

**A Chernobyl Fallout Transfer Study
with Risk Context and a RAD51 Follow-up Proposal**

Course: Experimental Design and Data Reproducibility

Team: Chernobyl Echo

Executive summary

Goal: connect atmospheric measurements to an exposure pathway narrative (air → soil → grass → cow → milk) and benchmark the resulting doses against commonly used radiological reference values, mapping dataset.

Methods: cleaned multi-country time-series of I-131, Cs-134, Cs-137; imputed missing values with Random Forest models; quantified temporal and geographic patterns; translated concentrations to inhalation dose (adult vs infant) using standard dose coefficients; added a simplified milk pathway model for infants converted dose to an illustrative lifetime cancer risk metric (ELCR).

Key finding: inhalation doses are sub-mSv across the analyzed European countries ($\leq \sim 0.34$ mSv for infants in our dataset). A simplified milk pathway scenario for infants can dominate inhalation ($\approx 9\text{-}19\times$ in our calculations), reaching a few mSv at the high end (≈ 2.7 mSv).

Interpretation: these modeled doses are small compared with reference levels used in emergency response, and far below thyroid absorbed doses observed in the most affected Chernobyl regions. Nevertheless, the pathway ranking is robust: for I-131, contaminated milk is the main concern for children if it is not controlled quickly.

Actionable recommendations: prioritize rapid food-chain controls (especially milk), targeted iodine thyroid blocking for children/pregnant women when directed by authorities, and transparent risk communication.

Future work: a DoE-driven lab validation line - test candidate radioprotectors and RAD51 pathway variation using factorial designs and DNA damage readouts.

1. Introduction and historical context

The 1986 Chornobyl accident released a complex mixture of radionuclides into the atmosphere. For public health, the early-phase isotope iodine-131 (I-131; half-life ≈ 8 days) is critical because it concentrates in the thyroid, especially in children, and can enter the body through inhalation and - most importantly - through ingestion of contaminated fresh milk.

In the most affected areas of Belarus, Ukraine, and parts of the Russian Federation, a large increase in thyroid cancer among those exposed as children or adolescents has been documented. Major syntheses (e.g., UNSCEAR and WHO) report more than $\sim 6,000$ thyroid cancer cases in those groups, with a substantial fraction attributable to radiation exposure.

In Western and Central Europe, radionuclide deposition was measurable and led to temporary food controls in several countries, but estimated thyroid absorbed doses to young children were far lower than in the most contaminated regions (often reported as $< \sim 20$ mGy for preschool children in most European countries).

1.1 Historical benchmark numbers (from UNSCEAR/WHO syntheses)

To anchor our modeled doses in the historical record, we summarize a few widely cited benchmark numbers. UNSCEAR estimates that average thyroid absorbed doses (mainly from milk I-131 intake in the first weeks) were about 500 mGy for evacuees; about 100 mGy for residents of contaminated areas; about 20 mGy for non-evacuated residents of the former Soviet Union; and about 1 mGy for residents of most other European countries. UNSCEAR also reports an average thyroid absorbed dose of ~ 4.8 mGy for Poland (often discussed as a case where rapid iodine prophylaxis and food controls limited thyroid exposure).

WHO/UNSCEAR syntheses report $> \sim 6,000$ thyroid cancer cases diagnosed among those exposed as children or adolescents in Belarus, Ukraine and the Russian Federation, with a substantial fraction attributed to radiation exposure.

These numbers matter for interpretation: a “few mSv” effective dose in Europe is not in the same risk regime as hundreds of mGy thyroid absorbed dose in the most affected regions.

2. Conceptual pathway: air → ground → milk

Figure 1 summarizes the exposure chain we model conceptually and (in simplified form) quantitatively.

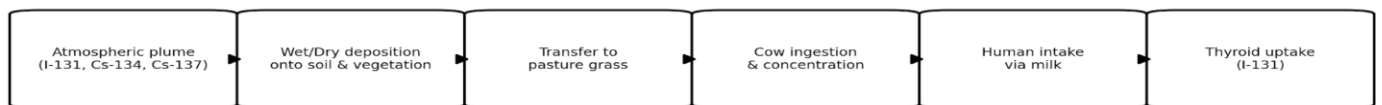


Figure 1. Conceptual transfer chain from atmospheric plume to thyroid-relevant intake via milk.

To support spatial intuition, we also include a static snapshot derived from the interactive map. The full interactive visualization is provided as Supplementary.

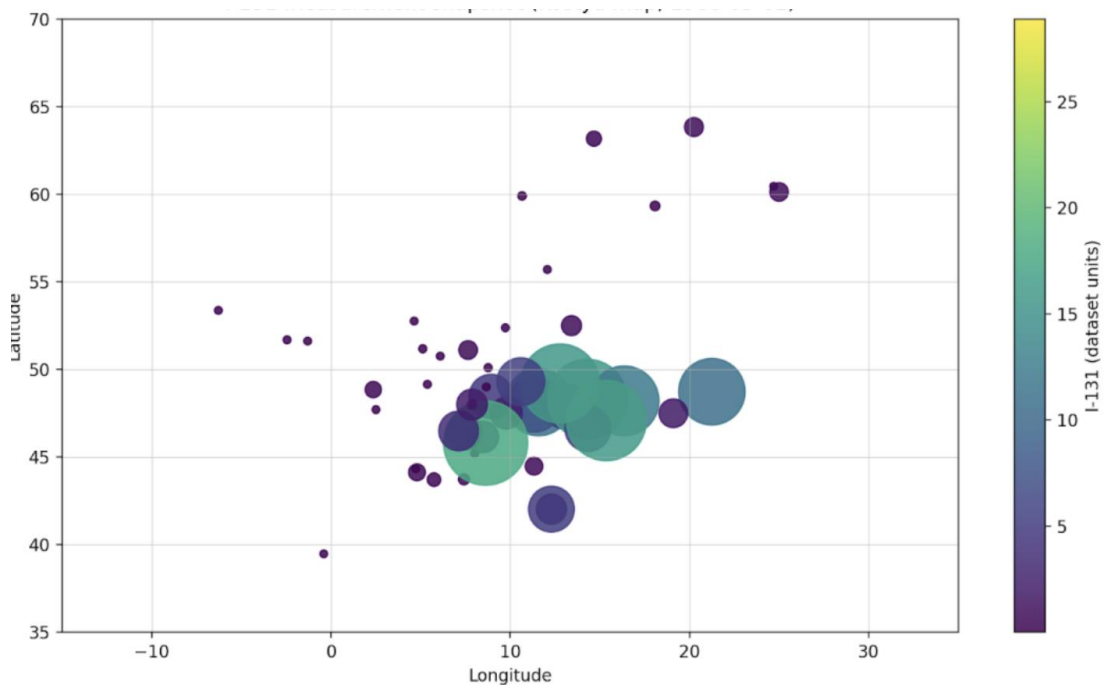


Figure 2. Static snapshot derived from the interactive Plotly map (date label shown on figure).

