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Radiogenomics: Linking Imaging Biomarkers and Cancer Genomics – A Comprehensive Review

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***Abstract*-- Radiogenomics integrates quantitative imaging (radiomics) and tumor genomic data to noninvasively infer molecular characteristics of cancers. This approach promises to overcome biopsy limitations and capture spatial tumor heterogeneity. We review recent advances (2018–2026) in radiogenomics for major cancers, summarizing methodologies, key findings, and clinical implications.**

Methods we surveyed the literature on oncologic radiogenomics, focusing on studies correlating imaging-derived features (from CT, MRI, PET, etc.) with genetic alterations or expression profiles. We considered data types (imaging modalities, genomic assays), feature extraction (handcrafted radiomics, deep features), and analytical methods (statistical and machine-learning models) used. We synthesized results by cancer type (lung, breast, glioma, prostate, etc.), noting specific gene–image associations, effect sizes, and reproducibility.

The Result is Numerous studies have identified imaging signatures predictive of driver mutations or molecular subtypes. For example, CT and PET radiomics can predict EGFR, KRAS or ALK mutations in lung cancer (often AUC ≈0.8); MRI features correlate with ER/PR/HER2 status and Oncotype scores in breast cancer (AUC ≈0.7–0.9). In gliomas, MRI-based radiomics distinguishes IDH-mutant vs. wild-type tumors and identifies EGFR, PTEN, NF1, TP53 mutations. For prostate cancer, quantitative MRI features (e.g. ADC, perfusion) associate with PTEN loss and ERG rearrangement. In each cancer type, imaging–genomic associations are often validated in independent cohorts, with effect sizes (Cohen's D) generally larger when considering tumors with single driver alterations. However, small sample sizes, retrospective designs and heterogeneous methods limit reproducibility.

Conclusion of this study is Radiogenomics shows promise for noninvasive tumor genotyping and prognostication, leveraging routine imaging to predict molecular features. To realize clinical utility, studies must use standardized imaging/radiomic protocols, larger multi-center cohorts, and robust validation. Ethical and regulatory frameworks for handling combined imaging and genomic data (privacy, informed consent, data sharing) are essential. We provide recommendations on best practices (study design, validation, sharing) to accelerate reliable radiogenomic biomarker discovery and translation into precision oncology.

I. Introduction

Radiogenomics is an emerging discipline that links high-throughput imaging features ("radiomics") with tumor genomic data to improve cancer characterization. Radiomics computes quantitative descriptors (shape, texture, intensity) from medical images, enabling objective capture of tumor phenotype beyond visual inspection. Genomic profiling (mutations, gene expression, methylation) reveals the molecular basis of oncogenesis, but typically requires invasive tissue sampling and may miss spatial heterogeneity. Radiogenomics hypothesizes that tumor genetics are reflected in imaging phenotypes, so that image-derived biomarkers can noninvasively predict molecular features. This integration could guide targeted therapy, stratify patients, and monitor disease without repeated biopsies. Figure 1 (see recommended workflow below) outlines the typical pipeline: collecting imaging and genomic data, extracting features from images and sequencing data, and applying statistical/machine-learning models to associate imaging biomarkers with gene alterations.

The field has grown rapidly. Recent reviews highlight key applications in oncology (breast, lung, brain, prostate, etc.) and describe general approaches. Here we update and expand on these reviews by focusing on work from 2018–2026. We examine the types of imaging and genomic data used, feature extraction and modeling methods, and the robustness of reported associations. We compare findings across cancer types, noting which imaging features correlate with specific gene alterations, and synthesize effect sizes and reproducibility. We conclude with a discussion of biological interpretations, clinical potentials, limitations (technical and ethical), and recommendations for future radiogenomic research.

II. Literature Review

Imaging Modalities and Radiomic Features

Radiogenomic studies use diverse imaging modalities – primarily computed tomography (CT), magnetic resonance imaging (MRI), and nuclear medicine (PET/SPECT), with emerging use of ultrasound or digital pathology images.

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Each modality highlights different tissue properties (e.g. CT attenuation, MRI relaxation/ perfusion, PET tracer uptake) providing complementary information. Radiomic features are extracted from segmented tumor regions (and often peritumoral areas) at multiple scales. Handcrafted features quantify tumor shape, first-order intensity (histograms), and texture (gray-level co-occurrence, run-length, etc.). Deep learning (DL) approaches are increasingly used to derive features automatically from images, often outperforming or augmenting handcrafted radiomics. Some studies incorporate "prior-based" radiomics, embedding known tumor biology (e.g. anatomical connectomics or functional regions) into features.

