

## The properties and health hazards from early nuclear weapon fallout: The Castle Bravo incident revisited

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**Abstract** – Early fallout is defined as the fallback to the earth’s surface of radioactive particles shortly after a nuclear detonation (often arbitrarily defined within 24 h). At the difference of wide spreading global fallout, early fallout mainly consists of larger particles that are often visible. The initial mixture is rich in short- and very short-lived radionuclides associated with a very high initial activity that decreases rapidly (in 7 h, the dose rate is reduced by 90%). The main danger of early fallout results from external irradiation by highly penetrating gamma-radiation that may cause acute radiation sickness. Only in the case of the thyroid, internal irradiation by the incorporation of radioiodine may prevail. The bombings of Hiroshima and Nagasaki are examples of airbursts with many fatalities by prompt effects (blast, burns, and initial ionizing radiations), but they produced little fallout. The nuclear test code-named Castle Bravo on the Marshall Islands (1954) did not have casualties by its blast, thermal or initial radiation effects, but the inhabitants of the nearby islands and the crew of a Japanese fisherboat (Lucky Dragon) were affected by large amounts of fallout. For the inhabitants of the Rongelap Atoll, the average dose from external irradiation was assessed at 1.6 Gy. From a clinical point of view, based on hematological data using the METREPOL classification system, the acute radiation syndrome can be categorized as mild (H1). Blood transfusions were not required, and antibiotics were not administered for prophylaxis or therapy of infections related to irradiation. The equivalent dose received by the thyroid resulted mainly from internal irradiation with 7.6 Gy. The major late effects were thyroid abnormalities, including thyroid failure, nodules, and malignant tumors. The 23 Japanese crewmen seem to have been irradiated by higher doses (2.9 Gy). Compared to the hematological data of the Rongelap victims, the evolution pattern over time is quite similar. Still, the absolute values of the cell counts are lower, and on average, the acute radiation syndrome can be categorized as rather moderate (H2). Considering the individual cases, data show a large interindividual variability, and the clinical severity category ranges from “no alterations” (H0) to severe (H3). Victims were treated with repeated blood transfusions and antibiotics. Several of them developed jaundice, and one of them died six months after the incident showing symptoms compatible with subacute liver failure. A radiochemical organ analysis revealed that only the bones were clearly contaminated with fission products. In the 1990s, many surviving crewmen were diagnosed with hepatitis C, incurred probably from blood transfusions that were often contaminated at the time, and died from hepatocellular carcinomas. Thyroid dysfunctions were not reported. The Castle Bravo case permits to study the health hazards resulting from early fallout independently from the prompt effects of a nuclear detonation. The prevailing external irradiation was confirmed, except for the thyroid with a higher dose resulting from radioiodine incorporation mainly caused by ingestion. As shown for the Japanese fishermen, the risks incurred by medical treatments must be carefully weighed against the benefits of the therapeutic intervention. The cause of death of the only short-term fatality is not fully elucidated, but is consistent with liver failure due to transfusion hepatitis rather than radiation effects.

**Keywords:** nuclear weapons / fallout / acute radiation sickness / thyroid dysfunction / Castle Bravo / Lucky Dragon

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## 1 Fallout from nuclear detonations

The energy released by the detonation of a nuclear weapon is distributed to the blast and shock wave, thermal radiation, initial ionizing radiation (“prompt effects”), and residual ionizing radiation (Glasstone and Dolan, 1977). The residual radiation is due to neutron activation products from the initial radiation on the ground in the area near ground zero, but mainly to radioactive fallout. Fallout is defined as “the fallback to the earth’s surface of particles contaminated with radioactive material from the radioactive cloud.” The early (or local, up to roughly 500 km) fallout is arbitrarily defined as the fallout within 24 h after the nuclear detonation. It consists mainly of larger particles, whereas global fallout with its smaller particles is brought back to earth over large areas and extended periods of time (months to years) (Greene, 1965; Simon *et al.*, 2006).

The weapon debris with the fission products are the main constituents of this radioactive mixture (Glasstone and Dolan, 1977). Shortly after detonation, these products consist of more than 300 nuclides from about 36–40 elements (Glasstone and Dolan, 1977; Wohni, 1995). The initial mixture contains a majority of short- and very short-lived nuclides responsible for the very high initial activity in the nuclear cloud (Glasstone and Dolan, 1977; Buddemeier, 2018). This results in a rapid change in the composition of the mixture over time and a rapid decay of activity (7–10 rule of thumb: every seven h-fold factor of time, there will be a 10-fold decrease of the dose rate) (Buddemeier, 2018). This is an important difference between early and global fallout or the release of radioactive material from a nuclear plant accident with much less short-lived nuclides and thus a lower activity. The main danger of early fallout results from external irradiation by highly penetrating gamma-radiation with a high dose rate that may cause acute radiation sickness (Buddemeier, 2018). In contrast, the main hazard from global fallout results from radioactivity incorporation leading solely to stochastic radiation damages with long-term health effects like the occurrence of leukemia or solid cancers (Simon *et al.*, 2006).

Early fallout occurs in particular after surface or near-surface bursts, with the fireball touching the ground. Large amounts of earth material will be vaporized and drawn into the nuclear cloud by a chimney effect and mixed with the radioactive debris (Glasstone and Dolan, 1977). The bombings of Hiroshima and Nagasaki are examples of airbursts at relatively high altitudes for the yields of the bombs and thus produced only little fallout that was washed out by rain (Simon *et al.*, 2006). It is estimated that the contribution of this “black rain” exposure to the health effects incurred by the victims was rather minor. In Hiroshima and Nagasaki, many fatalities showed combined injuries (mechanical trauma + burns + initial nuclear radiation exposure), and studying the relatively small contribution of fallout to health damages seems almost impossible. In contrast, a 1954 nuclear test in the Marshall Islands (code name “Castle Bravo”) resulted in a massive fallout exposure of victims without blast injuries or burns, and without exposure to initial radiation (Simon *et al.*, 2006; Henriksen *et al.*, 2014). This case allows for a better understanding of the isolated health effects of early radioactive fallout.

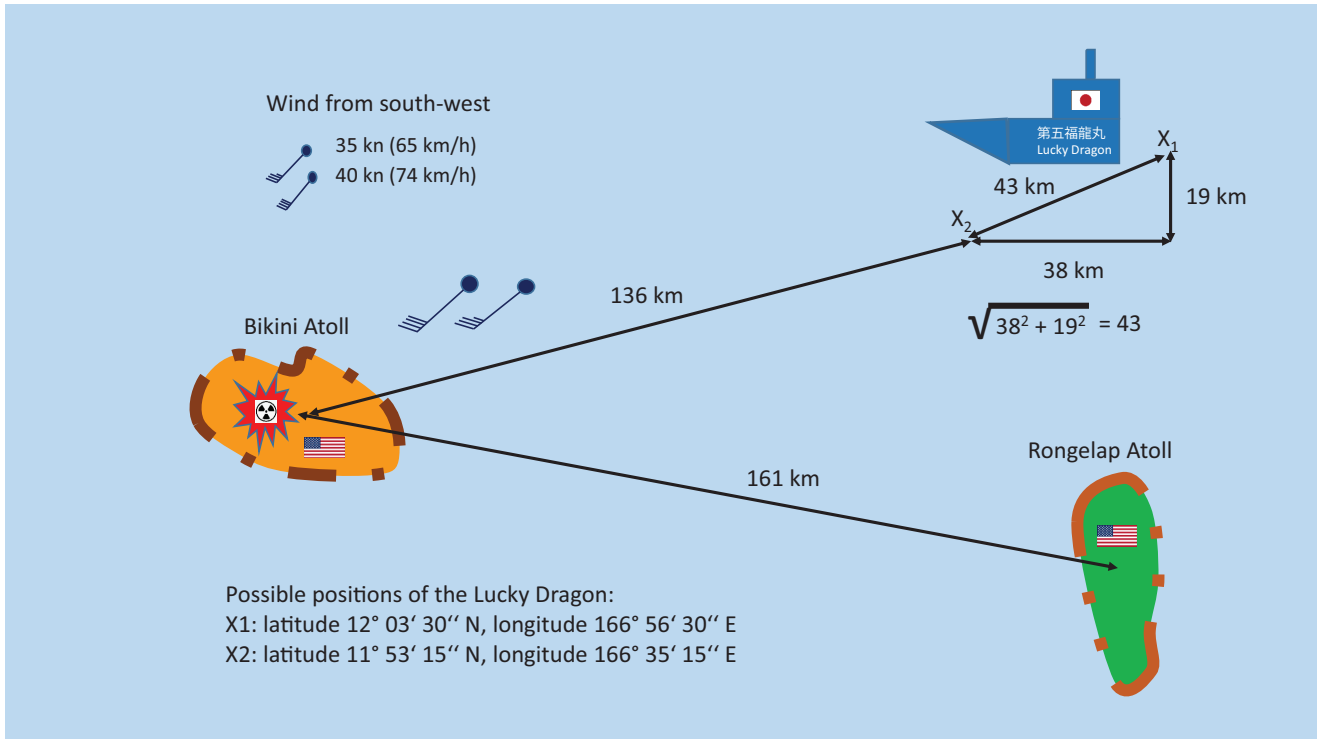
## 2 The Castle Bravo incident: general description

From 1946 to 1958, 66 nuclear tests were conducted in or near the Bikini Atoll (Simon *et al.*, 2010a). It seems that 20 tests have resulted in the deposition of measurable fallout on inhabited islands. The total explosive yield corresponds to roughly 100 Mt TNT equivalents and thus is much higher than at the Nevada test site. The largest test conducted was the detonation of a 15 Mt thermonuclear device on March 1, 1954 (code name Castle Bravo). The detonation yield was 2–3-fold greater than anticipated (Robbins and Adams, 1996). There were no casualties caused by the blast, thermal burns or the initial ionizing radiation. But due to the bursting point near the surface and unusual and unexpected meteorological conditions, the radioactive plume was driven in an eastern direction and led to an intense radioactive fallout exposure of the population of the northern atolls of the Marshall Islands, in particular the Rongelap Atoll with 64 inhabitants, Sifo Island (Ailinginae Atoll) with 18 inhabitants, the Rongerik Atoll with 28 American servicemen, but also the more distant Utirik Atoll with 157 inhabitants. Moreover, 23 fishermen of a Japanese vessel (daigo fukuryumaru, “Lucky Dragon number 5”) present in the area were heavily affected by the fallout (Oishi, 2011). In Japan, this incident has sometimes been called the “third atomic bombing of mankind” (Reischauer, 1988).