To connect the monitoring measurements to the broader atmospheric dispersion context, we include a reference plume/transport visualization for the same time period. This panel is provided for qualitative spatial intuition (direction/timing of transport) only and is not used for quantitative dose calculations in our pipeline.

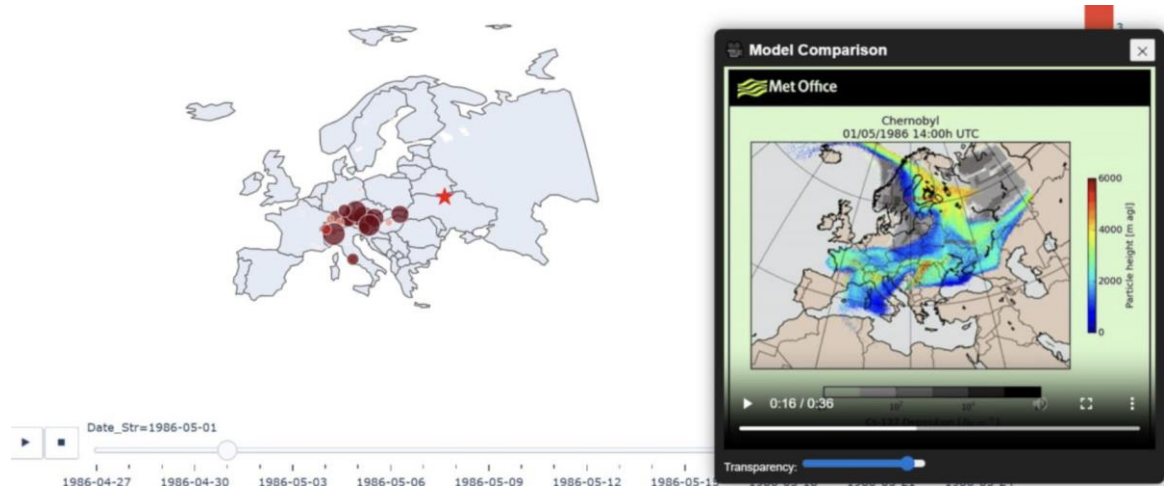


Figure 3. Linking observations to transport context. Left: I-131 monitoring measurements (snapshot from our interactive map; date shown on the panel). Right: reference atmospheric transport/plume visualization (Met Office; date/time shown on the panel).

3. Data and methods

3.1 Dataset and preprocessing

We worked with a multi-country dataset of atmospheric activity concentrations (Bq/m^3) for I-131, Cs-134, and Cs-137 across April-May 1986. Data were cleaned to correct non-numeric entries, aligned in time, and grouped by country and date.

3.2 Missing data imputation

Because early-phase monitoring data are incomplete, we imputed missing radionuclide values using Random Forest regression, trained on correlated radionuclides and temporal features. Model quality was assessed using held-out data with R^2 scores.

3.3 Dose translation (inhalation)

We translated time-integrated atmospheric concentrations into illustrative committed effective doses for two receptor types:

- Adult (representative breathing rate and dose coefficients)
- Infant (higher dose coefficients per intake and different breathing rate)

Important: effective dose is a whole-body risk metric. For I-131, thyroid absorbed dose is the organ-specific metric most directly linked to thyroid cancer risk; our milk pathway module is meant to approximate the route that drives thyroid dose in children.

3.4 Milk pathway module (infants)

We used a simplified transfer model: atmospheric I-131 → deposition to pasture/grass → cow ingestion → milk concentration → infant intake including I-131 decay. The goal is not perfect reconstruction, but a transparent “order-of-magnitude” comparison between inhalation and ingestion via milk.

3.5 Risk contextualization

We converted dose to an illustrative excess lifetime cancer risk (ELCR) using standard

nominal risk coefficients. This is provided only as a benchmark; epidemiological inference requires far riar information.

4. Results

4.1 Imputation model performance

The Random Forest imputation achieved good predictive performance, particularly for I-131 and Cs-134.

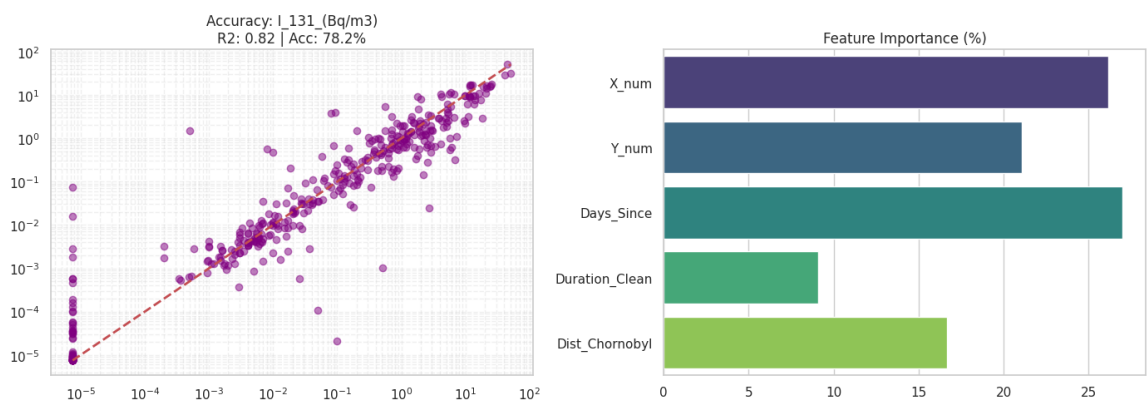


Figure 4. I-131 imputation: observed vs predicted (R^2 shown on plot).

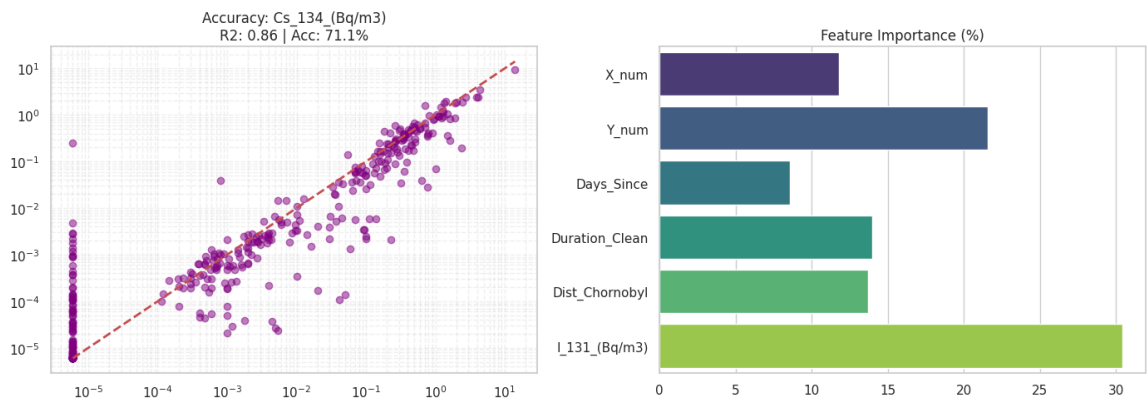


Figure 5. Cs-134 imputation: observed vs predicted (R^2 shown on plot).

