

Lung Cancer: Epidemiology, Diagnosis, Treatment, and Deep Learning–Based Segmentation Models

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Abstract—

Lung cancer continues to cause more deaths than any other malignancy worldwide, even as smoking rates decline and early-detection and treatment technologies improve. This review synthesizes current knowledge across four areas: disease epidemiology and risk factors, diagnostic and staging pathways, the rapidly shifting treatment landscape, and computational methods for segmenting tumors and lung anatomy in computed tomography (CT) scans. On the clinical side, we trace how screening, minimally invasive surgery, molecular diagnostics, and liquid-biopsy monitoring are reshaping patient management, alongside the expansion of targeted agents, immunotherapy, and antibody-drug conjugates. On the computational side, we examine architectures spanning convolutional U-Net variants, attention-augmented networks, self-configuring frameworks such as nnU-Net, and transformer-based models, comparing their reported accuracy across public and institutional datasets. Across the literature, whole-lung and lobe segmentation is essentially a solved problem, with Dice scores routinely above 0.95, whereas segmenting individual tumors and multiple synchronous lesions remains considerably harder and continues to attract active research.

Index Terms—

lung cancer, epidemiology, TNM staging, immunotherapy, targeted therapy, image segmentation, U-Net, nnU-Net, deep learning, computed tomography.

I. INTRODUCTION

Lung cancer originates from the epithelial lining of the bronchi, bronchioles, or alveoli and is broadly divided into two clinical categories: non-small cell lung cancer (NSCLC), which accounts for roughly 85% of diagnoses, and small cell lung cancer (SCLC), a biologically more aggressive form that tends to metastasize early. For several decades it has held the grim distinction of being the world's leading cause of cancer mortality, a burden historically attributable to tobacco use but increasingly shaped by non-smoking exposures such as air pollution and occupational carcinogens.

This review draws together three closely related clinical threads: (i) the epidemiology and risk factors that shape who develops the disease, (ii) the diagnostic pathway and staging framework used to characterize it once found, and (iii) the therapeutic landscape, both well-established and newly emerging, including minimally invasive surgery and liquid-biopsy-guided monitoring. Because computer-aided detection has become integral to catching disease early, we also examine the deep learning segmentation models used to delineate tumors and lung structures on CT, covering a representative architecture, attention-based refinements, and a side-by-side comparison of results reported across the literature.

II. EPIDEMIOLOGY AND RISK FACTORS

A. Global and U.S. Burden

For 2026, the American Cancer Society projected roughly 229,410 new diagnoses of lung and bronchus cancer in the United States, alongside close to 124,990 deaths [1]. The disease burden is far larger worldwide, with an estimated 2.2 million new cases and 1.8 million deaths occurring each year [2]. An average American faces a lifetime risk of about 5.2%, and diagnoses cluster most heavily among adults between 65 and 74 years old [1]. Encouragingly, age-adjusted incidence has been falling by roughly 2% annually over the last decade, and mortality among men has dropped by more than 60% relative to 1990 levels [1], a trend largely attributable to declining smoking rates and improvements in early detection.

B. Tobacco and Secondhand Smoke

Cigarette smoking remains by far the most significant risk factor and underlies the large majority of cases diagnosed worldwide. Even among people who have never smoked, exposure to secondhand smoke raises lung cancer risk by an estimated 20-30% relative to unexposed nonsmokers [3], underscoring why smoke-free workplace and public-space policies remain a public health priority.

C. Environmental and Occupational Exposures

In the United States, radon gas exposure ranks as the second-leading cause of lung cancer after smoking [3]. Asbestos is implicated in an estimated one-quarter to one-third of lung cancer deaths, and the risk rises sharply in individuals with combined asbestos exposure and a smoking history [4]. Additional occupational hazards linked to elevated risk include diesel exhaust, arsenic, chromium, and nickel, most notably among workers in rubber manufacturing, roofing, and mining [4]. Outside the workplace, ambient fine particulate matter (PM_{2.5}) has become an increasingly important driver of lung cancer, particularly in regions where smoking prevalence is low; global burden-of-disease analyses attribute a substantial rise in PM_{2.5}-attributable lung cancer deaths between 1990 and 2017 to this exposure pathway [5]. Household air pollution from solid-fuel cooking and heating contributes a comparable non-smoking-related risk in many low- and middle-income settings, though it

receives less epidemiological attention than ambient PM_{2.5} [5].