The Japanese vessel “Lucky Dragon” with 23 fishermen seems to have been struck by fallout earlier than the Rongelap Atoll (about 2–3 h after detonation) due to its position and wind direction (List, 1955; Lessard *et al.*, 1985; Yamamoto, 2009; Oishi, 2011) (Fig. 1). This is compatible with the finding that the mean particle size of the ashes falling down on the Lucky Dragon was larger than in the Rongelap Atoll (size at median activity 370  $\mu\text{m}$  versus 150  $\mu\text{m}$ ) (Suito *et al.*, 1956; Lessard *et al.*, 1985). The ship stayed on site for about 6 h, unaware of radioactivity, and then returned to Japan where it arrived after two weeks (Yamamoto, 2009; Oishi, 2011). The whole crew began to feel sick in the evening of the day of fallout exposure. After its return in Japan, the whole crew was admitted at hospitals in Tokyo with a diagnosis of acute radiation sickness (Kato, 1984). The radioactive contamination on the Lucky Dragon and the available Bikini ashes were analyzed to get information on the fallout composition (ASN, 2008; Yamamoto, 2009) (Tab. 1).

## 3 Health impact on the local inhabitants of the Marshall Islands

Among the exposed inhabitants of the Rongelap Atoll, about two-thirds developed anorexia and nausea within 48 h, and about 10% had vomiting and diarrhea (Conard *et al.*, 1980). Children under 5 years were more severely affected than adults. These symptoms lasted about two days. One-fourth of the victims complained of itching and burning of the exposed skin, eye irritations, and burns appeared after about two weeks in 90% of the cases. In 15%, ulcerations developed. Epilation was seen in about one-third of the adults and in almost all children (Robbins and Adams, 1996). The lesions healed rapidly with residual hyperpigmented plaques. The



**Fig. 1.** Schematic representation of the Bikini Atoll (detonation of the nuclear device), the Rongelap Atoll and the possible positions of the Japanese fisherboat Lucky Dragon. The latitudes and longitudes for the centers of the two Atolls were read from google maps. For the possible positions of the Lucky Dragon, the information given by [Oishi \(2011\)](#) was used. The distances between the points were calculated using the internet calculator of the National Hurricane Center and Central Pacific Hurricane Center (n.d.). The wind direction from south-west (showed by the staff parts) to north-east (showed by the dot end) and its speed (one full barb: 10 knots) are given for a height of 10–12 km using the standard symbols on weather maps.

relatively high proportion of victims showing skin symptoms is probably mainly due to superficial contamination with beta-emitting radionuclides. In addition, the caustic effects of the alkaline calcium oxides and carbonates may have contributed to the early symptomatology ([Conard \*et al.\*, 1980](#)).

The first blood samples were examined after evacuation on the 3rd day after the start of fallout exposure. Control values from 115 Marshallese from the Majuro Atoll were used. Lymphocytes dropped to 50% of controls by the third day among Rongelap inhabitants. Neutrophils dropped by 20 to 30% during the second postexposure week and reached their lowest values after about five to six weeks (–50%, lowest value < 1000/ $\mu$ l). Thrombocytopenia (1/3 of controls, lowest value 35 000/ $\mu$ l) was maximal after four weeks. Erythropoietic depression was not detected ([Conard \*et al.\*, 1980](#); [Robbins and Adams, 1996](#)).

The time course of the mean values of the white blood cells and thrombocytes among the inhabitants (over 5 years of age) of the Rongelap Atoll during the first weeks after fallout exposure is shown in [Figure 2](#). Lymphocyte values remained depressed over the whole time. The fluctuations of the total white blood cell count reflect the changes of the neutrophils reaching a minimum in the 6th week. Thrombocytes reached their lowest values in the 4th week. It must be mentioned that the mean counts calculated for each individual in the peak depression period showed a large variability (between day 39 and 51: lymphocytes: 1260–3550/ $\mu$ l; neutrophils: 1450–5170/ $\mu$ l; between day 26

to 30: platelets: 45 000–200 000/ $\mu$ l) ([Cronkite \*et al.\*, 1954](#)) (these data are means and should not be confused with single minima).

The hematological data were used to assess the severity of the acute radiation syndrome using the METREPOL system (Medical Treatment Protocol for Radiation Accident Victims) developed by [Fliedner \*et al.\* \(2001\)](#). This is a scheme for the categorization of acute radiation syndromes with the gradings: H1 (mild), H2 (moderate), H3 (severe), and H4 (fatal) based on granulocyte, lymphocyte, and thrombocyte counts in peripheral blood and the time course of these changes. This evaluation tool is typically applied to individual cases. As detailed data for individual victims were not available to us for the victims on the Rongelap Atoll, we used the mean values as shown in [Figure 2](#). According to the METREPOL rules, the acute radiation syndrome can be categorized as mild (H1, viewing neutrophile and lymphocyte values) to moderate (H2, based on thrombocytes). A near-normalization of blood counts occurred within two years of the accident ([Conard \*et al.\*, 1980](#); [Robbins and Adams, 1996](#)).

There were no bleeding episodes requiring transfusion or infections that could be related to the hematopoietic syndrome caused by irradiation and needing antibiotic therapy. In some patients of the Rongelap group, antibiotics were administered. Still, treatment would have been indicated even if they had not been irradiated (*e.g.*, cystitis, traumatic gangrene of the foot) ([Cronkite \*et al.\*, 1954](#)). No prophylactic antibiotics were

**Table 1.** Radionuclides detected in the fallout ashes on the Lucky Dragon (26 March 1954). (Source: Yamamoto, 2009).

| Nuclide | Activity (%)  | Radiation          | $T_{1/2 \text{ phys}}$ | $T_{1/2 \text{ eff}}$ | Absorption fraction  |                        |                      |
|---------|---------------|--------------------|------------------------|-----------------------|----------------------|------------------------|----------------------|
|         |               |                    |                        |                       | $f_{\text{default}}$ | $f_{\text{insoluble}}$ | $f_{\text{fallout}}$ |
| Sr-89   | 1.0           | $\beta^-$          | 50.7 d                 | 50 d                  | 0.1                  | 0.01                   | 0.05                 |
| Sr-90   | 0.02          | $\beta^-$          | 28.2 a                 | 4.6 a                 | 0.1                  | 0.01                   | 0.05                 |
| Y-90    | 0.02          | $\beta^-$          | 2.7 d                  | 2.7 d                 | 0.0001               | 0.0001                 | 0.00001              |
| Y-91    | 8             | $\beta^-$          | 58.51 d                | –                     | 0.0001               | 0.0001                 | 0.00001              |
| Zr-95   | 5             | $\beta^- + \gamma$ | 64 d                   | 64 d                  | 0.002                | 0.002                  | 0.002                |
| Nb-95m  | –             | $\beta^-$          | 3.61 d                 | 3.59 d                | 0.01                 | 0.01                   | n.a.                 |
| Nb-95   | 3             | $\beta^-$          | 34.98 d                | 33.44 d               | 0.01                 | 0.01                   | n.a.                 |
|         | $\Sigma = 15$ |                    |                        |                       |                      |                        |                      |
| Ru-103  |               | $\beta^- + \gamma$ | 39.3 d                 | 38.5 d                | 0.05                 | 0.01                   | 0.001                |
| Ru-106  |               | $\beta^- + \gamma$ | 372.6 d                | 268 d                 | 0.05                 | 0.05                   | 0.001                |
| Rh-106  |               | $\beta^-$          | 29.8 s                 | –                     | 0.05                 | 0.05                   | n.a.                 |
| Te-127m |               | $\beta^-$          | 109 d                  | –                     | 0.1                  | 0.01                   | 0.2                  |
| Te-127  |               | $\beta^-$          | 9.35 h                 | –                     | 0.1                  | 0.01                   | 0.2                  |
| Te-129m |               | $\beta^-$          | 33.6 d                 | –                     | 0.1                  | 0.01                   | 0.2                  |
| Te-129  |               | $\beta^-$          | 69.6 min               | –                     | 0.1                  | 0.01                   | 0.2                  |
| Te-132  |               | $\beta^- + \gamma$ | 3.26 d                 | –                     | 0.1                  | 0.01                   | 0.2                  |
| I-131   |               | $\beta^- + \gamma$ | 8 d                    | 7.5 d                 | 1.0                  | 0.01                   | 1.0                  |
| I-132   |               | $\beta^- + \gamma$ | 2.3 h                  | 2.298 h               | 1.0                  | 0.01                   | 1.0                  |
| Ba-140  | 5             | $\beta^- + \gamma$ | 12.8 d                 | 12.7 d                | 0.1                  | 0.01                   | 0.05                 |
| La-140  | 5             | $\beta^- + \gamma$ | 40 h                   | 40 h                  | 0.0005               | 0.0005                 | 0.00001              |
| Ce-141  | 7             | $\beta^-$          | 32.5 d                 | 32 d                  | 0.0005               | 0.0005                 | 0.00001              |
| Ce-144  | 2             | $\beta^-$          | 284 d                  | 280 d                 | 0.0005               | 0.0005                 | 0.00001              |
| Pr-143  | 16            | $\beta^-$          | 13.6 d                 | 13 d                  | 0.0005               | 0.0005                 | 0.00001              |
| Pr-144  | 2             | $\beta^-$          | 285 d                  | 280 d                 | 0.0005               | 0.0005                 | 0.00001              |
| Nd-147  | 9             | $\beta^-$          | 10.98 d                | –                     | 0.0005               | 0.0005                 | n.a.                 |
| Pm-147  | –             | $\beta^-$          | 2.6 a                  | 2 a                   | 0.0005               | 0.0005                 | n.a.                 |
| S-35    | 0.05          | $\beta^-$          | 87 d                   | 16.3 d                | 0.1                  | 0.01                   | n.a.                 |
| Ca-45   | 0.2           | $\beta^-$          | 163 d                  | 161 d                 | 0.1                  | 0.01                   | n.a.                 |
| U-237   | 20            | $\beta^-$          | 6.75 d                 | 4.66 d                | 0.02                 | 0.002                  | 0.002                |
| Pu-239  | 0.0004        | $\alpha$           | $2.4 \times 10^4$ a    | 50 a                  | 0.0005               | 0.00001                | 0.00001              |

$T_{1/2 \text{ phys}}$ : physical half-life;  $T_{1/2 \text{ eff}}$ : effective half-life (if available);  $f_{\text{default}}$ : recommended default value for the gastro-intestinal absorption fraction if the chemical compound ingested is unknown;  $f_{\text{insoluble}}$ : absorption fraction for insoluble compounds (type “S”, slow);  $f_{\text{fallout}}$ : absorption fraction from local fallout and coral material as matrix as used for the calculations of the internal doses absorbed by fallout victims on the Marshall Islands. Sources: ASN (2008), IMBA database and Ibrahim *et al.* (2010).  $T_{1/2 \text{ eff}}$  of U-237 was calculated assuming a biological half-life of 15 d. n.a.: not available.

administered because of the fear of the possible development of bacterial resistances (Conard, 1992).

