

# Factors Governing the Individual Response of Humans to Ionising Radiation

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The draft report of ICRP TG111

## Human Studies on Cancer

9 April 2026

**ICRP**

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# Background

**Radiation-related cancer risk is a central issue in radiological protection.**

- **Current understanding largely derived from epidemiological studies:**
  - Atomic bomb survivors (Life Span Study ~ 100,000)
  - Occupational exposure (e.g., nuclear workers)
  - Medical exposure (radiotherapy, CT)
  - Environmental exposure
- **Consistent dose–response relationship → supports a likely causal link.**
- **Substantial risk variation between individuals**
  - Critical for radiological protection & decision-making (e.g. medical).

# Approach

- **Potential effect modifiers**

- Age
- Sex
- Smoking
- Other lifestyle factors (alcohol, reproductive history/hormonal factors, ...)
- Underlying conditions (diabetes, chronic inflammation, ...)
- Environmental factors (UV/sunlight exposure, ...)
- Genetic and epigenetic factors

- **Risk quantified by Excess Relative Risk & Excess Absolute Risk**  
(ERR) (EAR)

- **Significant evidence is largely from Life Span Study (LSS).**

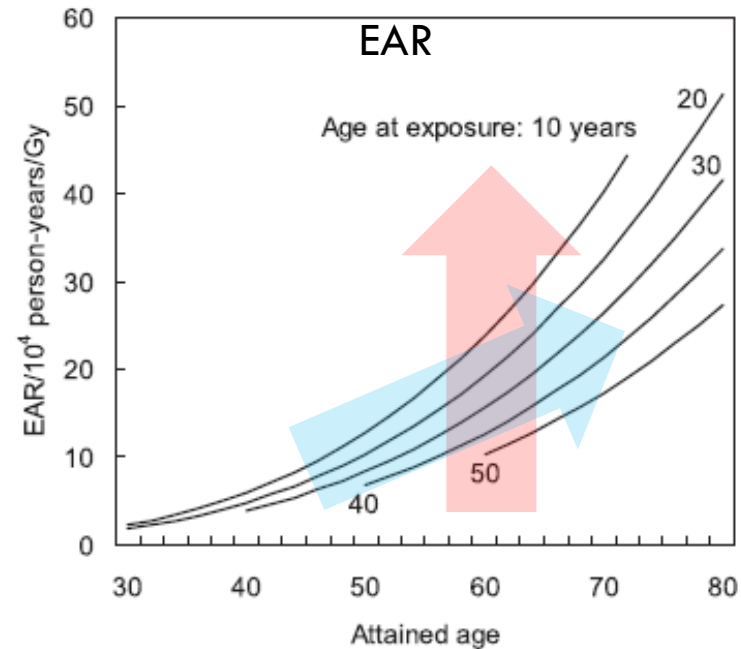
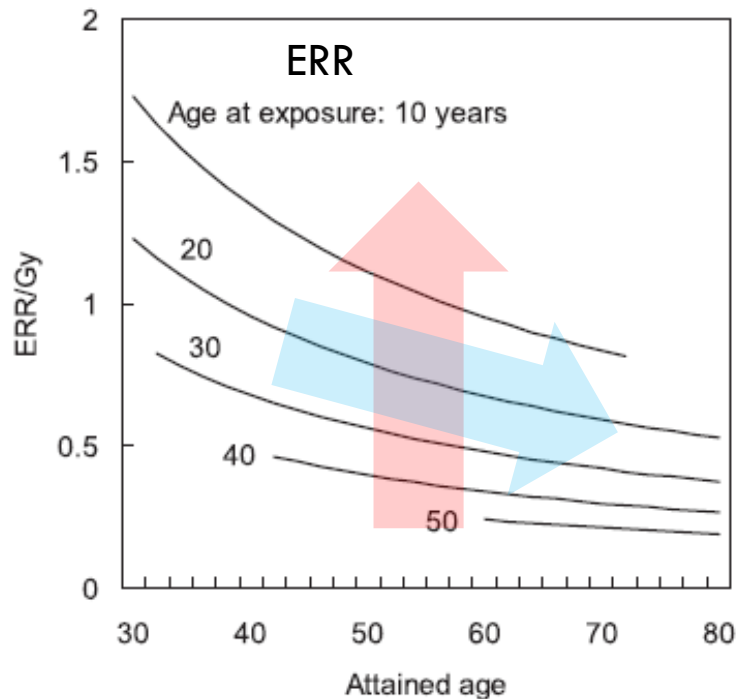
- A large, well-characterized cohort with accurate dosimetry and long-term follow-up.