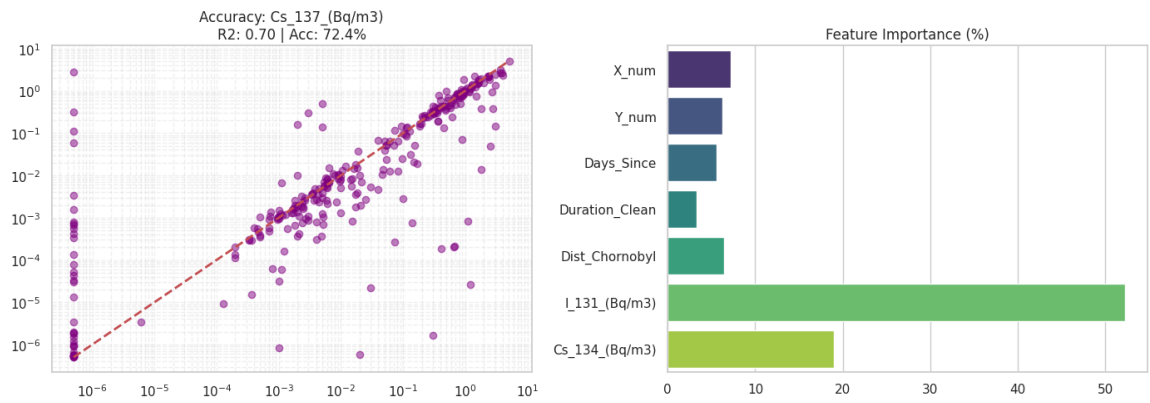


Figure 6. Cs-137 imputation: observed vs predicted (R^2 shown on plot).

4.2 Temporal evolution of airborne activity

Across countries, I-131 shows a pronounced early-May peak consistent with decay and changing plume transport.

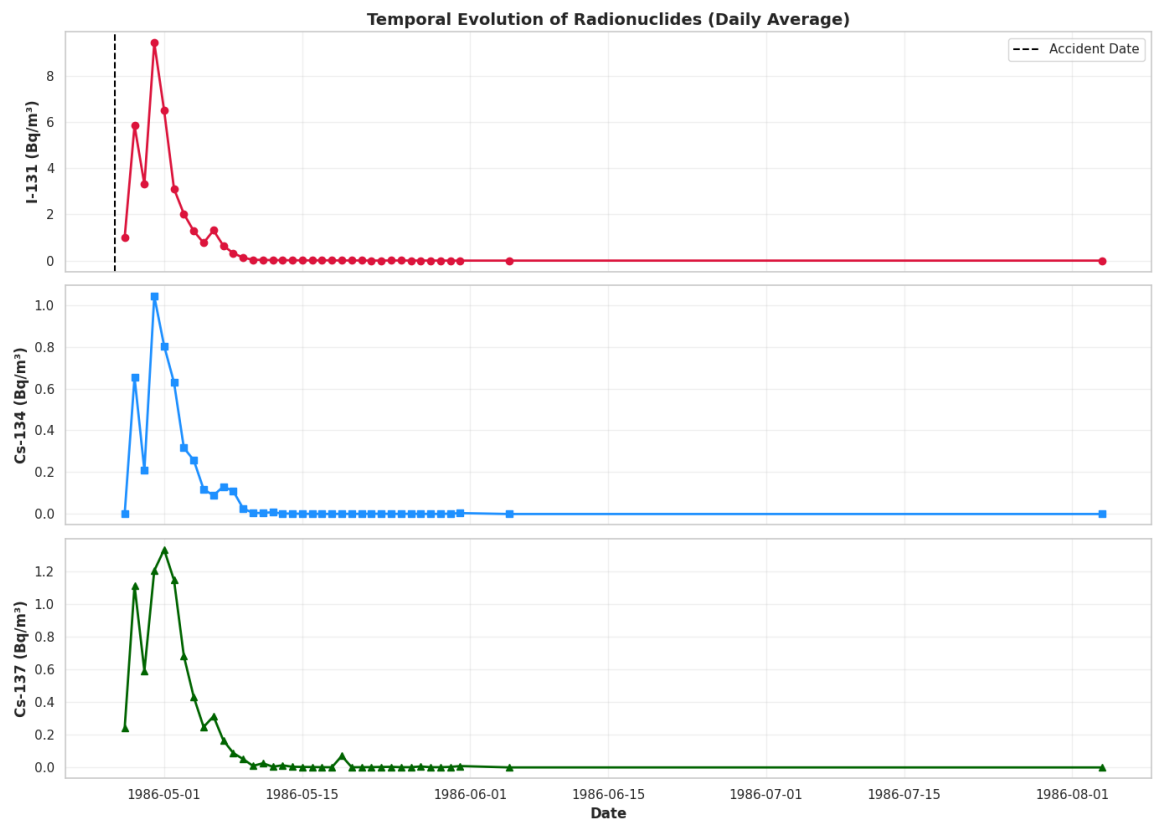


Figure 7. Time series of mean I-131 activity by country (with uncertainty band).

4.3 Country-to-country differences

Distributions show heterogeneity: some countries exhibit higher central tendency and upper tails of I-131 measurements.

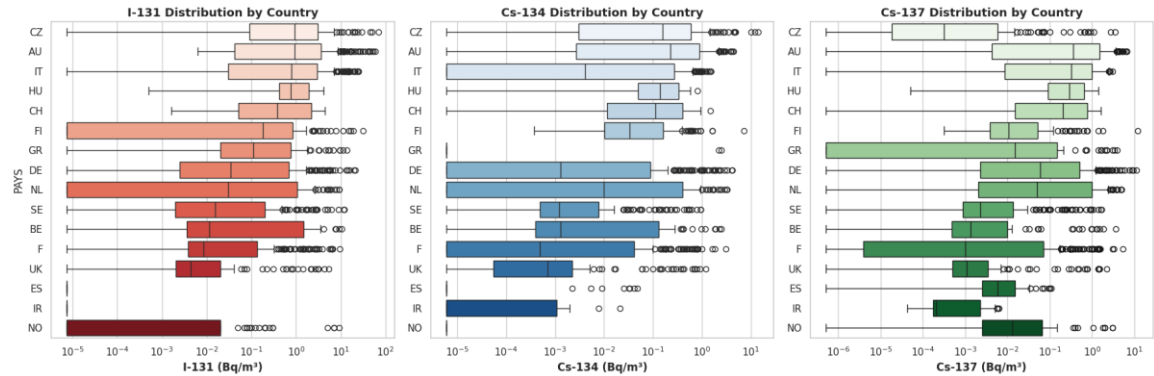


Figure 8. Distribution of I-131 measurements by country.

