

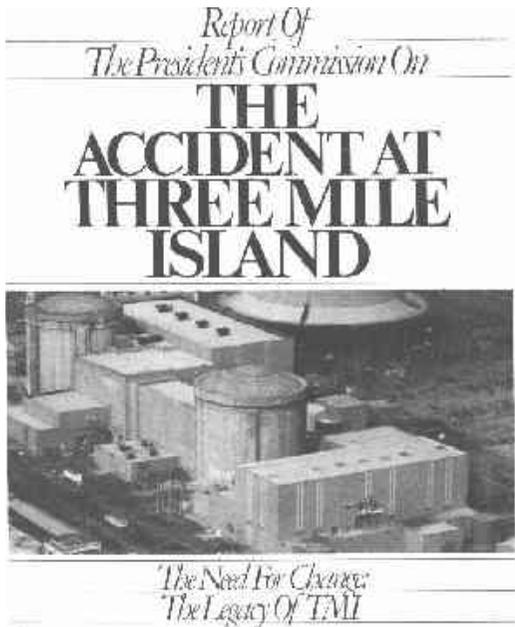
# The Fukushima accident: source term and consequences

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# Icons of the nuclear age



28 March 1976



26 April 1986



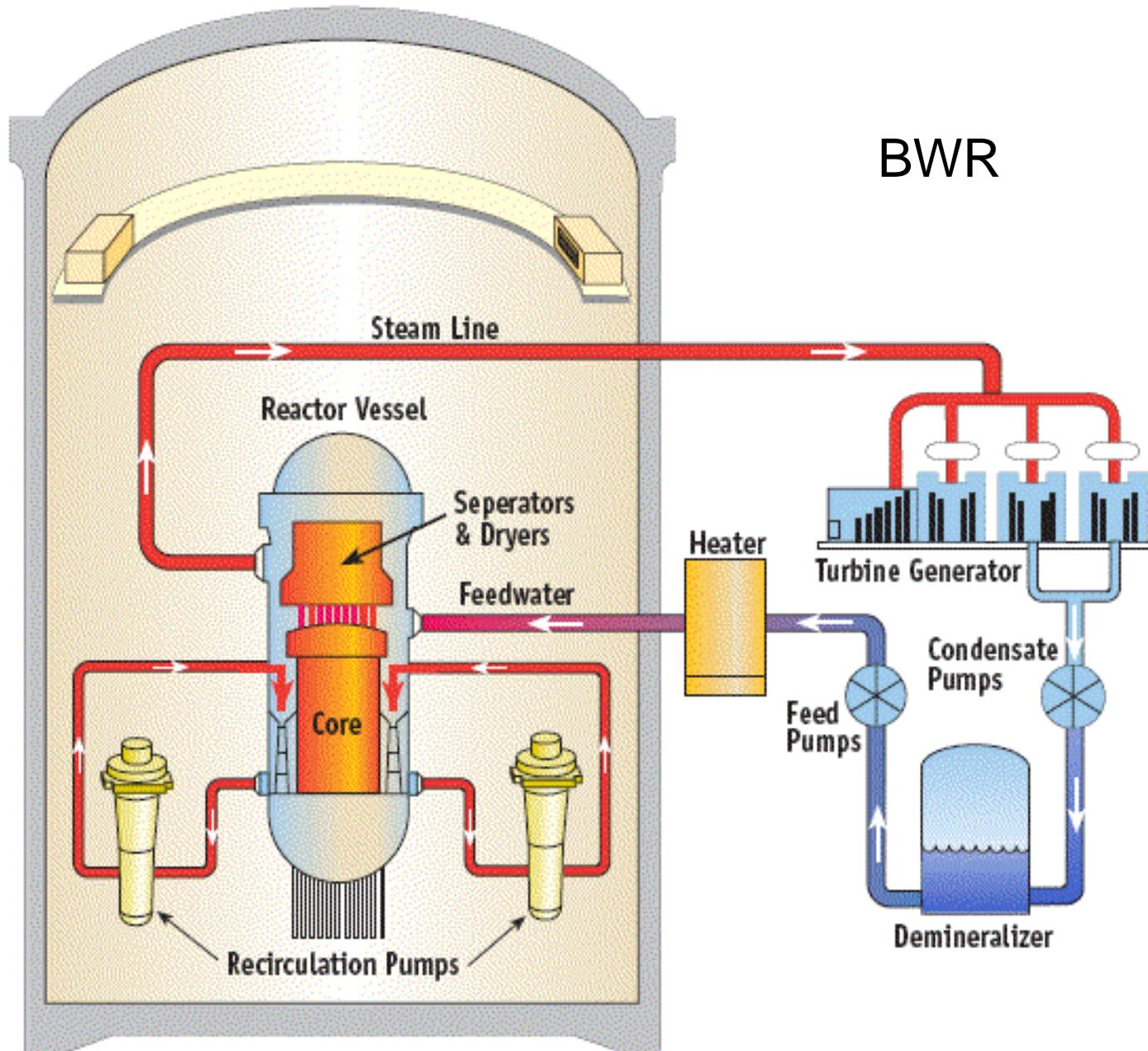
11 March 2011

	<b>TMI-Unit 2</b>	<b>Chernobyl-Unit 4</b>	<b>Fukushima Unit 1</b>	<b>Fukushima-Unit 2</b>	<b>Fukushima Unit 3</b>
<b>Type</b>	<b>PWR</b>	<b>RMBK</b>	<b>BWR</b>	<b>BWR</b>	<b>BWR</b>
<b>Power in GWe</b>	0.902	<b>3.2</b>	0.439	0.783	0.783
<b><sup>133</sup>Xe EBq</b>	(9 – 40) x10 <sup>-8</sup>	6.5	<b>16.7</b>		
<b><sup>137</sup>Cs EBq</b>	~0	<b>0.085</b>	<b>0.035</b>		
