

Radiation Therapy through Genetic Profiling: A Conceptual Framework for Radiogenomic Risk Stratification and Normal-Tissue Radioprotection

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ABSTRACT

Radiation therapy remains a central modality in cancer treatment, but normal-tissue toxicity and long-term stochastic effects vary substantially among patients. This conceptual review examines how genetic profiling and radiogenomic risk stratification may support more individualized radiotherapy planning. The paper synthesizes mechanisms of radiation-induced DNA damage, DNA repair, oxidative stress, hypoxia, inflammation, and inherited radiosensitivity, then proposes a clinically cautious framework for incorporating these data into treatment decisions. Particular attention is given to investigational strategies such as transient modulation of DNA repair pathways, plasmid or episomal expression systems, and recombinant viral delivery of radioprotective genes. These approaches are presented as future research directions rather than current standards of care because broad enhancement of DNA repair may protect tumor cells, preserve genomically unstable cells, or increase unintended long-term risk. A practical translational framework is proposed in which tumor sequencing, germline risk assessment, dosimetry, clinical history, toxicity monitoring, and ethical safeguards are combined into multivariable decision models. The paper concludes that radiogenomics may eventually improve the therapeutic ratio of radiotherapy, but only if candidate biomarkers and gene-modulation strategies are prospectively validated, tissue-specific, equitable, and integrated with established clinical judgment.

1. INTRODUCTION

Radiation therapy is one of the most widely used local treatment modalities in oncology. It is used as

definitive therapy, adjuvant therapy, neoadjuvant therapy, palliation, and organ-preserving treatment across malignancies such as breast, prostate, lung, head and neck, gynecologic, hematologic, and central nervous system cancers. The therapeutic principle is to deposit ionizing energy within tumor tissue in a manner that creates lethal DNA damage, cell-cycle arrest, mitotic catastrophe, apoptosis, or loss of reproductive capacity. Modern treatment planning uses image guidance, intensity modulation, stereotactic delivery, particle therapy, brachytherapy, and increasingly adaptive planning to improve dose conformity and spare organs at risk [1, 2].

Despite these advances, radiation therapy remains limited by normal-tissue toxicity. Acute effects such as dermatitis, mucositis, pneumonitis, cystitis, enteritis, fatigue, marrow suppression, and localized inflammation can interrupt treatment or reduce quality of life. Late effects such as fibrosis, vascular injury, infertility, lymphedema, neurocognitive change, organ dysfunction, and secondary malignancy may arise months to years after treatment. The occurrence and severity of these toxicities vary considerably even among patients receiving similar doses to similar tissues. This variability suggests that clinical and dosimetric factors alone do not fully explain patient radiosensitivity [3, 4].

Genetic profiling and radiogenomics offer a possible means of explaining some of this variability. Radiogenomics, in this context, refers to the study of inherited and tumor-acquired genomic features that influence radiation response, normal-tissue toxicity, and treatment outcome. Germline variants may alter DNA damage recognition, repair efficiency, oxidative stress response, inflammatory signaling, vascular response, immune modulation, or tissue regeneration. Tumor-specific alterations may influence whether malignant cells are radiosensitive or radioresistant. A more holistic approach to radiation oncology therefore requires separating two related but distinct goals: improving tumor control and protecting normal tissue.

The objective of this article is to propose a conceptual framework for integrating genetic and radiogenomic information into radiotherapy planning. Rather than suggesting that genetic profiling should immediately replace current clinical standards, this paper argues that genomic information may eventually function as a risk-stratification tool when combined with tumor histology, stage, imaging, dosimetry, prior therapy, baseline organ function, comorbidities, and longitudinal toxicity monitoring. The revised focus is therefore translational and forward-looking: how might genetic profiling, validated biomarkers, and carefully controlled gene-modulation strategies one day reduce normal-tissue toxicity while preserving or improving tumor control?

