SOME PERSPECTIVES ON ACTION LEVELS IN AREAS CONTAMINATED WITH RADIOACTIVE FALLOUT

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Maximum permissible radiation levels for planned activities must be distinguished from action levels as applied to protect people and animals after nuclear accidents. The essential thing with action levels is the optimal balance between the danger of the radiation and the harm involved in the counter measures themselves. Among other things these counter measures are dependent on the magnitude of the fallout, i.e. on the apprehended doses to man and animals.

Massive fallout covering large areas

Extensive fallout that covers large areas constitutes a danger of more or less acute effects on man and animals, effects which we somewhat improperly call non-stochastic. Let me start with a discussion of the situation for domestic animals. The "critical" animals in Sweden are reindeers and cows, simply because they are grazing very large areas every day. We can do very little to protect reindeers but cattle can be put into stables and/or be fed with non-radioactive fodder at least for short periods of time. But sooner or later, they must be pastured on contaminated ground. I shall try to say something about when and for how long times this can be done.

Firstly, we have to take the doserate dependence into consideration. A certain radiation dose is more biologically effective when delivered during a short period of time than in cases where the same dose is accumulated during longer periods. The radiation from fission products, deposited on the ground or incorporated into the animals does always lead to a prolonged, irradiation with decreasing doses. It is thus necessary to take the doserate dependence into consideration when evaluating the risk for the cattle. I have based my calculations of the doserate dependence on clinical experiences and found a surprisingly good agreement between these semi-theoretical calculations and the observed doserate dependence as reported in the literature for several types of disparate damage in different animals, including the cow. By applying my calculated

figures to the grazing cow, it has been possible to obtain a scheme for acceptable grazing periods in areas that have been contaminated by fission products as released from a nuclear power plant (Tab. 1). This scheme is based on measurements with an instrument that is well accessible all over Sweden from military and civil defence units. The instrument is called Intensimeter and gives the radiation intensity in mR/hr.

The left column of Tab. 1 gives five different figures of the time that has elapsed since the accident. Let me give two examples how to use the scheme. If the radiation intensity, as measured one meter above the ground is less than 68 mR/hr, 2.4 hours after the accident, the cows may graze for one day. If the intensity is less than 11 mR/hr, the cows can be released without restrictions. If the cows are fed with non-contaminated fodder for two weeks after the accident, they can be released for one day if the intensity is less than 110 mR/hr, and without restrictions if it is less than 19 mR/hr. It must, however, be pointed out that these estimations are very rough and the validity of the various parameters and assumptions underlying the calculations must be controlled by a competent radiophysicist, i.e. the scheme can only be used as a first approximation.

Analyses show, that if the animals can be pastured on contaminated areas without any severe detrimental effects, man can live in the areas without any risks of such effects and he can also drink the milk from the cows as well as water from open wells.

All this estimations have ignored the risk of cancer and genetic damage to the animals as well as to the milk concumer. After high radiation doses such risks are fairly low as compared to those of more acute kinds. After massive contaminations of vast areas, problems may arise with regard to the general supply of provisions. In particular, we should bear in mind that milk is an essential calcium source for children. This problem is less after low amounts of fallout, particularly if the contamination is local, and the interest is shifted from the

animals to the consumer of animal products. Restrictions as to consumtion become less severe for the general health and contaminated food may be replaced by food from less contaminated areas. Simultaneously the basic risk is shifted from non-stochastic to stochastic effects, particularly to cancer.

Problems in assessments of low-dose risks

Estimations of cancer risks involve many problems, particularly in the low-dose region. Does it, for instance, exist a threshold for radigenic cancer and if not, is it possible to determine the risk at very low doses? Let me once say, that many epidemiological data do not demonstrate a clear-cut threshold dose, i.e. a dose under which we for certainty can say that the risk of cancer is zero. In my opinion, this implies that we are not entitled to state that the risk is zero even at extremely low doses. But how should we quantify the low-dose risk? For planned activities, the usual method is to assume a direct proportionality between dose and expected cancerfrequency. But for non-planned activities, the action levels must be compared with the actual harm that might be involved in the counter measures themsleves. How should this be made if the radiation effects can not be quantified?

