RADILOGICAL CONSEQUENCES IN NEW ZEALAND OF A NORTHERN-HEMISPHERE DOMINATED NUCLEAR WAR

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ABSTRACT

The doses delivered to the New Zealand population as a result of a postulated nuclear war are estimated. The postulated war is dominated by northern hemisphere exchanges with some detonations also over Australia; New Zealand is spared direct attack. The doses are estimated conservatively using models from the literature and are of similar order (a few mSv) from both the northern hemisphere conflict and Australian attacks. The impact of the latter supposes a near worst-case prevailing meteorology. The typical somatic effects of such doses are a few hundred cancer inductions protracted over half a century, and perhaps a significant incidence of thyroid disorders if no countermeasures prevent the production and consumption of contaminated milk.

KEYWORDS:

*NUCLEAR WARFARE; NUCLEAR WEAPON DEBRIS, FALLOUT RADIATION DOSAGE; HEALTH PHYSICS; MALIGNANT NEOPLASMS; *NEW ZEALAND.
INTRODUCTION

A major nuclear conflict between superpowers would have a profound effect upon New Zealand. Such a conflict could devastate much of the combatant territories and their peoples, a fate which NZ could escape if not targeted. This report addresses the radiological impact upon the NZ population due to the atmospheric dispersal of large quantities of radioactive fission products from the nuclear weapons detonated outside NZ. The unavailability of pharmaceuticals and medical supplies due to interrupted trade with the northern hemisphere would impair the normal treatment of radiogenic disorders and perhaps significantly exacerbate the health impact.

This report is prepared at very short notice (approximately three weeks) at the request of the "Nuclear Impacts Study" consultative group under the auspices of the NZ Planning Council. The short notice precludes all but the most cursory research in the pertinent literature. In particular, the recent SCOPE 28 report [Pittock et al., 1986; Harwell and Hutchinson, 1986] has not received the scrutiny deserved of it.

A SCENARIO OF THE CONFLICT

I am provided with a vague qualitative scenario ("When Deterrence Fails") of the postulated nuclear war. It would be dominated by northern hemisphere (NH) conflict but includes a small proportion of nuclear detonations in the southern hemisphere (SH). The scenario assumes no NZ targets, and two alternative sub-scenarios are supplied in respect of detonations over Australia. Both sub-scenarios are qualitative and are presented as the news media might conceivably report them.

Qualitative scenarios by themselves cannot yield quantitative impact assessments. I have therefore extended the scenario to enable quantification of the radiological impact:

(a) The total yield of fission explosive detonated in the NH is of order 1 Gt (= 1000 Mt). (1 Mt is equivalent to 1 million tonnes of TNT explosive). The pattern of SH fallout should resemble that detected after the atmospheric bomb tests; the latter, with major yields in 1961-2, were also NH dominated but had different airburst/groundburst ratios than typical war scenarios would postulate. Inter-hemispheric
transport is slow, so only long-lived fission or activation products are important. See Annex 1 for details.

(b) The much smaller fission yield of ground-bursts or low-altitude bursts over Australia is of order 1 Mt. NZ is downwind of SE Australia, and pessimistic assumptions plausibly maximise the fallout over NZ. See Annex 2 for details.

(c) Debris from large (exceeding about 1 Mt) or high-altitude detonations over Australia mostly enter the stratosphere or upper troposphere. Such material behaves much as that of NH origin, though since fallout is faster than inter-hemispheric mixing the fallout per Mt exceeds that of NH origin (by perhaps a factor of 3).

As a perspective, total world arsenals exceed 10Gt of total yield (fission + fusion) [Pittock et al., 1986, Table 2.1]. The US National Academy of Sciences postulated in 1975 a war scenario in which 10Gt was exploded (500x10Mt, 5000x1Mt weapons), a scenario now considered outdated by today's "modern" tactical arsenal of "small" warheads (typically 20-200Kt). The AMBIO [1982] scenario considered all large cities and military targets to be hit with a total of 5742Mt, 3% of which would be exploded in the SH. A recent review of nuclear winter by Turco et al. [1984] and the SCOPE 28 report [Pittock et al. 1986, Table 2.2] both considered these and several other war scenarios; for all but a limited city excursion (1000 x 0.1 Mt = 100 Mt) exchanges of several Gt of nuclear explosive (fission + fusion) were postulated.

The impacts of NH and of Australian detonations are assessed in Annexes 1 and 2 respectively. Quantitative estimates are cited "per Gt" and "per Mt" of fission respectively. Such scaling with fission yield is approximate only: the greater the yield or altitude of each detonation, the greater is the fraction of fission products which is injected directly into the stratosphere to become unavailable for short-term fallout. In SCOPE 28 Pittock et al. [1986, Table 7.3] cite external doses delivered by deposited radionuclides originating from a single groundburst of specified magnitude; the doses, based empirically on bomb-test data, show non-linearity with bomb yield particularly in the hemisphere of detonation. For an airburst SCOPE 28 recommends that those same doses be doubled, reflecting an expectation that half the fission products from a groundburst (together with ground activation products) are fused to large entrained particles and fall out close to ground zero. However, the scaling of consequences with total
yield should be dependable if scenarios differ only in the number of detonations and not in the mix of individual yields.

THE RADI OLOGICAL IMPACT

The exposure of the NZ population to the scenario's fallout is low, and the consequences therefore not severe. I limit consideration to the somatic consequences - mainly cancer. Acute lethal effects (non-stochastic) require exposure to a gray or more of dose within about an hour which is nowhere near realised for a NZ resident. Genetic effects are expected to be of less impact than somatic effects.

Activity is measured in becquerels (1 Bq = 1 decay per second = 27 pCi). The unit of absorbed dose (energy absorbed per unit tissue mass) is the gray (1 Gy = 1 J/kg = 100 rad), and the unit of dose equivalent (usually just called the dose) is the sievert (1 Sv = 100 rem). For low LET radiation (e.g. X-rays, Y-rays, all but the very lowest energy electrons) 1 Sv = 1 Gy, while for high LET radiation (neutrons, pions, α-particles and other ions) 1 Sv > 1 Gy. For radionuclides considered herein sievert and gray are treated as synonymous.

The assessment of somatic effects of NH and of Australian detonations in Anexes 1 and 2 are on a fairly ab initio basis, and are based on the fission products (from presumed fission of 239Pu) of Table 1. Data compiled for recent UNSCEAR reports [1977; 1982] and in the course of a study of nuclear-reactor safety [USNRC, 1975, Appendix VI] are extensively used. Some comparisons are made with data tabulated in the SCOPE 28 report. The activation nuclide 14C is also given cursory analysis in Annex 1.

Radioactive materials can also be released to the atmosphere from targeted nuclear facilities. In Annex 1 it is suggested that targeted commercial reactors whose cores are fully dispersed should be considered equivalent to nuclear detonations, with approximately 30 Mt to be associated with each GW(e) of generating capacity.

