Reprint from

"BIOLOGICAL EFFECTS
OF LOW-LEVEL RADIATION"

INTERNATIONAL ATOMIC ENERGY AGENCY

EVALUATION OF LOW-LEVEL EFFECTS IN THE JAPANESE A-BOMB SURVIVORS AFTER CURRENT DOSE REVISIONS AND ESTIMATION OF FALLOUT CONTRIBUTION

I. SCHMITZ-FEUERHAKE, P. CARBONELL
Department of Physics,
University of Bremen,
Bremen, Federal Republic of Germany

Abstract

EVALUATION OF LOW-LEVEL EFFECTS IN THE JAPANESE A-BOMB SURVIVORS AFTER CURRENT DOSE REVISIONS AND ESTIMATION OF FALLOUT CONTRIBUTION.

An evaluation of the physical conditions and biological effects in the A-bomb survivors who were far away from the explosions or not in city (NIC) demonstrates, as other authors have stated, that the contribution of fallout to the low-dose groups in Hiroshima and Nagasaki must not be neglected. Therefore the former controls, i.e. the groups 0–9 rad T65D and NIC of the ABCC-RERF Life Span Study including 81 500 persons present themselves as the largest investigated collective showing significantly low-LET effects in the low-dose range. The mean internal dose was estimated to be above 4 rad and below 25 rad. Risk estimates to be gained are 30–300 $\times 10^{-6}$ rad^{-1} for leukaemia and 180–1800 $\times 10^{-6}$ rad^{-1} for all cancer mortality.

1. INTRODUCTION

Low-level effects in the A-bomb survivors have formerly been attributed to the neutrons of the Hiroshima bomb. Recognizing these as low-LET effects after current dose revisions, the question of a fallout contribution to the doses is new. If the fallout cannot be neglected it must be concluded that irradiated controls were used in the ABCC-RERF sample, i.e. the groups NIC (not in city), <1 rad T65D,[1] or 0–9 rad T65D, the latter at last being chosen as a control by the BEIR III Committee [1]. Evaluating the late effects since 1974 the RERF used another method to estimate the excess cases per rad [2, 3]: the observed rates in several dose classes were fitted to a dose-response model including a dose-independent background. This will again lead to a suppression of low-dose effects and underestimation of risks if an additional influence of fallout is valid.

The contribution of fallout can be demonstrated by several biological effects observed in the lower dose classes of the survivors. To derive values for the fallout

[1] 1 rad = 1.00 $\times 10^{-2}$ Gy.

doses three sources of information were used: (1) physical considerations, (2) conclusions by the dose response observed for prominent late effects and (3) internal dose estimations by chromosome aberrations.

2. INDICES SHOWING THE CONTRIBUTION OF FALLOUT

2.1. Typical late effects in the groups 0–9 rad T65D and NIC

As was pointed out by Baum [4] there are significant effects in all low-dose groups of A-bomb survivors if compared with the Japanese national rate. (This is true also in the case of Nagasaki, where a situation of pure low-LET effects was suggested; see Fig.1.) A profile of standardized mortality and incidence rates related to the national rate is shown for 0–9 rad and NIC of the Life Span Study in Fig.2. Effects known to be typically radiation-induced such as leukaemia and cancer of the breast and lungs show significant elevation; extremely elevated are thyroid carcinomas.

The upper part of Fig.2 shows values gained by the ABCC report of Moriyama and Kato [5–10] which refers to the investigations up to 1972. After that date expected values for cancer derived by the national statistics were not published by the ABCC-RERF. The national rate refers to tumour registers in Japan of 1962–64 and 1966 carried out in two regions of 3.4 million inhabitants altogether.

The group NIC shows no significant elevation in the case of leukaemia but only 4 500 of 26 500 persons were early entrants (entered the cities within three days after explosion). Six cases of leukaemia were found in the early entrants up to 1978, which is about twice the normal rate (see point "EE" in Fig.2). (The

