

Computational Modeling of Energy-Dependent Radiation-Induced Cellular Damage using an Enhanced Linear–Quadratic Framework for Radiology Optimization

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ABSTRACT

The increasing application of ionizing radiation in diagnostic imaging has raised concerns regarding radiation-induced cellular damage and the need for biologically informed radiological optimization. This study developed an enhanced computational Linear–Quadratic (LQ) framework to investigate the combined effects of photon energy and absorbed dose on cellular survival and damage in mammalian cells. The model incorporated energy-dependent radiosensitivity parameters and evaluated X-ray photon energies from 10 to 150 keV over absorbed doses of 0–10 Gy. Cellular survival fraction and damage index were calculated using the LQ formalism, while regression and contour analyses were used to characterize energy–dose relationships. The results showed a progressive decrease in radiosensitivity from 0.75 at 10 keV to 0.33 at 150 keV, indicating greater predicted biological effectiveness at lower photon energies. At 1 Gy, survival fraction increased from 0.438 to 0.696 as photon energy increased from 10 to 150 keV, whereas the corresponding damage index decreased from 0.562 to 0.304. At 4 Gy, survival fractions ranged from 0.015 to 0.158, while at 10 Gy, survival approached complete cellular inactivation across all investigated energies. Regression analysis demonstrated increasingly strong relationships between photon energy and damage index with increasing dose, with R^2 values ranging from 0.618 at 1 Gy to 0.903 at 6 Gy. Contour analysis further demonstrated greater predicted cellular damage at lower photon energies, particularly at intermediate doses. The proposed framework provides a quantitative approach for evaluating energy-dependent radiobiological responses and supporting radiological optimization and radiation protection strategies.

Keywords:

Linear–Quadratic model,
Radiobiology,
Cellular Survival,
Photon Energy,
Absorbed Dose,
Radiation Damage.

INTRODUCTION

The increasing use of ionizing radiation in medical imaging, particularly computed tomography (CT), fluoroscopy, and other advanced imaging modalities, has raised concerns regarding radiation exposure and its potential biological effects on human tissues (Auwal *et al.*, 2025a; Brenner & Hall, 2007; Seeram, 2023). Although these techniques provide valuable diagnostic information, exposure to ionizing radiation may induce DNA damage, chromosomal aberrations, and other cellular alterations associated with adverse biological outcomes (Hall & Giaccia, 2019; ICRP, 2022). Furthermore, variations in scanner technology, imaging protocols, operator experience, and quality assurance

practices contribute to differences in patient radiation doses across imaging centres (IAEA, 2024; Rehani *et al.*, 2020; Balashazy *et al.*, 2009). These challenges are particularly relevant in developing countries, including Nigeria, where limitations in equipment maintenance, quality control, and implementation of optimization strategies may contribute to elevated patient exposures during radiological examinations (IAEA, 2024; WHO, 2023; Balashazy *et al.*, 2009). Consequently, improving the prediction of radiation-induced biological effects while maintaining diagnostic image quality remains an important objective in medical physics.

Computational radiobiology provides an important approach for investigating the relationship between

radiation exposure and cellular response. The Linear–Quadratic (LQ) model is widely used to describe cell survival as a function of absorbed dose through its linear and quadratic components, thereby providing a mathematical basis for evaluating radiation-induced cell killing (Hall & Giaccia, 2019). However, the classical LQ framework does not inherently account for photon-energy-dependent variations in biological effectiveness unless additional radiation-quality parameters are incorporated. Recent computational and experimental investigations have demonstrated the relevance of radiation quality to biological response. McElligott *et al.* (2024) used Monte Carlo simulations to estimate the relative biological effectiveness of clinical kilovoltage photon beams for DNA double-strand-break induction, while Habelt and Dörr (2023) investigated the biological effectiveness of low-energy X-rays in mutant p53 cancer cells. Similarly, Li *et al.* (2020) evaluated differences in biological effectiveness between 160 kVp and 6 MV X-rays using DNA dosimetry. These studies highlight the importance of considering photon energy alongside absorbed dose when investigating radiation-induced biological effects. In addition, Li (2023) discussed limitations of the LQ model as a predictive description of cell survival, particularly at high doses, emphasizing the need for careful interpretation and continued development of radiobiological models. Broader computational approaches to biological systems, including multiscale and molecular simulation frameworks, further demonstrate the value of integrating physical and biological processes in radiation-response investigations (Luthey-Schulten *et al.*, 2022; Yusufaly *et al.*, 2025; Umegaki *et al.*, 2024).

