home during spare-time activities, represent an equivalent of about 100 days lost and lifetime heavy tobacco consumption equals a loss of more than six years.

Conclusions

Increased public awareness to radiation induced risks due to technological pro cesses has resulted in a largely emotional rather than factual discussion of the threat of cancer induction.

Based on human epidemiological review as well as laboratory data from exposed animals and theoretical modelling the results of independent scientific review committees indicate that per million exposed persons theoretically about 12 additional lethal cases will be caused by a dose of one millisievert (mSv). This is the equivalent of about 0.01% of the "normal" cancer cases that will have been observed within a follow-up period of 30 years. This result is based on worst case assumptions, such as linear extrapolation from effects observed at high doses down to low environmental doses.

Comparing the risks from radiation with several other "accepted" risks from every-day hazards reveils that, e. g. the typical dose experienced due to natural radionuclides in the environment equals a loss of eight days from the mean life expectancy, whilst the habit of heavy cigarette consumption equals a loss of over six years as compared to a loss of 2 days for an assumed nuclear rector accident.

Whilst a sensibilisation of the public by media and environmentalists to man-made risks is generally desirable, it should be stressed, that the decision making process about acceptance or rejection of certain technologies should be based on quantitative objective arguments rather than emotions in order to enable an optimised solution for the resulting impact on man and the environment.

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FIG. 1

ANNUAL EMISSION OF POLLUTANTS IN SALZBURG, AUSTRIA.

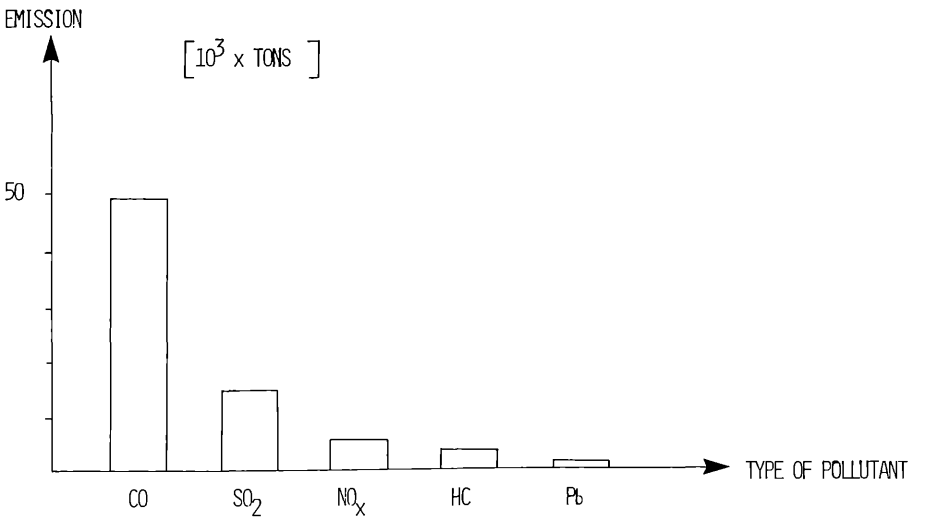


FIG. 2

COMPARATIVE RATES OF MORTALITY FROM CANCER PER 100 000 INHABITANTS IN DIFFERENT REGIONS BETWEEN 1900/1902, 1928/1930 AND 1958/1960.