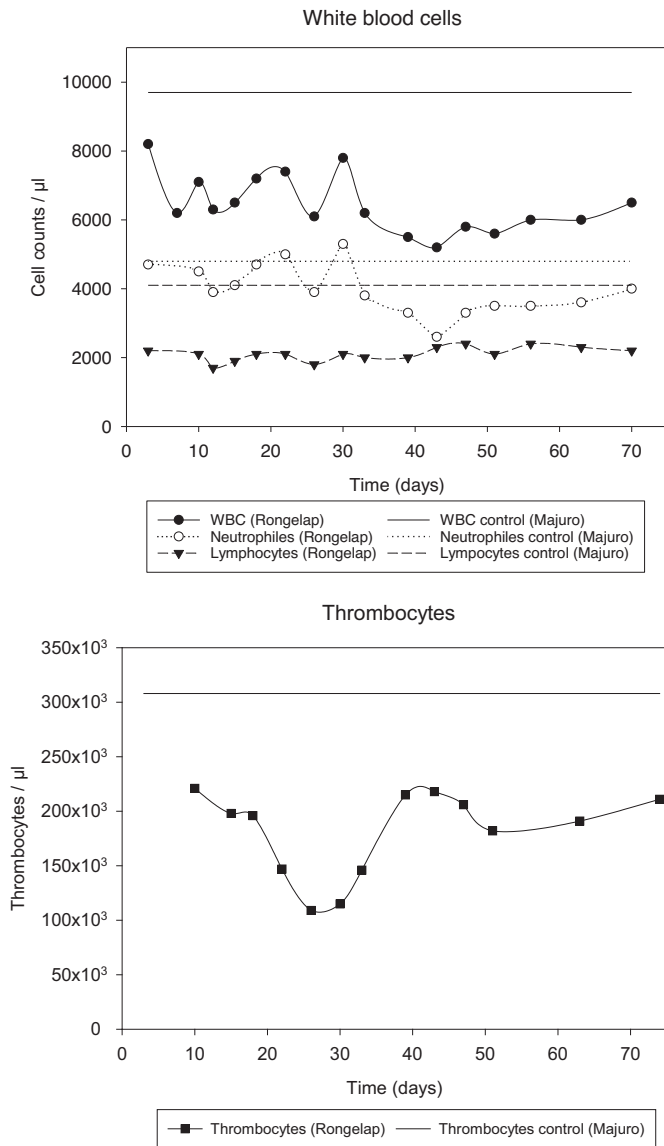
The major late effects of radiation exposure were thyroid abnormalities, including thyroid failure, nodules, and malignant tumors. The first observed pathology was thyroid atrophy leading to growth failure in two boys and further cases of hypothyroidism. Thyroid nodules appeared beginning nine years after the accident and were particularly frequent in victims exposed to radiation as children below 10 years of age. People were, in part, treated with thyroid hormones (Conard, 1992). An excess of thyroid cancers classified as papillary carcinoma was observed among Rongelap inhabitants, with the first case diagnosed 11 years after radiation exposure.

Further possible late effects could be a fatal acute myelogenous tumor in a 19-year-old man exposed as an

infant at the age of 1 year (1972), a basal cell epithelioma removed from a healed skin lesion (1986) as well as further single cancer cases with an uncertain relation to the radiation exposure.

Estimation of cancer risks by fallout exposure was based on the total population of 24 783 exposed inhabitants of the whole Marshall Islands. It must be mentioned that doses from external irradiation as well as acute intake of radionuclides were far lower in the southern compared to the northern Atolls, and the exposure pattern may have been different from a quantitative but also qualitative point of view. The total fraction of cancers due to radioactive fallout exposure (cancers that occurred till now and will occur in the future) has been estimated with about 20% for the thyroid, about 5% for leukemia, and about 1% for all other cancers (Simon *et al.*,





**Fig. 2.** Time course of the mean of the cell counts of the total white blood cells, neutrophils, lymphocytes, and thrombocytes in the inhabitants of the Rongelap Atoll. The first blood sample was taken after evacuation three days after the beginning of fallout exposure. Source of the data: Cronkite *et al.*, 1954.

2010a). On the bottom line, it seems that thyroid diseases have been the major late health effect (Robbins and Adams, 1996).

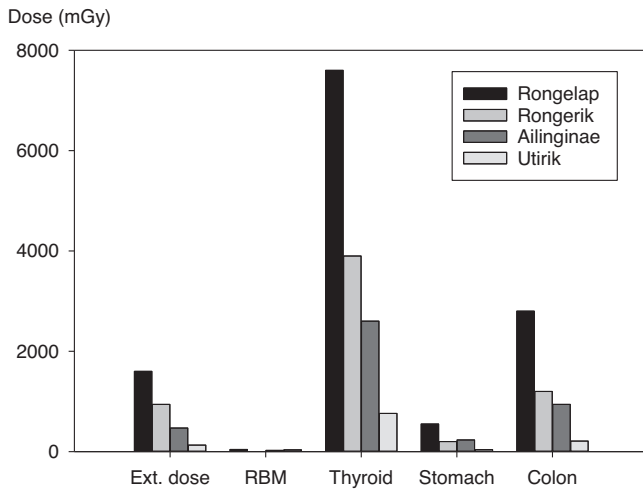
For the Rongelap Island community, the total external dose has been estimated on average with 1600 mGy (5–95% uncertainty range: 1100–2200 mGy). The Bravo test was by far the most important contributor to this total dose (Simon *et al.*, 2010a). Doses absorbed by victims on neighboring Atolls are estimated to be less (Rongerik 940 mGy, Ailinginae 470 mGy, and Utirik 130 mGy) (Simon *et al.*, 2010a).

Radiotoxicological analyses of urine showed that most of the  $\beta$ -activity in the first weeks (77% on day 46) was due to strontium-89, barium-140, and rare earth (in particular lanthanum-140 in equilibrium with barium-140). Depending on the assumptions made, the initial body burden for

strontium-89 was estimated with 1.6  $\mu$ Ci (skeletal body burden) or 2.2  $\mu$ Ci, and for barium-140 with 2.7  $\mu$ Ci (skeletal body burden) or 0.34  $\mu$ Ci (Cronkite *et al.*, 1954). Based on urine data of day 82 (corresponding to a body burden of strontium-89: 0.19  $\mu$ Ci, barium-140: 0.021  $\mu$ Ci for the collection day), we calculated the initial body burden using the internal dosimetry software package “Integrated Modules for Bioassay Analysis” (IMBA) (Birchall *et al.*, 2007) that amounted to 3.11  $\mu$ Ci for strontium-89 and 23.5  $\mu$ Ci for barium-140. According to our computations, the incorporation of strontium-89 would be responsible for a committed effective dose (50 years) of 0.36 mSv with the red bone marrow absorbing 1.83 mSv of equivalent dose, the bone surface 2.28 mSv and the liver 0.075 mSv. The incorporation of barium-140 would have caused 2.78 mSv committed effective dose, 4.76 mSv equivalent dose to the red bone marrow, 6.79 mSv to the bone surfaces, and 0.401 mSv to the liver. Although our activities determined with IMBA software were slightly higher than the values estimated by Cronkite *et al.* (1954), especially for barium-140, the resulting radiological doses (summed over 50 years) are quite low compared to the annual natural radiation load.

In seven Rongelap individuals that were considered to have incorporated a relatively large amount of radioactivity, it was attempted to speed up elimination out of the body by administering Ca(EDTA) (ethylene diamine tetraacetic acid) (1 g/25 lb body weight daily given orally for three days). Although the daily-excreted activity with the urine was increased 2.5 times compared to the days before treatment, the total body burden was not reduced to a relevant extent, as the value of the total excretion rate was very low (0.1% in 24 h, *i.e.*, rate constant roughly  $0.001 \text{ d}^{-1}$ ) (Cronkite *et al.*, 1954). Ca (EDTA), like Ca(DTPA), favored for decorporation today, is poorly absorbed by the gut, so that in case of oral administration high therapeutic efficacy is not to be expected. Moreover, we have no information about the time point the decorporation treatment was started. But in the case initiation is late, most of the incorporated radionuclides may already have entered “deep” pharmacokinetic compartments not accessible to EDTA that distributes in the extracellular space. According to official French guidelines, decorporation therapy should start within 2 h after radionuclide incorporation (ASN, 2008). Simulations have shown that there is a time window ranging from several hours to several days, depending on the radionuclide, the physicochemical properties of the compounds, and the invasion pathway, that should be used for optimum therapeutic efficacy (Rump *et al.*, 2016, 2017; Yan *et al.*, 2019).