<b><sup>131</sup>I EBq</b>	5x10 <sup>-7</sup>	<b>1.76</b>	<b>0.01 - 0.7</b>		
<b>Ratio <sup>131</sup>I/<sup>137</sup>Cs</b>	na	15 - 33	0.29 - (6.2) <sup>[1]</sup> - 20		

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[1] Measured ratio at litate

# BWR



In addition in Unit 4 there was damage from the explosion at Unit 3 to the spent fuel container leading to a loss of coolant and probable melting and possible criticality and further release of nuclides.

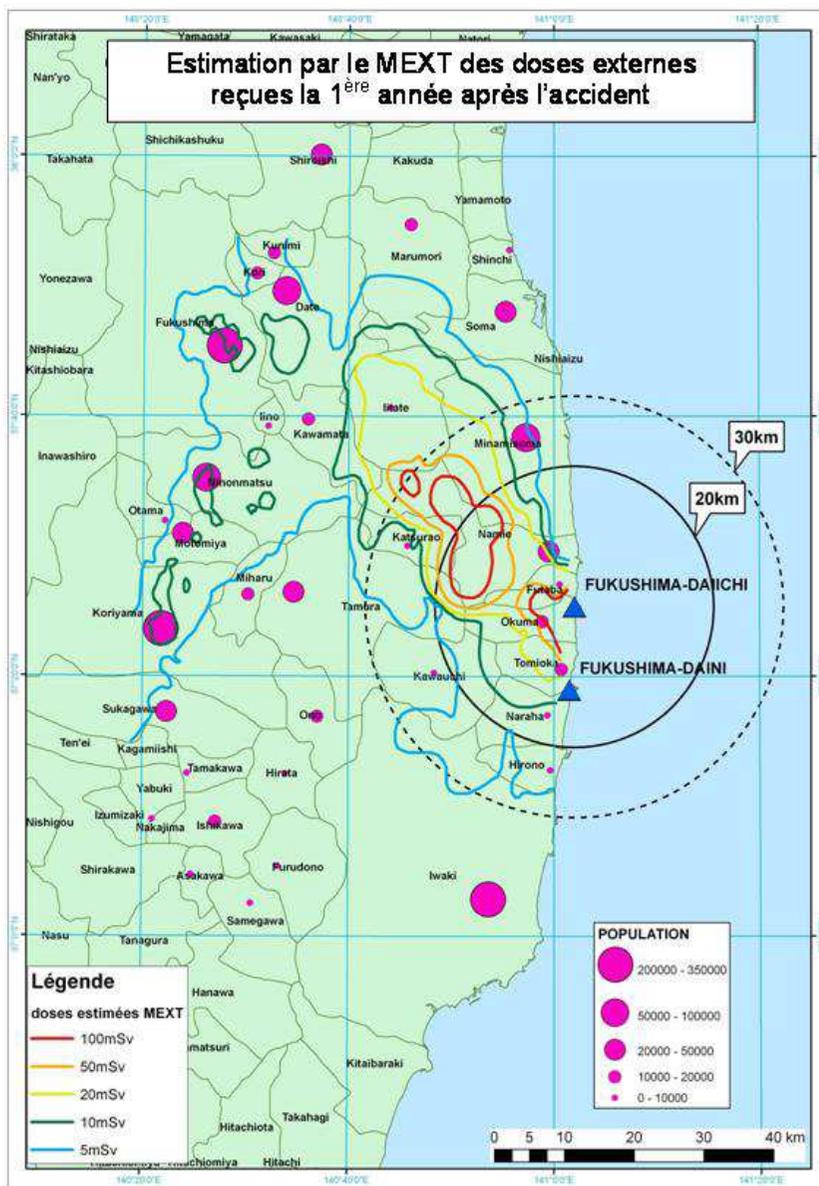
There are therefore several components to the source term that need to be considered. They include:

- Aerial release and deposition on land

- Releases directly to ocean but probable retention in coastal waters

- Radioactive debris (including fuel rods containing uranium and plutonium) dispersed by the explosions

- Neutrons (local problem early on)



Japanese estimates of external doses for the first year

**Within the red contour 100mSv**

**Within the orange contour 50mSv**

**Within the yellow contour 20mSv**

**Within the green contour 10mSv**

**Within the blue contour 5mSv**

Taken from IRSN Report DRPH/2011-10

## CAUTION:

There is a good deal of interpretation between these values and the actual measurements made in March/April by aerial survey.

The points to bear in mind here are:

These are estimates only of the external dose component and probably include generous allowance for shielding by buildings and time spent indoors

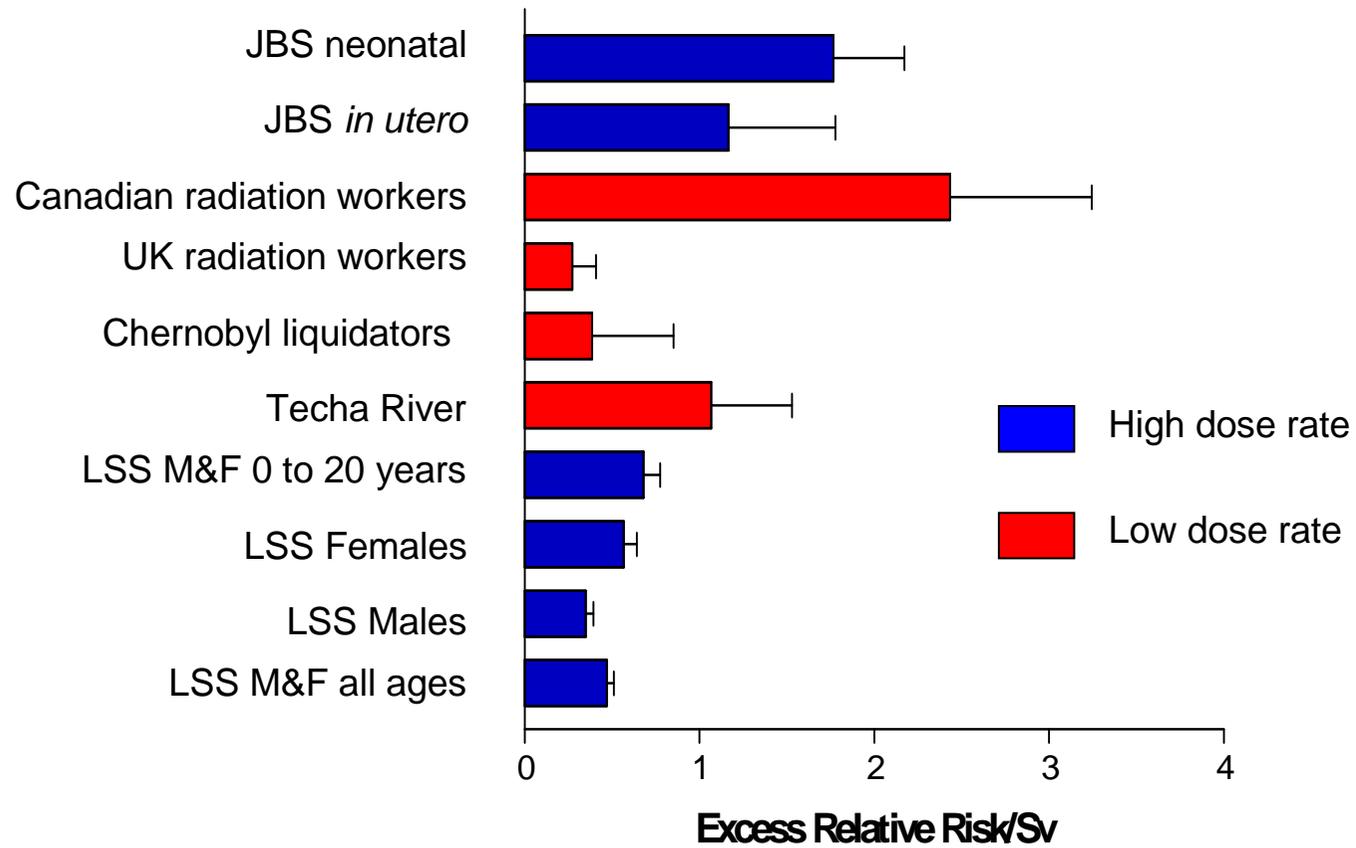
No estimate of the internal doses has been included and in areas where food is locally produced the total dose is typically double the external dose.