The "absolute-risk" model of radiogenic cancer commonly adopted assumes a linearity of somatic response and is characterised by a risk factor for each exposed tissue (fatal cancers induced per unit of tissue dose per million persons exposed). Such linearity is by no means well established
[BEIR, 1980, Chapter V], and so estimates of radiogenic cancer induction are subject to considerable uncertainty. According to the model, the period of risk follows a latent period (typically 10-30 yr) during which no radiogenic cancers are expressed. Those cancers then become manifest over succeeding decades (perhaps up to 50 yr). The risk factors adopted here for fatal cancers are taken from ICRP publication 40 [ICRP, 1984, Table A2]. The total radiogenic cancer incidence is about treble that for fatal cancers [BEIR, 1980, Chapter V].

The particular case of radiogenic thyroid disorders deserves special mention. There is some evidence that a thyroid dose delivered internally by $^{131}$I is considerably less effective in inducing thyroid nodules (tumors or lesions, benign and malignant) than is the same dose delivered by external $\gamma$-irradiation [USNRC, 1975, Section 9.3.5 and Appendix H to Appendix VI; BEIR, 1980, Appendix A to Chapter V]. Accordingly, the "Reactor Safety Study" [USNRC, op cit] recommended dividing thyroid doses from $^{131}$I by ten. The reasons for the different effectivenesses are unknown but are believed to be related to the relative microdosimetric efficacies of $\gamma$ and $\beta$ irradiation of the thyroid. Thyroid doses calculated herein from internal $^{131}$I are not mitigated in this way, so that the estimated incidence of thyroid disorders may be significantly over-estimated.

The estimated number of radiogenic cancers should be compared with the "background" incidence endemic in the NZ population. There are approximately 5000 cancer fatalities each year in NZ (21% of all deaths), with 9500 cancers notified annually. An increase in cancer fatalities of less than 1% would be barely detectable unless the excess cancers were of a distinctive form (e.g. predominantly leukemias).

Fallout from NH detonations (Annex 1) is estimated to induce fewer than 170 fatal cancers per Gt of fission yield over the succeeding 50 yr, more or less equally caused by exposure to deposited $^{137}$Cs and by ingestion of contaminated NZ-grown foodstuffs.

Fallout from small-yield Australian detonations at low altitude would be responsible for as many as 70 fatal non-thyroid cancers per Mt of fission yield, mainly as a result of external exposure to deposited nuclides. That this number is so large is a consequence of pessimistic assumptions about the meteorology which enhance the role of short-lived radionuclides.
(notably $^{140}$Ba and $^{95}$Zr and their daughters). Milk products contaminated by $^{131}$I could be responsible for about 3000 cases of thyroid nodules per Mt of fission protracted over 50 yr. All of these nodules should be medically - if not surgically - examined to identify the one third which could prove malignant. With contemporary medicine 10% of thyroid cancers prove fatal. However, countermeasures to minimise such exposure to $^{131}$I are available: cattle can be supplied stored food; contaminated milk can be withheld from distribution (eg reserved for milk powder or cheese production); iodide tablets taken in advance can block radioiodine retention.

In short, even allowing for the uncertainty in the estimates, it is unlikely that radiogenic cancers will become detectable among the indigenous NZ population, as long as NZ remains untargeted. The increase in thyroid disorders may be statistically detectable in the absence of effective countermeasures. Moreover, the consequences of thyroid dysfunction and other radiogenic morbidities may be exacerbated by the diminished availability of NH-sourced maintenance drugs.

CONCLUSIONS

The scenarios as presented are unlikely to give rise to a significant health hazard among the NZ population. This conclusion should be unaffected by details of the postulated conflict, provided that no detonations occur within 2000 km upwind of NZ. It is also assumed that no "nuclear winter" effect radically alters the atmospheric circulation patterns to subject the SH to more fallout of NH origin. Such a conclusion is in qualitative accord with that of McEwan [1985] who presents little detail of the calculations employed but appears to have used latitudinally-averaged data similar to tabulations in SCOPE 28 [Pitcock et al., 1986, Chapter 7].

The assessments presented here take no account of "hot spots". These could arise from atypically high local rainout of Australian-sourced air pollutants. The scope for this is limited because the meteorological postulates of Annex 2 already assume NZ as a $10^5$ km$^2$ "warm spot"; however, NZ rainfall is orographically influenced and certainly not uniform. Rainfall variabilities could conceivably produce hot spots in areas of high population density or in regions of importance to the food chain (e.g. due to orographic rainfall either on the pastures of lower alpine slopes, or accumulating as runoff in rivers or lakes with fish stocks important to a
sub-population). It is noted in Annex 1 that forecasted $^{137}$Cs contamination is comparable to levels in Europe which followed the Chernobyl accident - indeed is comparable to hot spots in Britain after Chernobyl. Such contamination is sufficient to cause concern (though not an immediate health hazard) especially in respect of agricultural production (including milk), and especially again in Taranaki where cesium levels in plants may persist at a higher level than elsewhere.

Notwithstanding the forecasted minor health impact, the possibility cannot be discounted that some overlooked reconcentration of a hazardous radio-nuclide could occur to contaminate a local food resource or food-chain component of importance to a sub-population. The reconcentration could be due to a physical channeling (high local rainout and runoff) or to a metabolic sorption by plant or animal species. Practically all of the data on food-chain reconcentration is sourced in the NH, so that chains of unique importance to NZ or to a sub-population could escape attention.

It is clear from Annex 2 that estimates of fallout of Australian source depend heavily upon the postulated weather patterns over the Tasman Sea. However, relatively little is known about trans-Tasman weather trajectories and rainfall incidences [T.S. Clarkson (NZ Meteorological Service), personal communication]. Trans-Australasian meteorology thus provides a major uncertainty in assessing fallout over NZ of Australian-sourced pollutants.

Little work seems to have been done to document the properties of NZ soils in respect of their response to cesium (in particular), iodine and strontium contamination. The only source of information on the anomalously weak cesium-fixing capacity of those soils and its variability comes from monitored $^{137}$Cs levels (by NRL) following the 1960s' bomb tests and from NRL's follow-up [Baltakmens and Gregory, 1977]. Any contingency planning (such as determining emergency reference levels) for hazardous radiocesium contamination of NZ pastureland should take account of this variability in cesium fixation for which Taranaki soils are especially anomalous - resulting in a relatively high transfer of soil-borne cesium to pasture grasses (and to other plants?).
ACKNOWLEDGEMENTS

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ANNEX 1: CHRONIC EFFECTS OF NORTHERN HEMISPHERE DETONATIONS

Fallout over NZ from the postulated huge yield of northern hemisphere (NH) detonations would dominate that from the much smaller yield of southern hemisphere (SH) detonations after a few months. This was noted in fallout monitoring programmes in the 1960s: air concentrations of fission products over NZ peaked in 1964, one year after the NH peak (and a factor of about 3.5 smaller), following the major detonation yields of 1961-2.

The scenarios as supplied do not quantify explosive yields. In this cursory analysis I consider a reference total fission yield of 1Gt (Giga-tonne), equal to 1000 Mt (Megatonne) of TNT equivalent. I assume an approximate scaling of NZ consequences with NH total fission yield; this scaling will be most dependable if the total number of detonated warheads rather that the mix of warhead sizes contrast different war scenarios. NZ consequences can then be cited per Gt of total fission yield.

