Commentary: Low dose-rate exposures to ionizing radiation

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Current radiation protection recommendations\(^1\) are mainly based on risk estimates from studies of populations with relatively high doses received at high dose rates, notably the survivors of the atomic bombing in Hiroshima and Nagasaki;\(^2\) together with extrapolation models to predict risks associated with lower dose protracted or fractionated exposures. The health effects of low doses of radiation, and particularly the possible existence of a threshold below which there may be no effect, have, however, been the subjects of controversy for decades.\(^3\),\(^4\)

Direct information on the effects of low dose-rate and protracted exposures to ionizing radiation can in principle be derived from epidemiological studies of populations with such exposures. For studies to be informative, however, they must fulfill a number of important criteria. They should include observation of large populations followed up over many years. The follow-up must be non-differential (i.e. not only restricted to persons who are ill or who have received high doses), sufficiently complete and the information on outcome (diagnosis) should be accurate. Precise and accurate individual dose/exposure level estimates must also be available for all persons in the study.\(^5\)

A population of particular interest to estimate directly the effects of low-dose protracted exposures is that of the persons living along the Techa River in the Southern Urals. In 1949, the Mayak Production Association started operations, producing and separating plutonium for the USSR nuclear weapons programme. Waste from the plant was released into the river and the population was also exposed to accidental and gaseous releases in the period 1949–56. The population residing in the villages along the river received protracted exposures, both external and internal due to incorporation of radionuclides (\(^{89}\)Sr & \(^{90}\)Sr, \(^{137}\)Cs, \(^{103}\)Ru & \(^{106}\)Ru, \(^{92}\)Zr & \(^{95}\)Nb), at low dose rates. The follow up of this population, which includes subjects with different ethnic backgrounds (Slavs and Tatar/Bashkir), is thus important for improving our understanding of radiation effects.

In this issue, Krestinina and collaborators\(^6\) report on cancer incidence among the 17 433 members of the expanded Techa River Cohort (a cohort of approximately 30 000 individuals, born before 1950 who resided in any of 41 exposed...
villages along the Techa River during the period of releases7) who reside in the catchment area of the Chelyabinsk regional oncology centre. The analyses are based on over 47 years of follow-up and over 1800 cases of cancer. They report a significant radiation-related increase in solid cancer incidence in this population (with about 3% of the cancer cases attributable to radiation). The estimated excess relative risk (ERR) of solid cancer is 1.0/Gy of radiation dose [95% confidence interval (CI) 0.3, 1.9], very similar to the estimate these authors obtained based on cancer mortality in the entire expanded Techa River Cohort8 (Table 1). These estimates are higher than, though statistically compatible with, comparable estimates derived by extrapolation from the atomic bomb survivors study (gender-averaged ERR estimate at age 65 for a person exposed at age 25—the approximate mean ages at exposure and diagnosis in the current study—0.6/Gy).

There are a number of concerns about the Techa River dosimetry, however,9 and work is underway to improve the dosimetry system. Current consensus is that dose estimates for 90Sr and 137Cs are reasonable. The role of short-lived isotopes in internal and external doses is under review: as there was little systematic monitoring before July 1951, there may be errors in the estimated amount of activity released and in the radionuclide composition, and this could affect the magnitude of the risk estimates.

Direct information about the effects of low-dose protracted exposures also comes from a recent 15-country collaborative study of nuclear industry workers.10 The solid cancer risk estimate from this study is similar to that seen in the Techa River Cohort study (Table 1). Analyses of smoking and non-smoking-related causes of death indicate however that smoking may play a role in the increased risk of all cancers excluding leukaemia, although it is unlikely to explain all of the increased risk observed in the 15-country study.10

Table 1  ERR/Gy and 95% CI for solid cancers—comparison of risk estimates

<table>
<thead>
<tr>
<th></th>
<th>Size of cohort</th>
<th>Average dose (mGy)*</th>
<th>Average number of years of follow-up</th>
<th>Solid cancers</th>
<th>ERR/Gy (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Techa River cohort</td>
<td></td>
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<td></td>
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<tr>
<td>Mortality follow</td>
<td>29 873</td>
<td>30</td>
<td>29.0</td>
<td>1842</td>
<td>0.92 (0.2, 1.7)</td>
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<tr>
<td>Incidence follow</td>
<td>17 433</td>
<td>40</td>
<td>25.6</td>
<td>1836</td>
<td>1.0 (0.3, 1.9)</td>
</tr>
<tr>
<td>15-Country Nuclear workers study</td>
<td>407 391</td>
<td>19.4</td>
<td>12.7</td>
<td>4770</td>
<td>0.87 (0.03, 1.9)</td>
</tr>
</tbody>
</table>

*Doses in the Techa River analyses are the sum of estimated internal and external doses to the stomach lagged by 5 years; estimated colon dose lagged by 10 years was used in the nuclear workers study.

References