4.4 Radionuclide correlations and fractionation signals

We assessed relationships among radionuclides and indicators of fractionation (e.g., I-131/Cs-137 decreasing with distance).

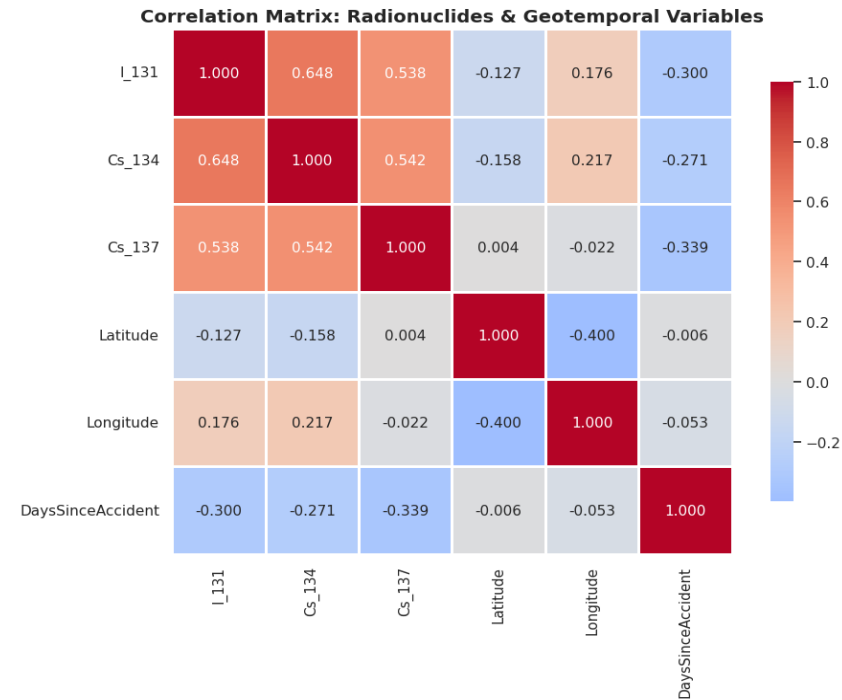


Figure 9. Correlation matrix between radionuclides and distance.

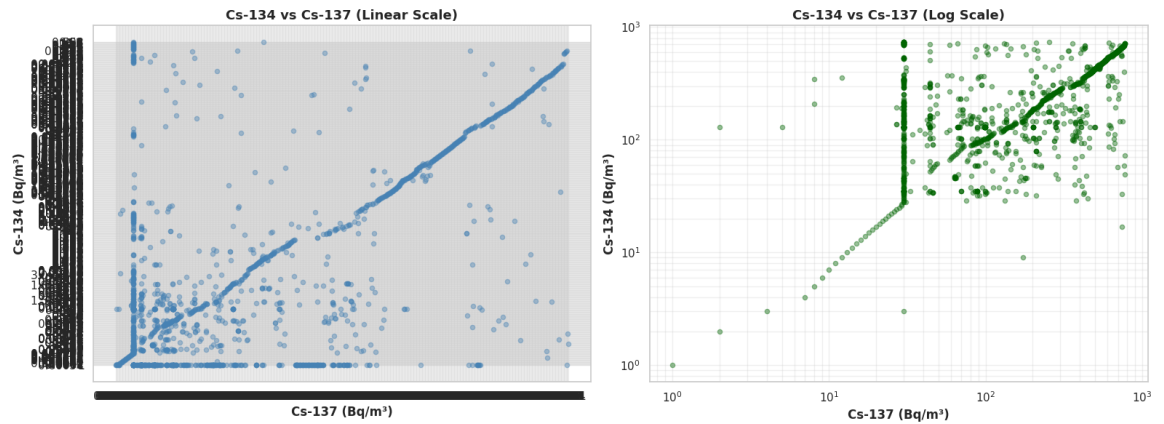


Figure 10. Cs-134 vs Cs-137 relationship (shared source signature).

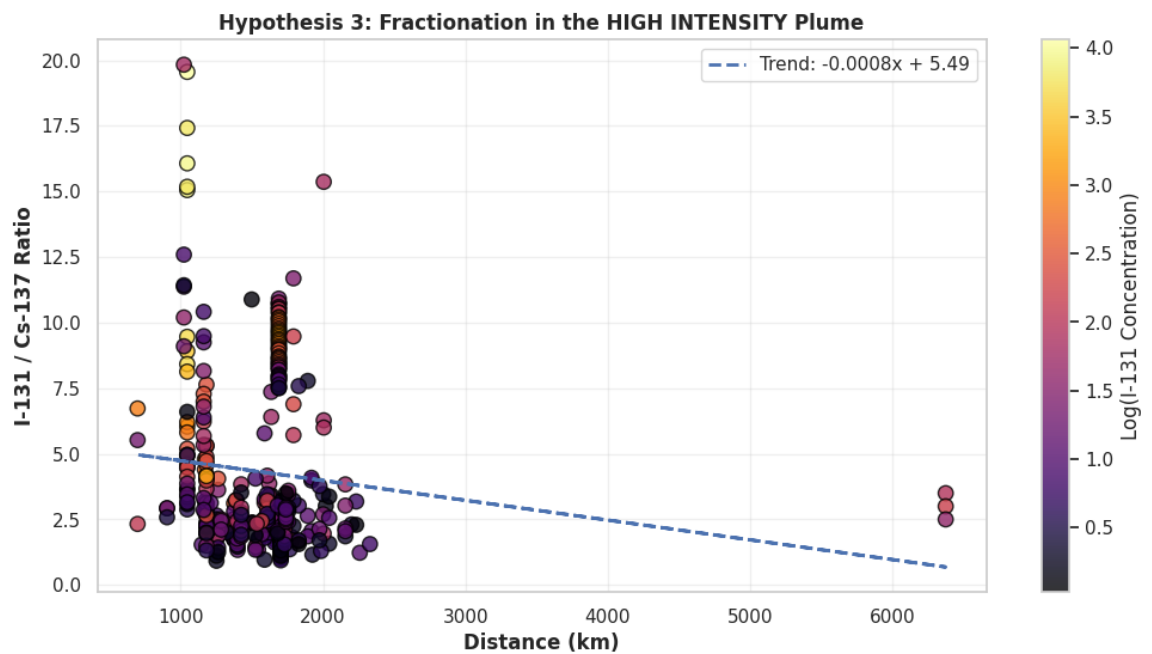


Figure 11. I-131/Cs-137 ratio vs distance (illustrative fractionation trend).