Despite these advances, a gap remains in the development of a computational framework that explicitly incorporates photon-energy-dependent radiosensitivity into an LQ-based prediction of cellular survival and damage across a broad low-energy X-ray range and varying absorbed doses. In particular, the combined influence of photon energy and dose on a mathematically defined cellular damage index requires further systematic investigation. Addressing this gap, the present study develops an enhanced computational LQ framework to evaluate the effects of photon energies from 10 to 150 keV and absorbed doses from 0 to 10 Gy on predicted cellular survival and damage in a generic mammalian cell. The model incorporates an energy-dependent scaling of radiosensitivity parameters, while regression and contour analyses are used to characterize energy–dose relationships. The resulting framework is intended to provide a quantitative basis for investigating radiobiological response and informing radiation protection and radiological optimization strategies. Its potential relevance includes supporting medical physicists, radiologists, and radiographers in understanding energy-dependent biological effects,

while contributing to broader efforts to improve patient radiation safety and optimize diagnostic imaging practices.

MATERIALS AND METHODS

A computational model was developed to quantify radiation-induced cellular damage in a generic mammalian cell exposed to X-ray photons. The model integrates photon energy, absorbed dose, and biological response using established radiobiological principles.

Materials

- i. Computer 8th generation (Octa-Core)
- ii. Python Software 3.5.2

Radiation Parameters

- i. Photon energy range: 10–150 keV
- ii. Absorbed dose range: 0–10 Gy

These ranges are representative of diagnostic and low-energy radiation exposure conditions.

Radiobiological Model

Cell survival was estimated using the Linear–Quadratic Model, expressed as:

$$S(D, E) = e^{-[\alpha(E)D + \beta(E)D^2]} \quad (1)$$

Where: S surviving fraction, D : absorbed dose (Gy), $\alpha(E)$, $\beta(E)$: energy-dependent radiosensitivity parameters

Energy Dependence of Radiosensitivity

To incorporate photon energy effects, a scaling factor was introduced:

$$\eta(E) = 1 + 0.3 \left(\frac{50}{E} \right) \quad (2)$$

$$\alpha(E) = \alpha_o \eta(E), \quad \beta(E) = \beta_o \eta(E)$$

With baseline parameters: $\alpha_o = 0.30 \text{ Gy}^{-1}$ and $\beta_o = 0.03 \text{ Gy}^{-1}$

Damage Index

The degree of cellular damage was quantified using:

$$DI = 1 - S(D, E) \quad (3)$$

Where: $DI = 0$: no damage and $DI = 1$: complete cell death

Regression Analysis of Energy vs Damage

$$DI = aE + b \quad (4)$$

Where; DI = Damage Index, E = Photon Energy, a = Slope (Energy dependence) and b = Intercept. Regression analysis was performed to test the relationship between photon energy and cellular damage index using equation (4).

Contour Visualization

A two-dimensional contour plot was generated with:

- i. X-axis: Photon energy (keV)
- ii. Y-axis: Absorbed dose (Gy)
- iii. Contours: Damage index levels

The computational implementation was carried out using Python

RESULTS AND DISCUSSION

Table 1: Results of the developed computational radiobiological model from the photon energy values

Photon Energy (keV)	$\alpha (E)$	Radiosensitivity Level
10 keV	0.75	Very high
30 keV	0.45	High
50 keV	0.39	Moderate
150 keV	0.33	Lower

