

GGFI-Net: Genomics-Guided Cross-Modal Imputation for Multimodal Survival Analysis with Missing Modality

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Abstract. Survival prediction modeling has shown promise in capturing diverse tumor phenotypes from multimodal data, such as radiomics and genomics. However, its clinical application is often hampered by two major constraints: missing modalities in fragmented datasets, and severe overfitting caused by high-dimensional features in small-sample regimes. To address these challenges, we propose a Genomics-Guided Cross-Modal Feature Imputation Framework (GGFI-Net) for survival analysis. Specifically, we introduce a Lightweight Cross-Modal Mapper (LCM) that learns CT-embedding-aligned auxiliary representations from genomic features, providing auxiliary radiological embeddings when CT is unavailable without the computational overhead of heavy generative models. Furthermore, we design a Genomics-Driven Survival Distillation (GDSD) module. This teacher-student architecture transfers survival-relevant discriminative knowledge from complete genomic profiles to supervise the latent radiomic representations, effectively mitigating overfitting and ensuring the prognostic fidelity of the imputed features. Unlike prior fusion or generative imputation methods, GGFI-Net explicitly models cross-modal survival-consistent feature alignment. Experiments on public pancreatic cancer cohorts show empirical performance improvements. Notably, on the modality-deficient TCGA-PAAD dataset, GGFI-Net improves over genomics-only baselines, increasing the C-index from 0.645 to 0.679. It also achieves competitive performance on the modality-complete CPTAC-PDA dataset (C-index: 0.709). These results suggest that GGFI-Net is a promising framework for incomplete multimodal survival modeling under data scarcity.

Keywords: Survival prediction · Cross Modality · Knowledge Distillation · Radiogenomics.

1 Introduction

Prognostic assessment, particularly survival analysis, is crucial in tumor treatment; it empowers clinicians to devise personalized treatment plans and enable timely interventions, thereby ultimately prolonging patient survival[27]. In overall survival (OS) prediction, diverse biological modalities offer complementary perspectives on tumor progression. This synergy suggests that different biological dimensions offer a mutually reinforcing and holistic representation of tumor heterogeneity. Such complementarity has catalyzed the development of multimodal survival prediction models, which aim to combine characteristics of different modalities for enhanced prognostic accuracy.

Conventional multimodal approaches primarily focus on feature fusion strategies [9, 7, 3, 14]. However, these methods strictly require paired and well-aligned data, which contradicts the fragmented nature of clinical data. Consequently, addressing the missing modality is crucial for improving multi-modal prognosis. One of the solutions is to restore modal alignment by synthesizing missing modalities. For example, using Generative Adversarial Networks (GANs) [8, 26] or diffusion models [12]. However, these generative approaches typically rely on high-capacity architectures, which necessitate massive training cohorts and are highly susceptible to overfitting in small-sample regimes. Feature imputation and Knowledge Distillation (KD) offer a lightweight path for modeling incomplete data [16]. Feature-level imputation focuses on directly acquiring meaningful latent representations, effectively bypassing the computationally intensive and often unstable step of full-modality reconstruction. Such imputation strategies can be grounded in either unimodal priors [10, 20] or shared latent manifolds [25, 1], where the imputed features are compatible with downstream decoders [23]. KD can effectively recover missing information and maintain prognostic consistency, even when certain inputs are absent, by distilling the high-level prognostic representations from the teacher model. [17, 15, 5]

Inspired by these advancements, we propose a Genomics-Guided Cross-Modal Feature Imputation Framework (GGFI-Net) for survival prediction, specifically optimized for fragmented small datasets. Unlike conventional generative approaches, GGFI-Net employs a cross-modal imputation strategy that bypasses the instability of full-modality synthesis. By integrating Genomics-Driven Survival Distillation (GDSD), the framework ensures that the imputed features retain high prognostic fidelity, effectively maximizing the utility of fragmented clinical data under data scarcity. Our main contributions are:

1. We proposed GGFI-Net, a novel survival prediction approach tailored for small-sample and unpaired training data, showing empirical improvements in scenarios with modality missingness.
2. We introduced a lightweight cross-modal mapper (LCM), which learns CT-embedding-aligned auxiliary representations from genomic features, enabling efficient feature-level imputation without the computational overhead and overfitting risks of heavy generative models.
3. We designed a Genomics-Driven Survival Distillation method (GDSD), a teacher-student architecture that transfers survival-relevant discriminative

knowledge from complete genomic profiles to supervise the latent radiomic representations, thereby ensuring the prognostic fidelity of the imputed features.