Genomic and Multi-omics Assays

"Genomics" in radiogenomics includes DNA mutations, copy number, gene expression, methylation, proteomics, etc. Tissue biopsy (surgical or needle) is the traditional source, analyzed by high-throughput methods (targeted NGS panels, whole-exome/genome sequencing, RNA-seq, microarrays). Emerging spatial genomics adds location-specific gene maps, potentially aligning with imaging heterogeneity. Liquid biopsies (circulating tumor DNA, CTCs, exosomes) offer a noninvasive way to obtain tumor genomic material; although promising, integration with imaging data is nascent. Proteomic and epigenomic profiling (IHC, mass spec, methylation arrays) are also used to define molecular subtypes. In practice, radiogenomic studies most often link imaging to key genomic markers (e.g. hotspot mutations, transcriptional subtypes) rather than exhaustive multi-omics, though multi-omics data (transcriptomics, metabolomics) are recognized as valuable for deeper insights.

Analytical Approaches: Statistics and Machine Learning

The core analysis in radiogenomics is to associate imaging features with molecular variables. Early studies used univariate or multivariate statistics (correlations, ANOVA, regression) to find significant image–gene links. Contemporary work relies heavily on machine-learning (ML) models. Common pipelines include feature selection (LASSO, mRMR, random forest ranking) followed by supervised classifiers (logistic regression, SVM, random forest, XGBoost) to predict mutation status or gene expression signature. Deep learning models (CNNs) enable end-to-end learning from images to gene labels without handcrafting features. Model performance is evaluated by metrics such as area under the ROC curve (AUC), accuracy, sensitivity and specificity.

Cross-validation (often k-fold or train/test split) and independent cohorts are used to test generalizability. Radiogenomic analyses may use simple correlation (e.g. Pearson) for feature–gene associations or build integrative models combining both data types. Some advanced techniques (e.g. canonical correlation, graph neural networks) have been proposed to fuse heterogeneous data, but most studies remain in a supervised classification/regression framework.

Recent reviews summarize that radiogenomic studies show broad methodological variety but share common steps: image preprocessing (normalization, registration, segmentation), feature extraction, model building and validation. Figure 2 (below) illustrates the evidence landscape by cancer type.

Key Findings by Cancer Type (2018–2026)

Lung Cancer: A large literature focuses on lung adenocarcinoma and non-small-cell lung cancer (NSCLC). Many CT radiomic studies report significant differences between EGFR-mutant and wild-type tumors. Multivariate models combining multiple features typically achieve AUCs in the range ~0.75–0.90 for EGFR prediction. KRAS and ALK mutations have also been predicted, though often with lower accuracy. Some studies use dual-time-point CT or PET/CT radiomics, and report that perfusion or dual-energy features improve mutation prediction. For example, a recent review noted that DL models using preoperative CT scans achieved ~80–87% AUC for predicting EGFR status. PD-L1 expression and tumor mutational burden (TMB) have been linked to imaging as well, enabling immunotherapy decision support in NSCLC (reviewed in He et al., 2024). Models are often validated on external cohorts or TCGA-derived data, with reproducible performance reported.

Breast Cancer: Multiparametric MRI has been widely used to radiogenomically predict receptor status and genomic scores. Radiomic features from dynamic contrast-enhanced (DCE) MRI have been shown to predict ER, PR, HER2 status and Ki-67 index. For HER2, DL radiogenomic models achieved AUC≈0.85 in one study. Luminal vs. triple-negative subtypes, and Oncotype/EndoPredict recurrence scores, have also been predicted by MRI features in combination with genomic markers. For instance, a LightGBM radiogenomic model using contrast MRI and variant allele frequencies reached AUC~0.87 for pCR prediction in TNBC, significantly outperforming imaging alone. Overall, breast studies demonstrate that combining imaging with gene-expression signatures yields stronger prognostic and predictive models than either alone. The *Convergence of Radiology and Genomics* review

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(Demetriou et al., 2024) and others emphasize integration of multi-omics (gene mutations, mRNA panels, immune markers) with MR radiomics in breast cancer.