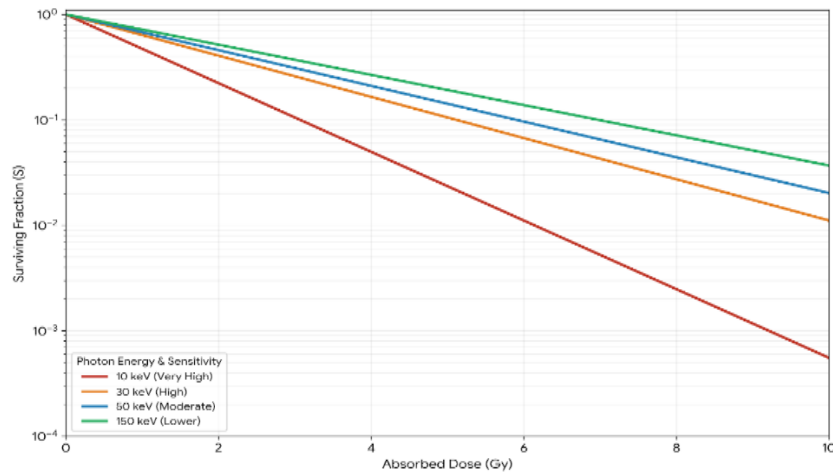


Figure 1: Cell Survival Curve using Computational Radiobiological Model

Table 2: Investigation of cell survival index and cell damage of photon energy at low Dose (1 Gy)

Energy (keV)	Survival (S)	Damage (DI)
10 keV	0.438	0.562
30 keV	0.610	0.390
50 keV	0.651	0.349
150 keV	0.696	0.304

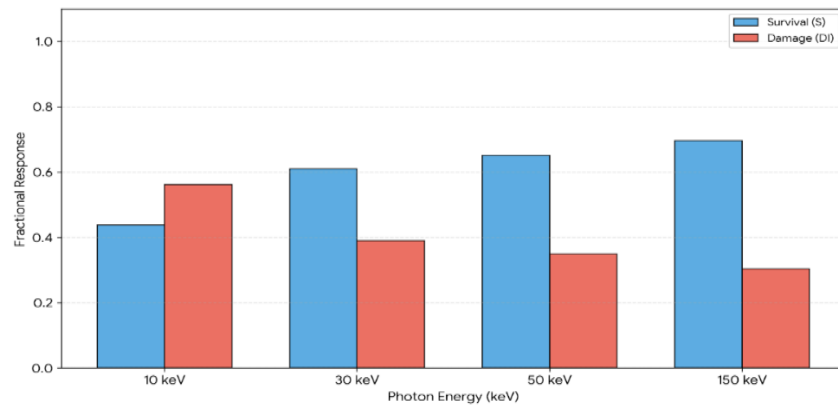


Figure 2: Relationship between cell survival index and cell damage of photon energy at low Dose (1 Gy)

Table 3: Investigation of cell survival index and cell damage of photon energy at moderate Dose (4 Gy)

Energy (keV)	Survival (S)	Damage (DI)
10 keV	0.015	0.985
30 keV	0.080	0.920
50 keV	0.113	0.887
150 keV	0.158	0.842

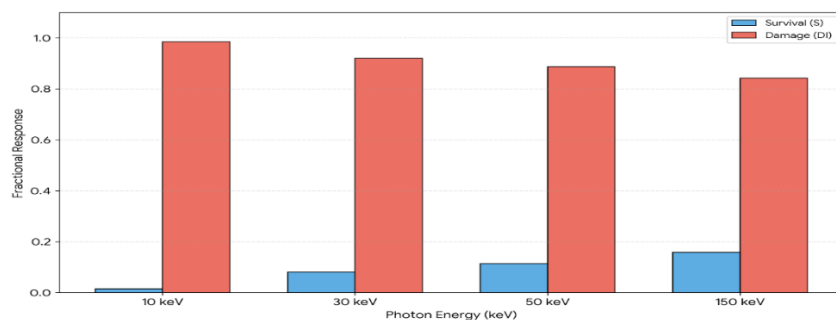


Figure 3: Relationship between cell survival index and cell damage of photon energy at low Dose (4 Gy)

Table 4: Investigation of cell survival index and cell damage of photon energy at high Dose (10 Gy)

Energy (keV)	Survival (S)	Damage (DI)
10 keV	0.0000003	0.9999997
30 keV	0.000123	0.999877
50 keV	0.000410	0.999590
150 keV	0.001360	0.998640

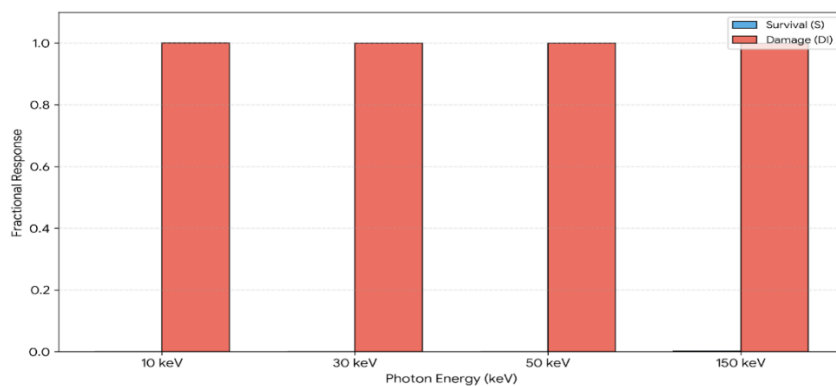


Figure 4: Relationship between cell survival index and cell damage of photon energy at high Dose (10 Gy)