4.5 Dose results and pathway comparison

Inhalation dose (effective dose) remains below 1 mSv across countries in this dataset for both adults and infants; infants have higher committed dose per unit intake. A simplified milk pathway model suggests that ingestion through milk can dominate infant exposure in the first month after deposition, typically by an order of magnitude.

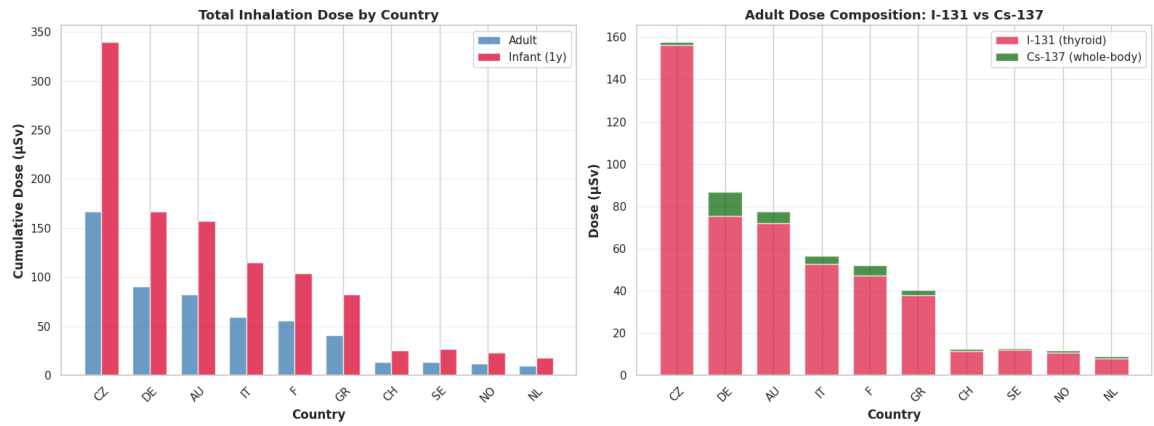


Figure 12. Inhalation dose by country (adult vs infant).

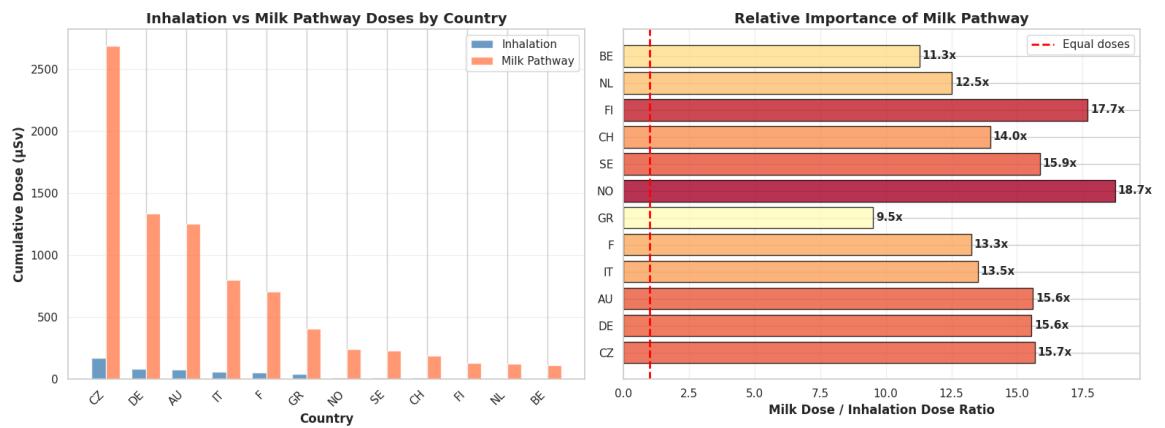


Figure 13. Inhalation vs milk pathway doses and milk-to-inhalation multipliers (infant scenario).

4.6 Illustrative risk contextualization

We translated effective dose to an illustrative ELCR, primarily to compare magnitudes across countries and pathways.

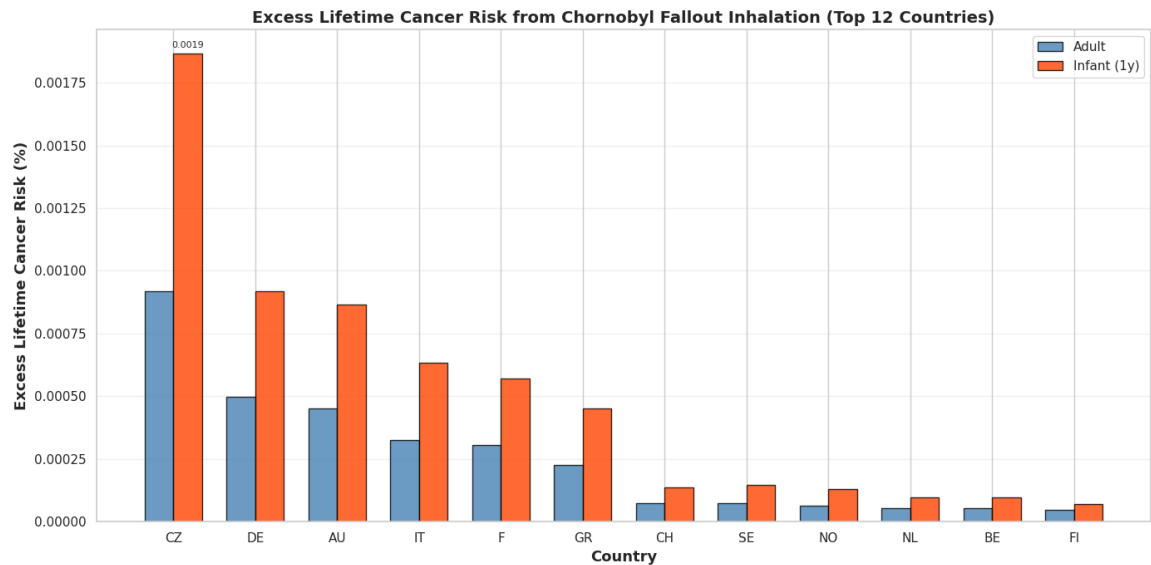


Figure 14. ELCR by country (adult vs infant) based on nominal risk coefficients.

5. Discussion: is this a “big” dose?

A useful way to interpret dose is to benchmark it against: typical natural background radiation (order of a few mSv per year), dose limits for planned exposures of the public (1 mSv/year), and emergency reference levels (tens of mSv and above).

In our dataset, inhalation doses are well below 1 mSv, and thus small in absolute terms. The milk pathway scenario for infants can reach a few mSv in the upper tail - still modest, but no longer “negligible” compared with background.

Crucially, Chernobyl’s observed thyroid cancer signal in affected regions was driven by thyroid absorbed doses from I-131 ingestion (often hundreds of mGy to several Gy), which are orders of magnitude larger than the doses modeled here for most of Europe. This supports the conclusion that for the studied European countries, the modeled exposures would be unlikely to create a detectable thyroid cancer signal - while still justifying rapid food-chain controls as a precaution in early-phase releases.