Gliomas and Brain Tumors: Radiogenomics is well established in glioma. Numerous studies report MRI radiomic signatures of IDH1 mutation, 1p/19q co-deletion, and MGMT methylation status in gliomas, with pooled AUCs often in the 0.8 range (meta-analyses). For GBM (IDH-wildtype glioblastoma), a recent multi-cohort study (Kazerooni et al., 2024) identified distinctive MRI signatures for key driver mutations (EGFR, PTEN, NF1, TP53). In that study, EGFR-mutant tumors showed higher perfusion (increased rCBV) and vascular proliferation, whereas TP53-mutant tumors exhibited increased vascular permeability (higher percent signal recovery). The deep-learning models achieved high reproducibility: using conventional MRI features, predictive models for each gene had robust AUCs even in independent cohorts. Importantly, effect sizes (Cohen's D) for discriminating mutant vs. wild-type were larger in "exclusive" cases (tumors with only one driver mutation) than in mixed-mutation tumors, suggesting imaging signals are clearest when genomic complexity is lower. Overall, radiomic features reflecting cellularity, edema and angiogenesis have been linked to glioma molecular subtypes, aiding noninvasive diagnosis and prognostication.

Prostate Cancer: Multiparametric MRI (mpMRI) radiomics has been used to infer molecular risk markers in prostate cancer. One study found that MRI-visible lesions were significantly more likely to harbor PTEN loss or ERG gene rearrangements than MRI-invisible lesions. In another analysis, tumors with PTEN inactivation had lower DWI-ADC values on mpMRI. Radiomic features from T2-weighted and diffusion images have correlated with Decipher and cell-cycle gene scores (proliferation signatures). Machine learning models integrating radiomics with gene panels (e.g. Prolaris, Decipher) achieved moderate AUCs (~0.7–0.8) for predicting high-grade disease or lymph node involvement. For example, Hectors et al. (2019) reported AUC≈0.84 for predicting a high-risk Decipher score from multiparametric MRI features. These studies are early and limited by small cohorts, but they suggest MRI biomarkers (e.g. volumetric growth rates, gland texture) correlate with aggressive genotypes (PTEN, TP53 alterations) in prostate cancer. Comprehensive reviews (Conti et al., 2021) highlight PTEN as the most studied gene – fast MRI contrast uptake and heterogeneous T2 signal both tend to indicate PTEN loss.

Other Cancers: Radiogenomics has also been explored in colorectal, liver, kidney and other cancers. For instance, CT radiomics of colorectal liver metastases predict KRAS/BRAF status and CD73 expression, while PET radiomics correlate with mismatch repair deficiency. In renal cell carcinoma and thyroid cancer, imaging features have been linked to VHL, PBRM1 mutations or immunohistochemical markers (cytokeratins). While oncology is the main focus, non-oncologic disease (e.g. stroke genetics) is emerging but outside our scope.

III. Methods In Radiogenomic Studies

Data Collection and Preprocessing

Radiogenomic studies typically acquire standard-of-care imaging and genomic data from the same patients. Imaging modalities include CT (often contrast-enhanced for thoracoabdominal cancers), multiparametric MRI (T1/T2, diffusion, perfusion) for CNS and prostate, and PET/CT (FDG or specific tracers). Preprocessing steps often involve tumor segmentation (manual, semi-automated or automated) to define regions of interest. Images may be normalized (intensity, voxel size) and, if multi-site, harmonized with algorithms to reduce scanner variance. Coregistration across sequences (e.g. aligning DWI to anatomical MRI) allows fusion of features. On the genomics side, tumor tissues are sequenced (targeted panels or whole exome) or assayed for expression signatures. Data quality control (checking for motion artifacts, sequencing coverage) is critical.

Feature Extraction

From the segmented images, radiomic features are computed: **shape** descriptors (volume, surface, irregularity), **first-order** intensity metrics (mean, variance, skewness of voxel intensities), and **texture** features (e.g. gray-level co-occurrence matrix, wavelet transforms). Features may be drawn from the tumor core and from peritumoral regions (to capture invasion patterns). Handcrafted radiomic feature sets follow standardized definitions (e.g. as in Pyradiomics or IBSI standards). In addition, deep learning approaches (CNNs or autoencoders) are used to extract features or directly classify images with no manual selection. For example, convolutional neural network "signatures" have been learned for gene prediction in glioblastoma. Priors such as brain connectomics or organ architecture have also been used to craft specialized features that embed known biology.