The plain answer is that this is impossible on grounds of principle. Let me develop this conclusion a little. Cancer is an extremely complicated process which takes place in many steps, some of which involving stochastic changes in the genoms. The vulnerability to a carcinogenic agent varies from one individual to another due to differences in heredity, living habits, exposures to other carcinogens etc. The assessments of such risks are essentially an epistemological problem. Let me illustrate this statement with an example from the physics. In mechanic and thermodynamic systems, knowledge of positions and speeds of the single molecules does not tell us anything about the system in its entirety. But we can achieve such a knowledge by statistical methods, provided that the system is closed, i.e. that the entropy of the system is constant. This is not the case with a human being or a human

population. Such systems are necessarily open and subjected to an unbroken increase in entropy, i.e. an increase in "informative disorder". Because of this difficulty I prefer to talk about dominant and non-dominant doses or concentrations of biologically active substances instead of high and low radiation doses or concentrations. A non-dominant dose is defined as a dose that is too low to give rise to more extensive effects than those that may be caused by all other agents or factors that we are meeting with in our daily lives. My point is, that we of practical reasons as well as on grounds of principle, can have no knowledge whatsoever about the possible effects of a non-dominant radiation dose or concentration of a substance. From a practical point of view, this is impossible since such a knowledge would demand a complete insight into the future of the exposed individual or population. Furthermore, such a knowledge is impossible on grounds of principle since the genetic changes involved in the tumorigenic development are stochastic by nature.

The action of an agent on a mammal is thus dualistic in the sense, that the agent firstly exerts a primary effect on the exposed tissue and secondly that this primary effect is followed by reactions of the exposed mammal to that challenge. The variability of this second branch of the dualism among different individuals implies that we can not speak about <u>the</u> dose-effect relationship as an independent concept, valid for all individuals and populations. This is not only true for low doses but the effect of variability in sensitivity can sometimes be seen after high doses too. One such an example is shown in Fig. 1. The curves in this figure show all mortal malignancies as observed in Hiroshima and Nagasaki after 1945. The curves are significantly different and we can today say nothing about why they are different. In fact, the data from Nagasaki do not demonstrate any increase at all for doses below about 250 rad kerma.

I shall not bother you with further examples. Let me only

point out that the assumption of a direct proportionality between radiation dose and cancerfrequency is - to say the least - a very uncertain assumption. The linear model may be used in establishments of maximum permissible dose levels but if so, we must not forget that the straight line does not represent any biological reality. Its use may be warrented in planned activities because it probably implies an over-safety and because we have no better alternatives, but it is for example, not acceptable for prognoses as to how many cases of cancer we may expect after exposures to non-dominant doses. And it can not be used in comparisons with other risks. From an epistemological point of view, the analogy between a complex biological process like cancer induction and a mathematical polynomial of the first degree is nothing but a scientific collapse.

Could we, in determination of action levels, possibly accept radiation risks that is not more extensive than all other risks which are accepted today without hesitation? This means an acceptance of non-dominant doses. This would certainly imply something of a paradigm-shift in radiation protection and such a recommendation can probably not be given by WAVPH alone, at least not at this conference. But the problem is essential and we should bear it in mind which, among other things, is necessary in order to avoid counter measures that might be more dangerous than the non-dominant radiation dose.

Let me give one example to illustrate the problem. Table 2 shows what would happen with regard to cancer risks if we permanently move from the counties around the three biggest cities in Sweden, Stockholm, Gothenburg and Malmö into the respective cities. The table has been obtained from the Swedish Cancer Registry for cancer incidencies diagnosed during 1982 per 100,000 persons after corrections for differences in age. The increases in cancer frequencies by moving into the cities are much higher than those to be expexted on basis of a linear dose-effect relationship in the most contaminated areas of Sweden as caused by the Chernobyl accident.

We have learned from USSR that people living around the Chernobyl area have been permanently evacuated to Kiev. Does this measure really reduce the risk of cancer for all evacuee?