Estimation of internal doses of the victims of the Atolls near the Bikini Atoll is based on the assumption that the incorporation of radionuclides results from ingestion (contaminated food, eating utensils, to a lesser degree contaminated water). To assess internal contamination, 63 radionuclides have been considered to estimate the acute intakes and five radionuclides for the chronic intakes (Simon *et al.*, 2010b). The 63 nuclides were chosen so that it can be reasonably expected that they are responsible for more than 98% of the total radiological dose absorbed. For iodine-131, the estimation of intakes was based on bioassay measurements of collected urine samples. For other radionuclides, the ground deposition density of the activity ( $\text{Bq}\cdot\text{m}^{-2}$ ) was estimated, and



**Fig. 3.** External and internal doses absorbed by different organs and tissues in the victims of the Rongelap Atoll. RBM: red bone marrow. Data from [Simon \*et al.\* \(2010a\)](#).

it was assumed that intake is directly proportional to this deposition density for each radionuclide. The ratio between intake and deposition density was derived from the iodine-131 data. Moreover, the time of intake, which is important in particular for short-lived radionuclides and depends on the fallout's arrival time, was taken into account ([Beck \*et al.\*, 2010](#)). Radiological doses were then calculated as the product of the average intake and the ingestion dose coefficient ( $\text{Gy Bq}^{-1}$ ) for individual radionuclides. The dose coefficients are based on the ICRP biokinetic models, but for the early intakes, the intestinal absorption fractions were adjusted to values specific for radionuclides ingested from fallout particles that are (only in part) lower than the values of the ICRP ([Ibrahim \*et al.\*, 2010](#)). This may result from the fact that the absorption is controlled by the dissolution of the fallout particle material for some nuclides. That is also the reason why the material of the earth's surface at the detonation point should be taken into account (absorption fractions have been determined for "continental soil" from nuclear land tests and for coral material from tests on the Marshall Islands) ([Tab. 1](#)) ([Ibrahim \*et al.\*, 2010](#)). This adjustment was not required for chronic intakes, as it could be reasonably assumed that food contamination results mainly through root uptake. Therefore, the radioactive materials are in a soluble form ([Simon \*et al.\*, 2010b](#)).

The internal doses estimated substantially differ among the organs and tissues. Still, as expected for early fallout exposure, they are reported to exceed the doses by external irradiation only for the thyroid (Rongelap community: total 7600 mGy, uncertainty range 1500–23 000 mGy; 94% of the total dose by the Bravo test) and the colon (2800 mGy, uncertainty range 560–8500 mGy; 90% from the Bravo test) ([Simon \*et al.\*, 2010a](#)). The estimated doses are lower for the victims of the other affected Atolls and in particular for the more distant Utirik community (760 mGy for the thyroid; 210 mGy for the colon) ([Fig. 3](#)).

It is thought that the total red bone marrow dose is mainly due to external irradiation, and only to a lesser extent (Rongelap community: 42 mGy, range 8.4–130 mGy; 83%

from the Bravo test) to the incorporation of radioactivity. Considering the relatively small dose absorbed by the red bone marrow and the results of urine analyses, compared to the radioactivity present in the fallout, it seems that the systemic absorption of most metal radionuclides has not been a major issue. This is consistent with the low gastrointestinal absorption fractions of many of the major contributors of the fallout ([Tab. 1](#)).

The high internal doses absorbed by the gastrointestinal tract may be seen as the consequence of continuing ingestion of contaminated food or drinking water during the first days after the detonation before evacuation. Many of the compounds containing fission products are highly insoluble ([Simon \*et al.\*, 2010a](#)). So, they are not systemically absorbed but remain in the gut till the time of excretion through feces. The particularly large dose absorbed by the colon (2800 mGy) ([Fig. 3](#)) is probably the consequence of the longer mean residence time compared to the more proximal parts of the gastrointestinal tract (rate constant of the transport from the stomach to the small intestine:  $24 \text{ d}^{-1}$ ; rate constant out of the lower large intestine  $1 \text{ d}^{-1}$ ) ([ICRP, 1979](#)).

In clinical toxicology, the administration of cathartics alone is usually considered to have no role in the treatment, and in combination with charcoal, results are conflicting ([American Academy of Toxicology, 2004](#)). If used, osmotic agents like saccharide or saline cathartics should be favored (*e.g.*, sorbitol, magnesium citrate, etc.). It could be shown that the gastrointestinal transit time of charcoal could be reduced from roughly 22 h to 7 h by magnesium citrate ([Neuvonen and Olkkola, 1986](#)) and even to less than 1 h by sorbitol ([Krenzelok \*et al.\*, 1985](#)). In radiobiology literature, cathartics are mentioned as a possibility to decrease the radiological doses absorbed by intestinal tissues, in particular after ingestion of poorly soluble compounds ([Waller \*et al.\*, 2002](#); [Hormann and Fischer, 2009](#)). Using IMBA software, we found that by doubling the rate constants describing the intestinal transit in the ICRP model, the doses absorbed by the small intestine and the upper and lower large intestine by ingestion of a poorly soluble plutonium-239 compound (type "S") could be reduced by roughly 50%. Whereas 90% of the radioactive material is eliminated from the gut in 3.3 days, this time is shortened to 1.7 days when the transit rate constants are doubled. Thus, considering the radiological load, especially of the colon in the Rongelap victims, it may be helpful to administer laxatives in the case the consumption of contaminated food or water is suspected after local fallout exposure. It remains to be determined how fast after ingestion this measure must be taken to have a satisfactory efficacy. It is however important to emphasize that this measure is of secondary importance compared to protection against external irradiation.

For the thyroid, the internal dose caused by radioiodine intake (mean 7600 mGy, up to 23 000 mGy) was much higher than the dose absorbed by external irradiation ([Fig. 3](#)). Thyroid doses were exceptionally high in children ([Conard \*et al.\*, 1980](#)). Interestingly, considering the composition of the fallout and the probable intake of radioactivity (five iodine isotopes), most of the absorbed thyroid dose (estimation 80–90%) seems to have been caused by short-lived iodine and tellurium isotopes (I-132, 133, 134, 135 and Te-131m, 132) and not by iodine-131 ([Robbins and Adams, 1996](#)). So, data and findings should be used cautiously and must not be transferred to other

scenarios, such as nuclear power plant accidents. If the fallout exposure sites are more distant from the source, iodine-131 is expected to be the most important radioactive iodine isotope.

The thyroid doses absorbed by the victims on the Rongelap Atoll must be put in perspective to doses achieved in therapeutic thyroid irradiation. An activity of 6 MBq (160  $\mu$ Ci) iodine-131 per gram thyroid weight has been reported to cause iatrogenic hypothyroidism in 20–40% of the patients after one year and 50–80% after ten years (Hagen *et al.*, 1967; Suhail *et al.*, 2001). The thyroid equivalent dose corresponds to 44.6 Gy (own calculations with the IMBA software). This is even a low value as in patients treated with iodine-131 for Grave's disease, the median dose causing hypothyroidism has been determined with 220 Gy, and there is no statistically significant effect below 50 Gy (Peters *et al.*, 1997; Reiners *et al.*, 2020). After external beam radiation therapy of the cervical region, the 50% intercept of the dose-effect curve is lower and corresponds to 45 Gy (Vogelius *et al.*, 2011; Reiners *et al.*, 2020). In adults, a threshold level of 10–20 Gy has been proposed to induce hypothyroidism. Even this latter value is above the doses described for the victims on the Rongelap Atoll. So, at first sight, it is not apparent to expect the occurrence of hypothyroid dysfunctions. However, for children exposed to iodine-131 after the accident in Chernobyl (cohort from Belarus: range 0–26.6 Gy, mean 540 mGy), a dose threshold of 3–5 Gy for an enhanced risk of hypothyroidism has been suggested (Ostroumova *et al.*, 2013; Reiners *et al.*, 2020), and this is also in line with other findings reported for atomic bomb survivors of Hiroshima and Nagasaki, although in the latter case, the thyroid doses were estimated as quite low (0–4 Gy, mean 182 mGy) (Imaizumi *et al.*, 2017). So, on the bottom line, the observations on the population of the northern Marshall Islands Atolls seem consistent with other findings described in the literature.

Different mechanisms are involved in radiation-induced hypothyroidism (Reiners *et al.*, 2020). It may be due to deterministic radiation damages leading to necrosis or apoptosis of thyrocytes and atrophy of the gland. On the other side, radiation may also induce autoimmune processes targeting the gland and causing thyroiditis with subsequent hypothyroidism. This latter effect may be stochastic in nature without a threshold level. The large differences in equivalent doses causing hypothyroidism in different settings are nevertheless not fully understood.

Comparing the incorporation in the early exposure phase with the chronic intake after resettlement indicates that acute radionuclide incorporation has contributed more to the total internal dose than chronic radionuclide contamination, particularly for the thyroid (7600 mGy *versus* 14 mGy) and the colon (2800 mGy *versus* 17 mGy) (Simon *et al.*, 2010a). This is easily understandable considering the short decay half-life of radioiodine and the relatively short decay of activity as a whole in early fallout (7/10 rule) (Fong, 2007).

#### 4 Health impact on the Japanese fishermen of the Lucky Dragon

The crewmen of the Lucky dragon started to develop symptoms the evening of the detonation day that were described as pain, headache, nausea, dizziness, diarrhea, red

eyes, and itching (Oishi, 2011). Skin lesions began on the third day: the faces turned dark (*i.e.*, erythema), and painless small blisters looking like burns developed on the parts of the body that had been in contact with fallout particles. These skin lesions were later diagnosed as  $\beta$ -burns, particularly on the head, the neck, the hands, and the feet. Hairs began to fall out in bunches after one week.

On arrival in their home port, the fishermen still complained of nausea, headaches, bleeding from the gums, and skin burns. Because of the remaining radioactive contamination of the hairs and nails, the crewmen had their hair shaved by city employees as barbers refused to serve them.