In Western Europe, contamination with iodine-131 occurred mainly through ingestion of contaminated milk, and raw vegetables. The contribution of short-lived isotopes is negligible. Some products considered to have a radioactive burden higher than a "safety

threshold" were not approved for sale by local authorities. Thyroid doses are not always correlated with the level of radioactive deposit. They also depended on whether cows were on pasture, on dietary habits, and on the avidity of the thyroid for iodine, being higher in countries with low iodine in diet.

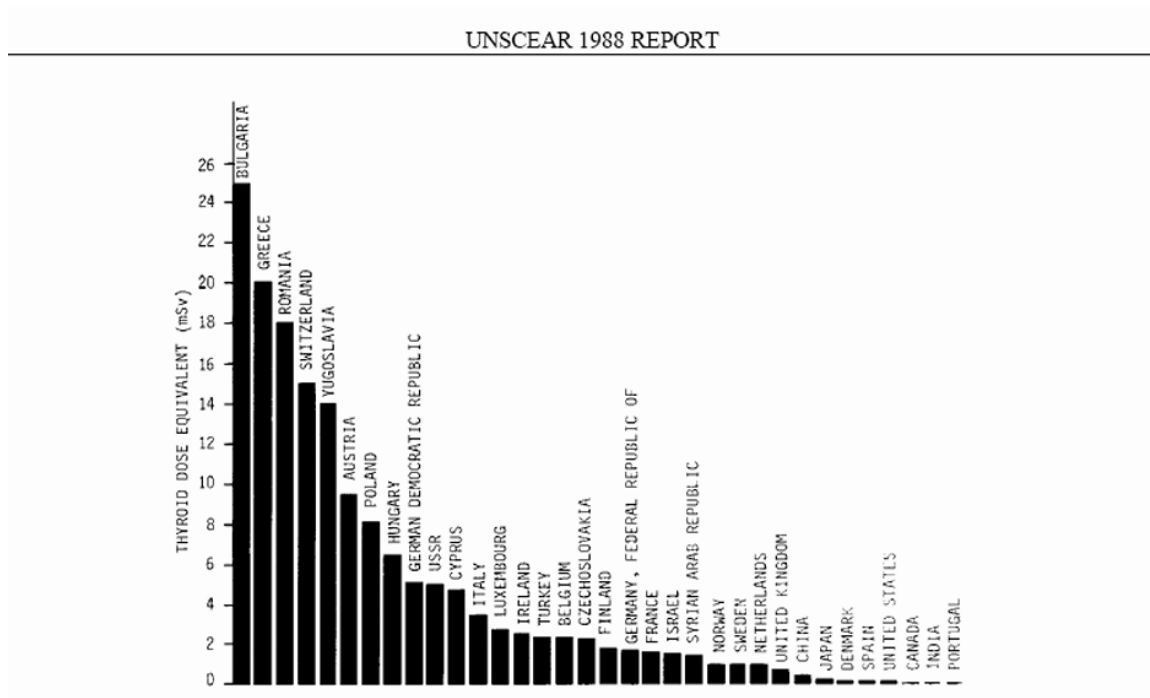


Figure XV. Country-wide average infant thyroid dose equivalents from the Chernobyl accident

6. Public health / safety strategy

If a similar early-phase release occurred today, the priority actions should follow the exposure chain logic:

1. Rapid monitoring and transparent reporting: airborne concentrations, deposition, and food measurements (milk first).
2. Food-chain controls: temporary restriction/monitoring of fresh milk and leafy vegetables in affected zones; ensure safe feed for dairy cattle.
3. Targeted iodine thyroid blocking (stable iodine) for vulnerable groups (children, pregnant/lactating women) only when advised by public health authorities and timed appropriately.

4. Risk communication: explain that milk controls and thyroid protection are the main levers; avoid panic language but be explicit about uncertainty and thresholds.
5. Longer-term: environmental remediation where needed (soil management), health registries, and focused thyroid screening for high-exposure groups.

6. Future work: radioprotectors and RAD51

7.1 Motivation: RAD51 and radioprotection

RAD51 is a central homologous recombination protein that repairs DNA double-strand breaks. Because ionizing radiation induces DNA damage of this type, RAD51 activity (and the integrity of the HR pathway) is a plausible modifier of radiation sensitivity. In this project, RAD51 is introduced as a mechanistic bridge: our exposure-pathway modeling motivates prevention (especially for I-131 and the milk pathway), while RAD51-based radioprotector ideas motivate future laboratory validation at the DNA-repair level.

7.2 Radioprotector concept

Two complementary layers should be clearly separated in a reproducible risk-mitigation strategy:

- Public health countermeasures (population-level): e.g., thyroid blocking with stable iodine for radioiodine exposure; rapid food-chain controls such as restricting fresh milk and contaminated feed.
- Cellular radioprotectors (research-level): candidate compounds intended to reduce DNA damage or enhance DNA repair after exposure. These are not substitutes for public health measures; they are mechanistic interventions to be validated experimentally.

7.3 Experimental design

7.3.1 Research question and hypotheses

Primary question: Does a RAD51-modulating candidate (radioprotector) measurably mitigate radiation-induced cellular damage, and does the effect depend on dose and timing of administration?

- H0: No difference between candidate and vehicle controls across outcomes at matched dose/time.
- H1: The candidate changes outcomes (e.g., reduces DNA damage markers and/or increases survival), potentially with Dose x Compound and Timing x Compound interactions.

7.3.2 Experimental system and units

Experimental unit: a well (or dish) of cultured cells treated under a defined condition. To support generalization, use at least two biologically relevant cell models (e.g., a thyroid-derived epithelial model and a non-thyroid normal cell model such as dermal fibroblasts). All wet-lab work must be conducted in certified facilities under radiation safety oversight.

7.3.3 Factors, levels, and controls

Suggested factorial structure (example; exact levels should follow lab constraints and safety protocols):

- Factor A - Radiation dose: 0 (sham), low, medium, high (e.g., 0, 0.5, 1, 2 Gy).
- Factor B - Compound condition: vehicle control; RAD51 stimulator candidate (e.g., RS-1); optional comparator (e.g., a RAD51 inhibitor such as B02) for mechanism-checking.
- Factor C - Timing: pre-treatment vs post-treatment (or early vs delayed post-treatment).
- Factor D - Concentration: low vs high (predefined from literature or pilot).
- Blocking factor (recommended): experimental day / batch (captures drift in cell state, irradiation conditions, staining).

Control groups to include in every batch: (i) sham irradiation + vehicle, (ii) irradiation + vehicle, and (iii) compound-only (no irradiation) to detect toxicity or baseline effects.