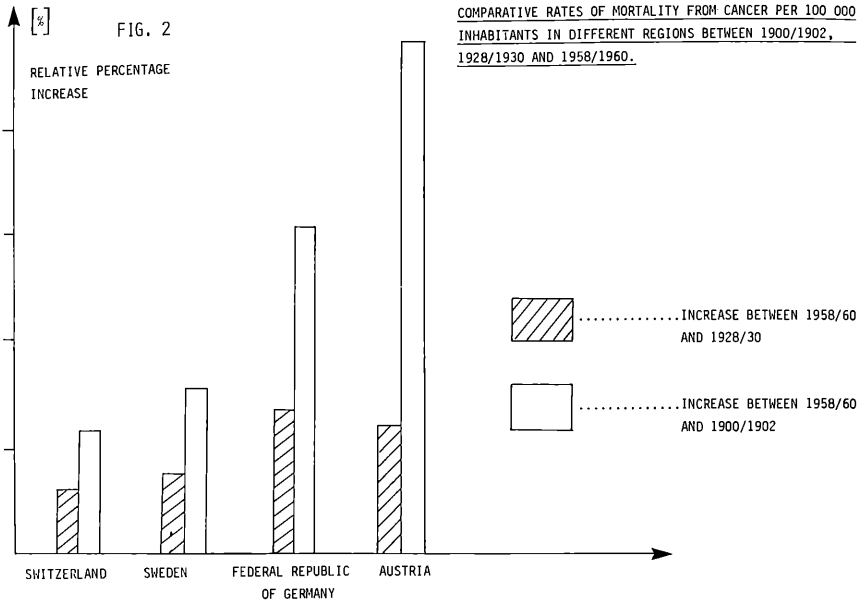
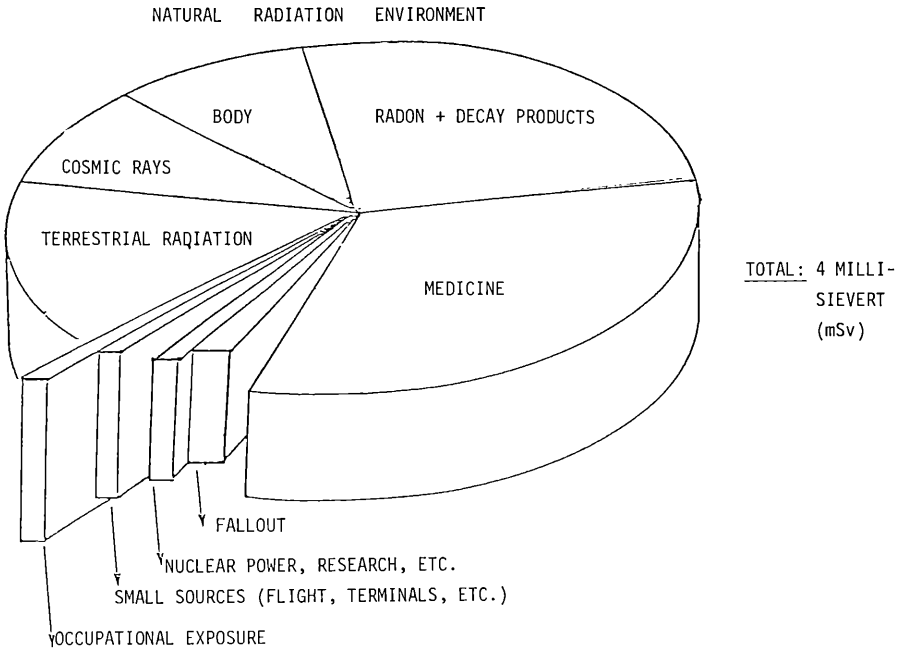


FIG. 3

MEAN ANNUAL DOSE OF AUSTRIAN CITIZEN (EFFECTIVE EQUIVALENT DOSE)