2 Methodology

2.1 Overview

As shown in Fig.1, the framework consists of two integral components. The first component is the LCM, which learns CT-embedding-aligned auxiliary representations from genetic features. The second is the GDSD to enforce prognostic consistency. Specifically, a uni-modal teacher network, which is trained on complete genomic profiles provides stable supervision to guide the student network, ensuring the model yields reliable survival predictions with both real and imputed radiomic features.

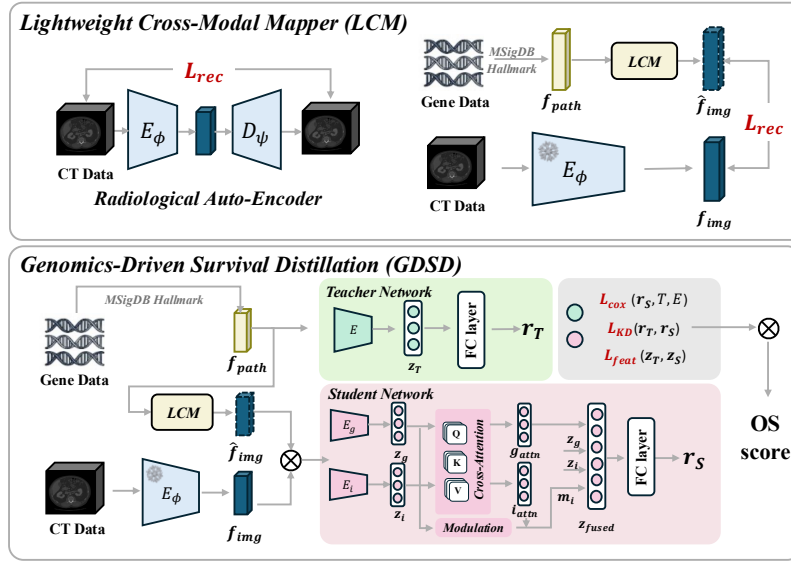


Fig. 1. The overview of the proposed framework

2.2 Lightweight Cross-Modal Mapper (LCM)

In this section, we propose a module designed to bridge the semantics from genomics to radiomics. Its primary objective is to learn CT-embedding-aligned auxiliary representations from genomic features, thereby providing auxiliary radiological embeddings during inference when CT is unavailable.

Training of the radiological encoder. To ensure the mapper learns biologically meaningful radiological imaging patterns, we train a 3D radiological autoencoder. Its encoder E_ϕ , initialized with MedicalNet (ResNet-10) [4], incorporates an appended linear bottleneck layer that compresses the native 512-dimensional output into a 128-dimensional embedding to mitigate downstream overfitting. Paired with a lightweight decoder D_ψ , the network is fine-tuned to capture tumor-specific heterogeneity via a standard reconstruction loss $\mathcal{L}_{rec} = \|\mathbf{x}_{img} - D_\psi(E_\phi(\mathbf{x}_{img}))\|_2^2$, where x_{img} denotes the input CT volume. Once optimized, E_ϕ is frozen to extract the compact ground-truth features $\mathbf{f}_{img} = E_\phi(\mathbf{x}_{img})$ for subsequent cross-modal mapping.

Training of the LCM. To train the LCM, we utilize a paired multi-modal dataset $\mathcal{D} = \{(\mathbf{x}_g^i, \mathbf{x}_{img}^i)\}_{i=1}^N$, where $\mathbf{x}_g^i$ and $\mathbf{x}_{img}^i$ denote the genomic profile and CT volume for the i -th patient, acquired synchronously. For the image branch, we extract the ground-truth image embeddings $\mathbf{f}_{img}^i = E_\phi(\mathbf{x}_{img}^i)$ using the frozen radiological encoder E_ϕ . For the genomic branch, processing raw RNA-seq data is a challenge due to its high dimensionality ($> 60,000$ genes). To address this, we perform biological dimensionality reduction. Specifically, we project the raw gene expression space into compact pathway activity scores using Gene Set Variation Analysis (GSVA) based on the MSigDB Hallmark gene sets [2]. This process effectively reduces the feature dimensionality from over 60,000 raw genes to a compact, 50-dimensional pathway embedding $\mathbf{f}_{path}^i \in \mathbb{R}^{50}$. This dense vector serves as the biological prior input to the LCM, enabling efficient and lightweight cross-modal mapping. The mapper M is then trained to minimize the reconstruction loss $\mathcal{L}_{map} = \|M(\mathbf{f}_{path}^i) - \mathbf{f}_{img}^i\|_2^2$.