Table 5: Evaluation of Photon Energy and Cellular radio sensitivity effect

Energy (keV)	$\alpha(E)$	Biological Effect
10	0.75	Very high damage
30	0.45	High damage
50	0.39	Moderate
150	0.33	Lower damage

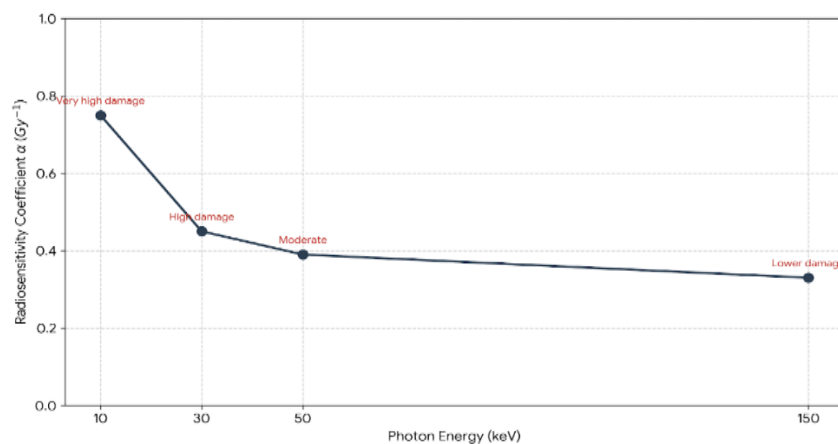


Figure 5: Graph of Photon Energy and Cellular radio sensitivity effect.

Table 6: Regression Analysis between Photon energy and Radio-biological damage

Dose (Gy)	Slope (a)	Intercept (b)	R ²	Interpretation
2	-0.00142	0.478	0.618	Moderate correlation
4	-0.00165	0.751	0.673	Stronger correlation
6	-0.00089	0.957	0.797	Strong relationship
10	-0.00028	0.995	0.903	Very strong relationship

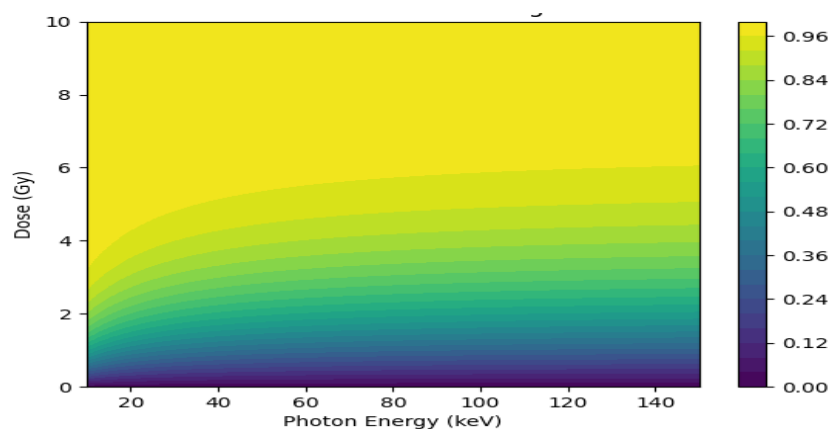


Figure 6: Contour Plot of Cellular Damage Index

Table 7: Model-derived RBE at Matched Surviving Fractions using 150 keV as the reference radiation

Matched survival	Energy	Dose for same survival (Gy)	Model-derived RBE
50%	10 keV	0.851	2.099
50%	30 keV	1.359	1.315
50%	50 keV	1.539	1.161
50%	150 keV	1.787	1.000
10%	10 keV	2.460	1.933
10%	30 keV	3.740	1.272
10%	50 keV	4.161	1.143
10%	150 keV	4.756	1.000
1%	10 keV	4.288	1.836
1%	30 keV	6.312	1.247
1%	50 keV	6.949	1.133
1%	150 keV	7.871	1.000