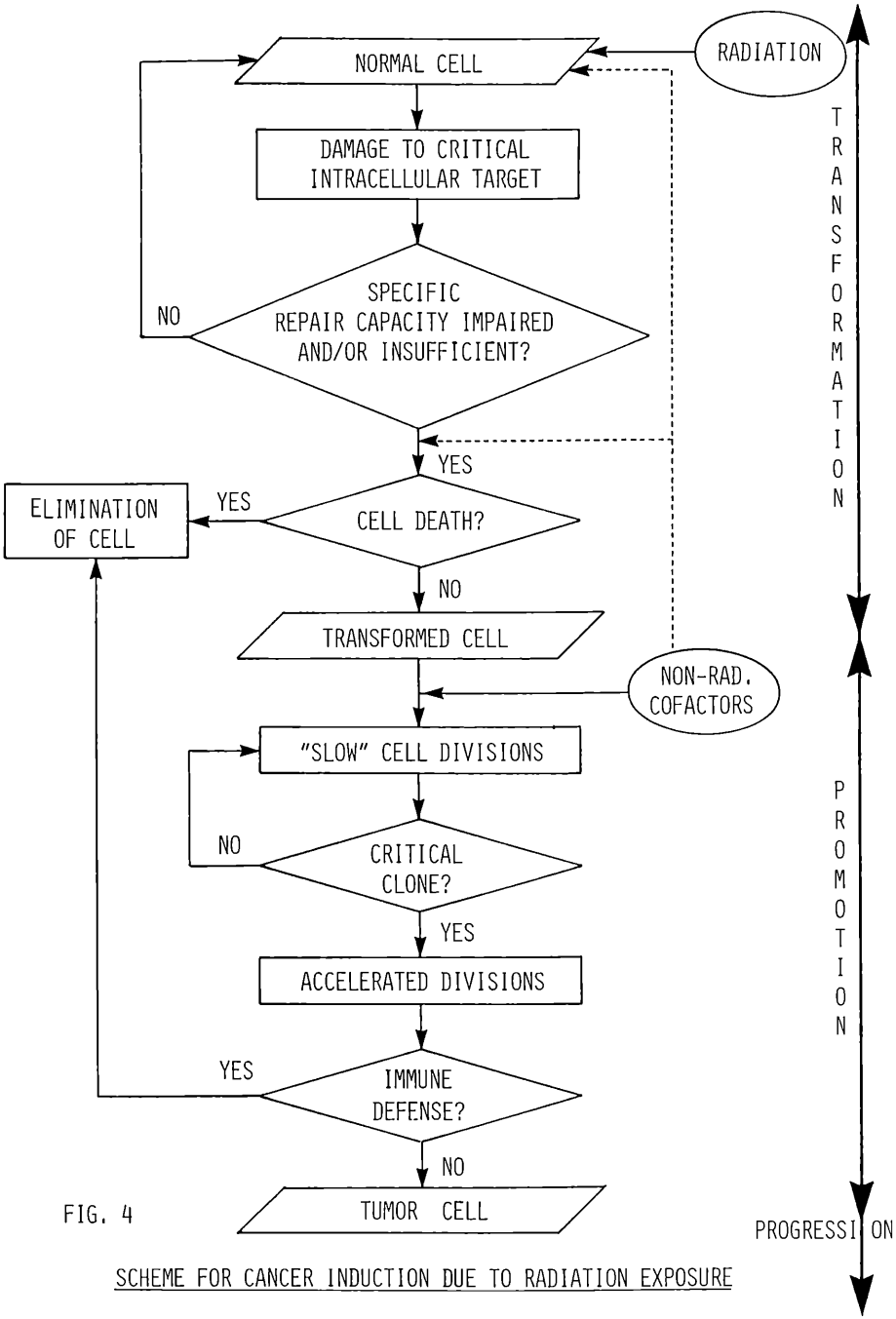
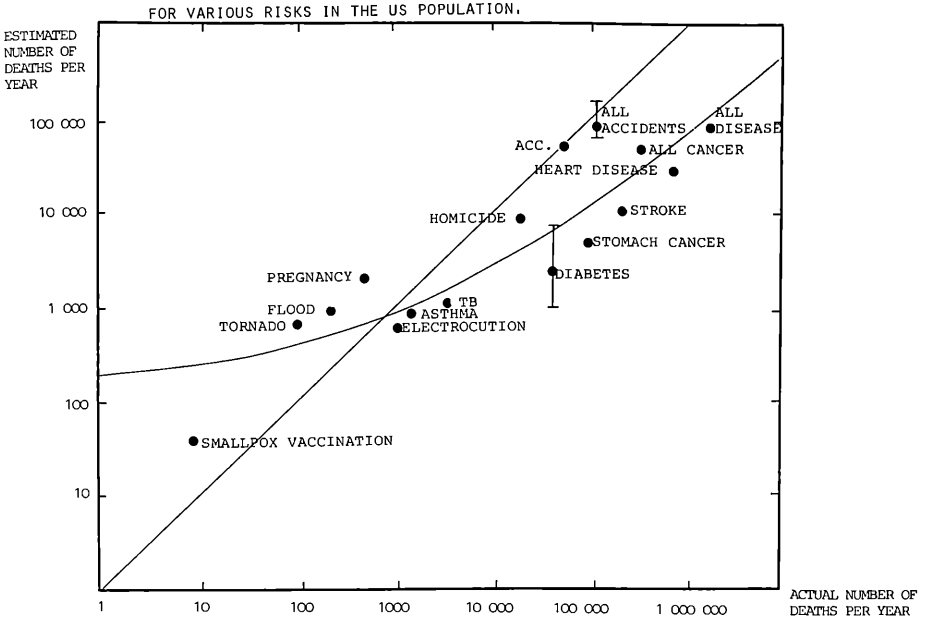


