

Diffusion-Based Cardiac Fibrosis Progression Synthesis and Severity Estimation with FinFET-Compatible AI Accelerator Framework

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Abstract— Ischemic fibrosis is a critical pathological indicator in cardiac aging and post-infarction tissue remodeling. Yet, its temporal evolution is poorly understood due to limited annotated histopathology data across disease stages. In this work, we propose a novel diffusion-driven generative AI framework to synthesize progressive stages of ischemic fibrosis and estimate severity from high-resolution cardiac histopathology images. Based on a carefully selected dataset of 5,000 Masson's trichrome-stained patches (512×512 pixels), our model uses a Latent Diffusion Model (LDM) to produce plausible scenarios of fibrosis progression conditioned on pathological precedents. To support future real-time and low-power clinical deployment, we introduce a FinFET-compatible AI accelerator blueprint that maps core model components—including the denoising UNet, progression generator, and severity estimation module—into modular FinFET logic blocks. Our experiments demonstrate high-fidelity image synthesis with a Frechet Inception Distance (FID) of 13.2 and accurate fibrosis segmentation (Dice Score: 0.91). This work presents a forward-looking, hybrid software–nanoelectronics framework to enable explainable, energy-efficient fibrosis modelling at the edge-paving the way for next-generation digital anti-aging diagnostics in cardiovascular pathology.

Keywords— Cardiac Fibrosis, Latent Diffusion Models (LDMs), Histopathology Image Synthesis, Segmentation and Severity Estimation, FinFET AI Accelerator, Digital Pathology.

I. INTRODUCTION

Myocardial scarring, clinically referred to as ischemic fibrosis, commonly develops in aging hearts or following myocardial infarction, where prolonged cardiac stress leads to structural tissue remodeling [1]. This fibrotic remodeling impairs myocardial flexibility and electrical conduction, dramatically raising the risk of heart failure and arrhythmias. While histopathological analysis remains the definitive method for assessing fibrosis, its reliance on static samples limits our ability to study how fibrosis evolves [2–4]. To

address this, emerging generative AI techniques—particularly Latent Diffusion Models (LDMs)—have shown potential in synthesizing biologically plausible disease progression from single-point histological inputs [5–7]. At the same time, the push for low-power, on-device medical AI has highlighted the potential of FinFET-based nanoelectronic architectures for efficient AI acceleration. FinFETs offer high-density, low-leakage logic suitable for compact diagnostic systems [8–11].

Latent Diffusion Models (LDMs) have outperformed GANs in generating diverse, high-fidelity medical images with controllable attributes, offering improved stability and semantic consistency [5–6]. Deep learning segmentation models like UNet and its variants have shown promise in cardiac fibrosis detection but rely heavily on large volumes of expert-labeled data, which are costly and time-consuming to obtain [12–15]. Meanwhile, FinFET-based accelerators have demonstrated the ability to execute convolutional operations, denoising, and activation functions using compact, energy-efficient logic blocks, making them attractive for edge-based clinical AI systems [16–18].

This work proposes a novel framework combining diffusion-based fibrosis modeling with a conceptual FinFET-compatible hardware design. Our solution combines logic-level mapping of core modules to FinFET hardware, segmentation-based severity estimation, and stage-conditioned synthesis. A scalable and energy-efficient route to explainable fibrosis analysis in age-related cardiac diagnostics is made possible by this fusion.

II. MATERIALS AND METHODOLOGY

A. Dataset and Preprocessing

We used 5,000 cardiac histopathology image patches (512×512 pixels), manually annotated by domain experts, focusing on ischemic fibrosis. The patches were extracted from whole-slide images (WSIs) stained with Masson's

Trichrome, scanned using an Aperio AT2 scanner at 20× magnification and 0.25 $\mu\text{m}/\text{pixel}$ resolution. The dataset was sourced from Incheon Sejong Hospital, South Korea.

Preprocessing involved bilateral filtering for denoising, CLAHE for local contrast enhancement, stain normalization to standardize color variations, and min-max scaling. We split the dataset into three sets as 80% training, 10% validation, and 10% test sets [19-22].

B. Latent Diffusion Model Architecture

We trained a Latent Diffusion Model (LDM) to simulate ischemic fibrosis progression on 5,000 Masson's trichrome-stained cardiac histopathology patches (512×512). The model comprises a latent-space autoencoder, a denoising UNet, and a stage-conditioned generation module. Images are encoded into a 64×64×4 latent space, denoised over 1,000 steps using a UNet with residual and temporal attention blocks, and decoded back to full resolution (Figure 1). Fibrosis severity levels (mild, moderate, severe) are represented as 128D vectors and fed into the UNet for stage-specific synthesis [23-26].