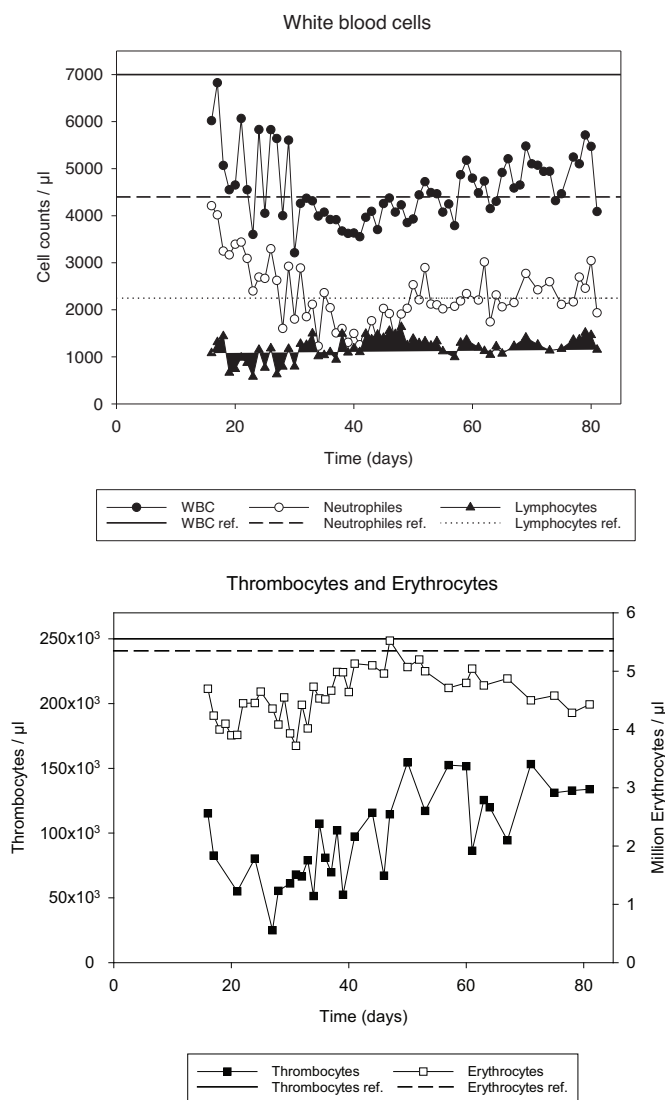
The first blood samples for blood cell counts were taken after the crew returned to Japan two weeks after fallout exposure on day 16. Lymphopenia was noted in all victims from the 2nd to the 8th week. Total leucocyte counts reached a minimum in the 4th to 8th week: counts lay below 2000/ $\mu$ l in five cases, and in one case, it was depressed to 800/ $\mu$ l. In thirteen cases, it lay between 2000 and 3000/ $\mu$ l and between 3000 and 4000/ $\mu$ l in five cases. Thrombocytes reached a minimum between the 4th and 7th week (Kumatori *et al.*, 1965).

We extracted the hematological data of all 23 fishermen from the database SEARCH available at our institute. This database contains data from more than 800 patient case histories of radiation accidents (Friesecke *et al.*, 2000). The time course of the mean values of the white blood cells, thrombocytes, and erythrocytes during the first weeks after fallout exposure is shown in Figure 4. Compared to the data of the Rongelap victims, the evolution pattern over time is quite similar. Still, the absolute values of the cell counts are lower (*e.g.*, lowest mean neutrophils count 1300/ $\mu$ l in Japanese 2600/ $\mu$ l in Rongelap victims, lymphocytes 630/ $\mu$ l *versus* 1700/ $\mu$ l and thrombocytes count 25 000/ $\mu$ l *versus* 109 000/ $\mu$ l). Again, it must be mentioned that a large variability was observed among the Japanese group (range of the individual minimal values: WBC: 800–3300/ $\mu$ l; neutrophils: 95–1593/ $\mu$ l; lymphocytes: 26–924/ $\mu$ l; thrombocytes: 3000–80 000/ $\mu$ l).

To assess the acute radiation syndrome's severity, we again used the METREPOL system, which could be applied individually to all 23 cases. Based on mean blood cell count values (for comparison with the Rongelap group), the severity of the acute radiation syndrome can be categorized as mild (H1, based on neutrophils) to moderate (H2, based on lymphocytes and thrombocytes). Considering the individual cases, data show a large interindividual variability ranging from H0 (no alteration) to H3 (severe) (Tab. 2). The time courses of blood values for the mildest and the most severe case are displayed in Figure 5.

The bone marrow was hypoplastic or aplastic in severe cases (Kumatori *et al.*, 1965). Clinically, fever, bleeding from the nose and gums, subcutaneous bleeding, bloody stools, and persistent diarrhea were reported (Oishi, 2011). The victims were treated with antibiotics (including penicillin, aureomycin, achromycin) and repeatedly with blood transfusions and dry plasma from April to May (Oishi, 2011). In June, the hematological conditions improved, but 17 of the 23 victims are reported to have developed liver dysfunction, with jaundice in seven or eight patients (Oishi, 2011).

After discharge from the hospital in May 1955, it was tried to examine the victims annually at the National Institute of



**Fig. 4.** Time course of the mean of the cell counts of the total white blood cells, neutrophils, lymphocytes, erythrocytes, and thrombocytes in 23 crewmen of the fisherboat Lucky Dragon. The first blood sample was examined on March 16, 1954 (*i.e.*, 16 days after fallout exposure). Source of the data: SEARCH database. Besides measured cell counts, lines indicating the mean normal values (*ref.*: reference value) of the respective cell lineages are shown.

Radiological Sciences, established in 1957. Routine physical examinations revealed that in 1964, *i.e.*, ten years after the incident, peripheral blood examinations were almost normal. Residual beta burns were diagnosed in 13 of the 18 cases examined as well as lens opacities in six cases. In ten cases, the liver was palpable, but no cirrhosis was diagnosed. Glutamate-pyruvate transaminase (GPT) and glutamate-oxalacetate transaminase (GOT) levels were in a normal range, except in one case (GOT:70) (Kumatori *et al.*, 1965). No thyroid abnormalities have been described (Conard, 1992).

Starting checks on hepatitis C in 1991, it is reported that among 15 surviving crew members, 12 were confirmed to be infected. This has been related to the transfusion of blood products during hospitalization. Up to 1995, eight crew

**Table 2.** Classification of the severity of the acute radiation syndrome observed in the 23 crewmen of the fisherboat Lucky Dragon based on the counts of the neutrophils, the lymphocytes, and the thrombocytes using the METREPOL-system.

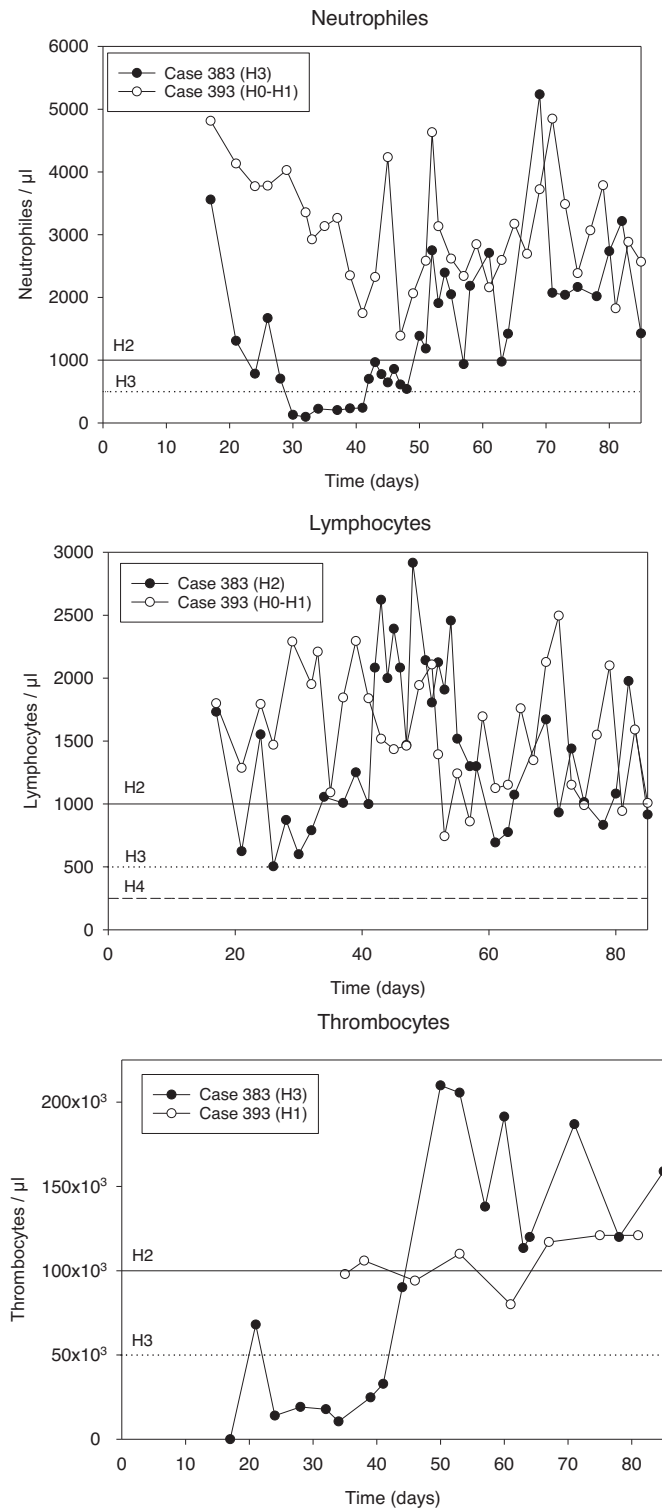
| Case | Neutrophils | Lymphocytes | Thrombocytes |
|------|-------------|-------------|--------------|
| 383  | H3          | H2          | H3           |
| 384  | H1          | H1          | H2-H3        |
| 385  | H3          | H3          | ?            |
| 386  | H3          | H2          | H3           |
| 387  | H1          | H2          | ?            |
| 388  | H0-H1       | H2          | H2           |
| 389  | H1          | H2          | H2           |
| 390  | H0-H1       | H1          | H2           |
| 391  | H1          | H1          | H1           |
| 392  | ?           | H0-H1       | H2           |
| 393  | H0-H1       | H0-H1       | H1           |
| 394  | H0-H1       | H0-H1       | H2           |
| 395  | H1-H2       | H1-H2       | H2           |
| 396  | H1          | H1          | H1           |
| 397  | H0-H1       | H2          | H1-H2        |
| 398  | H1-H2       | H2          | H2           |
| 399  | H0-H1       | H2          | H2           |
| 400  | H0-H1       | H2          | H2           |
| 401  | ?           | ?           | H0-H1        |
| 402  | H1          | H2          | H1-H2        |
| 403* | H2          | H2          | H3           |
| 404  | H1          | H1          | H1-H2        |
| 405  | H1          | H2          | H1-H2        |

H0: no alterations; H1: mild; H2: moderate; H3: severe; H4: fatal. In some cases indicated by “?” data seemed conflicting and a reasonable categorization did not seem possible. Nr 403: \*Categorization of the chief radio officer, as the only short-term fatality of the crew. Source of the blood data: SEARCH database with case numbers as registered.

members (not including Mr. Kuboyama, see Section IV.4) of the Lucky Dragon had died, six of them from liver cancer or hepatic cirrhosis (Oishi, 2011).