Atmospheric testing of nuclear weapons since the mid 1950s has detonated a total of about 550 Mt of explosive including 217 Mt of fission yield [UNSCEAR, 1982, p227], mostly in the NH. The fallout measured over NZ from these tests should then provide an indication of the fallout to be expected from a NH-dominated war, and any effects peculiar to NZ are then built in. Consequently I rely on the fission-product monitoring programme conducted by the National Radiation Laboratory (NRL) in Christchurch throughout the 1960s and 1970s, dividing the results by 0.217 to express per Gt of fission yield. Nevertheless, this scaling from bomb tests to nuclear warfare is only approximate [McEwan, 1985]. Most recent scenarios [AMBI0, 1982; Pittock et al., 1986, Table 2.2] tend to postulate (i) a mix of weapon sizes somewhat different (smaller on average) from those detonated in atmospheric tests, and (ii) a larger proportion of surface bursts (aimed at missile silos) for which about about 50% of the fission products fall out locally. Thus forecasted long-range fallout scaled from weapon-test fallout might be overestimated by a factor of perhaps 2 to 10.

I consider just two modes of chronic exposure: protracted external exposure to fallout deposits (groundshine mode), and exposure to fallout incorporated into ingested foodstuffs, especially cow's milk (ingestion mode). Because these chronic effects are small, I need only be approximate. Special consideration is given to the activation product $^{14}$C. I consider some exacerbating effects of targeted commercial nuclear facilities.
Groundshine

The radionuclide of dominant concern for all but the first few months post-detonation is $^{137}$Cs with half-life 30 yr. Because of the lag between NH detonation and SH fallout (due to long stratospheric residence times) only this nuclide need be considered.

Because rainout predominates over dry deposition as the major deposition mechanism, deposition levels are proportional to local rainfall. The NRL has routinely reported levels of $^{90}$Sr in local rainwater, reporting these as deposition levels (in MBq/km$^2$); these are highest in the West Coast. A graphical summary is presented in the review for 1981-1985 [NRL, 1986]; rain since 1973 is almost totally free of $^{90}$Sr indicating that fission-product rainout is complete within a decade. The time-integrated countrywide average deposition level over the nuclear-testing era is 740 MBq ($^{90}$Sr)/km$^2$; this is deduced from individual NRL Annual Reports with linear interpolation from zero in 1955 to the earliest reported result in 1960. The cited level is uncorrected for post-deposition radioactive decay and so represents the deposits to which inhabitants have been (and are being) exposed.

The radionuclides $^{137}$Cs and $^{90}$Sr have very similar half lives and their measured activity ratio is consistently near 1.6 [UNSCEAR, 1982, p220]. Based on the monitored $^{90}$Sr in rainfall the average integrated $^{137}$Cs fallout over NZ has been 5.5 KBq/m$^2$/Gt of fission.

To put this deposition into perspective, European deposits following the Chernobyl reactor accident have been reported (preliminarily) as follows. A New Scientist report [Webster, 1986] places Glasgow "among the areas with the highest levels of radionuclide deposition" (in Britain?) at about 2 KBq/m$^2$ of $^{137}$Cs in one day (3 May 1986) accumulating to around 9.5 KBq/m$^2$ by 9 May. The US Environmental Measurements Laboratory reported some Swedish $^{137}$Cs depositions exceeding 200 KBq/m$^2$ in "hot spots", but more typically a few KBq/m$^2$ which is near the weapons-fallout background [USEML, 1986]. A report by the World Health Organisation [WHO, 1986] indicates model-simulated levels of about 5 KBq($^{137}$Cs)/m$^2$ throughout most of Europe except at hot spots of high rainout of up to 140 KBq/m$^2$.

As a further perspective, the U.K. National Radiation Protection Board has proposed "derived emergency reference levels" (DERLs) for deposited $^{137}$Cs
of 180 to 890 MBq/m² [White, 1986]. These levels are trigger points for emergency evacuation in the absence of which 14-day doses of 100 to 500 mSv would be delivered.

Deposits are weathered into soil surfaces which then provide inhabitants with some shielding from decay gamma rays. Cesium is strongly retained in the upper few centimetres due to its fixation in most soil minerals [e.g. Baltakmens and Gregory, 1977] and so is likely to remain in the upper few centimetres of unirtled soils for its natural lifetime. (However, Taranaki soils are notably anomalous in their poor cesium-fixing capacity). Fallout on paved surfaces is likely to be removed by rain-induced or hosed runoff.

UNSCEAR [1977, p150] has deduced a whole-body "dose conversion factor" for $^{137}$Cs deposition which relates the dose delivered over all time to the whole body of an inhabitant (external irradiation is not very tissue-specific) to the exposed fallout level. The dose conversion factor 0.39 mGy/(KBq/m²) accounts for the effect of shielding from weathered soils by presuming exponential penetration to a mean depth of 3cm. For the scenario considered the dose commitment is thus 2.1 mGy/Gt (0.21 rad/Gt). This estimate compares favourably with the SCOPE 28 result [Pittock et al., 1986, Table 7.3] for groundshine in the latitude band 30-50°S from a single mid-NH latitude summer groundburst. The latter cites a 50-yr dose (unshielded) which increases with yield from 0.02 rad/Gt (for a 0.1 Mt burst) to 0.4 rad/Gt (20 Mt burst). The increasing dose per Gt is predominantly due to the increasingly more efficient delivery of fission products to the SH (via the stratosphere) for the higher yield bursts.

Using a risk factor of 12.5 fatal cancers per million exposed per mGy of dose to the whole body [ICRP, 1984, Appendix A], a groundshine dose of 2.1 mGy/Gt to a population of 3.3 million would induce about 87 fatal cancers per Gt of total fission yield over some 50 yr. The total number of induced cancers would be treble that number — viz, about 300 per Gt.
Ingestion

The radionuclides of concern are $^{90}$Sr and $^{137}$Cs. I consider their contamination of milk, probably the single most important foodstuff as a pathway of these environmental radionuclides to the human body.

The NRL monitoring programme has reported milk-contamination levels $M(t)$ at time $t$ throughout and since the 1960s. The contaminating mechanisms are twofold: direct fresh fallout onto pasture grasses, and the slower and less efficient contamination due to uptake from soils contaminated in the root zone by fallout years or decades earlier. For the 217Mt of fission yield from atmospheric bomb tests the measured milk contamination (country-wide average) integrated over the nuclear era is:

$$\int_{1955}^{1985} M_{90}^{\text{Sr}}(t) \, dt = 110 \text{ SU-yr},$$

$$\int_{1955}^{1985} M_{137}^{\text{Cs}}(t) \, dt = 384 \text{ CU-yr}.$$

The data is from individual NRL annual reports (see a graphical summary in NRL [1986]) with linear interpolation from zero in 1955 to the earliest record. The units SU (strontium units) and CU (cesium units) are pCi/g(Ca) and pCi/g(K) respectively. Assuming calcium and potassium contents of milk given by 1.2g(Ca)/l and 1.4g(K)/l, and a per capita intake of 0.7 l/day (conservative estimate for a child, significant overestimate for an adult) the time-integrated activity ingested via milk is 5.8 KBq($^{90}$Sr)/Gt and 23 KBq($^{137}$Cs)/Gt.