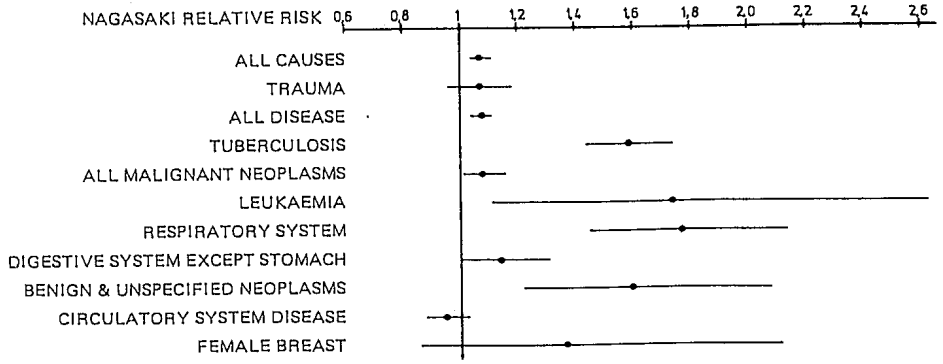

FIG.1. *Standard mortality and incidence rates for cancer and other causes compared to national rates for group 0–9 rad T65D in Nagasaki 1950–72, from* [5].

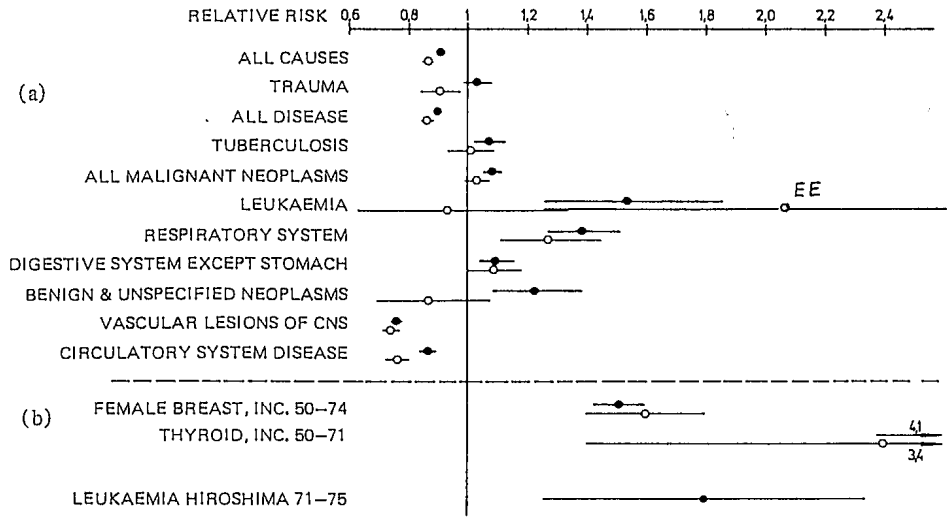

FIG.2. Standard mortality and incidence rates for cancer and other causes compared to national rates for controls in Hiroshima and Nagasaki:
—●— group 0–9 rad T65D; —○— group NIC.
(a) mortality up to 1972 (and standard deviations) from ABCC [5], *except EE;*
(b) breast cancer incidence [6], *thyroid cancer* [7, 8], *leukaemia 1971–75* [9];
EE = leukaemia in early entrants.

RERF authors, however, compare these cases to the groups <1 rad and the late entrants and maintain they find no increase.)

Japanese investigators found elevated leukaemia rates in a greater collective of early entrants [11, 12]. The data of Hirose [12] are taken from the tumour registries of Hiroshima and Nagasaki up to 1968. Forty-five cases of leukaemia were found in 25 798 early entrants of Hiroshima, which corresponds to about 3.7 times the Japanese normal rate.

2.2. Lack of correlation between estimated T65D and effects in the case of thyroid carcinoma

All kinds of prominent radiation-induced cancer in the A-bomb survivors show a strong correlation to T65D except for thyroid carcinoma; see values in Fig.3 gained from [7]. This is in contradiction to other findings about thyroid cancer induced by low-LET radiation where an approximately linear dose response can be derived between 7 and 1000 rad [13].

2.3. Chromosome aberrations in the groups <1 rad T65D and NIC

Chromosome studies in A-bomb survivors have been carried out since 1967. Sasaki and Miyata [14] derived dose values and compared them with the T65D

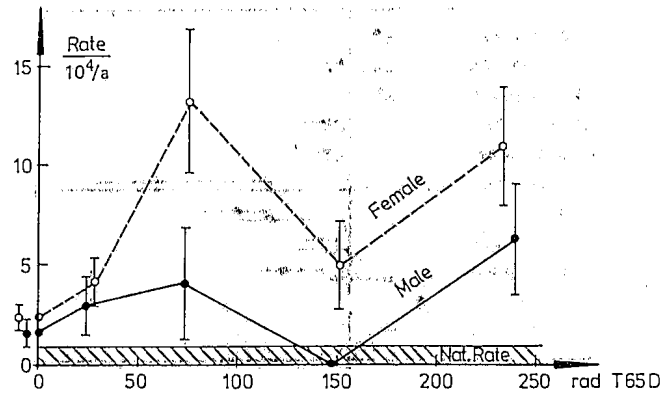

FIG.3. Incidence of thyroid carcinoma in A-bomb survivors, Hiroshima and Nagasaki 1958–71, from [7].