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Genomic and Molecular Data Processing

Tumor DNA is analyzed via next-generation sequencing. Key genetic variables include driver **mutations** (e.g. EGFR, TP53), **chromosomal alterations** (1p/19q codeletion in glioma), **microsatellite instability/TMB**, and **gene expression subtypes** (e.g. intrinsic breast subtypes). Clinical-grade assays (e.g. Oncotype DX in breast, Decipher in prostate) produce genomic risk scores, which have been correlated with imaging. Often genomic data is binarized (mutant vs wildtype) or discretized (high vs low expression). Some studies build polygenic risk scores (PRS) or pathway scores to reduce dimensionality. Liquid biopsy data (ctDNA variant allele frequencies) have been used in only a few proof-of-concept studies, combining temporal imaging changes with ctDNA levels for monitoring (beyond scope of most reviewed papers).

Statistical and Machine Learning Models

After preprocessing and feature extraction, radiogenomic associations are analyzed. Common steps include: (1) **Feature selection** – reducing high-dimensional radiomic data via methods like LASSO regression, recursive feature elimination, or maximum-relevance-minimum-redundancy (mRMR) to avoid overfitting. (2) **Model training** – supervised classifiers/regressors (logistic regression, support vector machines, random forests, gradient boosting) are trained to predict a genomic endpoint (e.g. mutation status, expression level) from imaging features. (3) **Deep learning** – some studies employ convolutional or fully-connected neural networks to learn complex imaging–genomic mappings end-to-end. (4) **Validation** – performance is assessed by cross-validation or on held-out test sets; metrics include area under the ROC curve (AUC), accuracy, and precision/recall. Many recent papers use independent external cohorts (e.g. TCGA-NSCLC data or multi-center MRI datasets) for validation. Radiogenomic models are also compared to "null" models (imaging or genomics alone) to demonstrate added value of integration.

In advanced analysis, imaging and genomic data may be fused (concatenated into one input vector) to jointly predict clinical outcomes (e.g. survival, treatment response) or to cluster tumors into integrated subtypes. Statistical correlation analyses (Spearman/Pearson) are also performed post-hoc to identify which radiomic features are most associated with specific genes or pathways. Overall, statistical and ML approaches in radiogenomics mirror those in radiomics and computational genomics, adapted to the paired data context.

IV. Results Synthesis

Imaging–Genomic Associations Across Cancer Types

- *Lung (NSCLC):* Multiple studies report that CT radiomic features predict driver mutations. For EGFR, consensus image features include ground-glass components and texture heterogeneity. In large cohorts, multivariate CT models attained AUCs ~0.8 for EGFR mutation prediction. KRAS and ALK have been less consistently predicted, with some studies showing modest AUC (<0.75). Radiogenomic models also predict PD-L1 expression and tumor mutational burden, potentially guiding immunotherapy. Cross-modality (CT+PET) radiomics has shown incremental improvement. Notably, Shariaty et al. (2025) review the NSCLC radiogenomic pipeline (image acquisition→segmentation→feature extraction→modeling), reflecting mature workflows with validated case studies.
- *Breast:* MRI radiomics can stratify receptor-defined subtypes. Recent work shows MRI texture and kinetic features can identify HER2-positive vs negative tumors, and estimate Ki-67 or Oncotype scores. For example, a study reported AUC ≈0.85 for predicting pathologic response in HER2+ patients using MRI radiomic features combined with genomic data. In triple-negative breast cancer, a LightGBM model incorporating dynamic contrast MRI features and circulating tumor DNA variant allele frequencies achieved AUC ~0.87, significantly outperforming radiomics alone. Models for distinguishing Luminal A vs B vs TNBC subtypes have reached AUC>0.8 in some reports. These high accuracies reflect strong imaging–genomic links in breast cancer, but external validation remains limited to a few studies.
- *Gliomas:* Numerous radiogenomic studies of glioma have yielded reproducible biomarkers. Meta-analyses report pooled AUC ~0.84 for MRI radiomics to predict IDH mutation status across ~3000 patients. Individual studies find that ADC texture and perfusion metrics are highest in IDH-mutant gliomas, reflecting their altered metabolism. For 1p/19q codeletion, radiomic signatures (including shape and margin irregularity) achieved AUC ~0.8. In GBM specifically, the recent study by Kazerooni et al. (2024) demonstrated highly significant imaging signatures: for example, EGFR-mutant GBMs showed elevated relative cerebral blood volume (rCBV) in enhancing tumor ($p<0.001$) and markedly higher perfusion than EGFR wildtype.