What are the doses delivered to critical body tissues, and the somatic effects of those doses, as a result of $^{90}$Sr and $^{137}$Cs ingestion? As a conservatism I assume that the doses delivered are as if the lifetime intake were ingested on the first day; in practice the ingestion rate declines rapidly over a decade or so post-deposition. The doses are delivered internally in a manner dictated by the timescale and sites of internal redistribution of the radiocontaminant - i.e. by strontium and cesium metabolism. The cumulative dose per unit ingestion has been assessed for several critical tissues. Cesium becomes redistributed fairly uniformly throughout muscle tissue and is excreted within a few years. With no preferential target tissue, the critical tissue becomes the "Whole
body" as for external irradiation, and the time-integrated dose is about 15 mGy/MBq ingested [USNRC, 1975, Table VI 8-4], or for the scenario here, 0.35mGy/Gt (fully delivered within a decade of ingestion). Strontium, on the other hand, is a bone seeker and the beta dose is delivered predominantly to the bone over some 30-40 yrs accumulating to 290 mGy/MBq ingested [USNRC, 1975, Table VI 8-4], or 1.7 mGy/Gt.

Assuming risk factors of 12.5 and 0.5 induced fatal cancers per million exposed per mGy to the whole body and to the bone respectively [ICRP, 1984, Appendix A], the excess cancer fatalities per Gt of total fission yield over 50 years among an exposed population of 3.3 million persons is 14 and 2.8 (respectively) - a total of 17 per Gt of fission. Data for the USA presented in the "Reactor Safety Study" [USNRC, 1975, section E3.2] suggests that milk provides about 25% and 33% of a typical dietary input of $^{137}$Cs and $^{90}$Sr. Scaling in this way predicts excess fatal cancer inductions via ingestion of about 66 in total per Gt of fission yield. Inclusion of curable cancers would treble this number: 200 per Gt.

Note that the NRPB has proposed DERLs for dairy pastureland contamination of 35 to 350 KBq($^{137}$Cs)/m$^2$ as a trigger for emergency milk interdiction [White, 1986]. For a forecasted deposit of 5.5 KBq/m$^2$/Gt, the lower DERL limit would be realised for a NH conflict of 6 Gt (fission).

Carbon 14

UNSCEAR [1982, p242] estimates the dose commitment (dc) to the world population from atmospheric nuclear tests. The dominant contribution is 2.6 mSv from 14C but is protracted over the natural lifetime of $^{14}$C (mean life 8267 yr); of that dc 7%, 8% and 10% are delivered by the years 2000, 2020, 2050 respectively. Assuming most of the release during 1961-2, approximately 0.2 mSv, is delivered within 50 yr of $^{14}$C production, and the remaining 2.4 mSv is delivered over thousands of years to many generations.

Production of $^{14}$C is by the same mechanism as cosmogenic (natural) $^{14}$C; from neutron capture by atmospheric $^{14}$N. The production rate is thus proportional to the neutron flux liberated by the detonation into air, and thus depends upon both fusion and fission yields, the former being the major neutron source per Mt. Production of $^{14}$C also depends upon the proximity of the detonation to the surface; for a ground-burst half the
neutron flux is absorbed in the ground. Thus $^{14}$C production does not closely scale with fission, fusion or total yield, but as a guide the bomb-$^{14}$C production to date of 220 PBq [UNSCEAR, 1982, p214] is equivalent to about 400 PBq/Gt(total) or 660 PBq/Gt(fusion).

Assuming the more conservative proportionality of $^{14}$C production to total yield, the 50-yr dc is approximately 0.4 mSv/Gt(total). This dc would induce about 17 cancers per Gt(total) among NZ survivors of the war, and perhaps a dozen times this number over the succeeding 10,000 yrs.

Commercial Nuclear Facilities as Targets

If nuclear facilities (commercial reactors, fuel-reprocessing facilities, spent-fuel storage facilities) are targeted, then the contents of those facilities are exposed to the environment. I illustrate by considering a 1 GW(e) (= 1000 MW(e)) reactor as target, and consider the pessimistic prospect of the entire core becoming pulverised and airborne in just the same manner as the bomb debris; this prospect is of questionable likelihood. More detail is provided by Pittock et al. [1986, Appendix 7A] who also show that fuel-reprocessing and spent-fuel facilities may present more hazardous targets. Almost all commercial reactors are sited in the NH and so the importance of SH fallout can be gauged by looking at the single radionuclide $^{137}$Cs, together with any further nuclides not prevalent in bomb debris (for which we consider plutonium rather than uranium as the fissile material). A typical core inventory of $^{137}$Cs is 170 PBq/GW(e) [USNRC, 1975, Table VI 3-1], compared to bomb-produced $^{137}$Cs at a representative rate of 5.9 PBq/Mt(fission) [UNSCEAR, 1982, p228]. Thus for each GW(e) the core's inventory of $^{137}$Cs is equivalent to a 30 Mt fission weapon. Since the core of a 1 GW(e) reactor has a thermal energy production of 3.5 GW(th), an alternative equivalence is 10 Mt to 1 GW(th).

From the above consideration of groundshine the dose delivered from weathered deposits scales to 63 $\mu$Gy/GW(e) from $^{137}$Cs. To this should be added a contribution from $^{134}$Cs (half-life 2 yr) which is prevalent in uranium fission. By comparison, Pittock et al. [1986, Table 7A.1] cite a 50-yr dose commitment for the 30-50°S latitude of 3 mGy per 100 GW(e), or 30 $\mu$Gy/GW(e), without accounting for weathering or for local rainout.
Summary

Contamination of ground and of foodstuffs as a result of fallout from NH nuclear detonations is not likely to provide a detectable health hazard among a background cancer incidence of 9000 p.a. and mortality rate of 5500 p.a. Fewer than about 170 cancer fatalities over the succeeding half century are likely to result among the indigenous NZ population. The number of induced genetic disorders would be of comparable magnitude, but delivered over several generations. Such chronic effects might be enhanced as a result of a diminished supply of medical equipment and pharmaceuticals available in NZ after the NH-dominated war.
ANNEX 2: CHRONIC EFFECTS OF AUSTRALIAN DETONATIONS

Trans-Tasman traversal times for airborne pollutants are typically 80 hrs. Consequently in exploring the impact on NZ of a fission-product plume of Australian origin it is necessary to consider potentially hazardous nuclides with half lives as short as a day or less.