(Fig.3). Nineteen persons had been a greater distance than 2.4 km from the hypocentre, which means <1 rad after T65D and <5 rad after LLNL (Lawrence Livermore National Laboratory dose estimation 1980). The internal dose, however, was found to be from 2–30 rad (Fig.4), the mean being 8 rad.

Awa et al. [15] studied chromosome aberrations from 1968 to 1971. Four hundred and nineteen persons of the groups <1 rad T65D and NIC were used as controls. The aberration frequency in controls, however, was much higher than would be expected from worldwide measured spontaneous rates. A mean rate of $2.4 \pm 0.3 \times 10^{-3}$ was found for dicentrics, which is 4.4 times a control value of 0.55×10^{-3} recommended by Lloyd et al. [16].

2.4. Epilation and other acute effects in persons at greater distance from the explosion

Yamada and Jones demanded more exact investigations of fallout effects in Hiroshima and Nagasaki in 1972 [17]. They studied acute radiation effects in persons who had resided in the region of black rain. Persons at a distance greater than 1.6 km from the hypocentre were chosen as a control, which corresponds to <20 rad T65D. In these they registered epilation in 4.5% and so-called minor symptoms in 2.9% and major symptoms in 6.3% of cases. This may only be explained by a heavy burden of fallout.

2.5. Microcephaly

A very high rate of microcephaly was found in Hiroshima: 11% in the group <5 rad and 17% in the group 5–10 rad of dose received by the embryo estimated

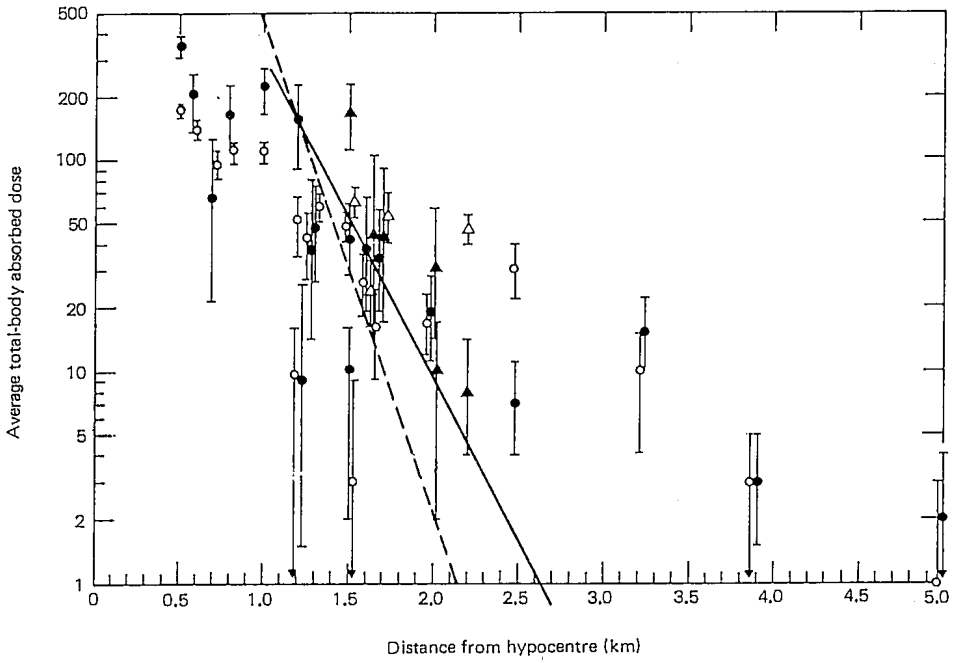

FIG.4. Whole-body dose by chromosome aberrations in A-bomb survivors in relation to the distance from hypocentre in Hiroshima, from Sasaki and Miyata [14]. *(In addition the Lawrence Livermore National Laboratory (LLNL) dose estimate of 1980 is shown.)*

from T65D [1]. This must be attributed to nearly pure low-LET radiation. Similar low-level effects were not seen in studies after medical irradiation [1].