2.3 Genomics-Driven Survival Distillation (GDSD)

GDSD is a survival prediction framework designed to maintain prognostic consistency across both complete and modality-incomplete scenarios. Rather than merely compensating for absent data, GDSD leverages the complete genomic profile to supervise and refine the features generated by the imputation module. By distilling discriminative survival knowledge into the auxiliary latent space, this strategy encourages the CT-embedding-aligned representations to retain prognostic relevance, thereby supporting multimodal survival modeling even under missing modality.

Teacher-Student Architecture. The uni-modal teacher network T takes the 50-dimensional hallmark pathway scores $\mathbf{f}_{path}$ as input. Since genomic data are clinically ubiquitous and complete in our setting, the teacher network serves as a stable supervisor. The teacher consists of a deep feature extractor that maps genomic inputs to a high-level latent space $\mathbf{z}_T \in \mathbb{R}^{64}$, followed by a linear layer to predict the risk estimate r_T .

The student network S is designed for multi-modal survival prediction. It comprises two encoders: a genomic encoder E_g and a radiological encoder E_i . To

handle missing modalities, E_i dynamically accepts either the combination of real CT embeddings $\mathbf{f}_{img}$ and generated embeddings $\hat{\mathbf{f}}_{img}$ from the frozen LCM with a ratio R , or only $\hat{\mathbf{f}}_{img}$ if the CT modality is missing. After encoders, we can get the genomic feature $\mathbf{z}_g$ and image radiological feature $\mathbf{z}_i$. We implement a Cross-Modal Attention mechanism to capture the bidirectional synergy between modalities. To compute the gene-guided image attention g_{attn} , the radiological feature $\mathbf{z}_i$ serves as the Query (Q) to aggregate relevant context from the genomic feature $\mathbf{z}_g$, which acts as the Key (K) and Value (V) via standard scaled dot-product attention. Symmetrically, the image-guided genomic attention i_{attn} is computed by using $\mathbf{z}_g$ as the Query and $\mathbf{z}_i$ as the Key/Value. To further refine the representations, we introduce a feature modulation layer inspired by Feature-wise Linear Modulation that linearly reshapes the image-enhanced features conditioned on the raw genomic profile: $\mathbf{m}_i = \text{Modulate}(\mathbf{i}_{attn}, \mathbf{z}_g)$. The final fused representation $\mathbf{z}_{fused}$ is derived by concatenating the original multi-modal features, the gene-guided attention, and the modulated features: $\mathbf{z}_{fused} = [\mathbf{z}_g; \mathbf{z}_i; \mathbf{g}_{attn}; \mathbf{m}_i]$.

Optimization and Distillation. The framework is optimized via a hybrid objective function. The primary task loss is the Cox Partial Likelihood Loss ($\mathcal{L}_{cox}$), which maximizes the concordance between predicted risk scores r_S and true survival labels (time T , event E). To ensure the student inherits the teacher’s robust genomic representation, we implement a multi-level distillation strategy. The logits-level distillation loss ($\mathcal{L}_{KD}$) is formulated to minimize the discrepancy between the student’s predicted risk scores r_S and the teacher’s predicted risk scores r_T :

$$\mathcal{L}_{KD} = \|r_S - r_T\|_2^2$$

Simultaneously, we introduce the feature-level Alignment loss ($\mathcal{L}_{feat}$) to constrain the student’s genomic latent space $\mathbf{z}_g$ within the manifold of the teacher’s robust feature space $\mathbf{z}_T$:

$$\mathcal{L}_{feat} = \|\mathbf{z}_g - \mathbf{z}_T\|_2^2$$

The total objective is formulated as:

$$\mathcal{L}_{total} = \mathcal{L}_{cox}(r_S, T, E) + \frac{1}{2} \exp(-s_{KD}) \mathcal{L}_{KD} + \frac{1}{2} s_{KD} + \frac{1}{2} \exp(-s_{feat}) \mathcal{L}_{feat} + \frac{1}{2} s_{feat}$$

where s_{KD} and s_{feat} are learnable parameters representing the log-variance of the homoscedastic uncertainty, which are optimized via backpropagation[13]. This dual-level constraint ensures that the student not only mimics the teacher’s final predictions but also aligns its internal genomic encoding with the teacher’s trained feature space.