Discussion

To quantitatively distinguish the energy-dependent LQ radiosensitivity coefficient from relative biological effectiveness (RBE), an iso-survival analysis was performed using the model parameters as presented in table 7. The $\beta(E)$ coefficients were obtained consistently with the reported 1 Gy survival fractions, yielding values of 0.07554, 0.04430, 0.03925, and 0.03241 Gy⁻² for 10, 30, 50, and 150 keV, respectively. Using 150 keV as the reference radiation, model-derived RBE values were calculated from the ratio of the reference dose to the dose required at each test energy to produce the same surviving fraction. At a surviving fraction of 0.50, the required doses were 0.851, 1.359, 1.539, and 1.787 Gy for 10, 30, 50, and 150 keV, respectively, corresponding to RBE values of 2.099, 1.315, 1.161, and 1.000. At a surviving fraction of 0.10, the corresponding doses were 2.460, 3.740, 4.161, and 4.756 Gy, yielding RBE values of 1.933, 1.272, 1.143, and 1.000. Similarly, at a surviving fraction of 0.01, the required doses were 4.288, 6.312, 6.949, and 7.871 Gy, giving RBE values of 1.836, 1.247, 1.133, and 1.000, respectively.

These results provide the quantitative bridge between the energy-dependent $\alpha(E)$ values and biological effectiveness that was not explicitly established in the original analysis. The model-derived RBE decreases systematically with increasing photon energy, with the largest enhancement predicted for 10 keV photons. Importantly, this result does not imply that the $\alpha(E)$ coefficient itself is equivalent to RBE. Rather, the RBE values arise from the dose ratios required to produce identical biological effects, thereby satisfying the conventional iso-effect definition of RBE. Table 7 presents the RBE values of 1.84–2.10 predicted for 10 keV relative to the 150 keV reference are of a similar order to the enhanced biological effectiveness reported for clinical kilovoltage photon beams by McElligott *et al.* (2024), who reported RBE values ranging from 1.03 to 1.82 for double-strand-break induction. The present comparison should nevertheless be interpreted cautiously because the reference radiation, biological endpoint, photon spectrum, cell system, and irradiation conditions differ between the studies. Consequently, the agreement should be regarded as supportive of the predicted direction and approximate magnitude of energy dependence rather than as direct experimental validation of the model.

Table 1 demonstrates a progressive reduction in radiosensitivity from 0.75 at 10 keV to 0.33 at 150 keV. This trend confirms the inverse relationship between photon energy and biological effectiveness. The high radio sensitivity observed at 10 keV suggests that low-

energy photons produce denser ionization clusters within cellular structures, resulting in greater probabilities of irreparable DNA double-strand breaks (DSBs). The observed trend agrees with recent Monte Carlo and micro-dosimetric studies showing that kilovoltage photons induce more complex DNA lesions than megavoltage photons due to increased photoelectric interactions and localized energy deposition. McElligott *et al.* (2024) reported RBE values ranging from 1.03 to 1.82 for kilovoltage photon beams, confirming that lower-energy photons produce greater biological effects per unit absorbed dose. Similarly, Habelt and Dörr (2023) demonstrated enhanced biological effectiveness for 25 kV X-rays in mutant cancer cell lines, emphasizing the increased radio sensitivity associated with low-energy photons. The decreasing radio sensitivity observed between 30 and 150 keV suggests a transition from highly localized ionization events to more sparsely distributed energy deposition patterns. This behavior is consistent with the reduced probability of clustered DNA damage at higher photon energies reported by (Yusufaly *et al.*, 2025; Luthey-Schulten *et al.*, 2022).