3. ESTIMATION OF FALLOUT RADIATION AND INTERNAL DOSE

3.1. Physical considerations

The fallout dose for the group 0–9 rad T65D by external radiation is estimated to be 10–20 rad according to published data about local dose-rate measurements and rainout conditions [18]. Measurements of incorporated nuclides have not been carried out for the years before 1969 in the Nishiyama region of Nagasaki, where the highest contamination occurred [19]. The doses were found to be negligible at that time with regard to an influence of the dose response. Therefore, indirect sources of information may be more relevant to judge a possible contribution of internal radiation.

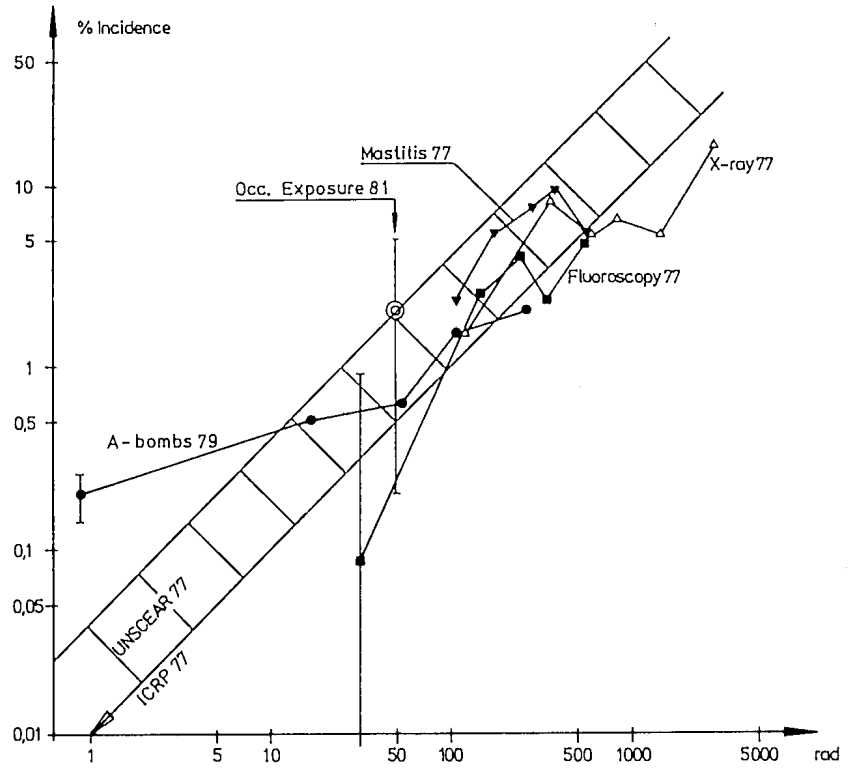

FIG.5. Radiation-induced breast cancer; age of women at irradiation >10 a: from Refs [25–28].

3.2. Dose-response extrapolation

The excess against the national rate for cancer in the cohort 0–9 rad T65D demonstrated in Section 2.1 leads to an over-linearity of the dose response in the lower dose range for all prominent malignancies [20]. An example is shown in Fig.5 for breast cancer, which is generally accepted to show a linear non-threshold dose dependency. This was recently confirmed by findings of Baverstock et al. [21] for dial painters irradiated by chronic low γ-doses (see "Occ. Exposure" in Fig.5). In order to estimate an internal dose by dose-response extrapolation for the group 0–9 rad T65D an analysis for four age classes of <40 a ATB (for which linearity was confirmed [6]) was made. This results in a mean breast dose of 10–20 rad.

For the case of leukaemia we refer to Gofman [22], who derived an increase of 17% per rad (T65D). This will lead to a marrow dose of 4–10 rad in the group 0–9 rad T65D.

3.3. Chromosome aberrations

Chromosome aberrations could be suggested to be the most reliable source of information about a fallout burden because the internal dose is measured directly. The investigated sample, however, was small and the time between irradiation and measurement very long.