FIG. 4

SCHEME FOR CANCER INDUCTION DUE TO RADIATION EXPOSURE

FIG.5 COMPARISON OF ACTUAL AND ESTIMATED NUMBER OF DEATHS PER YEAR FOR VARIOUS RISKS IN THE US POPULATION.



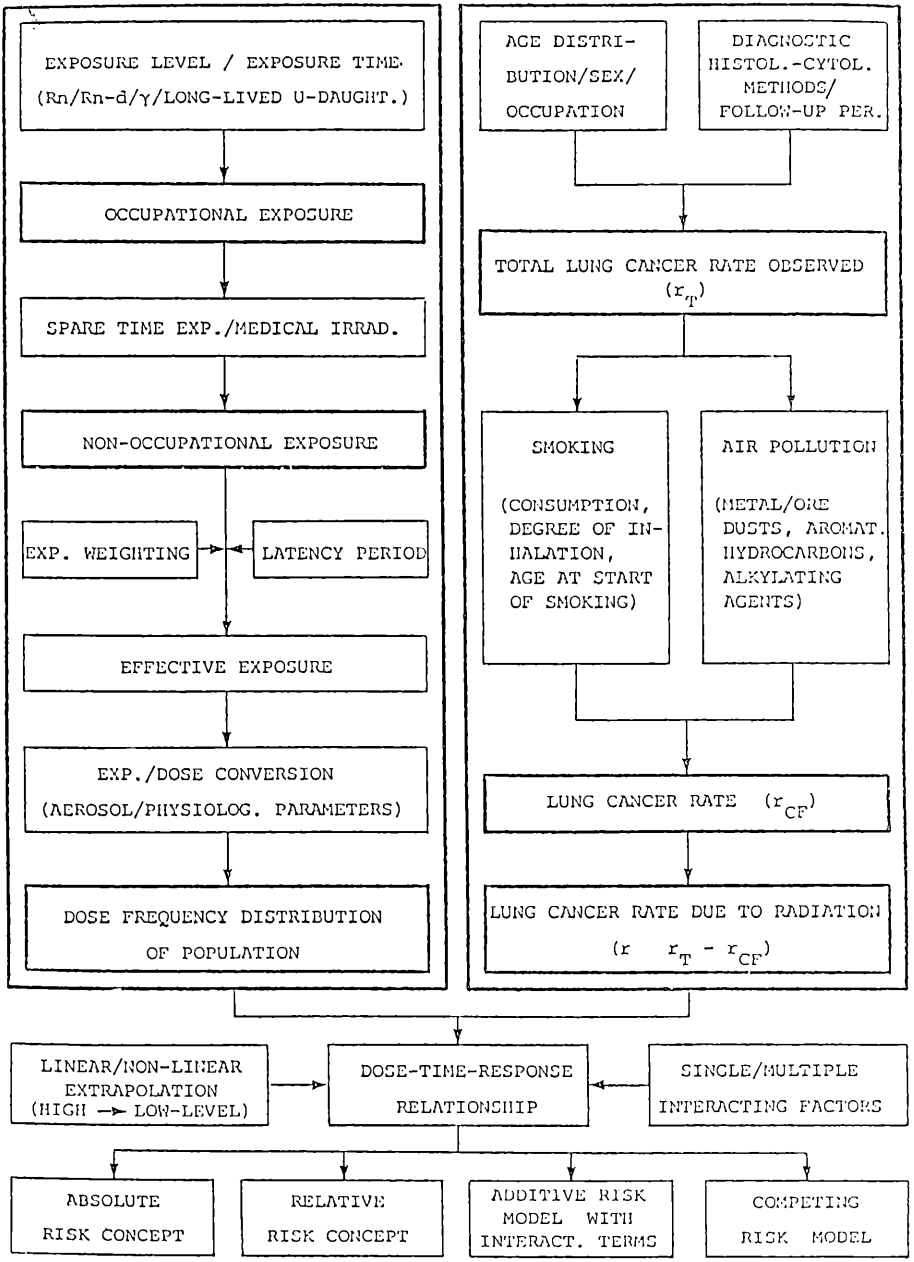


FIG. 6: SCHEME OF RADIOLOGICAL, DEMOSCOPICAL AND MEDICAL DATA NEEDED FOR CONTROL AND TEST POPULATIONS FOR RISK ASSESSMENT OF LUNG CANCER INDUCTION DUE TO RN DAUGHTER EXPOSURE.