Except for "small" near-surface detonations (eg, less than 100 Kt), significant quantities of debris will be injected directly into the stratosphere where they remain resident for several years. The proportion so injected depends upon both yield and altitude of the detonation, and so upon the specifics of the postulated nuclear attack. My approach is that, of all low-altitude detonations over Australia subjected to westerly weather patterns, half the fission yield follows the normal pattern of global fallout (including stratospheric residence, etc.) with NZ a fallout destination on equal terms with all other destinations in its latitude. The other 50% of fission debris are presumed to enter the lower troposphere, and their effects are assessed pessimistically in the light of NZ's downwind position. Since the proportion of tropospheric injection per detonation does not scale with its megatonnage (eg see Pittock et al. [1986, Table 7.3]) this approach will be inappropriate for cumulative yields exceeding a few Mt (assuming fewer than about 10 low-altitude detonations in southern Australia). Quantitative results are cited per Mt of fission (for low-altitude detonations) even though such scaling with yield is at best approximate. I use the abbreviation "t.i. fraction" for the fraction of tropospherically-injected fission products.

Stratospherically injected debris seems to fall out predominantly in the hemisphere of entry [UNSCEAR, 1982, p214]. Judging from the world-wide pattern of $^{90}\text{Sr}$ fallout, some 75% falls out in that hemisphere (a simple guesstimate noting the UNSCEAR result that fallout of $^{90}\text{Sr}$ in the NH and SH is in the ratio 3.1). Thus fallout sourced in Australia and depositing on NZ after prolonged stratospheric residence produces effects about 1.5 times those noted in Annex 1 for NH detonations; this factor comprises 3 (for fallout in the same hemisphere as entry) and 0.5 (the complement of the t.i. fraction). Thus quotients expressed per Mt are $1.5 \times 10^{-3}$ times those expressed per Gt in Annex 1 and as such do not warrant separate consideration.

There is a dearth of information on the behaviour of plumes traversing the
Tasman Sea - the extent of crosswind spreading and of rainout (even of rainfall), etc. [T.S. Clarkson (NZ Meteorological Service), personal communication]. One quantitative estimate of the impact upon NZ of Australian sourced air pollution is the study of acid rain over NZ by Holden and Clarkson [1986]. These investigators sought to gauge the scope of the acid rain problem in NZ caused by sulphur injected (as $SO_2$) into the SE Australian atmosphere. Holden's and Clarkson's quantitative input to the problem, where poorly known, erred on the conservative side. I follow that philosophy.

One difference between modelling the impact of a single episode or short sequence of episodes (such as herein) and the modelling of a pervading situation (such as sulphur transport from an ongoing source) is that the latter can afford to employ time-average parameters in order to deduce average or cumulative deposition levels. For an episodic event one considers instead a scenario and proceeds conservatively, acknowledging that the simulated impact most surely over-estimates most realisable impacts.

For transport of fission-product within the Australasian troposphere I make the following conservative assumptions:

(a) no airborne radionuclides are lost from the plume(s) via rainout en route to NZ (i.e. it doesn't rain during transit to NZ);
(b) maximum plausible deposition of the plume contents occurs over NZ;
(c) deposition over NZ is presumed uniform, so that no "hot spots" occur in inhabited regions or in areas of importance to the food chain;
(d) despite maximal loss by rainout from the plume, air concentrations over NZ are computed as at the western coast as if no depletion had occurred.

Put simply, I assume that the plume is not rained out until it reaches NZ, whereupon the full plume contents are rained out uniformly over the country.

Holden and Clarkson [1986] adopt a "box model" (earlier presented by Fisher [1983]) to the trans-Tasman situation. The rectangular box has opposite sides bounded by the Brisbane-Adelaide and Kaitaia-Invercargill transects, and is 2200 km long and 1500 km wide, with mid-line connecting Sydney and Wellington. The box encloses all of SE Australia (where most of Australia's major sources of sulphur pollution lie, save Mt Isa which is subject to prevailing easterly winds). For such SE Australian pollution sources the trans-Tasman depletion due to dry deposition alone is -
\[ C = \frac{Q \alpha}{(\Lambda \mu a)}, \quad \alpha = f \exp(-\beta x/u) \]

where:
- \( C \) = time-integrated air concentration over NZ (eg, in Bq-day/m³);
- \( Q \) = source strength in Australia of tropospheric injection (eg, in Bq);
- \( \alpha \) = trans-Tasman depletion factor;
- \( f \) = fraction of air trajectories connecting SE Australia and NZ (0.40);
- \( \beta = \nu/\varepsilon \) = removal rate by dry deposition;
- \( \nu \) = dry-deposition velocity (1.0 cm/s);
- \( a \) = mixing height (1000m);
- \( u \) = wind speed (8 m/s);
- \( L \) = width of box (1500 km).

The values recommended by Holden and Clarkson are shown above. The sources \( Q \) are presumed to be 50% (the t.i. fraction) of fission production (Table 1).

The pollutant soon spreads into a uniform vertical profile up to the mixing height, which depends strongly upon the prevailing weather pattern - typically from 200m on still nights with stable temperature gradients to 1500m on sunny days with temperature gradients inverted by high insolation. A diurnal dependence should strictly be incorporated [Fisher, 1984], but the data does not support this refinement, and a typical average value of 1000m is selected. The wind speed of 8 m/s corresponds to an 80 hr trans-Tasman crossing. The value \( f=0.40 \) is a conservative guesstimate by Holden and Clarkson in the almost total absence of pertinent data (save some back-trajectory analyses from a Ninety-Mile Beach site). The total SE Australian source is thus presumed to disperse into a cross-wind width of \( L/f = 4000 \) km at 2200 km downwind (approx. 100° sector).

The above numerical estimates imply that \( \alpha = 2.6 \times 10^{-2} \) and \( C/Q = 2.5 \times 10^{-17} \) day/m³.

For sources in south-western and southern Australia the same broad approach can be followed; they can be conservatively treated as if they were all in SE Australia. Sources in northern Australia (and probably central Australia too) are subjected to prevailing easterly conditions and so reach NZ only after almost circum-navigating the hemisphere; they are best treated by latitudinal averaging whereupon effects are insignificant compared to fallout of NH origin.
The plume, depleted to a fraction $\alpha$ en route to NZ, is presumed to be rained out over NZ; this can be accomplished to within 8% by the plausible rainout coefficient $10^{-4}$ s$^{-1}$ (assuming a NZ "width" of 200 km). The country-wide average deposition level would be:

$$D = \frac{\rho A C_{\alpha}}{A} = \alpha \frac{Q A}{A}$$

where $A$ is the total surface area ($2.7 \times 10^{11}$ m$^2$), so that $D/Q = 1.0 \times 10^{-13}$ m$^{-2}$. Thus, noting the 50% tropospheric injection, a fraction $5 \times 10^{-14}$ of the SE Australian source (corrected for 80 hr of decay) is assumed to deposit on each m$^2$ of NZ territory. From the fission yields of Table 1, the NZ-wide deposition of $^{90}$Sr would then be 0.19 kBq/m$^2$/Mt. This may be compared for credibility with monitored $^{90}$Sr fallout accumulated to 1980 (taking account of decay) in mid latitudes of the same (northern) hemisphere as the detonations, of $3.23/217 = 0.015$ kBq/m$^2$/Mt [UNSCEAR, 1982, p230]. The factor of 12 discrepancy is due to the conservative assumptions made in recognition of NZ's downwind position.