The collective of higher size was investigated by Awa [23]. Symmetrical aberrations (dicentrics and rings) showing an effect in the group <1 rad and NIC, however, are known to decrease with time. The excess would correspond to a dose of 0.3 rad after Lloyd et al. [16] if there had been no delay. Randolph and Brewen [24] estimated a reduction factor of 79 after 23.5 years, which would mean a dose of 24 rad. Uncertainties, of course, are high in this case. Recent conclusions of Awa continuing the former investigations are that the dose of survivors of the distances 2–4 km – i.e. <1 rad T65D – amounts to some rad [23].

Sasaki and Miyata (Fig.4) derived the internal doses by using a time-independent ratio of aberrations. Only a few persons <1 rad were examined, but the tendency of the whole dose response allows a rough estimation about the supposed excess dose at great distances (about 10 rad).

More exact conclusions from aberration frequencies in survivors are to be expected. Published data serve as proof in this context that the estimated additional doses from 3.1 and 3.2 may not be exceeded tremendously.

4. LOW-LEVEL, LOW-LET AND LOW-DOSE-RATE RISK ESTIMATES FROM THE GROUP 0–9 rad T65D AND THE HIROSHIMA/NAGASAKI TUMOUR REGISTRIES

From considerations in Section 3 it is concluded that the mean internal dose for the survivors of the group 0–9 rad T65D (20 rad after LLNL) probably exceeds 4 rad and lies below 25 rad.

The NIC group is not included for risk evaluation because it is inhomogeneous and the fallout conditions (and residual radiation from neutron activation in the centre of the cities) are difficult to judge.

In the RERF sample 70 cases of leukaemia were registered in the period 1950–78 [10] for the group 0–9 rad, which leads to a relative risk against the national rate of 1.8 and an absolute risk of 761×10^{-6} per dose. The absolute risk for the mentioned dose range would then lie from $30–190 \times 10^{-6}$ rad^{-1}.

If one assumes a six-fold rate for all cancer mortality compared to leukaemia after UNSCEAR 1977 [25], an all-cancer mortality of $180–1140 \times 10^{-6}$ rad^{-1} results, which corresponds to the values of the BEIR III report [1] using linear non-threshold extrapolation.

[12] HIROSE, F., Acta Haematol. Jap. **31** (1968) 765.
[13] SCHMITZ-FEUERHAKE, I., MUSCHOL, E., BÄTJER, K., SCHÄFER, R., "Risk estimation of radiation-induced thyroid cancer in adults", Late Biological Effects of Ionizing Radiation (Proc. Symp. Vienna, 1978) Vol. 1 (1978) 219.
[14] SASAKI, M.S., MIYATA, H., Nature (London) **220** (1968) 1189.
[15] AWA, A.A., SOFUNI, T., et al., J. Radiat. Res. **19** (1978) 126.
[16] LLOYD, D.C., PURROTT, R.J., et al., Int. J. Radiat. Biol. **28** (1975) 75.
[17] YAMADA, H., JONES, T.D., An Examination of A-bomb Survivors Exposed to Fallout Rain and a Comparison to a Similar Control Population, Oak Ridge Nat. Lab. Rep. ORNL-TM-4017 (1972).
[18] CARBONELL, P., Dissertation, University of Bremen, in preparation.
[19] OKAJIMA, S., TAKESHITA, K., "Effects of the radioactive fallout of the Nagasaki atomic bomb", Atomic Bomb Casualty Commission, Hiroshima, Rep. 12–75 (1975).
[20] SCHMITZ-FEUERHAKE, I., BÄTJER, K., MUSCHOL, E., Fortschr. Röntgenstr. **131** (1979) 84.
[21] BAVERSTOCK, K.F., PAPWORTH, D., VENNART, J., Lancet, Feb. 21 (1981) 430.
[22] GOFMAN, J.W., "Radiation and human health", San Francisco (1981).
[23] AWA, A.A., personal communication, 1982.
[24] RANDOLPH, M.L., BREWEN, J.G., Radiat. Res. 82 (1980) 393.
[25] UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION, Sources and Effects of Ionizing Radiation, 1977 Report to the General Assembly, United Nations, New York (1977).
[26] BOICE, J.D., MONSON, R.R., J. Natl. Cancer Inst. **59** (1977) 823.
[27] SHORE, R.E., HEMPELMAN, H., et al., J. Natl. Cancer Inst. **69** (1977) 813.
[28] BARAL, E., LARSSON, L.E., MATTSON, B., Cancer (Philadelphia) **40** (1977) 2905.

Printed by the IAEA in Austria