# Age

- **Key Evidence**

- **Age at exposure:** Younger age → higher cancer risk
- **Attained age:** ERR decreases with age while EAR increases with age

All solid cancer mortality data in LSS (1950-2003):



Ozasa, K., et al., Radiat Res, 2012.

# Age

- **Key Evidence**

- **Age at exposure:** Younger age → higher cancer risk
- **Attained age:** ERR decreases with age while EAR increases with age
- Magnitude of the age effects varies across cancer sites.
  - Stronger dependence for e.g., leukaemia, thyroid and breast cancers.

- **Key Message**

- ✓ Age is one of the strongest and most consistent modifiers.
- ✓ Risk depends on both age at exposure and attained age.

# Biological Sex

- **Key Evidence**

- In the Life Span Study
  - Differences clearer in ERR (female > male), but less consistent in EAR
  - Varies by cancer site
    - female > male (e.g., stomach, lung, thyroid)
    - male > female (e.g., leukaemia EAR)
- Limited or inconsistent evidence from environmental/occupational cohorts.

- **Key Messages**

- ✓ Sex is a robust modifier but the underlying mechanisms remain unclear.
- ✓ Plausible contributors: sex variations in baseline rates, age, cancer types, hormonal and biological factors.

# Smoking

- **Key Evidence**

- Smoking & radiation both independently increase cancer risk.
- Strongest impact observed for lung cancer.
- Interaction between smoking and radiation for lung.
  - Rather complicated pattern observed in the LSS.
  - In other studies, observations are inconsistent or limited for interaction.
- For most other cancer sites, smoking has little impact as a risk modifier.

- **Key Message**

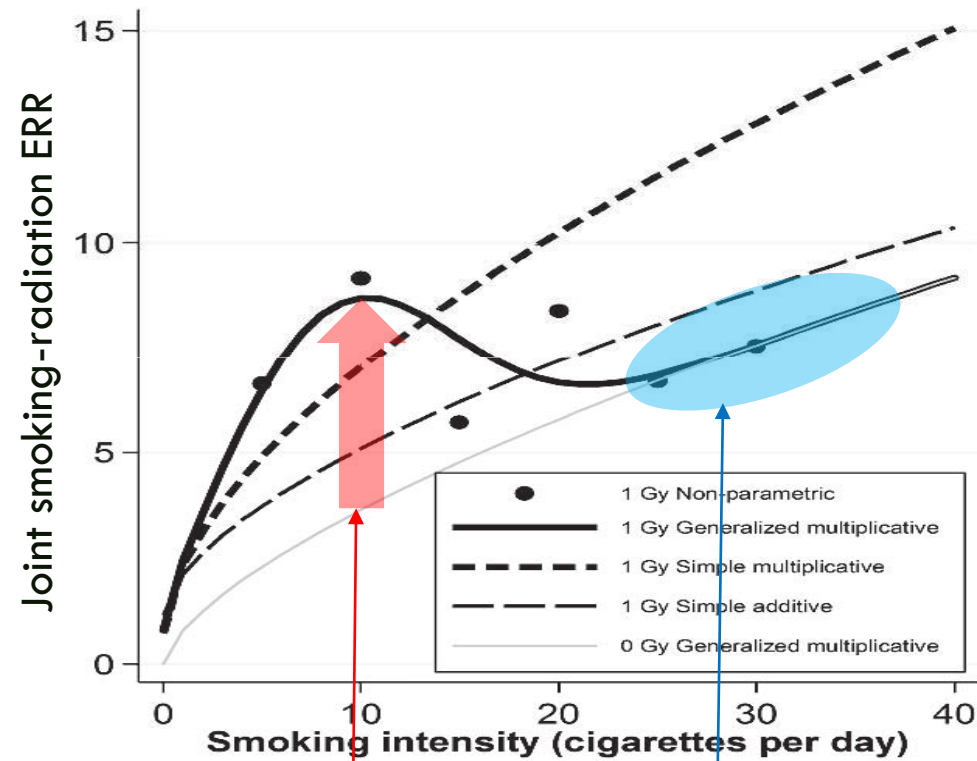
- Smoking is a major, well-established modifier.
- But interaction with radiation is complex and not fully understood.