2. METHODOLOGICAL APPROACH

This manuscript is structured as a conceptual narrative review and translational framework rather than an original clinical trial or systematic review. The analysis synthesizes peer-reviewed literature in radiation biology, DNA damage response, radiogenomics, normal-tissue toxicity, and investigational radioprotective gene therapy. The framework is organized around four questions. First, which biologic pathways plausibly influence radiosensitivity? Second, how could genomic information be applied at specific points in the radiation treatment timeline? Third, which investigational methods might protect healthy tissue from radiation injury without protecting tumor cells? Fourth, what confounders, ethical concerns, and evidence gaps must be addressed before clinical implementation?

The proposed framework intentionally avoids single-gene determinism. Radiosensitivity is multifactorial and depends on interactions among inherited genotype, tumor genotype, epigenetic regulation, tissue oxygenation, vascularity, immune microenvironment, inflammatory state, mitochondrial function, microbiome, dose distribution, fractionation, prior systemic therapy, and host factors. Therefore, the methodology emphasizes pathway-level interpretation and multivariable risk modeling rather than isolated marker interpretation.

The clinical application considered here includes three phases: pretreatment assessment, treatment planning and delivery, and post-treatment surveillance. Pretreatment assessment would include tumor sequencing when clinically indicated, germline risk assessment when justified by patient history or high-risk treatment fields, baseline organ function testing, and evaluation of prior therapies. Treatment planning would integrate

this information with dose constraints and organ-at-risk priorities. Post-treatment surveillance would assess acute and late toxicity using standardized toxicity scales, patient-reported outcomes, imaging, and biomarker monitoring.

3. BIOLOGICAL BASIS OF RADIATION-INDUCED DAMAGE

Ionizing radiation damages cells through direct and indirect mechanisms. Direct action occurs when radiation deposits energy in DNA or associated molecular structures, producing base damage, single-strand breaks, double-strand breaks, crosslinks, or clustered lesions. Indirect action occurs primarily through radiolysis of water, generating reactive oxygen species such as hydroxyl radicals, superoxide, and hydrogen peroxide. These reactive species damage nucleic acids, proteins, lipids, mitochondria, and cell membranes. Because human tissue is water-rich, indirect oxidative injury is a major component of low-linear-energy-transfer radiation injury.

Double-strand breaks are especially important because they are difficult to repair accurately. Cells respond through damage-sensing kinases, checkpoint activation, chromatin remodeling, apoptosis pathways, homologous recombination, non-homologous end joining, and alternative end-joining mechanisms. Tumors with impaired homologous recombination or defective checkpoint control may be more sensitive to radiation, whereas tumors with robust repair, hypoxia, altered apoptosis, or enhanced antioxidant capacity may be more resistant. Normal tissues may also vary in their ability to repair sublethal injury between fractions, which is one reason fractionated radiotherapy can spare normal tissue while accumulating lethal injury in tumor cells.

Linear energy transfer affects the pattern of injury. Low-LET modalities such as X-rays and gamma rays deposit energy more sparsely and produce a larger indirect oxidative component. High-LET modalities such as alpha particles, neutrons, and heavy ions create dense ionization tracks and more complex clustered damage that is less easily repaired. Proton therapy has a distinct physical dose distribution because of the Bragg peak, allowing dose to stop more precisely at depth, but biological effectiveness and tissue response still depend on multiple patient- and tumor-specific variables.

4. RADIOGENOMIC MARKERS AND PATHWAY-LEVEL INTERPRETATION

Several gene families and molecular pathways are repeatedly discussed in relation to radiation response. These include DNA damage recognition and repair genes such as ATM, ATR, BRCA1, BRCA2, XRCC1, ERCC1, ERCC2, RAD51, TP53, and genes involved in non-homologous end joining. They also include oxidative stress and mitochondrial defense pathways such as SOD2, GPX1, glutathione metabolism, and NRF2-regulated transcription. Inflammatory and fibrotic pathways such as TGF-beta, IL-6, TNF-alpha, and NF-kappaB may influence late tissue injury, while hypoxia-associated pathways such as HIF-1alpha and VEGF signaling influence tumor resistance and vascular response.