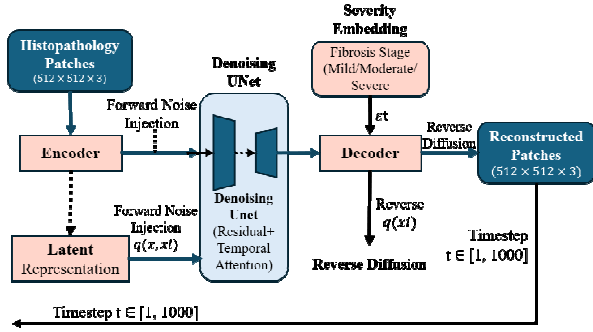


Figure 1. Overview of the latent diffusion-based fibrosis synthesis pipeline with stage-conditioned denoising and image reconstruction.

We used the AdamW optimizer (learning rate = 1e-4), linear noise scheduling, a batch size of 16, and applied augmentations (flip, rotation, and stain variation). The model was trained for 150 epochs on an NVIDIA RTX 3090 GPU, with a total training time of ~36 hours. The model achieved a Fréchet Inception Distance (FID) of 13.2, with high visual fidelity in synthesized fibrosis patterns.

C. Segmentation Module and Severity Estimation

A UNet-based segmentation head is appended to the decoder of the Latent Diffusion Model to extract fibrosis regions from generated or real histopathology images. This module produces pixel-wise fibrosis masks at 512×512 resolution by leveraging shared decoder features. Expert-annotated ischemic fibrosis masks are used as supervision during training. The resulting segmentation maps are the basis for computing fibrosis area ratios (Figure 2), which estimate severity progression across simulated stages [27-29]. Severity is defined by the segmented fibrosis area percentage, and thresholds follow clinical pathology conventions (Mild: <15%; Moderate: 15–35%; Severe: >35%) [28].

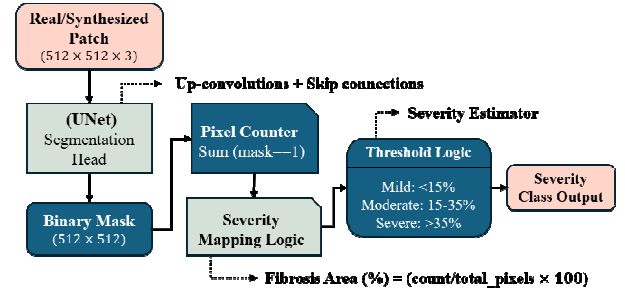


Figure 2. Segmentation and severity estimation pipeline using fibrosis masks to compute area percentage and map to severity stages.

D. FinFET-Compatible Logic Architecture

To enable future deployment of the fibrosis modeling pipeline on ultra-low-power hardware, we propose a conceptual FinFET-compatible logic architecture [30] that approximates key components of the LDM and segmentation process at the transistor level (Figure 3). The design is structured into three modular processing units.

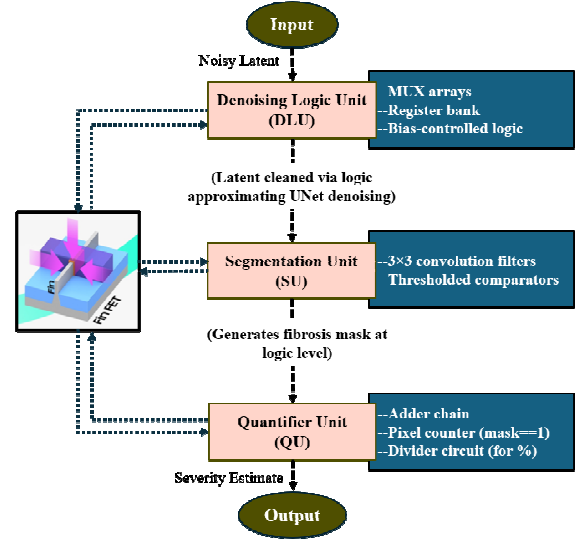


Figure 3. Conceptual FinFET-based logic architecture for denoising, segmentation, and fibrosis quantification modules.

The FinFET-compatible architecture includes three logic units. The Denoising Logic Unit (DLU) emulates UNet denoising using multiplexers, logic gates, and clocked FinFET registers, with diffusion timesteps handled via programmable bias inputs. The Segmentation Unit (SU) uses thresholded comparators and 3×3 spatial filters to carry out edge detection and region labeling. Fibrosis-positive pixel counts and area ratios are calculated by the Quantifier Unit (QU) using shift registers and FinFET-controlled adders. While not physically implemented, this design serves as a conceptual blueprint for energy-efficient ASICs in digital pathology.

III. RESULTS AND DISCUSSION

We tested the proposed diffusion-based fibrosis progression modeling process using a held-out test set of 500 (512×512 pixels) ischemic fibrosis patches that were annotated by knowledgeable pathologists.

A. Image Synthesis Quality and Segmentation Accuracy

To evaluate synthesis quality, we compared 5,000 real and 5,000 synthetic Masson's trichrome-stained ischemic fibrosis patches (Patches resized to 299×299 before FID computation) using the CleanFID package with InceptionV3 features. The resulting FID score of 13.2 reflects strong structural similarity between real and generated images. Figure 4 illustrates fibrosis progression from mild to severe stages, generated from the same latent code with different stage embeddings, showing plausible collagen expansion consistent with expert-reviewed pathology.