Australia has a research reactor (at Lucas Heights on the outskirts of Sydney) with power 10 MW(th). As derived in Annex 1, in the event of its destruction and dispersal, the resulting release of $^{137}$Cs is equivalent to a 100 Kt detonation. The $^{131}$I content of the core is proportionately smaller and equivalent to a 2 Kt detonation.

Three modes of exposure are considered as of potential importance: exposure to deposited $\gamma$-emitters (groundshine), exposure to radionuclides inhaled directly from the plume (inhalation mode), and exposure to ingested radionuclides (ingestion mode).

**Groundshine**

The nuclides of concern are shown in Table 2 along with dose-conversion factors for accumulated exposure over 7 and 30 days post-deposition and over all time. The conversion factors (dose delivered per unit initial deposit) are computed from the extensive tabulations by Kocher [1980] who presents dose rates 1m above an infinite smooth plane of unit deposition. The conversion factors of Table 2 take account of all $\gamma$-decays from parent and daughter(s). Table 2 also relates the doses to a reference Mt of fission using the yields of Table 1 and taking account of decay during the 80-hr traversal of the Tasman Sea and of the t.i. fraction.
The relatively important role of short-lived radionuclides (compared to $^{137}\text{Cs}$) is apparent. In particular, significant radionuclides are $^{140}\text{Ba}$ and $^{95}\text{Zr}$; in both cases their daughters ($^{140}\text{La}$ and $^{95}\text{Nb}$ with half-lives 40 hr and 35 day) are important contributors to dose. The relative downplaying of the role of $^{137}\text{Cs}$ arises because the assumed atmospheric residence (80 hr) is much shorter than is typical.

The dose integrated over all time, without assuming mitigation due to weathering or shielding, is about 1.6 mSv/Mt, about half of which is delivered in the first month. With the conservative assumptions made this has the potential to exceed the dose from delayed fallout from NH detonations, and could be responsible for as many as 66 fatal cancers (200 in total) induced in NZ over 50 yr per Mt of fission over southern Australia. This calculation assumes a risk factor of 12.5 radiogenic cancers fatalities per million exposed per mGy of exposure [ICRP, 1984, Appendix A].

By comparison, SCOPE 28 [Pittock et al., 1986, Table 7.3] reports fallout averaged over the 10-30° latitude band in the same hemisphere as a single detonation at 40° of between 0.4 and 0.02 mGy/Mt, decreasing with increasing yield.

The groundshine dose per Mt of SE Australian detonations is of similar magnitude to that per Gt of NH detonations. However, the conservative assumptions inherent in the former estimate surely result in its overestimation.

Inhalation

As an indication of the importance of inhalation as an exposure mode, I consider only the doses delivered to the lung and to the thyroid following retention of inhaled radionuclides. The radionuclides important for lung irradiation are listed in Table 3. Only $^{131}\text{I}$ is considered in assessing the thyroid dose. The air concentrations are those predicted by the box model (i.e. C) undepleted by rainout.

Assume a normal adult respiratory rate of $R=20\text{ m}^3/\text{day}$. The total activity of each nuclide inhaled per person is then $RC$, which equates to a fraction $2.5\times10^{-16}$ of the SE Australian source after 80 hr airborne. These activities are recorded in Table 3 along with dose conversion factors for the lung which take account of metabolic redistribution and elimination of the
inhaled species. The dominant contributions are from $^{106}$Ru and $^{144}$Ce as a result of their strong retention in the lung. The total lung dose is of order $100 \mu$Sv/Mt, protracted over 50 yr. Ignoring this protraction (i.e. assuming the entire dose to be delivered at the time of inhalation) and assuming a risk factor of 2 induced fatal lung cancers per million persons exposed per mSv dose to the lung [ICRP, 1984, Appendix A], 0.7 cancers are so induced over 50 yr among a population of 3.3 million for each Mt of fission yield.

A similar calculation for the thyroid dose proceeds as follows. The $^{131}$I activity inhaled after 80 hr airborne is $0.79 \text{ KBq/Mt}$ and, for a dose-conversion factor of $300 \mu$Sv/KBq [USNRC, 1975, Table VI D-2], a dose of $230 \mu$Sv/Mt is delivered to the thyroid. Adopting a risk factor of 0.5 fatal cancers (or 5 total cancers) per million exposed per mSv to the thyroid [ICRP, 1934, Appendix A], there results 4 thyroid cancers per Mt, 10% of them fatal, expressed over some 50 yr.

Inhalation is thus not a critical exposure pathway.

Ingestion

I focus upon contamination of milk since (i) milk is an important dietary foodstuff - particularly for children, and (ii) the dairy cow represents an efficient "vaccuum cleaner" as it grazes, reconcentrating with high efficiency certain contaminants dispersed over the pastureland.

Pasture grasses can be contaminated by two mechanisms: direct fallout onto foliar surfaces and retention thereon; and uptake of soil-borne contaminants. The former is of short-term importance and the latter of long-term, and I refer to them as the "direct-deposition" (d.d.) and "root-uptake" (r.u.) mechanisms.

Assessment of milk contamination via d.d. follows the approach of the "Reactor Safety Study" (RSS) [USNRC, 1975, Appendix VI E-3]. It is assumed that 50% of the depositing fallout is intercepted by foliar surfaces, the remaining 50% penetrating the grass cover onto the soil surface. The actual intercepted fraction depends upon grass densities but 50% is a reasonable average. The intercepted material is weathered from the foliage onto the soil with a decline which RSS models as exponential with half-time 14 days. The fraction of the fallout available to the grazing cow is thus
0.5 \exp(-0.693t/14) \text{ where } t \text{ is the post-deposition time in days. This conceptual model of the process has been refined by Lassey [1982] at a cost of increased mathematical complexity.}

The above considerations are supplemented by the following assumptions [USNRC, op cit]:

(a) fresh pasture is continually being grazed (at 45 \text{ m}^2/\text{day/cow}) during the month or so that d.d. is operative;
(b) there is a 3-day delay between milking the cows and consuming the milk;
(c) empirical "transfer functions" relate the metabolic secretion into milk of contaminants ingested by the cow;
(d) a critical consumer continues to drink 0.7 litre of milk daily, thereby ingesting via milk a total activity \( M \) over the few weeks after deposition.

USNRC [op cit] cite a concentration factor (CF) as the quotient \( M/D \) (where \( D \) is the fallout level). Although the details of that calculation may now be somewhat obsolete [e.g. Ng et al., 1977] and contains minor errors [Lassey, 1980] it is dependable enough for present purposes. For the key radioisotopes of strontium, iodine and cesium the CFs for d.d. are reported in Table 4. The large values for cesium reflect the high efficiency of cesium secretion to milk (about 2% is so secreted). Iodine secretion is less efficient (about 1.2%) but peaks within a day. Less than 0.1% of dietary strontium is secreted into dairy milk.

In deducing CFs for the r.u. pathway I utilise the analyses of UNSCEAR [1977, Annex C] and of Lassey [1979]. Only the isotopes of strontium and cesium need be considered.