Figure 1 illustrates the dose-response behavior predicted by the computational model. The survival curves exhibit the classical exponential decline expected from the Linear-Quadratic framework. The steepest decline occurs for 10 keV photons, indicating maximum biological effectiveness, whereas the shallowest decline occurs for 150 keV photons. The model predicts that survival decreases dramatically as absorbed dose increases. At approximately 4 Gy, the survival fraction for 10 keV photons falls below 2%, while higher-energy photons retain comparatively larger surviving fractions. This result aligns with experimental clonogenic survival studies conducted by (Shamsabadi, 2024; Yusufaly *et al.*, 2025; Luthey-Schulten *et al.*, 2022) which demonstrates increased cell killing efficiency for low-energy X-rays. DNA dosimeter investigations comparing 160 kVp and megavoltage photons reported significantly greater DNA damage and reduced cellular survival following exposure to lower-energy beams, despite identical absorbed doses (Li *et al.*, 2020). The near-linear appearance of the survival curves on a semi-logarithmic scale confirms that cell inactivation follows exponential kinetics, supporting the applicability of the Linear-Quadratic (LQ) formalism within the investigated dose range. However, recent literature has highlighted limitations of the classical LQ model at very high doses where additional biological mechanisms may influence survival outcomes (Li, 2023). Table 2 and figure 2 gives low dose exposure scenario at 1 Gy, this makes substantial differences in biological response among photon energies. The survival fraction

increases from 0.438 at 10 keV to 0.696 at 150 keV, while the damage index decreases correspondingly from 0.562 to 0.304. These findings suggest that biological response is highly sensitive to photon energy at low doses. The pronounced damage observed at 10 keV reflects the greater ionization density and enhanced production of clustered DNA lesions associated with low-energy photons. At low absorbed doses, cellular repair systems remain active, and therefore energy-dependent differences become more apparent. This observation agrees with radiobiological studies conducted by (Shamsabadi, 2024; Yusufaly *et al.*, 2025; Umegaki *et al.*, 2024), the studies reported that low-energy photons produce disproportionately higher biological effects than predicted solely by physical dose considerations. Enhanced DNA damage complexity and increased probabilities of lethal chromosome aberrations have been identified as key contributors to this phenomenon.

The results in Table 3 and figure 3 indicate substantial cellular damage at 4 Gy across all photon energies. Survival fractions range from 0.015 for 10 keV photons to 0.158 for 150 keV photons, while damage indices exceed 0.84 in every case. The persistence of energy-dependent differences at this dose level indicates that photon energy remains an important determinant of biological outcome even when substantial cellular injury has occurred. The approximately tenfold difference in survival between 10 keV and 150 keV photons demonstrates that radiation quality continues to influence cell fate despite increasing dose magnitude. Comparable findings (Shamsabadi, 2024; Yusufaly *et al.*, 2025; Umegaki *et al.*, 2024) have been reported in Monte Carlo-based DNA damage simulations, where low-energy photon beams generated significantly greater yields of double-strand breaks and complex DNA lesions compared with higher-energy photons. These studies suggest that increased photoelectric interactions and secondary electron clustering contribute to enhanced biological effectiveness at lower energies.

From table 4 and figure 4, high dose of 10 Gy, shows that survival fractions become extremely small across all photon energies, approaching complete cellular inactivation. Although energy-dependent differences remain detectable, the dominant effect becomes absorbed dose. The model predicts survival values of only 3×10^{-7} at 10 keV and 1.36×10^{-3} at 150 keV. These results indicate saturation of biological damage, where the overwhelming number of ionization events exceeds the repair capacity of the cell. Similar saturation effects have been reported in experimental radiobiology studies (McElligott *et al.*, 2024; Umegaki *et al.*, 2024; Habelt,

2023; Li *et al.*, 2020), where survival curves converge at high doses due to extensive DNA fragmentation and catastrophic chromosomal damage. Under these conditions, absorbed dose becomes the primary determinant of outcome, while photon energy exerts a secondary influence.

Table 5 and Figure 5 collectively revealed a sharp reduction in radio sensitivity with increasing photon energy. The radio sensitivity coefficient decreases by approximately 56% between 10 and 150 keV. This trend reflects the well-established relationship between photon energy, linear energy transfer characteristics, and DNA damage complexity. Lower-energy photons deposit energy over shorter distances and generate densely clustered ionization tracks. These tracks increase the probability of producing multiple DNA lesions within a small volume, thereby reducing repair efficiency and increasing cell death. Recent studies (McElligott *et al.*, 2024; Li, 2023; Umegaki *et al.*, 2024) involving kilovoltage irradiation systems have similarly demonstrated elevated biological effectiveness relative to higher-energy photon beams. The increased effectiveness has been attributed to enhanced induction of complex DNA double-strand breaks and clustered lesions that are difficult for cellular repair pathways to resolve.