7.3.4 Randomization, replication, and blinding

Randomization: randomly assign conditions to wells within each plate; randomize irradiation order to avoid time-of-day effects; randomize staining/imaging order where applicable. Replication: use biological replicates (independent culture/irradiation days) and technical replicates (multiple wells per condition within a day). Blinding: whenever feasible, blind image scoring / downstream analysis to condition labels until QC is complete.

7.3.5 Outcomes and measurement plan

Primary outcomes (choose 1-2 as preregistered): (a) DNA damage marker readout such as gamma-H2AX foci per cell at an early post-exposure time; (b) clonogenic survival fraction at a fixed endpoint. Secondary outcomes can include micronuclei frequency, apoptosis fraction, cell-cycle distribution, comet assay metrics, or HR-reporter readouts. Record all protocol parameters (cell passage, confluence, incubation times, staining antibodies/batches, imaging settings) to support reproducibility.

7.3.6 ANOVA / mixed-effects model specification

For a factorial design with blocking by day, analyze primary outcomes with an ANOVA-style model (or linear mixed-effects model if day/batch is treated as random):

$$y = \mu + Dose + Compound + Timing + Concentration + Dose:Compound + Timing:Compound + Dose:Timing + \dots + (1 \mid Day/Batch) + error$$

Key tests: (i) Compound main effect (overall protection), (ii) Dose x Compound interaction (dose-dependent protection), (iii) Timing x Compound interaction (window-of-opportunity). Apply multiple-testing control if many secondary endpoints are explored.

7.3.7 Reproducibility checklist (course-aligned)

Minimum reproducibility deliverables:

- Pre-registration of the primary endpoint(s), factor levels, exclusion rules, and the statistical model.

- Version-controlled analysis (e.g., Git), environment specification (requirements.txt/conda), and fixed random seeds for any image-analysis ML steps.
- Raw data + metadata schema (plate map, batch, instrument settings), plus a fully reproducible pipeline that regenerates figures and tables from raw inputs.
- Reporting of deviations (protocol changes, failed runs) and inclusion of negative/positive controls to reduce researcher degrees of freedom.

Figures 15-18 summarize the RAD51 background and a preliminary in-silico docking line prepared by the team.

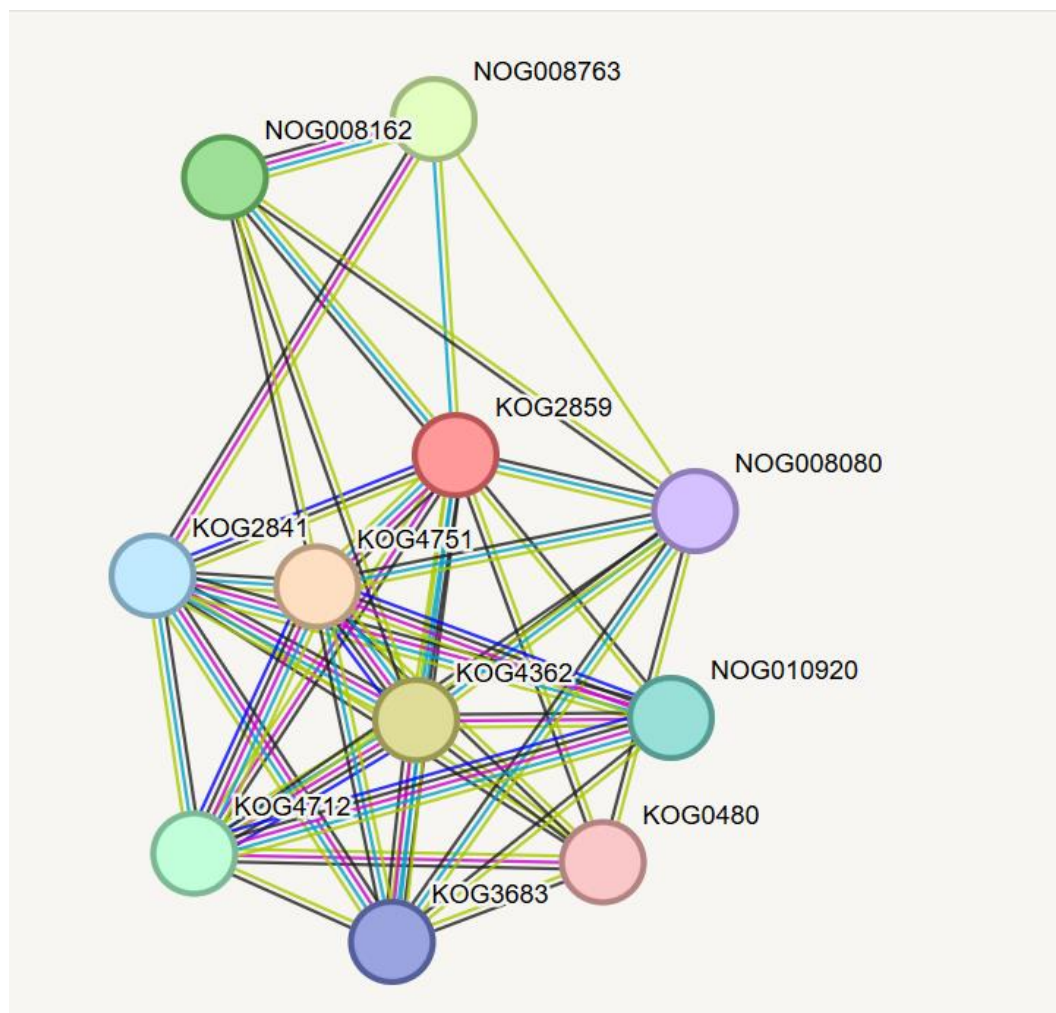


Figure 15. RAD51 interaction network and pathway context.

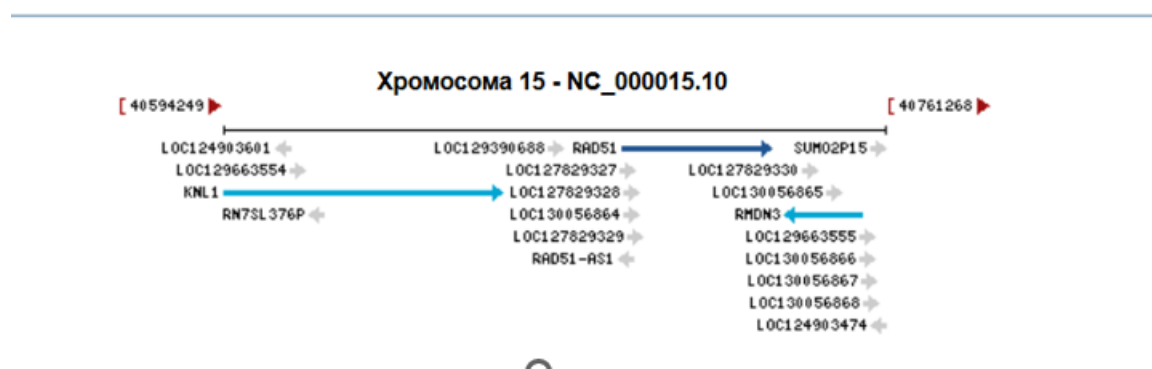


Figure 16. RAD51 genomic context and transcript overview.