In the case of the fishermen of the Lucky Dragon, it seems that the doses absorbed by external irradiation have been higher than for the inhabitants of the Rongelap Atoll. Doses have been estimated in the range of 200 R (roughly 2000 mGy) (Shiougawa, 1984), 1700–6000 mGy (min. 1000–4000, max. 5000–8000 mGy) (Nishiwaki, 1984; Simon *et al.*, 2006) or 1700–7000 mGy (Yamamoto, 2009). Based on estimates of the individual crewmen, the average whole-body gamma radiation dose would amount to roughly 2860 mGy (Kumatori *et al.*, 1965). Half of the total dose is expected to have been absorbed during the first day after exposure (Kumatori *et al.*, 1965; Simon *et al.*, 2006). It is speculated that the dose absorbed by the chief radio officer (Mr. Kuboyama), who died in September 1954, may have been particularly high because of the location of his cabin directly under the upper deck that remained contaminated despite cleaning (Nishiwaki, 1961). The specific activity of the initial fallout on March 1, 1954, was calculated to be in a range of  $3.7 \times 10^{10}$  Bq (Nishiwaki, 1961; Shiougawa, 1984). The reasons for the higher doses compared to the victims on the Rongelap Atoll





**Fig. 5.** Time courses of the cell counts of the neutrophils, lymphocytes, and thrombocytes in two cases of different severity among the 23 crewmen of the Lucky Dragon. Case Nr. 383 as the most severe, and case Nr. 393 as the mildest case according to the METREPOL categorization (see Tab. 2). Categories H0: no alterations, H1: mild, H2: moderate, H3: severe, and H4: fatal. The horizontal lines in the graphs show the limits of cell counts as one criterion for categorization (not to be interpreted as cut-off lines). The other criterion is the speed and duration of cell count drops and the time points of the nadirs. Source of the data: SEARCH database.

remain speculative but could possibly be explained by an earlier beginning of fallout (provided information on time is correct) combined with rain, increasing the downward transport of smaller particles. Both factors could explain the higher activity falling down on the Lucky Dragon.

The internal doses for the thyroid have been reported very cautiously, with a range between 100–10 000 mGy (Nishiwaki, 1984). Yamamoto (2009) estimates the thyroid dose with 200–1200 mGy. At least the latter value is less than the mean value reported for the Rongelap island community.

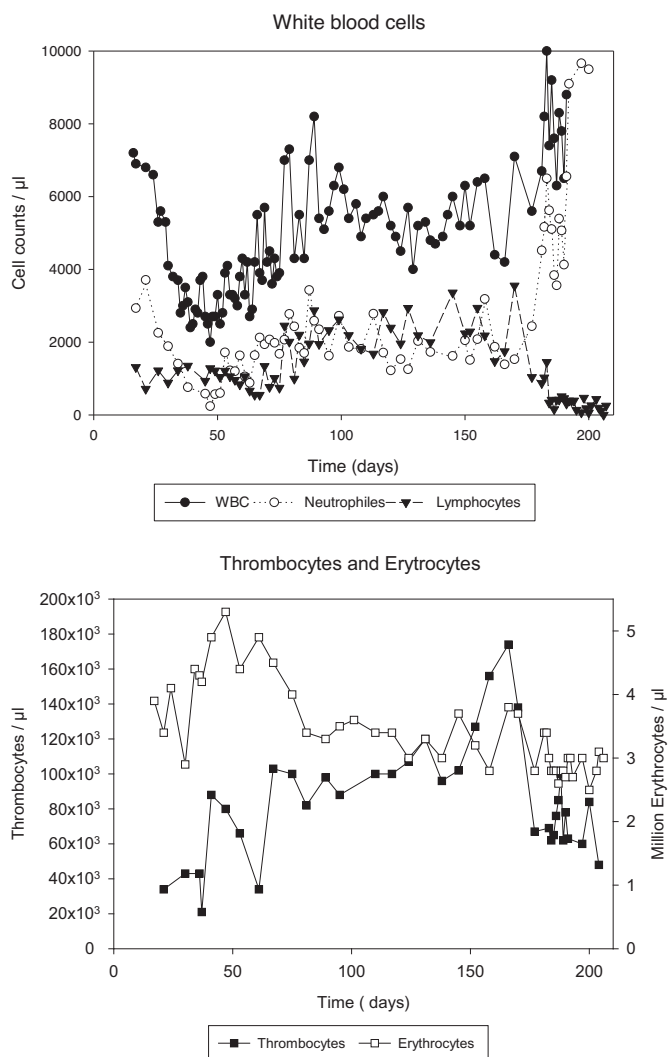
Urine samples analyzed four weeks after fallout exposure showed increased levels of radioactivity, with strontium-89 and barium-140 contributing by 4–20% (Kumatori *et al.*, 1965). A whole-body counting examination in 1964 (13 victims) and further radiotoxicological analyses of urine (10 victims) showed no significant increase in radioactivity compared to controls (Kumatori *et al.*, 1965).

## 5 Clinical course and death of the chief radio officer of the Lucky Dragon

The chief radio officer had been hospitalized at the Tokyo National Hospital and was treated with six other victims. It is reported that by the beginning of June, he had already got 11 blood (200 ml) and 51 dry plasma (100 ml) transfusions (Oishi, 2011). His condition rapidly worsened from August 20th, and he was treated with oxygen and got additional blood transfusions. He became confused, disoriented, and violent on August 29th, so he was isolated from the other patients. On August 30th, his condition further worsened, and he became unconscious and irresponsive. He remained in coma except for a short time on September 4th and died on September 23rd (Oishi, 2011).

Hematology data of the chief radio officer could be identified in the SEARCH database, and the time course till death is shown in Figure 6. Among the crew, he belonged to the patients with below-average leucocytes and neutrophils counts (minimal values WBC: 2000/µl and neutrophils 240/µl on day 47; thrombocytes 21 000/µl on day 37), suggesting that he might have absorbed a relatively high radiation dose or had a high radiation sensitivity. According to the METREPOL system, the acute radiation syndrome can be categorized as moderate (H2 based on granulocytes and lymphocytes) to severe (H3 based on thrombocytes) (see Tab. 2, case no.403). From end of August 1954, during the last weeks before his death, the total white blood cells count showed a sharp rise up to 25 000/µl concomitantly with the neutrophils reaching 15 330/µl (maximum values are not shown on Fig. 6). There was a sharp drop in lymphocytes values (down to 105/µl) and thrombocytes (from 174 000/µl to 48 000/µl). These laboratory results very probably reflect the development of a septic shock and are in line with the autopsy findings showing pneumonia (Chung *et al.*, 2015).

The autopsy performed showed as most important finding a liver atrophy (860 g) with inflammatory cell infiltration and slight cirrhosis. Pneumonia with mixed infection of *Aspergillus fumigatus* was diagnosed. Further findings included ascites (2600 ml), an atrophic spleen with congestion, a serious nephrosis, atrophic degeneration of the myocardium, degenerative atrophy, partial acute inflammation of gastroenteric



**Fig. 6.** Time course of the cell counts of the total white blood cells, neutrophils, lymphocytes, erythrocytes and thrombocytes in the chief radio officer of the fisherboat Lucky Dragon that passed away on September 23, 1954, as the only short-term fatality of the crew. The H-values give the categorization according to the METREPOL-system.

membranes, and partial recovery of the bone marrow from general myelopathia. It was concluded that radiation hepatopathy, induced by external and internal radiation exposure, should be considered as well as a virus hepatitis subsequent to the blood transfusions. It was suggested that the radiation hepatopathy may have at least accelerated the development of the virus infection (Ohashi *et al.*, 1955).

Radiotoxicological analyses of organ specimens on October 18, 1954 showed that activity was very weak, and compared to controls only the bones were clearly contaminated with fission products ( $20 \times 10^{-12}$  Ci/g weight) (Kimura *et al.*, 1956). Cerium-144 ( $\beta$ -radiation emitter, physical half-life 285 d) and its daughter praseodym-144 ( $\beta$ -radiation emitter, half-life 17.3 min) were identified as the main radionuclide(s). The internal dose absorbed by the bone was calculated with approximately eight rep (Röntgen equivalent physical, 1 rep = 9.3 mGy), *i.e.*, roughly 74.4 mGy till the time of death (Kimura *et al.*, 1956).

An internal dose estimation was also tried by administering Bikini ash to determine the time course of radioactivity due to the radionuclide mix in the inner organs (Miyoshi and Kumatori, 1956; Nishiwaki, 1961). It was shown that radioactivity accumulates not only in the bone but also in parenchymatous tissues like the liver or kidney. The effective biological half-life was about 10 to 15 days for the liver and 30–100 days for the bone (Nishiwaki, 1961). Despite the very small activities detected by the radiotoxicological analysis at the post-mortem examination of the chief radio officer, back extrapolation was used to calculate the initial radioactivity intake permitting dose estimations. The integral internal dose absorbed by the liver was estimated at 1 to 100 Gy and for the bone at 100 mGy to 3 Gy (Nishiwaki, 1961).