3 Experiments and Results

3.1 Dataset and Implementation Details

In this work, to evaluate the effectiveness of our framework in a high-heterogeneity scenario, we specifically focus on pancreatic cancer. Data were from multiple public repositories of pancreatic cancer and categorized into three cohorts. A CT

imaging cohort from the Medical Segmentation Decathlon (MSD) [21], with 420 pancreas tumor CT scans which is used to train the radiological encoder. The second one is 60 paired cases of CT imaging and RNA-sequencing (RNA-seq) data, sourced from the CPTAC-PDA collection in TCIA [6] and the CPTAC-3 project in the GDC Data Portal [30], respectively. The event rate (death) is 76.67%. The third one is a unimodal gene cohort, comprising 178 RNA-seq profiles from the TCGA-PAAD project [19], with an event rate of 60.67%.

All of the CT data were resampled with spatial resolution of $1.6 \times 1.6 \times 1.0 \text{ mm}^3$. Pancreatic ROIs were extracted based on the corresponding pancreas segmentation masks, which are obtained via the TotalSegmentator framework [28] and then resampled and zero-padded to a fixed size of $128 \times 128 \times 64$ voxels. CT intensities were clipped to $[-160, 240]$ Hounsfield Units (HU). RNA-seqs were standardized using Transcripts Per Million (TPM) to ensure inter-sample comparability, followed by log-transformed $\log_2(\text{TPM} + 1)$ for the subsequent analysis.

Model performance was assessed using the concordance index (C-index) [24] and the integrative area under the curve (iAUC). To ensure robust evaluation, we employed a 5-fold nested cross-validation strategy: an inner loop for hyperparameter tuning and an outer loop for unbiased testing. Final metrics are reported as the mean and standard deviation across the five independent outer test folds. Additionally, to evaluate overall survival risk stratification, Kaplan-Meier (KM) curves and Log-rank tests were generated by pooling the predicted risk scores across all outer test folds, with high- and low-risk groups split by the median predicted risk score.

3.2 Results

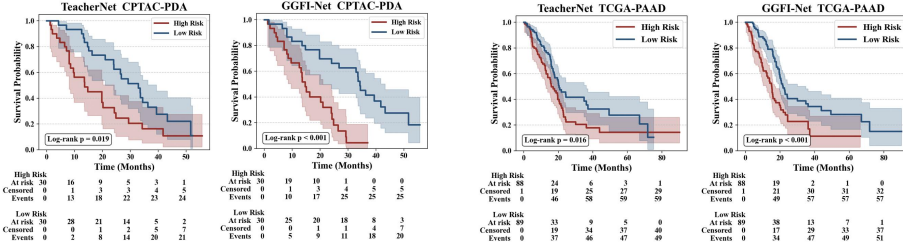
We benchmarked GGFI-Net against established clinical signatures (*KRAS* single-gene expression [29], 5-gene signature with *ADM*, *ASPM*, *DCBLD2*, *E2F7*, and *KRT6A* [18]) and genomics-only models (LASSO-Cox [22], DeepSurv [11], and TeacherNet). As shown in Table 1, GGFI-Net yields empirical improvements across both cohorts. On the modality-complete CPTAC-PDA dataset, it effectively synergizes multi-modal representations for enhanced prognosis (C-index: 0.709 ± 0.056). On the modality-deficient TCGA-PAAD cohort, which represents a transfer evaluation with possible cross-cohort shift, GGFI-Net improves the C-index from 0.645 ± 0.023 (TeacherNet) to 0.679 ± 0.022 . This suggests that LCM provides useful CT-embedding-aligned auxiliary representations when CT is unavailable. Furthermore, Kaplan-Meier (KM) analysis shows a clearer separation between high- and low-risk groups than TeacherNet, indicating improved survival risk stratification in this cohort.

3.3 Ablation Study

The impact of LCM-generated features. We evaluated the model’s performance by varying the ratio R of LCM-generated features during training. In the inference phase, the CPTAC-PDA dataset utilizes features extracted directly

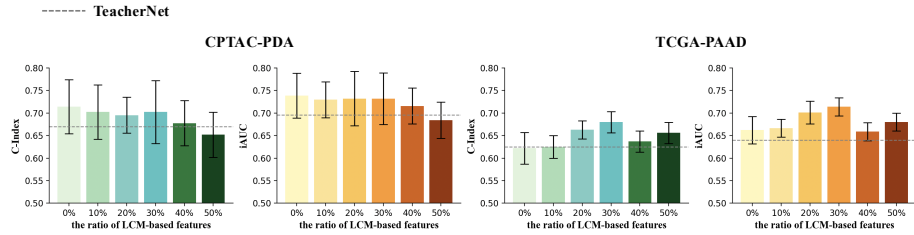
Table 1. Result comparison with different baselines