D. Genetic and Host Factors

Family history and inherited genetic susceptibility contribute modestly to individual risk. Certain patient groups, including younger individuals, never-smokers, and particularly women of East Asian descent, show a disproportionately high prevalence of specific oncogenic driver mutations such as EGFR alterations, a pattern that carries direct consequences for treatment selection, discussed further in Section IV [5]. Global Burden of Disease modeling shows that the relative weight of behavioral, environmental, and occupational risk factors has shifted considerably over the past three decades, with an increasing share of the non-smoking-attributable disease burden now concentrated in regions experiencing high levels of ambient air pollution [5], [18].

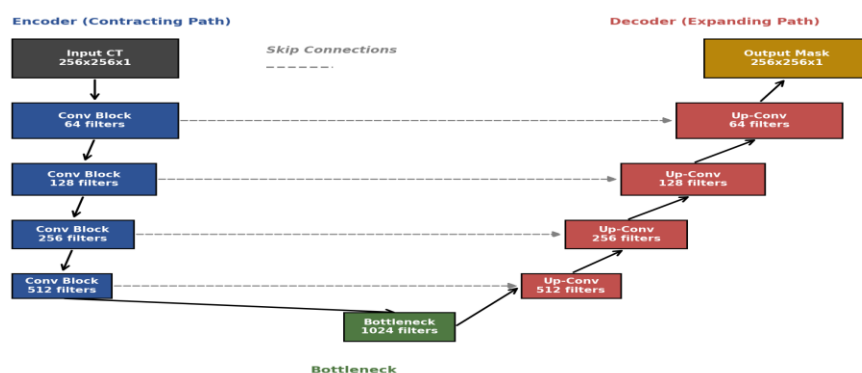


Fig. 1. U-Net encoder-decoder architecture with skip connections, used as the baseline model for lung CT segmentation.

III. DIAGNOSIS AND STAGING

A. Clinical Presentation

Lung cancer often develops without any warning; the lung parenchyma itself does not have many nerve fibres that are sensitive to pain, and by the time symptoms like a persistent cough, coughing up blood, chest pain, or unexplained weight loss occur, the disease may have already advanced. Traditionally, this sneaky advance has led to late diagnosis and short-term survival, with few options for cure, making for bad long-term results [7].

B. Screening

The strategy of annual screening with low-dose computed tomography (CT) was shown to be effective in reducing lung cancer death in randomized trials, including the National Lung Screening Trial and the NELSON study, which recruited current and former smokers with high lung cancer

risk. In line with these results, a large prospective cohort of patients that assessed LDCT screening in a diverse population of high risk patients found a screening detected cancer prevalence of approximately 2%, a number needed to screen of 49 and 97% sensitivity with the specificity at 12 months being greater than 95% [6]. Pilot screening programs elsewhere have shown that about two-thirds of cancers detected are at an early, potentially curable stage, as compared to the majority of cancers usually seen in unscreened populations [7]. Yet, screening uptake is inconsistent and different socio-economic groups are much less likely to access screening, highlighting ongoing low rates of insurance coverage, referral systems, and knowledge of eligibility criteria.

C. Diagnostic Workup and Biomarkers

A diagnostic work-up usually begins with contrast-enhanced CT of the chest and upper abdomen, then PET scanning to characterize and identify metabolic activity and evaluate for

the presence or absence of spread to other organs or tumors, and ends with the removal of the tissue by bronchoscopy, needle biopsy, or surgical biopsy. In adenocarcinoma, sequencing or analysis of the tumor's DNA is now a common part of the diagnosis, with actionable genetic changes found in the majority of cases (more than 60%) and helping to decide the treatment choice. For patients with limited tissue, degraded or compromised tissue or difficulty obtaining tissue, liquid biopsy, typically in the form of analysis of circulating tumour DNA (ctDNA) from a blood sample, is a more minimally invasive alternative and currently used as an adjunct or alternative to repeat tissue biopsy, and is growing in use to monitor developing drug resistance in patients whose tumor is already receiving targeted therapy [21].

D. TNM Staging

The origin, progression, and treatment planning of lung cancer depend on accurate diagnosis of the stage, therefore the lung cancer is classified using the Tumor-Node-Metastasis (TNM) system which is the 9th edition as of January 2025 [8]. Each tumor is categorized along three lines – the extent of local disease or invasion (T stage) ranging from T1a (tumor less than 1 cm) to T4 (large or locally advanced disease); regional lymph node involvement (N stage) ranging from N0 (no involvement) to N3 (locking of the node(s) by the tumor); and distant metastasis (M stage) of which there are no subdivisions. The three components are put together as an overall stage (0-IV) that influences almost all downstream treatment decisions [8].