The fact that the effect of an agent on a mammal is dualistic, i.e. also dependent on the variability in sensitivity to the agent, does imply that non-radiological risks too, may lack thresholds. This is, for example, the case with excess mortalities as functions of the sulfurdioxide in the air, as observed in Oslo, London and New York. But the maximum permissible concentration of sulfurdioxide is not based on the epidemiological findings we have from the three cities but on data from animal experiments and from healthy volunteers. The MPC for SO_2 in Sweden at long-term exposures (0.05 ppm) would imply an excess mortality of 5 - 9 % if the data from Oslo would have been applied, whereas the maximum permissible collective dose from nuclear power often is expressed as one man-rem per megawatt-year, which in Sweden would imply a cancerrisk of about 10^{-3}% (Fig. 2).

Let me summarize. We can, of grounds of principle, not have any knowledge about the biological effects of a non-dominant radiation dose or of a non-dominant concentration of a biologically active substance. This makes determinations of action levels very difficult after exposures to low radiation doses. But as scientists we have to admit this fundamental limitation of our knowledge and we must be very careful in choosing counter measures in cases of low levels of radioactive contamination. A fundamental demand on such comparisons of risks is that the criteria of judgement must be identical.

TABLE 2. CANCERRISKS AT MOVINGS TO SWEDEN'S THREE BIGGEST CITIES FROM THE SURROUNDING COUNTIES
The numbers within parentheses denote cancer frequencies excluding lungcancer

Place	Sex	Incidence $x10^{-5}$	Difference $x10^{-5}$	Increase %	Dose corresponding to the increase, mSv. *)
Stockholm county	M	449.7 (390.1)			
	F	349.2 (330.3)			
Stockholm city	M	499.7 (427.7)	50.0 (37.6)	11.1 (9.6)	25
	F	381.0 (363.3)	31.8 (33.0)	9.1 (10)	16
Gothenburg county	M	413.5 (371.9)			
	F	334.0 (315.9)			
Gothenburg city	M	476.0 (409.3)	62.5 (37.4)	15.1 (10.1)	31
	F	368.4 (352.6)	34.4 (36.7)	10.3 (11.6)	17
Malmö county	M	455.7 (399.9)			
	F	335.2 (324.2)			
Malmö city	M	684.0 (584.0)	228.3 (184.0)	50.1 (46.0)	114
	F	407.4 (379.8)	72.2 (55.6)	21.5 (17.1)	36

*) The dose is calculated under the assumption of a linear dose-effect relationship, i.e. that the incidence at long-term exposures is 2 % per Sv (UNSCEAR, 1977).

The figures in the table have been obtained from the Swedish Cancer Registry (National Board *of Health and Welfare. The Cancer Registry: Cancer Incidence in Sweden*, 1982.)

TABLE 1. Length of grazing periods as functions of measured intensities, 1 m above the ground at different times after a reactor accident.

Time between accident and start of grazing ↓	Grazing period → 1 day	7 days	14 days	More than 23 days
	mR/hr			
2.4 hrs	68	15	12	11
1 day	46	10	8	8
6 days	44	10	8	8
13 days	110	25	20	19
30 days	850	56	36	25

See text for description of the use of the table.