Table 6 revealed regression analysis of photon energy and cellular damage index, which demonstrates increasingly strong correlations between photon energy and damage index as dose increases. The coefficient of determination (R^2) rises from 0.618 at 1 Gy to 0.903 at 6 Gy. This progressive improvement indicates that the relationship between energy and biological damage becomes increasingly predictable at higher doses. The intercept values also increase from 0.478 to 0.995, suggesting that dose-dependent damage accumulation gradually overwhelms biological variability, similar trend shown by the researchers conducted by (McElligott *et al.*, 2024; Umegaki *et al.*, 2024). The strong correlation observed at higher doses supports the validity of the computational framework and confirms that absorbed dose remains the dominant predictor of biological response under severe irradiation conditions. Similar statistical relationships have been reported in computational radiobiology investigations utilizing Monte Carlo-derived DNA damage models and microdosimetric approaches by (Li, 2023; Umegaki *et al.*, 2024).

Figure 6 shows a contour analysis of cellular damage index, which provides a comprehensive visualization of the combined effects of photon energy and absorbed dose on cellular damage. Three distinct biological regions can be identified as low-damage region (<1 Gy) which is

characterized by effective DNA repair and high cellular survival across all photon energies. The Intermediate-damage region (2–5 Gy) is also characterized by rapid increases in damage, particularly at low photon energies where photoelectric interactions dominate. Moreover, the High-damage region (>6 Gy) is characterized by near-complete cellular inactivation regardless of photon energy, these assertions are in close to the findings of (Habelt, 2023; Umegaki *et al.*, 2024). The contour map highlights the synergistic interaction between absorbed dose and photon energy. While dose acts as the principal driver of biological damage, photon energy modulates the severity of damage by influencing ionization density and DNA lesion complexity.

This computational radiobiology studies have emphasized the importance of integrating energy-dependent biological parameters into predictive radiation models. Advanced multiscale simulations increasingly recognize that accurate prediction of radiation response requires simultaneous consideration of physical dose distribution and microscopic DNA damage processes. Collectively, the results demonstrate that low-energy photons possess significantly greater biological effectiveness than higher-energy photons within the investigated energy range. The enhanced cellular damage observed at lower energies arises from increased ionization density, greater production of clustered DNA lesions, and reduced repair efficiency. The study further confirms that while photon energy strongly influences biological response at low and moderate doses, absorbed dose becomes the dominant determinant of outcome at high-dose exposures. The developed computational framework therefore provides a valuable tool for optimizing radiological procedures, estimating biological risk, and improving radiation protection strategies. Incorporation of energy-dependent radio sensitivity parameters into radiobiological modelling may enhance the accuracy of dose-response predictions and contribute to safer and more effective clinical radiation applications.

CONCLUSION

This study developed an enhanced computational Linear–Quadratic (LQ) framework for evaluating the combined effects of photon energy and absorbed dose on cellular survival and radiation-induced damage over 10–150 keV and 0–10 Gy. The model predicted greater cellular damage at lower photon energies, with the radiosensitivity coefficient decreasing from 0.75 at 10 keV to 0.33 at 150 keV and survival at 1 Gy increasing from 0.438 to 0.696, while survival at 4 Gy declined to 0.015–0.158 and approached near-complete model-predicted inactivation at 10 Gy. These results indicate

that absorbed dose is the dominant determinant of severe cellular damage, while photon energy modulates the response at a given dose. However, the energy-dependent function $\eta(E)$, derived RBE values, and high-dose predictions remain computational and require experimental validation; the classical LQ model may also be less reliable at high doses and does not explicitly account for photon interactions, secondary-electron transport, microscopic energy deposition, DNA damage complexity, or cell-specific repair. Future studies should validate $\eta(E)$ and energy-specific α and β parameters using experimental clonogenic survival data, matched-survival RBE measurements, and Monte Carlo or multiscale DNA-damage modelling under clinically relevant conditions. Overall, the framework demonstrates a useful approach for incorporating photon-energy dependence into LQ-based cellular response modelling.