Current radiogenomic studies support the idea that inherited genetic variation contributes to radiation toxicity, but the effect is usually complex and polygenic. For example, the Radiogenomics Consortium has reported genome-wide association and meta-analysis data suggesting that common variants contribute to late toxicity after prostate radiotherapy and acute toxicity across prostate, breast, lung, and head and neck cancer cohorts [5, 6]. A large meta-GWAS of acute toxicity across 19 cohorts and 12,042 patients estimated shared single-nucleotide-variant-based heritability across cancer types and also emphasized tissue-specific mechanisms [6]. Head and neck cancer GWAS data have identified a replicated locus associated with mucositis, illustrating that toxicity may be organ- and endpoint-specific [7].

These findings are important but should be interpreted cautiously. Many variants have modest effect sizes, do not replicate consistently across populations, and may depend on treatment site, dose distribution, ancestry, toxicity endpoint, and follow-up duration. Therefore, the strongest near-term use of radiogenomics may be incorporation into multivariable risk models rather than single-marker treatment decisions. In clinical practice, a biomarker should not be considered actionable unless it improves prediction beyond established clinical and dosimetric factors and has been validated prospectively.

5. CONCEPTUAL CLINICAL TRANSLATION FRAMEWORK

A clinically realistic translation pathway would begin before radiation treatment. Patients could be stratified into standard-risk, elevated-risk, and very-high-risk groups using a combination of clinical history, tumor genomics, germline findings, baseline organ function, prior treatment exposure, and planned organ-at-risk dose. Very-high-risk patients might include those with known cancer predisposition syndromes, DNA repair syndromes, prior overlapping radiation fields, pediatric patients, or patients receiving treatment near highly sensitive organs.

In treatment planning, genetic risk would not independently determine dose. Instead, it could influence the weighting of existing planning decisions. For example, a patient with elevated predicted risk of radiation pneumonitis might receive more conservative lung dose constraints, consideration of proton therapy, adaptive planning, avoidance of concurrent pulmonary-toxic systemic therapy when feasible, and intensified post-treatment surveillance [8]. A patient with a tumor showing homologous recombination deficiency might be expected to have increased radiosensitivity, but this would need to be balanced against normal-tissue risk, systemic therapy interactions, and clinical trial evidence. A patient with Li-Fraumeni syndrome or another high-risk germline predisposition may require careful consideration of radiation avoidance or alternative modalities when oncologically appropriate.

Post-treatment, radiogenomic information could support surveillance intensity. Patients predicted to be at elevated risk of fibrosis, pneumonitis, mucositis, secondary malignancy, or organ dysfunction could undergo more frequent toxicity assessment, earlier rehabilitation referral, biomarker monitoring, and patient-reported outcome tracking. This surveillance concept can incorporate radiogenomic, toxicity-mechanism, and radiomic data [9-11]. This approach would treat genomics as one layer of a survivorship and toxicity-monitoring strategy rather than a stand-alone determinant of care.

6. INVESTIGATIONAL STRATEGIES FOR NORMAL-TISSUE RADIOPROTECTION

A more speculative but important future direction involves targeted protection of normal tissue during radiation therapy. These strategies must be approached cautiously because the central purpose of radiotherapy is to create lethal damage in tumor cells. Any intervention that broadly increases DNA repair, antioxidant activity, or cell survival could potentially protect tumor cells or allow damaged cells to survive with mutations. Therefore, a rational investigational strategy must be tissue-specific, tumor-sparing, transient, and measurable.

One theoretical approach is transient upregulation of DNA repair and stress-response pathways in normal tissue. Candidate mechanisms could include support of base excision repair, single-strand break repair, DNA ligase function, polymerase-mediated repair synthesis, homologous recombination, non-homologous end joining, mitochondrial antioxidant defense, and anti-inflammatory resolution. The intended application would be during fractionated therapy, when normal tissue repair between fractions is clinically beneficial. However, this approach is only plausible if delivery can be restricted to normal tissue and if expression is temporary. Persistent or systemic upregulation of repair pathways could reduce tumor control or increase survival of genomically unstable cells.