Method	Modality	TCGA-PAAD		CPTAC-PDA	
		C-index	iAUC	C-index	iAUC
Cox	single gene(<i>KRAS</i>)[29]	0.551±0.061	0.546±0.053	0.556±0.091	0.585±0.105
Cox	5-gene signature[18]	0.579±0.050	0.608±0.035	0.592±0.084	0.578±0.095
Lasso-Cox [22]	genetic pathway	0.633±0.038	0.648±0.056	0.657±0.065	0.665±0.056
DeepSurv [11]	genetic pathway	0.646±0.043	0.663±0.052	0.676±0.064	0.707±0.071
TeacherNet	genetic pathway	0.645±0.023	0.660±0.032	0.670±0.073	0.696±0.066
GGFI-Net	pathway+radiological	-	-	0.709±0.056	0.742±0.057
GGFI-Net	genetic+ imputed radiological	0.679±0.022	0.714±0.028	-	-

**Fig. 2.** KM analysis of GGFI-Net and TeacherNet on both cohorts

from CT scans via our radiological encoder, whereas the TCGA-PAAD dataset relies on LCM-generated features because of the missing imaging modalities.

As illustrated in Fig. 3, a 30% blending ratio during training provides an empirical validation-based setting in this study, enabling the LCM to compensate for missing radiological embeddings during inference while limiting interference with authentic imaging features in complete-modality cases.

**Fig. 3.** Impact of LCM-generated feature ratios on survival prediction performance

The efficacy of the multi-modal fusion mechanism. We compared several representative fusion mechanisms to evaluate the efficacy of the proposed multi-modality fusion mechanism in GGFI-Net. As summarized in Table 2, our GGFI-

Net, which combines Cross-Attention with Modulation, consistently outperforms all other configurations across both cohorts. Notably, when replacing our specific mechanism with a feature concatenation strategy, the C-index experienced a substantial drop from 0.679 to 0.648 on the TCGA-PAAD dataset, and from 0.709 to 0.685 on the CPTAC-PDA dataset. Furthermore, the complete GGFI-Net strictly outperformed partial fusion configurations, such as relying solely on cross-attention concatenation or isolated feature modulation, demonstrating the effectiveness of our integration design in GGFI-Net.

Effect of the loss functions. Experiments were also conducted to evaluate the effect of different loss functions. The C-index and iAUC values under different loss components are summarized in Table 2. The results demonstrate that both L_{KD} and L_{feat} contribute to the performance enhancement of GGFI-Net. Notably, L_{KD} yields a more substantial improvement, validating its pivotal role as the primary guidance in our knowledge distillation framework.

Table 2. Ablation study on multi-modality fusion mechanisms and different loss functions

Loss Function			Strategy of modality fusion		TCGA-PAAD		CPTAC-PDA	
$\mathcal{L}_{cos}$	$\mathcal{L}_{KD}$	$\mathcal{L}_{feat}$	Strategy of fusion		C-index	iAUC	C-index	iAUC
✓	✓	✓	GeneOnly (TeacherNet)		0.645±0.023	0.660±0.032	0.670±0.073	0.696±0.066
✓	✓	✓	Feature Concatenation		0.648±0.024	0.678±0.032	0.685±0.078	0.725±0.077
✓	✓	✓	Feature Modulation		0.655 ± 0.039	0.685 ± 0.037	0.692±0.071	0.732±0.069
✓	✓	✓	Cross-Attention Concatenation		0.649±0.034	0.681±0.030	0.695±0.064	0.730±0.059
✓			-		0.621±0.022	0.646±0.037	0.682±0.065	0.704±0.058
✓	✓		-		0.671±0.047	0.688±0.053	0.701±0.045	0.712±0.048
✓		✓	-		0.622±0.036	0.637±0.040	0.687±0.077	0.699±0.063
✓	✓	✓	Cross-Attention Modulation (GGFI-Net)		0.679±0.022	0.714±0.028	0.7093±0.056	0.742±0.057

4 Conclusion

We proposed GGFI-Net, a novel framework designed for survival prediction in scenarios characterized by small-sample sizes and modality missingness. By employing a cross-modal imputation strategy and genomics-driven knowledge distillation strategy, our method learns CT-embedding-aligned auxiliary representations while encouraging their prognostic relevance. GGFI-Net addresses the limitation of conventional multi-modal models that strictly require paired and aligned modalities. Experiments on TCGA-PAAD and CPTAC-PDA datasets show empirical improvements in both modality-complete and modality-missing scenarios. In conclusion, GGFI-Net provides a promising framework for multi-omics integration in real-world clinical settings where patients’ data are frequently fragmented and incomplete.

Disclosure of Interests

The authors have no competing interests to declare.

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