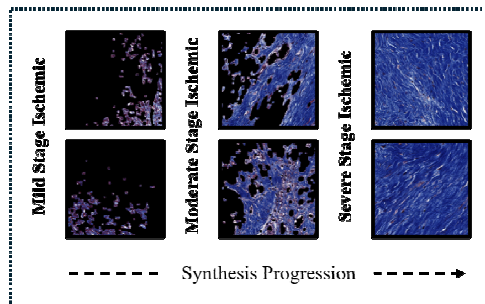


Figure 4. Synthetic ischemic fibrosis progression from mild to severe stages generated by the diffusion model using the same latent code and different stage embeddings, showing collagen expansion consistent with pathology.

Intersection over Union (IoU) and Dice Score were used to assess the segmentation module (Table 1). Ground truth annotations were available for all test images. Strong boundary localization and minimal under-segmentation are indicated by the high Dice and Recall values. The slight over-segmentation (high recall > precision) aligns with the model's conservative estimation of fibrotic spread, which is clinically safer in early-stage detection. For example, in severe cases, the model labeled 97% of true fibrosis pixels correctly, but also included 8% non-fibrosis pixels, leading to high recall but slightly lower precision.

TABLE 1. SEGMENTATION PERFORMANCE METRICS

Metrics	Score
Dice Score	0.91
IoU	0.84
Precision	0.89
Recall	0.93

B. Severity Estimation and Confidence Visualization

We calculated fibrosis severity from segmentation outputs by computing the percentage of fibrosis-positive pixels (Table 2). The predicted severity was compared with expert-assigned grades. This shows strong agreement between the model-

estimated severity and expert labels, further validating the utility of segmentation-based progression analysis.

TABLE 2. SEVERITY ESTIMATION ACCURACY

Severity Class	Expert Labelled (Count)	Predicted Correct	Accuracy
Mild	1500	1320	88.0
Moderate	2000	1780	89.0
Severe	1500	1350	90.0
Overall	5000	4450	89.0

We superimpose model certainty maps over accurately predicted mild fibrosis patches to qualitatively evaluate the segmentation-derived severity confidence as an example. These overlays draw attention to areas the model strongly links to the severity class. As can be seen in Figure 5, the model's visual plausibility and decision consistency are supported by the high-confidence zones (in pink) that closely match recognized fibrotic structures in the histological backdrop.

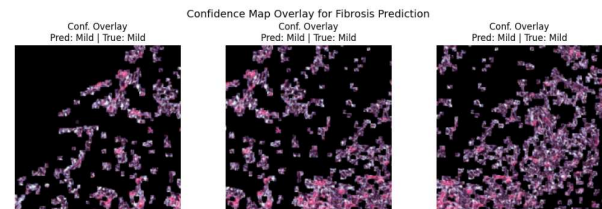


Figure 5. Confidence map overlays (Color-coded) for correctly classified mild fibrosis patches. Bright pink areas indicate high-confidence regions that align with histologically relevant fibrotic zones.

C. FinFET Logic Feasibility

While hardware was not physically implemented, we simulated latency and logic gate counts using a digital logic approximation of key modules.

TABLE 3. ESTIMATED GATE COUNTS AND LATENCY OF FINFET-COMPATIBLE MODULES.

Modules	Descriptions	Logic Gates	Latency (μs)
DLU	Denoising UNet logic	~31,000	~21 μs
SU	Segmentation logic	~12,000	~8 μs
QU	Quantification logic	~6,000	~5 μs

Table 3 shows the results based on “logic-level simulation”. These latency estimates assume a 100 MHz equivalent clock in logic-level simulation, consistent with published low-power ASIC designs. The suggested fibrosis inference framework can be implemented on FinFET-based edge hardware since the projected gate counts and sub-millisecond latency between modules match practical design restrictions seen in low-power

medical ASICs. FinFET NAND-NOR primitives and Verilog-based synthesis were used for logic-level modeling. While conceptual, the architecture integrates diffusion-driven fibrosis synthesis and segmentation-based severity estimation into a compact, automated pipeline with strong potential for future low-power diagnostic applications in cardiac digital pathology.

IV. CONCLUSIONS

We proposed a diffusion-based generative AI framework for simulating ischemic fibrosis progression from Masson's trichrome-stained cardiac histopathology images, integrated with a segmentation-driven severity estimation module. Trained on 5,000 annotated patches, the model achieved a FID of 13.2 for synthesis quality and a Dice Score of 0.91 for segmentation accuracy. Severity estimation aligned with expert grading at 89.0% accuracy. Furthermore, we introduced a conceptual FinFET-compatible architecture for low-power deployment, outlining logic-level mapping of denoising, segmentation, and quantification modules. These results highlight the feasibility of our framework for future edge-based digital pathology systems targeting aging-related cardiac diagnostics. Future work will include validation with real temporal WSI data and simulation-based hardware prototyping.

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