A second theoretical approach involves plasmid or episomal delivery of radioprotective gene expression factors. Episomal systems may provide temporary expression without permanent genomic integration. A possible use case would be localized delivery to normal tissues at high risk for injury, such as oral mucosa during head and neck radiotherapy, lung tissue during thoracic radiotherapy, bowel mucosa during pelvic radiotherapy, or salivary glands during treatment of oropharyngeal tumors. MnSOD/SOD2-based plasmid-liposome strategies and SOD mimetics have been investigated as radioprotectors and radiomitigators, providing a useful example of the type of pathway that could be explored further [12, 13].

A third approach involves recombinant viral vectors designed to introduce protective genes into selected normal tissues. Radiation-inducible gene-switch concepts also illustrate gene delivery approaches relevant to this area [14]. Viral delivery may improve transduction efficiency compared with plasmid systems but raises greater safety concerns, including immune response, insertional mutagenesis for integrating vectors, variable

expression duration, and difficulty preventing tumor uptake. Viral approaches may be most appropriate only for exceptionally high-risk contexts or tightly controlled clinical trials after rigorous preclinical validation.

These investigational approaches are not current standards of care. Their value lies in identifying a future research agenda: can normal tissue be protected without protecting tumor cells, and can this be done in a time-limited and organ-specific manner? Avasopasem manganese provides another relevant radioprotection example [15].

7. PROPOSED APPLICATION MATRIX

Table 1 summarizes how genetic and gene-modulation concepts could theoretically be applied across the radiation treatment timeline. The table is intended as a conceptual research framework, not as a clinical protocol.

Table 1. Conceptual framework for genetic and gene-modulation approaches in radiation therapy.

Approach	Treatment stage	How it may be applied	Scientific rationale	Expected result	Primary risks/monitoring
Radiogenomic risk stratification	Pretreatment	Combine germline and tumor markers with clinical history, organ function, imaging, and planned dose.	Inherited and tumor-specific variation can influence DNA repair, inflammation, hypoxia, and oxidative stress.	Improved risk prediction and more individualized planning.	Risk of overinterpretation; require validation, informed consent, and prospective toxicity tracking.
Dose/schema adaptation	Planning	Modify dose constraints, modality, fractionation, or surveillance intensity for high-risk patients.	Toxicity depends on dose distribution plus patient-specific susceptibility.	Reduced avoidable toxicity while preserving tumor control.	Must avoid undertreating curable tumors; monitor local control and toxicity endpoints.
Transient DNA repair support	Before/during fractions	Investigational tissue-specific upregulation of repair or stress-response genes in normal tissue.	Normal tissue recovery between fractions contributes to therapeutic ratio.	Potential reduction in acute injury and late fibrosis.	Could protect tumor cells or preserve damaged cells; require tissue targeting and limited expression.
Plasmid/episomal radioprotection	Localized pretreatment or concurrent	Local delivery of temporary radioprotective	Localized antioxidant or mitochondrial	Reduced mucositis, pneumonitis,	Variable expression, immune reaction,

	therapy	expression constructs to mucosa, lung, bowel, or salivary glands.	protection may reduce ROS-mediated injury.	xerostomia, or epithelial injury in high-risk fields.	off-target delivery; monitor toxicity, tumor response, and long-term outcomes.
Recombinant viral gene delivery	Future early-phase trials	Tissue-specific or inducible vector systems delivering radioprotective genes.	Higher delivery efficiency may permit organ-specific radioprotection.	Potentially broader protection for patients at exceptional toxicity risk.	Immune response, insertional risk, tumor uptake; require preclinical validation and regulatory oversight.

8. CONFOUNDERS AND LIMITATIONS

The major limitation of genetic personalization in radiation therapy is that radiosensitivity is not reducible to a single gene or pathway. Tumor type is a major confounder because the same alteration may have different implications in breast, prostate, lung, head and neck, or central nervous system cancers. Stage, tumor volume, histology, hypoxia, vascularity, immune infiltration, and tumor microenvironment may alter response independently of inherited genotype. A marker associated with normal-tissue toxicity in one treatment site may not predict toxicity in another.