REFERENCES

- Auwal Y. M., Meludu ,O. C., Pascal, T (2025a). Computational modeling and validation of indoor radon gas dynamics and accumulation using ansys fluent simulation 2025. *Communication in Physical Sciences*, 2025, 12(4): 1172-1183. <https://dx.doi.org/10.4314/cps.v12i4.3>
- Auwal Y. M., Meludu ,O. C., Pascal, T (2025b). Health Risk Assessment of Indoor Radon Exposure in Some Residential Dwellings Across North East, Nigeria. *Dutse Journal of Pure and Applied Sciences (DUJOPAS)*, Vol. 11 No. 2a June 2025. <https://dx.doi.org/10.4314/dujopas.v11i2a.18>
- Balashazy, I., Farkas, A., Madas, B. G., & Hofmann, W. (2009). Non-linear relationship of cell hit and transformation probabilities in a low dose of inhaled radon progenies. *Journal of Radiological Protection*, 29(2), 147–162. <https://doi.org/10.1088/0952-4746/29/2/003>
- Brenner, D. J., & Hall, E. J. (2007). Computed tomography—An increasing source of radiation exposure. *New England Journal of Medicine*, 357(22), 2277–2284. <https://doi.org/10.1016/j.apgeochem.2022.105534>
- Dror, R. O., Dirks, R. M., Grossman, J., Xu, H., and Shaw, D. E. (2012). Biomolecular simulation: A computational microscope for molecular biology. *Annu. Rev. Biophysics* 41 (1), 429–452. <https://doi:10.1146/annurev-biophys-042910-155245>

- Habelt, B., & Dörr, W. (2023). Relative biological effectiveness of low-energy X-rays (25 kV) in mutant p53 cancer cells. *Radiation and Environmental Biophysics*, 62(1), 161–170. <https://doi.org/10.57233/ijsgs.v5i2.146>
- Hall, E. J., & Giaccia, A. J. (2019). *Radiobiology for the radiologist* (8th ed.). Wolters Kluwer.
- International Atomic Energy Agency. (2024). *Radiation protection and safety in medical uses of ionizing radiation*. Vienna, Austria: IAEA.
- International Commission on Radiological Protection. (2022). *The 2020 recommendations of the International Commission on Radiological Protection*. ICRP Publication 145. Elsevier.
- Li, H. (2023). Invalidity of, and alternative to, the linear quadratic model as a predictive model for postirradiation cell survival. *Cancer Science*, 114(7), 2931–2938. <https://doi.org/10.1038/s41598-024-69147-3>
- Li, X., McConnell, K. A., Che, J., Ha, C. S., Lee, S. E., Kirby, N., & Shim, E. Y. (2020). DNA dosimeter measurement of relative biological effectiveness for 160 kVp and 6 MV X-rays. *Radiation Research*, 194(2), 173–179.
- Luthey-Schulten, Z., Thornburg, Z. R., and Gilbert, B. R. (2022). ‘Integrating cellular and molecular structures and dynamics into whole-cell models’. *Curr. Opin. Struct. Biol.* 75, 102392. <https://doi.org/10.1016/j.sbi.2022.102392>
- McElligott, O., Nikandrov, M., McCavana, P., McClean, B., & Vintró, L. L. (2024). Estimation of the relative biological effectiveness for double strand break induction of clinical kilovoltage beams using Monte Carlo simulations. *Medical Physics*, 51(5), 3796–3805.
- Rehani, M. M., Frush, D. P., Berris, T., & Einstein, A. J. (2020). Patient radiation exposure in medical imaging: Global trends and optimization strategies. *Radiology*, 295(2), 418–424. <https://doi.org/10.1016/j.R.2020.102277>
- Seeram, E. (2023). *Computed tomography: Physical principles, clinical applications, and quality control* (5th ed.). Elsevier.
- Shamsabadi, R., & Baghani, H. R. (2024). An inter-comparison between radiobiological characteristics of a commercial low-energy IORT system by Geant4-DNA and MCDS Monte Carlo codes. *International Journal of Radiation Biology*, 100(8), 1226–1235. <https://doi.org/10.4236/ijg201787051>
- Umegaki T, Moriizumi H, Ogushi F, Takekawa M, Suzuki T (2024) Molecular dynamics simulations of a multicellular model with cell-cell interactions and Hippo signaling pathway. *PLoS Comput Biol* 20(11): e1012536. <https://doi.org/10.1371/journal.pcbi.1012536>
- World Health Organization. (2023). *Radiation safety in medical imaging: Global perspectives and recommendations*. Geneva, Switzerland: WHO.
- Yusufaly, T., Abdollahi, H., Saboury, B., & Rahmim, A. (2025). Quantitative and computational radiobiology for precision radiopharmaceutical therapies. *arXiv Preprint*. <https://doi.org/10.1251/j.pcbi.101242>