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TP53-mutant GBMs showed increased MRI signal recovery (permeability marker) vs non-mutants. Importantly, when tested on an independent cohort, their DL and SVM classifiers maintained high AUCs (often >0.80). The study quantified effect sizes (Cohen's D): for instance, exclusive EGFR-mutant vs wild-type tumors showed D≈2.3, versus D≈0.6 for co-occurring multiple-mutation cases, indicating robust separability in cleaner samples.

- *Prostate:* Evidence is sparser but growing. A multi-institutional analysis (Eineluoto et al., 2020) found that MRI-visible prostate tumors had a much higher rate of PTEN loss (43.3%) than MRI-invisible ones (17.2%), implying that aggressive genotypes tend to be radiologically apparent. Another study reported that tumors with low PTEN expression had significantly lower ADC values (p=0.006). These findings suggest that ADC and contrast-enhancement kinetics may serve as surrogates for tumor suppressor loss. Radiogenomic models incorporating MRI radiomic features and gene-expression risk scores (e.g. Decipher or Prolaris) have achieved moderate performance (AUC≈0.75–0.85) in predicting adverse pathology or high-grade disease. However, sample sizes are small (typically N<200), and external validation is lacking.
- *Strength of Evidence and Reproducibility:* In general, radiogenomic associations exhibit moderate-to-large effect sizes when properly validated. As noted, exclusive gene-mutation cases yield stronger imaging correlations. Most studies report AUCs in the 0.7–0.9 range for key biomarkers, which is clinically promising. A few meta-analyses (glioma IDH, lung EGFR) confirm these accuracies across multiple cohorts. Importantly, reproducibility has improved: many recent works include independent test sets or multi-site data. For example, the glioblastoma study partitioned data into training (N≈270) and validation (N≈90) cohorts, with no significant drop in performance between them. He et al. (2024) also stress that radiogenomic models can be robust if standardized pipelines and feature harmonization are applied. Nevertheless, many reported associations still await multicenter confirmation, and "publication bias" toward positive findings is a concern.

V. Discussion

Biological Interpretation: Radiogenomic findings often align with known tumor biology. For instance, EGFR activation promotes angiogenesis, which explains the higher perfusion imaging signature in EGFR-mutant GBM. TP53 mutation tends to disrupt vascular integrity, consistent with its MRI permeability signature. Similarly, MGMT methylation in glioma (linked to treatment response) has been associated with more homogeneous contrast enhancement. In breast cancer, differences in vascular permeability and cellularity across subtypes lead to distinguishable DCE-MRI and diffusion features. In lung, tumors with lepidic (non-solid) growth and EGFR mutations often appear as ground-glass or subsolid nodules. These correlations suggest that imaging "phenotypes" (e.g. texture heterogeneity, angiogenic features) can serve as surrogates for molecular pathways. Radiogenomics thus provides insight into tumor ecology: it links macroscopic imaging (e.g. edema, necrosis) to microscopic genomics (e.g. hypoxia genes, proliferation indices).

Clinical Utility: The ultimate goal is to use radiogenomic biomarkers in patient care. Noninvasive prediction of actionable mutations could guide therapy when biopsy is risky or insufficient. For example, identifying EGFR-mutant lung cancer without biopsy could accelerate targeted treatment. In glioma, preoperative IDH and MGMT status predictions could refine surgical planning and adjuvant therapy. Radiogenomic risk scores might supplement existing nomograms for recurrence. The integration of imaging and genomics can also improve prognostic models: studies show that combined radiogenomic signatures outperform either modality alone for survival prediction. Moreover, as imaging is repeatable over time, radiogenomics enables monitoring of tumor evolution (e.g. emergence of resistant clones) without repeated invasive sampling.