We tried to reconstruct the internal doses absorbed departing from different assumptions using IMBA software. The decay of cerium-144 leads to praseodym-144 with a short physical half-life (17.3 min), and the dose resulting from this progeny decay is just added to the dose resulting from the decay of the mother nuclide by the software (“merger”). We assumed that all the radioactivity entered the blood (transfer compartment of the model) on the first day of fallout exposure (March 1, 1954,  $t=0$ ). The time between incorporation and the radiochemical analysis amounts to 231 days. Similar to Kimura *et al.* (1956), we assumed a total bone mass of 7000 to 8000 g so that the total activity of cerium-144 at the time of analysis was estimated at  $150 \times 10^{-9}$  Ci ( $7500 \text{ g} \times 20 \times 10^{-12}$  Ci/g). Departing from this value, we calculated the activity of cerium-144 in the skeleton at  $t=0$ , assuming the effective half-lives for bone tissue (30–100 days) experimentally determined by Nishiwaki (1961) (alternative 1). These values heavily differ from the biological half-life for cerium (3500 d) in the ICRP model (ICRP, 1989, 1993). In the latter case, the effective half-life and elimination out of the body are mainly determined by the physical decay half-life (285 d). This value was also used to estimate the bone cerium-144 content at  $t=0$  (alternative 2). According to the ICRP model, 30% of the cerium-144 intake deposits in the skeleton, and we derived the total cerium-144 amount in the body from this value. This amount corresponds to the bioavailable activity and not to an inhaled or ingested activity. Thus, using IMBA software, we computed the radiological doses absorbed by different organs and tissues for the period up to 50 years after incorporation. The IMBA software does not permit direct calculation of the fraction of the dose absorbed at different time points. In analogy to methods applied in pharmacology and toxicology, we used the area under the activity time curve (AUC) as a metric reflecting the total dose (Derendorf and Garrett, 1987; Rump *et al.*, 2019) to estimate the doses absorbed from the time of fallout exposure to death (Tab. 3).

Results must be considered an attempt to get very rough estimates of the order of magnitude of the internal doses. The effective half-lives determined experimentally using Bikini ashes by Nishiwaki (1961) differ markedly from the model parameters used by the IMBA software. As expected from the fraction deposited in the liver, computations confirm that it is the main target organ for cerium-144. Depending on the assumptions, the estimations of the radiological doses absorbed show a large variability: for the bone till death from 8.3 to 981 mGy (calculated 50 years values 21 to 2480 mGy) and for the liver from 24.5 to 2903 mGy (50 years

**Table 3.** Estimated internal doses absorbed by organs and tissues of the deceased radio officer of the Lucky Dragon depending on assumptions about the effective half-life ( $T_{1/2\text{eff}}$ ) of cerium-144 in bone: <sup>(1)</sup> Nishiwaki (1961), <sup>(2)</sup> ICRP model.

| Assumption $T_{1/2\text{eff}}$ in bone | Bone (mGy) |            | Liver (mGy) |            | Effective dose (mSv) |            |
|--|------------|------------|-------------|------------|----------------------|------------|
|  | 50 years   | Till death | 50 years    | Till death | 50 years             | Till death |
| 30 d <sup>(1)</sup>                    | 2480       | 981.0      | 7340        | 2903.3     | 932                  | 368.7      |
| 100 d <sup>(1)</sup>                   | 59.2       | 23.4       | 175.0       | 69.2       | 22.2                 | 8.78       |
| 3500 d <sup>(2)</sup>                  | 21.0       | 8.31       | 62.0        | 24.5       | 7.88                 | 3.12       |

**Table 4.** Comparison of the major similarities and differences between the two groups of victims of the Castle Bravo incident. Source for the absorbed doses of Marshall Islands inhabitants: Simon *et al.* (2010a); for the crewmen of the Lucky Dragon: <sup>a</sup> Kumatori *et al.* (1965), <sup>b</sup> Yamamoto (2009). Considering the available database, only an interval is given for the thyroid dose absorbed by the Japanese crewmen.

|                                 | Inhabitants of the Northern Marshall Islands Atoll                                  | Crewmen of the Lucky Dragon |
|---------------------------------|---|-----------------------------|
| Total victims                   | Rongelap: 64<br>Ailinginae: 18<br>Rongerik: 28<br>Utirik: 157                       | 23                          |
| Blast and thermal burn injuries | 0   | 0                           |
| Estimated total external dose   | Rongelap: 1600 mGy<br>Ailinginae: 470 mGy<br>Rongerik: 940 mGy<br>Utirik: 130 mGy   | 2860 mGy <sup>a</sup>       |
| Internal thyroid dose           | Rongelap: 7600 mGy<br>Ailinginae: 2600 mGy<br>Rongerik: 3900 mGy<br>Utirik: 760 mGy | 200–1200 mGy <sup>b</sup>   |
| Short-term fatalities           | 0   | 1 (September 23, 1954)      |
| Main long-term health impact    | Thyroid dysfunctions  | Liver diseases              |

values 62 to 7340 mGy) (Tab. 3). Thus, our values are in an order of magnitude compatible with the doses calculated by other authors for the bone, but rather lower for the liver (Kimura *et al.*, 1956; Nishiwaki, 1961).

Although computations based on the ICRP model may seem more trustful at first sight, as the parameters are explicitly given for cerium, whereas values given by Nishiwaki (1961) refer to Bikini ash as a mixture, it must be mentioned that toxicokinetic studies on animals are also the basis of the ICRP model. Still, the biokinetics of cerium in humans is hardly known (Keiser, 2011). The ICRP model cannot explain why cerium-144 and praseodym-144 could be clearly detected and quantified in bone but not in the liver of the chief radio officer. More activity would be expected to be deposited in the latter organ, and biological half-lives are identical for both tissues. Moreover, it must be emphasized that our computations are only based on cerium-144 and praseodym-144 detected in the bone six months after fallout exposure. It is not excluded that other radionuclides with a shorter effective half-life have also contributed to irradiation. Nishiwaki *et al.* (2000) emphasized that assuming the radioactive nuclides detected after the death of the chief radio officer are the only source of internal irradiation might lead to a great underestimation of the dose absorbed.

## 6 Similarities and differences between the two groups of victims

A precise comparison between the local inhabitants of the affected northern atolls and the crew of the Japanese fisherboat is challenging in so far as the information given in the available literature often relates to different laboratory findings and time points. Therefore, it is only possible to derive a general picture of the two victims groups (Tab. 4).

In the population of the Rongelap Atoll (the most affected compared to the other northern islands), anorexia and nausea were the most common prodromal symptoms (2/3 of the victims). In contrast, vomiting and diarrhea occurred only in 10% of the victims. Lymphocyte counts were at 50% of control values on the third day, corresponding to a mild radiation exposure of 0.8–1.5 Gy (Hall and Giaccia, 2019). A drop of thrombocytes to roughly 1/3 of controls (with the lowest value of 35 000/ $\mu\text{l}$ ) could be associated with a moderate exposure in a range of 2–4 Gy. Applying the METREPOL system confirms this evaluation. However, it must be emphasized that this categorization is based solely on biological parameters and should not be associated with clearly defined dosimetric intervals. Despite all uncertainties and interindividual

variability, based on prodromal symptoms and blood cell counts, the radiation exposure of the inhabitants of Rongelap can be described as mainly mild to possibly moderate.

Although the radiation exposure patterns are definitely different, the hematological changes in the victims of the Rongelap Atoll seem to show similarities to the group of Japanese victims of the bombings of Hiroshima and Nagasaki with definite clinical symptoms of radiation exposure but who survived at least in the first three or four months (LeRoy, 1950; Oughterson *et al.*, 1951; Cronkite *et al.*, 1954). In both groups, the early period after irradiation was characterized by considerable variation in white blood cell counts, mainly due to the neutrophil counts. There was a pattern of continued depression over several weeks (see Fig. 2). In addition, lymphocyte counts remained depressed in both groups at values around 2000/ $\mu\text{l}$  (see Fig. 2). In contrast, the victims of the Hiroshima and Nagasaki bombings with high dose exposures and mortality were reported to show an early depression of white blood cells with a definite nadir at four weeks (not shown in the Rongelap group nor in the crewmen of the Lucky Dragon). Thrombocyte counts in the Rongelap victims (and in the Lucky Dragon group) show a quite consistent pattern with a minimum after about four weeks. A well-founded comparison with blood data from the Japanese victims of the bombings of Hiroshima and Nagasaki is unfortunately not possible. Still, it should be mentioned that numerous purpura cases among the surviving bombing victims were reported during the 4th week, corresponding to the time of the lowest thrombocyte values in the Castle Bravo victims.

Among the fishermen of the Lucky Dragon, in the early phase (day 1–5), headache and fatigue were reported in more than 20 victims, anorexia in 17 to 18 victims, nausea, vomiting and diarrhea in seven to nine victims (Kumatori *et al.*, 1965). From the report of one crew member (Oishi, 2011), it can be inferred that the onset of symptoms may be estimated within a few hours (detonation at 6:45 am, begin of fallout after  $> 2$  h, pulling in the lines and cleaning the ship for 2 h, earliest end of the most intense exposure calculated about 16:00, first symptoms “beginning of the evening”) (Oishi, 2011). Thus, despite the uncertainties in the reported timelines, the latency period to the beginning of prodromal symptoms may lie in a range of about or even less than four h, which in conventional radiation injury triage systems is a criterion to classify the radiation exposure as clearly moderate rather than mild (vomit 1–4 h: 2–6 Gy) (Flynn and Goans, 2012). Prodromal symptoms, particularly diarrhea, seem to have been more frequent among the crew members of the Lucky Dragon than the inhabitants of the Rongelap Atoll. Early lymphocyte counts are not available as the ship reached its home port only two weeks after fallout exposure, with no medical care or diagnostics possible at an earlier time point. In the further course, the drop of leucocytes and granulocytes revealed to be more severe than in the victims of the Rongelap Atoll. Thrombocytes were also more depressed (mean value of the nadir 25 000/ $\mu\text{l}$  versus 109 000/ $\mu\text{l}$ ), and clinical signs of bleeding were observed. The information available on symptoms and blood counts is consistent with the higher doses estimated for the crewmen of the Lucky Dragon compared to the inhabitants of the Rongelap Atoll. This assessment is also confirmed by the METREPOL categorization, indicating a moderate rather than a mild clinical radiation syndrome.