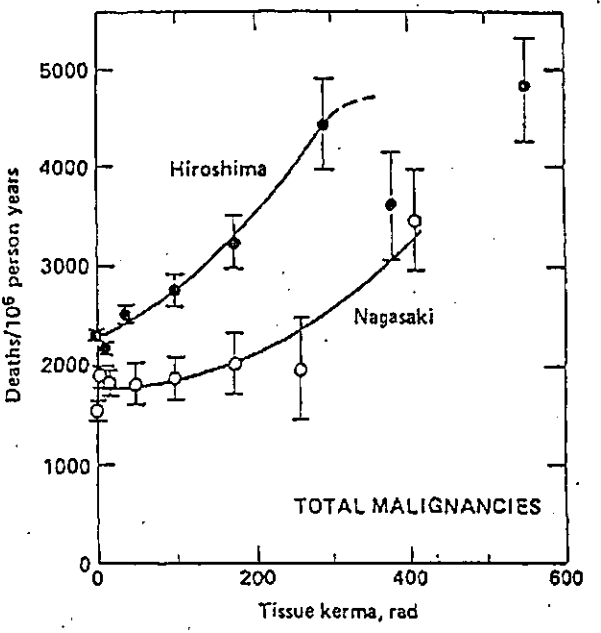

Figure 1. Mortality from total malignancies shown for Hiroshima and Nagasaki separately as functions of tissue kerma as estimated at Lawrence Livermore National Laboratory. The figure illustrates the fact that two populations may be significantly different in vulnerability to radiogenic carcinoma. (By courtesy of William E. Loewe).

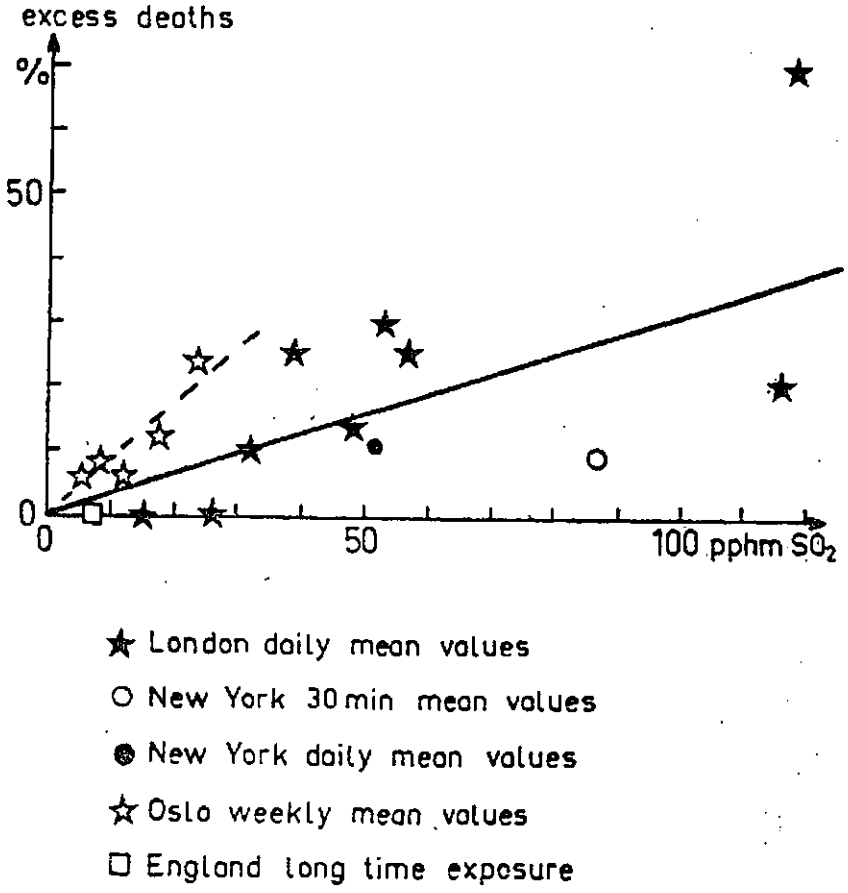

Figure 2. Excess mortality in Oslo, London and New York as functions of the concentrations of SO_2 in the air (expressed as pphm = parts per hundreth million). The curves from New York and London are based on daily means and are thus substantially dependent on "what happened the day before". This source of error is avoided by the weekly means from Oslo. The latter curve would imply an excess mortality of about 9% for the maximum permissible SO_2-concentration in Sweden (5 pphm), i.e. a risk that is about three orders of magnitude higher than the cancer-risk at the maximum permissible radiation dose from the nuclear plants in Sweden

The figure is obtained from: Air pollution across national boundaries. The impact on the environment of sulfur in air and precipitation. Sweden's case study for the United Nations Conference on the Human Environment. Royal Ministry for Foreign Affairs and the Royal Ministry of Agriculture. Stockholm 1971.