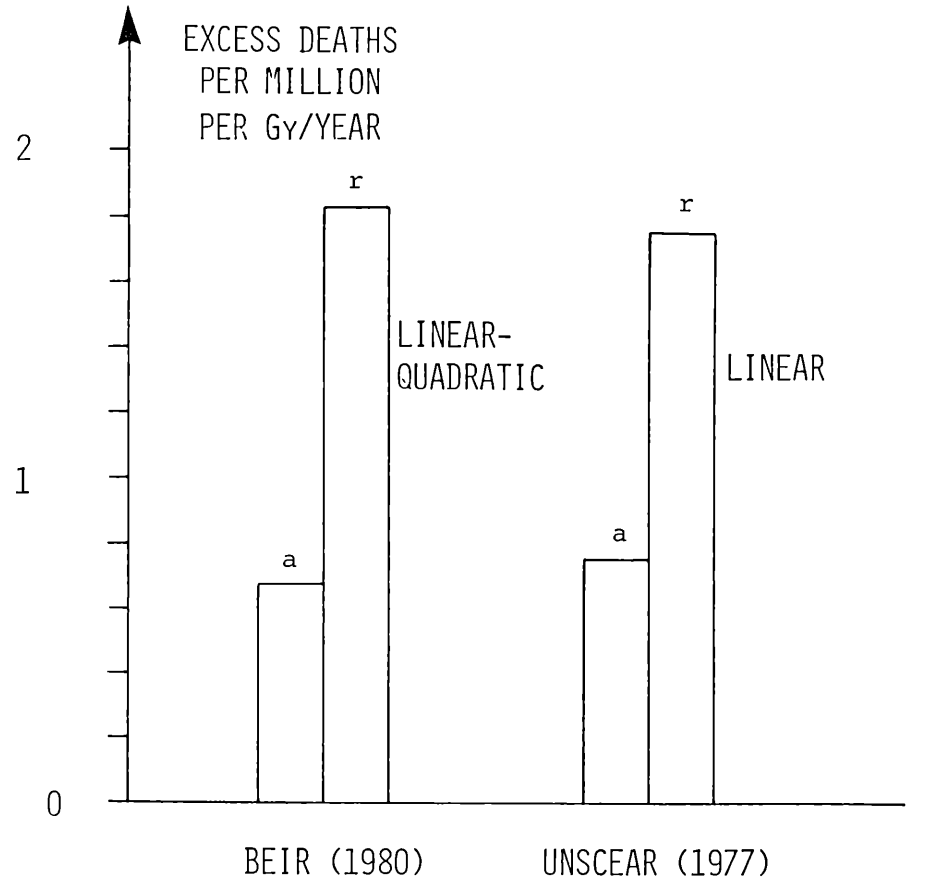


FIG. 7 COMPARISON OF LIFETIME RISK OF CANCER MORTALITY INDUCED BY LOW-LET RADIATION

a ABSOLUTE RISK CONCEPT

r RELATIVE RISK CONCEPT

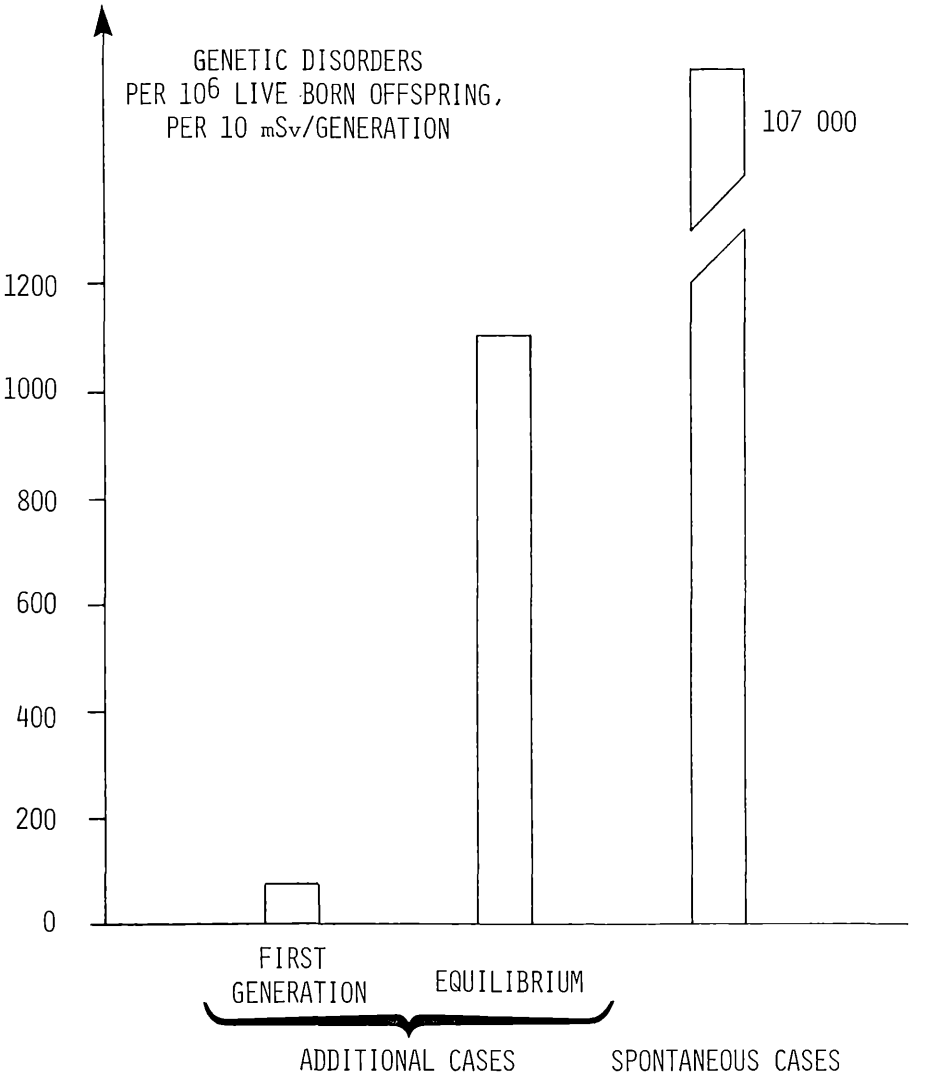


FIG. 8 GENETIC EFFECTS OF AN AVERAGE POPULATION EXPOSURE OF 10 mSv PER 30-YEAR GENERATION (BEIR, 1980)