Prior and concurrent therapies are also critical confounders. Chemotherapy, immunotherapy, targeted agents, surgery, hormonal therapy, smoking history, diabetes, autoimmune disease, infection, nutritional status, age, sex, body habitus, baseline organ function, and prior radiation exposure may influence toxicity risk. Dosimetric factors are equally important. Total dose, dose per fraction, treatment modality, motion management, image guidance, organ-at-risk constraints, and actual delivered dose must be interpreted alongside genomic data.

There are also methodological limitations in the current evidence. Many studies are retrospective, use heterogeneous toxicity endpoints, include limited ancestral diversity, and may lack long-term follow-up. Some associations are statistically suggestive but not genome-wide significant. Others are biologically plausible but have not been functionally validated. Rare high-penetrance variants may be clinically important but are not always captured by common-variant genome-wide association studies. Polygenic risk scores may improve prediction, but they can also perform poorly when applied to populations not represented in the discovery datasets.

Potential solutions include prospective registries, harmonized toxicity scoring, standardized patient-reported outcomes, serial imaging, longitudinal biospecimen collection, inclusion of diverse populations, and integration of genomic, radiomic, dosimetric, immune, and clinical variables. Clinical trials should evaluate whether genetic information changes treatment decisions and whether those changes improve meaningful endpoints such as tumor control, toxicity, survival, and quality of life.

9. ETHICAL, LOGISTICAL, AND CLINICAL CONSIDERATIONS

Genetic-based radiotherapy planning raises ethical and logistical concerns. Patients must understand what type of genetic information is being collected, whether testing is tumor-only or germline, whether incidental hereditary findings may be discovered, and who will have access to the results. Genetic discrimination, insurance implications, privacy protection, and unequal access to advanced genomic testing must be addressed before widespread implementation.

A further ethical concern is stratified access to treatment. If advanced radiogenomic testing is available

only in major academic centers or to patients with greater resources, precision radiation oncology could widen existing disparities. Conversely, if validated and implemented equitably, genomic risk assessment could help identify patients who need safer treatment approaches or more intensive follow-up. Implementation should therefore include genetic counseling pathways, transparent consent, data security, equitable testing access, and clear clinical thresholds for action.

Clinicians should also avoid therapeutic overconfidence. A genomic marker may support a hypothesis about risk, but it should not override established oncologic principles without validation. The decision to de-escalate, intensify, alter modality, or add investigational radioprotective therapy should be evidence-based and ideally conducted within clinical trials or registries.

10. FORWARD-LOOKING CONCLUSIONS

The future of radiation therapy will likely involve a more holistic model of precision radiation oncology. Genetic profiling may become one component of treatment planning, but its greatest value will come from integration with imaging, dosimetry, tumor biology, immune response, clinical history, and survivorship monitoring. The goal is not simply to increase DNA repair or reduce radiation injury in all cells. The goal is to selectively protect normal tissue while maintaining or improving tumor control.

Radiogenomic studies increasingly show that inherited variation contributes to radiation toxicity, but current evidence is not yet sufficient for broad routine dose modification based on single markers. The most realistic near-term path is development of validated multivariable models that combine clinical, dosimetric, genomic, radiomic, and patient-reported data. In parallel, investigational gene-modulation strategies such as transient DNA repair support, plasmid or episomal radioprotection, and recombinant viral delivery should be studied carefully as future adjuncts, with strict attention to tissue specificity, tumor sparing, expression duration, immune effects, and long-term stochastic risk.

If implemented responsibly, genetic profiling could help radiation oncologists identify patients at elevated toxicity risk, tailor surveillance, refine planning choices, and eventually guide new radioprotective interventions. However, translation will require prospective validation, ethical safeguards, diverse patient representation, careful regulation, and a continued commitment to balancing efficacy with patient safety.

CONFLICTS OF INTEREST

The author declares no conflicts of interest regarding the publication of this paper.

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