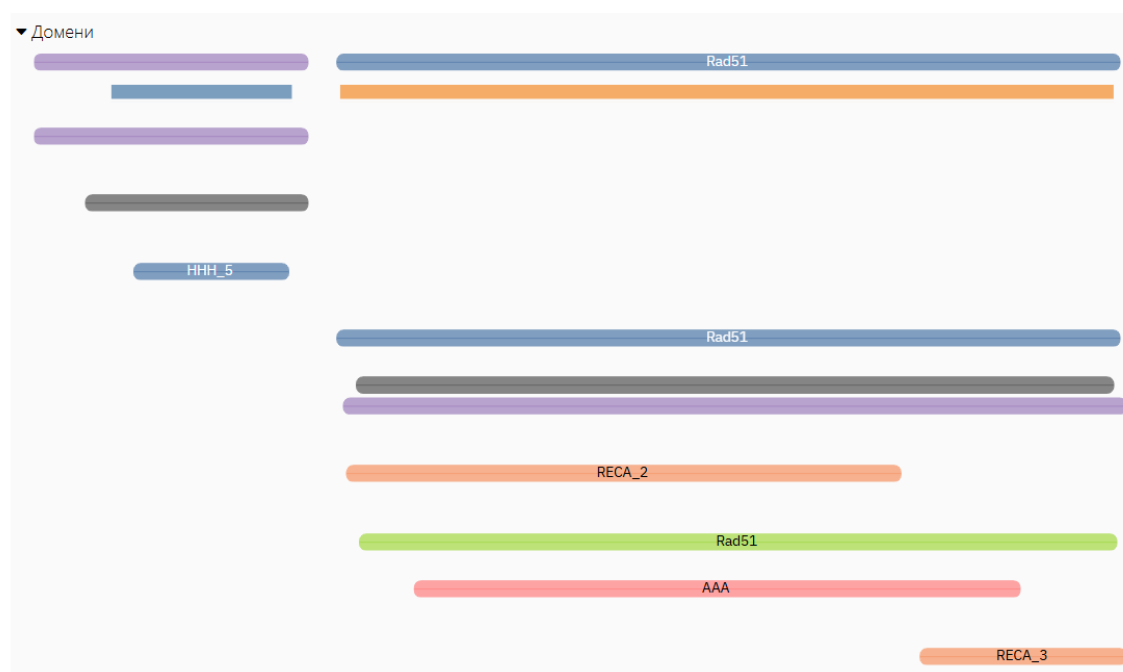


Figure 17. RAD51 protein domains and structural overview.

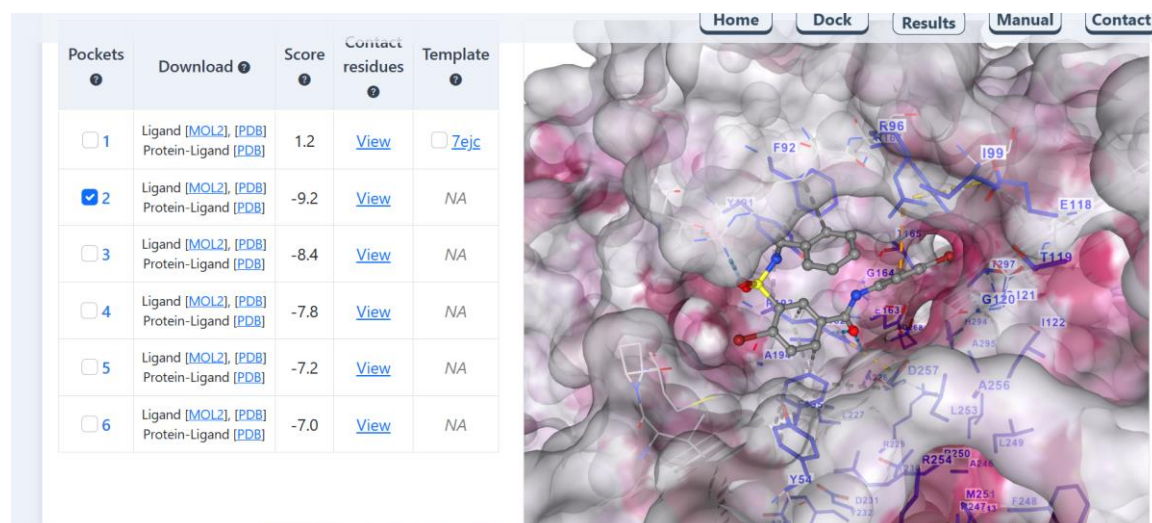


Figure 18. Example docking outputs for candidate ligands (in-silico screening)

References

1. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Sources and Effects of Ionizing Radiation: UNSCEAR 2008 Report to the General Assembly with Scientific Annexes. Annex D: Health effects due to radiation from the Chernobyl accident. United Nations (published in Volume II, 2011; PDF corrected 2016).
2. World Health Organization (WHO). Radiation: The Chernobyl accident - Q&A (summary of UNSCEAR conclusions). WHO, 2011.
3. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Sources and Effects of Ionizing Radiation: UNSCEAR 2000 Report to the General Assembly with Scientific Annexes. United Nations, 2000.
4. International Commission on Radiological Protection (ICRP). The 2007 Recommendations of the International Commission on Radiological Protection (ICRP Publication 103). Ann. ICRP 37(2-4), 2007.
5. World Health Organization (WHO). Iodine thyroid blocking: Guidelines for use in planning for and responding to radiological and nuclear emergencies. WHO, 2017.
6. Cardis E., Hatch M. The Chernobyl accident - an epidemiological perspective. Clinical Oncology, 23(4):251-260, 2011.
7. National Research Council. Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII Phase 2. The National Academies Press, Washington, DC, 2006. doi:10.17226/11340.
8. Jayathilaka K. et al. A chemical compound that stimulates the human homologous recombination protein RAD51. Proc Natl Acad Sci USA, 105(41):15848-15853, 2008. doi:10.1073/pnas.0808046105.

9. Huang F. et al. Identification of specific inhibitors of human RAD51 recombinase using high-throughput screening. ACS Chemical Biology, 6(6):628-635, 2011. doi:10.1021/cb100428c.

10. Mason J.M. et al. The RAD51-stimulatory compound RS-1 can exploit the RAD51 overexpression that exists in cancer cells and tumors. Cancer Research, 74(13):3546-3555, 2014. doi:10.1158/0008-5472.CAN-13-3220.

11. Kouvaris J.R., Kouloulis V.E., Vlahos L.J. Amifostine: the first selective-target and broad-spectrum radioprotector. The Oncologist, 12(6):738-747, 2007. doi:10.1634/theoncologist.12-6-738.