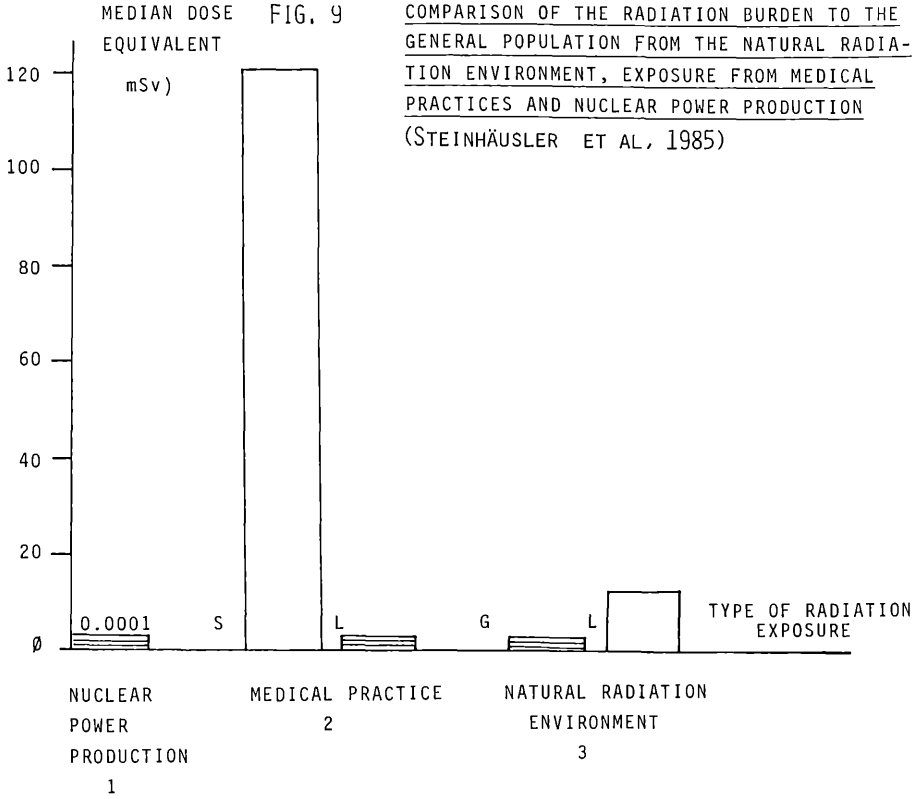


FIG. 9 COMPARISON OF THE RADIATION BURDEN TO THE GENERAL POPULATION FROM THE NATURAL RADIATION ENVIRONMENT, EXPOSURE FROM MEDICAL PRACTICES AND NUCLEAR POWER PRODUCTION (STEINHÄUSLER ET AL, 1985)

1 ANNUAL PER CAPUT VALUE, BASED ON 80 $GW_{(e)}$ ANNUALLY GENERATED NUCLEAR POWER.

2 DOSE PER CHEST X-RAY (RADIOGRAPHY) FOR SKIN (S) AND LUNG (L).

3 DOSE FOR CITIZEN OF SALZBURG (G= GONAD DOSE, L BASAL CELLS LUNG DOSE).

FIG.10 REDUCTION OF LIFE EXPECTANCY DUE TO RADIATION - INDUCED AND OTHER RISKS