UNSCEAR derived an empirical relationship between fallout levels and levels of \(^{90}\text{Sr}\) and \(^{137}\text{Cs}\) in milk. These take the form:

\[
C_i = b_1 F_i + b_2 F_{i-1} + b_3 \sum_{m=1}^{\infty} e^{-\mu m} F_{i-m},
\]

where \( C_i \) is the average contamination level in year \( i \) (expressed in SU or CU as defined in Annex 1) as a result of fallout \( F_i \) during that year and of fallout \( F_{i-1}, \text{ etc.}, \) during preceding years. Lassey [1979] showed how to apply these extensively-reported parameters \( (b_1-b_3, \mu) \) to predicting the impact of a single contaminating episode. For a deposition \( D \) of \(^{137}\text{Cs}\) or of \(^{90}\text{Sr}\) the level of milk contamination would be -
\[ C(t) = D b_3 \{\exp(-\mu t) - \exp(-\zeta t)\} \]

where \( \zeta \) is the rate of removal from pasture due to both grazing and radioactive decay. The effects of local conditions are therefore embodied in the parameters \( b_3 \) and \( \mu \). The formula can be applied to other isotopes of Cs and Sr by inserting the appropriate decay constants. The CF follows:

\[
\text{CF} = V \int_0^\infty \frac{C(t)}{D} \, dt = V b_3 (\mu^{-1} - \zeta^{-1})
\]

where \( V \) is the per capita consumption rate.

For \( ^{90}\text{Sr} \) fallout in milk products UNSCEAR [1977, p130 & p143] cites (an Australian average, the NZ value being unavailable):

\[
b_3 = 0.43 \text{ SU per mCi/km}^2 = 0.52 \times 10^{-3} \text{ m}^2/\text{y of milk},\]

\[
\mu = 0.09 \text{ per yr};
\]

for \( ^{137}\text{Cs} \) fallout (a NZ average):

\[
b_3 = 3.62 \text{ CU per mCi/km}^2 = 5.07 \times 10^{-3} \text{ m}^2/\text{y of milk},\]

\[
\mu = 0.51 \text{ per yr}.
\]

Setting \( V=0.7 \text{ l/day/person} \) for a critical age cohort and the mean pasture regrazing rate to 3 yr\(^{-1} \) (based on the national dairy herd and grazing area), the resulting CFs are as reported in Table 4. It is noteworthy that the r.u. pathway has the potential to deliver more \( ^{90}\text{Sr} \) into milk than does d.d., but the former is protracted over a time of order \( \mu^{-1} = 11 \text{ yr} \). For \( ^{137}\text{Cs} \) both pathways are of comparable importance.

It must be noted that the NZ value for \( b_3 \) is anomalously high by comparison with other regional averages, apparently due to the more ready availability for root uptake of soil-borne cesium. On a finer scale, NZ soils are quite variable and it is Taranaki soils in particular which display this anomalous feature very strongly. It is attributed to the poor cesium-fixing capacity of these soils [Lassey, 1979] as a result of which the greater mobility of cesium is evident [Baltakmens and Gregory, 1977]. During the 1970s when \( ^{137}\text{Cs} \) was detectable in milk, its concentration in Taranaki milk was about three times the national average even though local rainfalls are not excessive.
The CFs of Table 4 can be related to a dose delivered to critical tissues. Such doses are reported in Table 5. The activity ingested via other foodstuffs is variable [UNSCEAR, 1977, Annex C] but should not exceed that via milk by more than an order of magnitude (usually much less) for Sr and Cs; milk products are the dominant source of $^{131}$I.

Table 6 records the estimated incidence of induced radiogenic cancers per Mt of fission. These will be expressed over several decades after dose delivery. The expression of non-thyroid cancers is unlikely to be detectable until the fission yield exceeds several tens of Mt (whereupon the assumed t.i. fraction is questionable).

The fatality rate for thyroid cancers is approximately 10% [ICRP, 1984, Appendix A], so that thyroid cancer induction is about 1000 per Mt. Furthermore, thyroid nodules which prove benign (thyroid adenomas) have about twice the induction rate of malignant nodules [BEIR, 1980, Appendix A to Chapter V], so that some 3000 induced nodules per Mt would require medical treatment or surgical intervention over several post-war decades. However, the mean life of $^{131}$I on pasture is about 7 days (due to decay and weathering), so the dose can be largely avoided if dairy cattle are fed from stored food for the first few weeks and/or contaminated milk is detected and interdicted. The consumption of therapeutic doses of stable iodine (eg as iodide tablets stocked for this purpose) is believed to effectively block subsequent thyroidal absorption of (radio)iodine.

The dose delivered by $^{131}$I to the thyroid via milk consumption is 290 times that delivered via inhalation. That the former pathway is the critical one for exposure to $^{131}$I is well established. Burnett [1970] showed that for air concentrations and ground depositions equilibrated by a dry deposition velocity of 1.0 cm/s and with the dose conversions prevailing in the 1960s together with a milk consumption rate of 1.0 litre/day, the ratio of doses delivered via milk consumption and inhalation is 700:1.
Summary

The assumptions made for the source of short-term fallout are:
(a) the fission yield of all southern Australian low-altitude detonations
    is equally divided between stratosphere and troposphere; and
(b) 40% of the tropospheric content is airborne for 80 hr before being
    totally rained out over NZ.

Assumption (a) is probably appropriate for yields (fission + fusion) of
order 0.1 Mt per detonation at low altitude. For larger detonations or
high-altitude bursts the fallout over NZ is considerably over-estimated
since a decreased t.i. fraction would apply. Detonations in central or
northern Australia are most likely to be carried by easterly weather
patterns and contribute to the average atmospheric burden. Assumption (b)
embodies the most pessimistic meteorology.

External exposure to deposited fallout might be responsible for as many as
66 fatal cancers (200 cancers in total) per Mt of total fission yield,
expressed over about 50 yrs. Consumption of contaminated foodstuffs would
induce a much smaller number of non-thyroid cancers. In the absence of
countermeasures, the $^{131}$I content of milk in the few weeks after local
deposition might be responsible for up to about 3000 thyroid nodules
(expressed over several decades) per Mt of fission yield; all will require
medical treatment though only about a third will prove malignant and only
about 10% of those would normally prove fatal. Such medical treatment
depends upon the ready availability of normal medical diagnostic and
remedial action and of pharmaceuticals (including the drugs essential to
compensate for thyroid dysfunction).
REFERENCES


Ng Y.C., Colsher C.S., Quinn D.J. and Thompson S.E., "Transfer coefficients for the prediction of the dose to man via the forage-cow-milk pathway from radionuclides released to the biosphere", University of California (Lawrence Livermore Lab.) report UCRL-51939, 1977.