$^{137}\text{Cs}$  is the dominant nuclide and it has a 30 year half-life so doses are expected to remain high for decades to come.

To deal with this situation it has been proposed to raise the annual public dose limit to 20mSv/year, a 20 fold increase on the previous value. What does this mean in health terms?

Contrary to the position taken by the ICRP the risk of exposure to relatively low doses at low dose-rates is NOT lower and may well be higher, than that for the acute exposure to high doses experienced in Hiroshima and Nagasaki

### Selected ERRs/Sv for all cancer (incidence) by study



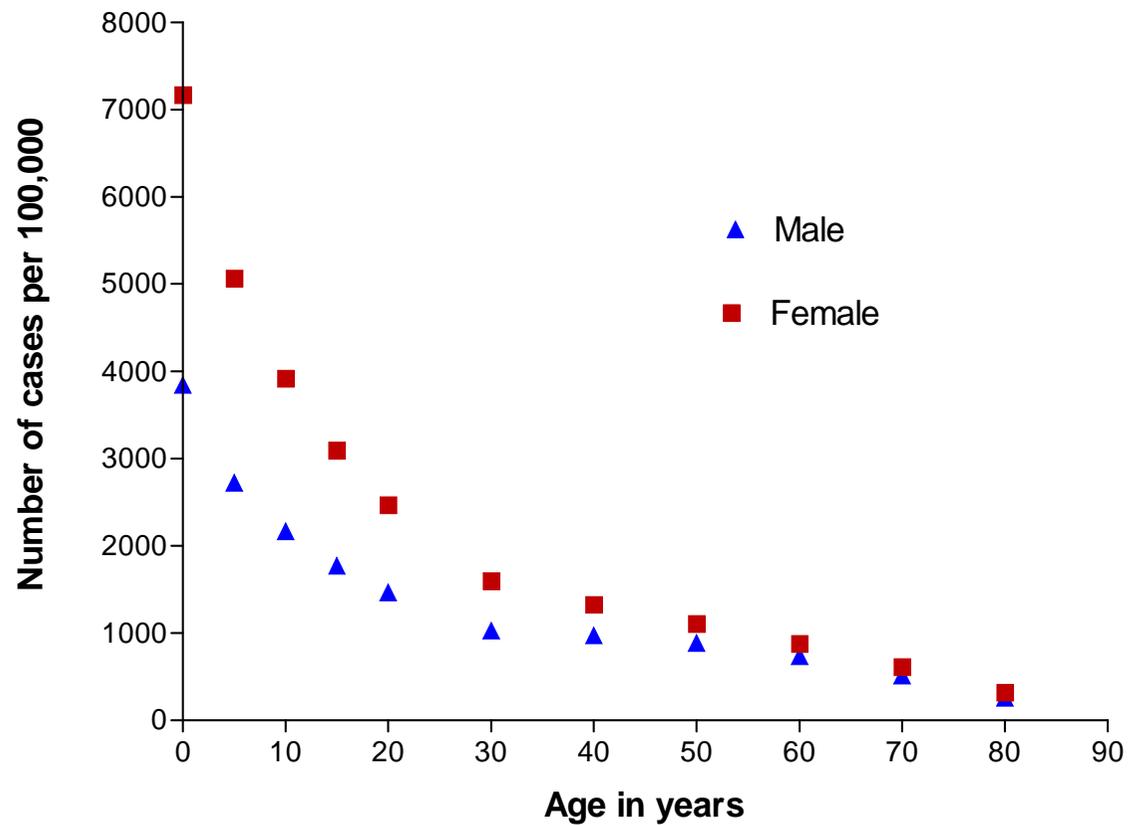
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However, those ERRs are for the average risk – do age and gender make a difference?