The situation of the crew of the Lucky Dragon is certainly not comparable to that of heavily irradiated victims such as for example, the two victims of the Tokaimura accident who had absorbed lethal radiation doses (8.5 Gy + 5.4 Gy or 4.5 Gy + 2.9 Gy of gamma-radiation + neutron radiation, respectively) and died several months after the accident of multiple organ failure (Hirama *et al.*, 2003). Although the circumstances are very different, a comparison might be dared with the third victim of the accident, who absorbed a lower dose of radiation (estimated 1.3 Gy gamma radiation, 0.81 Gy neutron radiation) and survived. He showed a drop in neutrophil counts to 1090/ $\mu\text{l}$  on day 20 and thrombocytopenia below 50 000/ $\mu\text{l}$  on day 16 without clinical signs of bleeding (Hirama *et al.*, 2003). At the difference from the victims of the Lucky Dragon, the nadirs of the values seem to have occurred earlier. The third Tokaimura victim had no stem cell transplant but he was treated with G-CSF from the 2nd day after the accident, showing a good response, and received thrombocyte transfusions several times. Although irradiation can be considered sublethal, a comparison of the clinical course with the victims of the Lucky Dragon is only possible to a very limited extent.

A further striking difference relates to thyroid diseases. Thyroid abnormalities were the main late effect in the irradiated Rongelap community, and victims irradiated as children were the most affected. This corresponds well with observations after other nuclear or radiological accidents like in Chernobyl (Hall and Giaccia, 2019), and viewing the thyroid equivalent doses absorbed (mean 7600 mGy), this is in line with the literature on radiation-induced thyroid damages. There are no reports about thyroid abnormalities among the crew members of the Lucky Dragon (Conard, 1992). This may be due to the simple fact that all crew members were adults at the time of exposure and so the gland was much less radiosensitive. Another explanation could be a lower level of radioiodine exposure. This would correspond to the lower thyroid equivalent doses in part reported for the Japanese victims (up to 1200 mGy), although the ranges are quite large. It must be mentioned that the estimation of thyroid doses done in Japan is based on external counting of the thyroid region in only four victims and four to seven weeks after exposure (Kumatori *et al.*, 1965), *i.e.*, after a period corresponding roughly to 4 to 7 decay half-times of iodine-131. Therefore, values should be considered cautiously. However, the lower equivalent thyroid doses may be real, as it is reported that the food and water consumed on the way back to Japan, except fresh fish, were taken from closed containers (Conard, 1992). Therefore, the nutritional intake of radioiodine, that is considered to be the main cause of incorporation after radioiodine exposure, may have been relatively low just by chance because the food was not contaminated. Another interesting possibility is that the Japanese crew members were relatively protected against radioiodine due to a regular high daily nutritional iodine intake characteristic of the Japanese diet. It was shown that the rate constant for the transport of iodide from serum into the thyroid is about half of the value in Caucasians (Matsunaga and Kobayashi, 2000, 2001). This may confer a relative thyroid protection in the case of radioiodine exposure compared to victims with an iodine-deficient diet. According to simulation results, however, this natural protection is very relative, and there is a clear indication for thyroid blocking after radioiodine exposure in Japanese as well as Caucasians (Rump *et al.*, 2021).



Another major difference is the occurrence of liver dysfunction and jaundice among several crew members of the Lucky Dragon several months following the radioactive exposure (Kumatori *et al.*, 1965). In contrast, liver disorders were not reported for the affected inhabitants of the Rongelap Atoll. The radiation-induced liver disease typically occurs four to eight weeks after radiotherapy but may also be seen as early as two weeks and up to six to seven months after irradiation (Kim and Jung, 2017; Toesca *et al.*, 2018). Therefore, the timeline of the clinical course in the Japanese victims would be consistent with radiation-induced liver toxicity. It seems that sinusoidal endothelial cell injuries and obliteration of small central hepatic vein branches by collagenous tissue lead to a veno-occlusive process with obstruction to liver blood flow (Toesca *et al.*, 2018). Radiological doses above 30–40 Gy to the whole liver have been associated with radiation-induced liver disease (Toesca *et al.*, 2018). But in radiation therapy, doses are usually fractionated, so threshold values should be used only very cautiously when assessing a single acute or continuous irradiation. The given threshold value for radiation-induced liver toxicity (30–40 Gy) is larger than the upper range of the whole external body doses reported for the crewmen of the Lucky Dragon (estimates up to 8 Gy) but within the range of integral internal dose absorbed by the liver of the deceased chief radio officer as proposed by Nishiwaki (1961) (1 to 100 Gy). However, the values at the upper limit of this huge range for the internal liver dose must be questioned for methodological reasons, but also particularly as liver dysfunctions were not a health issue among the victims of the Rongelap community, although fallout composition should be expected to be roughly comparable.

Besides direct radiation-induced tissue injury, additional indirect effects like immunosuppression may contribute to liver damage. It was shown that radiation therapy becomes a risk for the reactivation of viral hepatitis. Some patients' progression to liver failure was described (Cheng *et al.*, 2004; Toesca *et al.*, 2018). In the 1990s, hepatitis C was detected in several crew members of the Lucky Dragon, and it is reported that until 1995, six crewmen had died from liver cancer or cirrhosis (Oishi, 2011). The clinical course of the chief radio officer who died in September 1954 and the autopsy findings are consistent with subacute liver failure that could result from viral hepatitis. Among industrialized countries, Japan has one of the highest endemic rates of hepatitis C, particularly among the elderly (Chung *et al.*, 2010; Blaxell, 2014). In Japan, it is considered that hepatitis C, and not hepatitis B, is the major cause of hepatocellular carcinomas that is one of the most important causes of cancer death (Ikai *et al.*, 2007; Chung *et al.*, 2010). Until the beginning of the 1960s, post-transfusion hepatitis occurred in more than half of the transfused cases and the contaminated blood products causing jaundice have been termed “yellow blood” by the Japanese (Blaxell, 2014). A survey in the 1980s showed that among patients suffering from hepatocellular carcinoma, 23% had received blood transfusions (LCSGJ, 1990; Gersten and Wilmoth, 2002). Therefore, considering the repeated blood transfusions administered to the hospitalized crewmen of the Lucky Dragon, the high incidence of hepatitis is no surprise. The question of whether radiation damage to the liver and/or immune system has contributed to the lethal course of the chief radio officer cannot be definitely answered.

## 7 Conclusion and lesson's learned

Doctrines establishing the principles for managing nuclear accidents differ among countries, but a particular focus is often on nuclear power plant accidents (Schneider *et al.*, 2021; Bertho *et al.*, 2022). Whereas in Chernobyl and Fukushima, radioactivity was released over several days (Imanaka *et al.*, 2015; Eder *et al.*, 2020), but with radioactive plumes including less radionuclides with very rapid decay rates, the Castle Bravo incident involving a nuclear weapon detonation is of a different kind. There was an instantaneous release of large amounts of very high activity material that showed a rapid decay, but that fell down to earth rapidly. Because of the resurgent danger of nuclear weapon use, the lessons drawn from this accident should be considered in the development of nuclear accident management doctrines.

At the difference of radiation alone, the Castle Bravo case confirms that early heavy fallout is visible and can be recognized or at least suspected (“snow”, “ashes”), as locally nearby the detonation site, the fallout particles are quite large. Depending on the meteorological conditions, it is not automatically distributed in a concentric way around the hypocenter. Moreover, depending on burst conditions, the yield of the bomb and meteorology, early fallout may also come down relatively far, *i.e.*, hundreds of kilometers away from the hypocenter.

The major acute danger emanates from external irradiation, and thus sheltering is the most important protective measure, particularly in the early phase after detonation. The internal doses from radionuclide incorporation reported for most tissues and the red bone marrow among the Rongelap community victims were lower than by external radiation exposure. The internal contamination with radionuclides that was suspected to be the cause of a more severe clinical course in the Japanese victims and that was made responsible for the death of the radio officer was never proven. Overall, although in accidental settings with a small number of victims it may be considered, a decorporation treatment with Ca(DTPA) and Prussian Blue to speed up the elimination of systemically absorbed metal radionuclides does not seem to be a first priority after a nuclear detonation with fallout. Similarly, the administration of laxatives may be considered if ingestion of radioactively contaminated food or water is suspected, but this measure is definitely of secondary importance compared to the protection against external irradiation.

The incorporation of radioiodine however is of particular concern as it heavily concentrates in the thyroid. Although there is no immediate life-threatening risk, thyroid dysfunctions and cancers must be expected in the long run. If rapidly available, the early administration of potassium iodide (“iodine blockade”) is indicated. Most importantly, if achievable, contaminated food and drinks should be avoided. This is also the best way to reduce internal doses absorbed by the gastrointestinal tract, particularly in the colon. But again, protection against external irradiation by sheltering is the first priority.

Although the Japanese victims of the Lucky Dragon seem to have been more affected by irradiation, justifying a more aggressive and invasive therapy than the inhabitants of the Rongelap Atoll, the case shows that possible side-effects and risks of the treatments must be carefully weighed against the

real benefit of the intervention. This might be a difficult challenge when confronted with uncommon conditions, particularly if associated with fears and emotions like in radiological emergencies.

## Abbreviations

|          |   |
|----------|---|
| AUC      | Area under the curve                        |
| Ca(DTPA) | Calcium(diethylenetriaminepentaacetic acid) |
| Gy       | Gray  |
| ICRP     | International Commission on Radioprotection |
| IMBA     | Integrated Modules for Bioassay Analysis    |
| kt       | Kiloton                                     |
| rem      | Röntgen equivalent man                      |
| rep      | Röntgen equivalent physical                 |
| Sv       | Sievert                                     |
| TNT      | Trinitrotoluene                             |

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## Informed consent

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## Authors contributions

**A. Rump:** Conceptualization, methodology, investigation, and writing the original draft. **C. Hermann:** Conceptualization, methodology, and investigation. **A. Lamkowski:** Conceptualization, methodology, and investigation. **M. Abend:** Conceptualization, supervision, reviewing. **M. Port:** Conceptualization, supervision, reviewing.

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