Future of Radiation Protection

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Dose Response of Health Effects

Deterministic effects

Stochastic effects

Incidence vs. Dose
Deterministic Effects
(Tissue Reaction)
“Although uncertainty remains, medical practitioners should be made aware that the absorbed dose threshold for circulatory disease may be as low as 0.5 Gy to the heart or brain.”
Epidemiological Findings

• Excess risk observed in A-bomb survivors and some populations with occupational exposures

• Becomes apparent 10-20 years after exposure

• Heterogeneity among studies

• Considerable uncertainty about the shape of dose-response curve at low doses
Mechanistic Considerations

- Very late tissue reaction
- Include multiple classes of disease
- Involves different tissues within the organ
- Influenced also by changes in other organs
- Not organ-, but endpoint-specific mechanisms
- Radiation-related rather than radiation-induced
Practical Questions

- Is there a risk increase at low doses?
- Which classes of disease are associated with radiation?
- What are the target organs/tissues?
- What about relative biological effectiveness (RBE)?
- What about latency?
- Is the risk reduced in fractionated/protracted exposure?
- How does radiation interact with other risk factors?
Stochastic Effects
Definition of LNT Model

• Linear-non-threshold (LNT) model

  A dose-response model which is based on the assumption that, in the low dose range, radiation dose greater than zero will increase the risk of excess cancer and/or heritable disease in a simple proportionate manner.

• Linear dose response

  A statistical model that expresses the risk of an effect (e.g., disease or abnormality) as being proportional to dose.
Performance of LNT Model

• A simplest, robust statistical model in predicting the risks at low doses

• Describes representative epidemiological data fairly well

• Achieves additivity of dose with no reference to previous history of exposure

Properly validated for low dose-rate exposure?
Difference over DDREF

- Difference between cellular, animal and epidemiological data
- Difference between endpoints
- Difference between epidemiological studies
- Difference between tissues/organs
- Difference between low dose extrapolation factor (LDEF) and dose rate effectiveness factor (DREF)
Kinetics of Stem Cells

Stem cell competition for residence in the niche

Possible mechanism of risk modification

(ICRP Publication 131)
What To Recognize

• Tumorigenic process could differ between classes of cancer

• Analyses/experiments on the basis of cancer site/type emphasized

• More considerations on the mechanisms desirable

• Radiation risk influenced by many other factors
Evolution of RP System
Integration of New Findings

Accumulate data

Have different perspective

Revisit theoretical framework

Integrate into RP system

Develop meticulous, biologically-precise system
“Simplicity is not the absence of clutter, that's a consequence of simplicity. Simplicity is somehow essentially describing the purpose and place of an object and product. The absence of clutter is just a clutter-free product. That's not simple.”

— Jonathan Ive
Round Table Discussion

Futures of Health Risk Research and the System of Radiological Protection
Open Questions

• What is the future development of A-bomb studies?

• Is it possible that risk estimate for RP relies more on low-dose-rate epidemiological studies in the future?

• What kind of study is necessary or desirable regarding circulatory disease?

• Is the classification of health effects, deterministic and stochastic, still valid?

• How much do we know about risk modifiers, and how to reflect that knowledge in radiological protection?