<table>
<thead>
<tr>
<th>Nuclide</th>
<th>Half-life (day)</th>
<th>Production (PBq/Mt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$^{89}$Sr</td>
<td>50.5</td>
<td>590</td>
</tr>
<tr>
<td>$^{90}$Sr</td>
<td>$1.04 \times 10^{14}$</td>
<td>3.9</td>
</tr>
<tr>
<td>$^{90}$Zr</td>
<td>64.0</td>
<td>920</td>
</tr>
<tr>
<td>$^{103}$Ru</td>
<td>39.4</td>
<td>1500</td>
</tr>
<tr>
<td>$^{106}$Ru</td>
<td>368</td>
<td>78</td>
</tr>
<tr>
<td>$^{131}$I</td>
<td>8.04</td>
<td>4200</td>
</tr>
<tr>
<td>$^{136}$Cs</td>
<td>13.2</td>
<td>32</td>
</tr>
<tr>
<td>$^{137}$Cs</td>
<td>$1.10 \times 10^{4}$</td>
<td>5.9</td>
</tr>
<tr>
<td>$^{140}$Ba</td>
<td>12.8</td>
<td>4700</td>
</tr>
<tr>
<td>$^{141}$Ce</td>
<td>32.5</td>
<td>1600</td>
</tr>
<tr>
<td>$^{144}$Ce</td>
<td>284</td>
<td>190</td>
</tr>
</tbody>
</table>

*Taken from UNSCEAR [1982, p 228].
### TABLE 2

Unshielded groundshine doses accumulated after T day

<table>
<thead>
<tr>
<th>Nuclide</th>
<th>Dose conversion factor* $(\text{uSv/(KBq/m}^2))$</th>
<th>Dose per Mt (fission)$^\dagger$ $(\text{uSv/Mt})$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T=7</td>
<td>T=30</td>
</tr>
<tr>
<td>$^{95}$Zr</td>
<td>0.42</td>
<td>1.9</td>
</tr>
<tr>
<td>$^{103}$Ru</td>
<td>0.26</td>
<td>0.92</td>
</tr>
<tr>
<td>$^{131}$I</td>
<td>0.18</td>
<td>0.36</td>
</tr>
<tr>
<td>$^{137}$Cs</td>
<td>0.31</td>
<td>1.3</td>
</tr>
<tr>
<td>$^{140}$Ba</td>
<td>0.77</td>
<td>2.6</td>
</tr>
</tbody>
</table>

*Computed from dose-rate conversion factors of Kocher [1980], and includes effects of daughter products.

$^\dagger$Computed from dose conversion factors, the fission production rates of Table 1, the model result that a fraction $0.5 \times 10^{-19}$ of production is deposited on each m$^2$ of NZ after 80 hr airborne.

### TABLE 3

Doses to lung over 50 yrs via inhalation

<table>
<thead>
<tr>
<th>Nuclide</th>
<th>Dose conversion factor* $(\text{uSv/KBq inhaled})$</th>
<th>Activity inhaled$^\ddagger$ $(\text{KBq})$</th>
<th>Dose $(\text{uSv/Mt})$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$^{95}$Zr</td>
<td>35</td>
<td>0.23</td>
<td>7.8</td>
</tr>
<tr>
<td>$^{103}$Ru</td>
<td>15</td>
<td>3.8</td>
<td>5.2</td>
</tr>
<tr>
<td>$^{106}$Ru</td>
<td>1100</td>
<td>0.020</td>
<td>21</td>
</tr>
<tr>
<td>$^{140}$Ba</td>
<td>1.7</td>
<td>1.2</td>
<td>1.7</td>
</tr>
<tr>
<td>$^{141}$Ce</td>
<td>17</td>
<td>0.40</td>
<td>6.2</td>
</tr>
<tr>
<td>$^{144}$Ce</td>
<td>780</td>
<td>0.048</td>
<td>37</td>
</tr>
</tbody>
</table>

*From USNRC [1975, Table VI D-2].

$^\ddagger$Computed from the fission production rates of Table 1 and the model result that $2.5 \times 10^{-16}$ of production is inhaled per person after 80 hr airborne.
### TABLE 4

Concentration factors for contamination of milk

<table>
<thead>
<tr>
<th>Nuclide</th>
<th>CF, direct deposition*</th>
<th>CF, root uptake</th>
</tr>
</thead>
<tbody>
<tr>
<td>(^{89}\text{Sr})</td>
<td>0.28 Bq/(Bq/m(^2))</td>
<td>0.007 Bq/(Bq/m(^2))</td>
</tr>
<tr>
<td>(^{90}\text{Sr})</td>
<td>3.35 Bq/(Bq/m(^2))</td>
<td>1.4 Bq/(Bq/m(^2))</td>
</tr>
<tr>
<td>(^{131}\text{I})</td>
<td>0.69 Bq/(Bq/m(^2))</td>
<td>-</td>
</tr>
<tr>
<td>(^{136}\text{Cs})</td>
<td>1.4 Bq/(Bq/m(^2))</td>
<td>0.007 Bq/(Bq/m(^2))</td>
</tr>
<tr>
<td>(^{137}\text{Cs})</td>
<td>4.2 Bq/(Bq/m(^2))</td>
<td>2.1 Bq/(Bq/m(^2))</td>
</tr>
</tbody>
</table>

*Taken from USNRC [1975, Appendix VI E3].

### TABLE 5

Dose delivered by ingestion of milk

<table>
<thead>
<tr>
<th>Nuclide</th>
<th>Critical tissue</th>
<th>Ingested activity*</th>
<th>Dose conversion factor$^+$</th>
<th>Dose delivered</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(KBq/Mt)</td>
<td>((\mu\text{Sv}/\text{KBq ingested}))</td>
<td>((\mu\text{Sv/Mt}))</td>
</tr>
<tr>
<td>(^{89}\text{Sr})</td>
<td>bone</td>
<td>8.5</td>
<td>3.2</td>
<td>27</td>
</tr>
<tr>
<td>(^{90}\text{Sr})</td>
<td>bone</td>
<td>0.34</td>
<td>78</td>
<td>26</td>
</tr>
<tr>
<td>(^{131}\text{I})</td>
<td>thyroid</td>
<td>145</td>
<td>450</td>
<td>6.6x10(^4)</td>
</tr>
<tr>
<td>(^{136}\text{Cs})</td>
<td>whole body</td>
<td>2.3</td>
<td>2.4</td>
<td>5.5</td>
</tr>
<tr>
<td>(^{137}\text{Cs})</td>
<td>whole body</td>
<td>1.9</td>
<td>15</td>
<td>28</td>
</tr>
</tbody>
</table>

$^*$Computed from the concentration factors of Table 4, the fission production rates of Table 1, and the model result that 0.5x10\(^{-13}\) of production is deposited on each m\(^2\) of NZ after 80 hr airborne.

$^+$50-year doses taken from USNRC [1975, Table VI 8-4].
# TABLE 6

Somatic risks from radionuclides ingested via milk

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Risk factor*</th>
<th>Radiogenic cancers per Mt†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone (Sr)</td>
<td>0.5</td>
<td>0.09</td>
</tr>
<tr>
<td>Thyroid ($^{131}$I)</td>
<td>0.5</td>
<td>109</td>
</tr>
<tr>
<td>Whole body (Cs)</td>
<td>12.5</td>
<td>1.3</td>
</tr>
</tbody>
</table>

*Units: Fatal cancers per million persons exposed per mSv of tissue dose; Source: ICRP [1984, Appendix A].

†A population of 3.3 million is exposed to the doses of Table 5.