# Smoking

- Key Evidence

- Smoking
- Strong
- Interaction
  - Rather

LSS Lung cancer incidence 1958-2009 (Cahoon et al., 2017)



Interaction

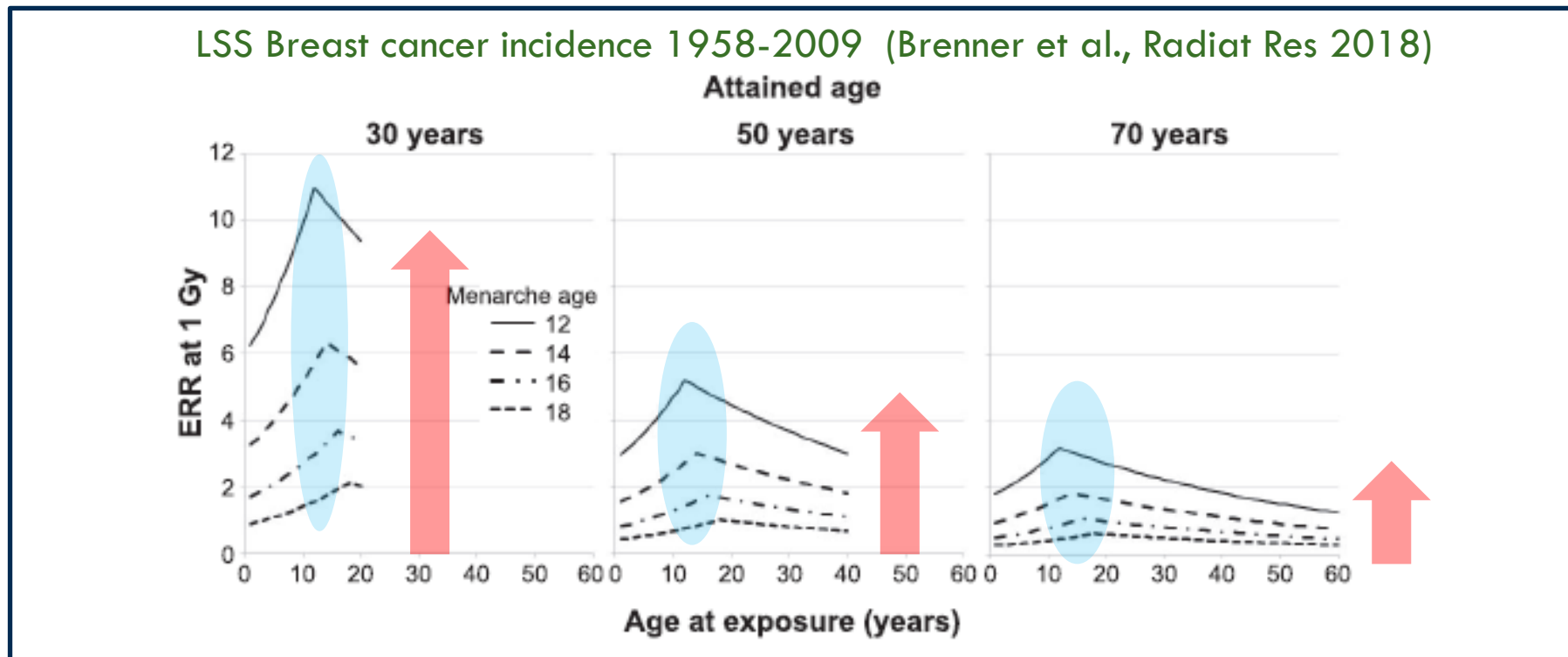
More than multiplicative  
for light-moderate smokers:

Little radiation-associated excess  
for heavier smokers:

# Reproductive Factors

- **Key Evidence**

- Reproductive history (e.g., age at menarche) modifies breast cancer risk
  - ERR and EAR  $\uparrow$  with age at menarche  $\downarrow$
  - Age at exposure effects are highest around menarche



# Reproductive Factors

- **Key Evidence**

- Reproductive history (e.g., age at menarche) modifies breast cancer risk
  - ERR and EAR↑ with age at menarche ↓
  - Age at exposure effects are highest around menarche
    - Highest breast sensitivity during puberty.

- **Key Message**

- ✓ Reproductive factors are important but site-specific modifiers
- ✓ Increased radiation sensitivity during hormonal development

# Other factors

- **Limited / Inconsistent Evidence**

- Lifestyle factors (alcohol, BMI, diet) → weak and non-reproducible findings
- Underlying conditions (diabetes, chronic inflammation)  
→ some signals, but not consistent
- Environmental factors (UV, chemicals) → sparse evidence, unclear effects
- Genetic/epigenetic factors → biologically plausible but limited epidemiologic support

- **Key Message**

- ✓ Many plausible modifiers, but
- ✓ Human evidence remains weak and inconclusive

# Conclusions

- **Only a few modifying factors have robust epidemiological support**
    - Age (age at exposure, attained age)
    - Biological sex
  - **Moderate evidence:**
    - Smoking for lung cancer
    - Reproductive factors for breast cancer
  - **Limited evidence for most of the other potential modifiers**
    - Possibly due to limited power, lack of complete data (measurement error, missing data), and heterogeneity.
- ➔ **Prediction of individual cancer risk remains a major challenge.**