Limitations: Despite progress, radiogenomics faces significant hurdles. **Data heterogeneity:** Radiomic features are sensitive to imaging protocols, reconstruction parameters, and segmentation variability. Lack of standardization (varying slice thickness, scanner models) can confound multi-center studies. The IBSI and other consortia are working on standard definitions, but many studies still use custom pipelines, making cross-comparison hard. **Small cohorts and overfitting:** Many papers have N<100 patients, risking spurious associations. Retrospective designs and absence of external validation limit generalizability. **Statistical pitfalls:** With hundreds of radiomic features, multiple testing and model overtraining are concerns. Some models report very high AUCs on internal data that drop in external validation.

Biological confounding: Imaging captures composite effects; e.g. a textural feature might reflect necrosis, edema, or stroma, not a single gene. Distilling causality is difficult. **Ethical and regulatory:** Integrating imaging and genomics raises privacy issues. Combined imaging/genomic data are highly sensitive and identifiable; robust de-identification and consent are required. There is also the question of incidental findings: for example, if a radiogenomic model predicts a high-risk mutation, should the patient be informed or undergo genetic counseling? Regulatory pathways for complex AI-based diagnostics are evolving; currently few radiogenomic tests are approved. Potential biases (racial, gender) in datasets must be addressed. Finally, harmonizing patient data sharing across institutions faces legal and ethical barriers.

VI. Recommendations

Based on the current evidence, we suggest the following best practices for radiogenomic research:

- *Standardize Imaging and Radiomics:* Adopt consensus protocols (e.g. fixed voxel size, calibration phantoms) and standardized feature definitions (IBSI guidelines) to improve reproducibility. Include test–retest analysis of radiomic features to select stable biomarkers.
- *Large, Multicenter Cohorts:* Pool data across institutions (and public repositories like TCIA/TCGA) to increase sample size and diversity. Radiogenomic models should be trained and validated on heterogeneous data to capture population variability.
- *Rigorous Validation:* Use nested cross-validation, hold-out test sets, and if possible prospective trials to confirm findings. Blindly applying models to independent cohorts is critical to assess real-world utility.
- *Biologically Informed Modeling:* Where possible, constrain models with known biology. For example, focus on features (or deep network activations) that map to interpretable traits (e.g. vascularity, cellularity). This enhances explainability and clinical trust.
- *Integrate Multi-omics and Clinical Data:* Combining radiomics with gene expression, proteomics or blood biomarkers may yield more robust signatures. Radiogenomic studies should consider integrating clinical variables (age, stage) and other omics in predictive models.
- *Open Science and Data Sharing:* Publish radiogenomic datasets and analysis code whenever feasible. Shared benchmarks (e.g. challenge competitions) can accelerate progress. Harmonized databases of paired imaging/genomics (with annotated segmentation) should be developed to facilitate replication.
- *Ethical Frameworks:* Ensure all studies have appropriate IRB approval, patient consent for data use, and adhere to regulations (HIPAA, GDPR). Consider equity: test models for bias and avoid discriminatory training data. Data sharing must balance openness with privacy (e.g. federated learning could allow model training without centralized patient data).
- *Regulatory Strategy:* For translation, engage regulators early. Document model development clearly (per TRIPOD/AQUA-AI guidelines), and plan prospective validation akin to a diagnostic trial. Work towards clinical trials demonstrating that radiogenomic-guided decisions improve outcomes.

Following these guidelines will enhance the quality and impact of radiogenomic research.

VII. Conclusions

Radiogenomics holds great promise for precision oncology by linking noninvasive imaging to underlying tumor genetics. Recent studies have demonstrated numerous robust correlations (e.g. CT features predicting EGFR mutations, MRI features predicting IDH status) across several cancer types. These findings suggest that radiomic biomarkers can serve as surrogates for genomic tests, guiding personalized therapy without additional biopsies. However, the field is still maturing: many reported associations need further validation, and technical standardization is urgently needed. Interpreting imaging signals in light of tumor biology (neoangiogenesis, cell density, stromal changes) will improve model transparency. Clinically, radiogenomic signatures could enable earlier and more precise treatment stratification, especially when tissue samples are limited.

Going forward, interdisciplinary collaboration (radiologists, oncologists, geneticists, bioinformaticians) is key. We advocate large-scale consortia and shared data initiatives to overcome current sample-size and heterogeneity challenges. Ethical, legal and social implications must be addressed – including patient privacy and the responsible use of predictive models.

If rigorously developed and validated, radiogenomic biomarkers may become routine tools in oncology, fulfilling the vision of image-guided personalized medicine.

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