The answer is YES even if only for the simple reason that exposure in childhood, compared to adulthood, allows much greater time for the risk to expressed. But over and above that children and especially girls are significantly more sensitive than the average.

### Lifetime incidence of all radiation induced cancer by age at exposure to 0.1Gy (100mSv)



Figures taken from BEIR 2007 with DDREF = 1

**Table 3** Risk factor-adjusted incidence and relative hazards of haemorrhagic stroke by radiation exposure

<b>Radiation exposure (Gy)</b>	<b>Incidence</b>	<b>Relative hazard (95% CI)</b>
<b>Men</b>		
<0.05	11.6	Reference
0.05 to <1	17.7	1.5 (0.8 to 2.7)
1 to <2	20.2	1.7 (0.7 to 4.1)
≥2	29.1	2.5 (0.8 to 7.3)
<b>p Value*</b>	0.009†	
<b>Women</b>		
<0.05	14.2	Reference
0.05 to <1.3	13.0	0.9 (0.6 to 1.4)
1.3 to <2.2	20.3	1.4 (0.6 to 3.7)
≥2.2	48.6	3.5 (1.4 to 9.0)
<b>p Value</b>	0.002	

Incidence rate per 10 000 person-years and relative hazards are adjusted for age, systolic blood pressure, body mass index, diabetes, total cholesterol, cigarette smoking, alcohol drinking and city.

\*The p value is a test for trend with dose modelled as a continuous variable. For women, a dose threshold model is used with a dose threshold at 1.3 Gy (95% CI 0.5 to 2.3 Gy).

†For men, risk of haemorrhagic stroke continued to rise with increasing doses <1 Gy (p=0.004).

# Summary

Full knowledge of the source term may never be acquired.

Considerable sparing of land deposition due to (mostly) favourable wind direction, nevertheless not inconsiderable long term contamination of substantial land areas.

Shortage of habitable and agriculturally productive land has forced an increase in the public dose limit (by a factor 20) and will increase life time risk of cancer and probably other diseases, especially for infants and children.

Possible trans-generational effects from inherited genomic instability.

Thyroid cancer likely to be less of a problem than after Chernobyl due to absence of iodine deficiency and a less dependence on milk in the diet.

Unknown quantities of radioactivity discharged to the sea may adversely affect coastal waters and lead to significant contamination of fish and sea food.

Pronounced psychosocial effect due to, among other things, unjustified assurances that there was no problem in the early days of the accident.

# Conclusions

Many of the lessons learned from the Chernobyl accident were either forgotten or ignored. As one consequence there is likely to be a largely avoidable increase in psychosocial illness.

All the evidence we have points to there being a significant impact on the health. Further follow-up of the Chernobyl populations as recommended by ARCH would provide valuable guidance for Fukushima.

The stricken reactors posed a lethal threat to workers trying to regain control: a private company cannot demand that employees lay down their lives in the greater societal interest; a National Government can and did.

Had this accident occurred in Europe the environmental and therefore health, consequences are likely to have been considerably more severe than in Japan where the Pacific Ocean absorbed much of the fallout.

The setting up of epidemiological studies should be a priority.

THANK YOU FOR YOUR  
ATTENTION