IV. TREATMENT APPROACHES

A. Surgery, Radiation, and Chemotherapy

For early-stage NSCLC, surgical resection remains the standard curative approach, frequently paired with adjuvant chemotherapy to lower the risk of recurrence. Minimally invasive approaches, video-assisted thoracoscopic surgery (VATS) and, increasingly, robot-assisted thoracic surgery (RATS), have largely displaced open thoracotomy for anatomically resectable tumors. A propensity-matched cohort comparing the two approaches found that RATS lobectomy was associated with less intraoperative blood loss (a median of 5 mL versus 12 mL) and a markedly lower conversion-to-thoracotomy rate, although at the cost of a longer median operative time (180 versus 144 minutes) [22]. For locally advanced, unresectable disease, concurrent chemoradiotherapy remains the standard of care, and platinum-based combination chemotherapy continues to anchor treatment for tumors that lack a targetable molecular alteration.

B. Targeted Therapy and Immunotherapy

Oncogenic driver mutations, including EGFR, ALK, ROS1, BRAF, MET, and KRAS alterations, are now managed with orally available small-molecule inhibitors such as osimertinib and alectinib, which deliver durable disease control with a more tolerable side-effect profile than conventional chemotherapy. Use of these agents has expanded into the perioperative setting: osimertinib and alectinib are now established for resected EGFR-mutated and ALK-positive disease, respectively, and osimertinib consolidation following chemoradiotherapy is established for unresectable, EGFR-mutated stage III disease [9]. Alterations that can be targeted continue to be expanded, and zongertinib is the newest addition for the treatment of HER2-mutant tumors, while capmatinib or tepotinib are options for the treatment of tumors with a MET exon 14 skipping alteration [9]. At the same time, immune checkpoint inhibitors targeting PD-1/PD-L1, including pembrolizumab, nivolumab and atezolizumab, have emerged as important drugs in the treatment of mNSCLC, early-stage NSCLC and locally advanced NSCLC [10]. Immunotherapy (IT) has yielded good trial outcomes when used in addition to chemotherapy (CT) for perioperatively treated cases, but the adoption of this approach into practice remains slower than treatment in clinical trials and mutation testing prior to surgical consultation has repeatedly emerged as a barrier to use [10].

C. Small Cell Lung Cancer and Antibody-Drug Conjugates

With its aggressive growth pattern and early systemic development, the treatment of SCLC remains largely based on chemotherapy and radiotherapy supplemented by immunotherapy as a standard first-line therapy. A bispecific agent targeting the DLL3 protein found on SCLC cells, tarlatamab, has been approved and is being further studied with companion imaging strategies designed to select patients most likely to be responsive to the bispecifics [10]. The new class of antibody-drug conjugates (ADCs) including datopotamab deruxtecan and telisotuzumab vedotin have developed into a validated class in NSCLC that carry toxic payloads directly to tumor cells [9].

D. Remaining Clinical Challenges

Nevertheless, this positive trend has not been matched by the speed of development of molecular testing facilities and many patients continue to receive treatment before full genomic information is available. Additionally, a fraction of patients are receiving postoperative treatment with adjuvants without any significant benefit, emphasizing the need for markers that can better predict who will benefit from treatment [9, 10].

V. SEGMENTATION MODELS FOR LUNG CANCER IMAGING

Segmenting the lung fields, individual lobes, or tumor regions is a foundational step in computer-aided detection pipelines for lung cancer, since it underpins downstream volumetric measurement, radiomic feature extraction, and malignancy classification.

A. U-Net and Convolutional Variants

In the field of lung CT segmentation, the commonly used U-Net architecture (Fig. 1), which features symmetric architecture between the encoder and the decoder as well as skip connections, is the standard baseline. A key aspect of the network's ability to trace irregular tumor edges is the fact that it combines the coarse features with high semantic value in the deep layers with high spatial resolution in the shallow layers, via skip connections. In practice, a U-Net decoder is often combined with a pre-trained encoder backbone (like ResNet50, VGG16 or Xception) achieving Dice scores above 0.94 for segmentation of cancerous and non-cancerous lung regions [11]. Extensions that are densely connected like U-Net++, U-Net 3+ and Wavelet U-Net++ introduce additional skip connections or feature in a wavelet domain to improve boundary accuracy [12]. Other variants, more recently, have tried to tackle the same boundary problem—this time the decoder is suppressed by channel- and spatial-attention modules that learn to ignore irrelevant background features before they get to the decoder. The channel-spatial attention network performed best of the baseline models, plain U-Net++ and Attention U-Net, on three public datasets of nodule images, with Dice scores of 92.69% on LUNA16, 97.17% on the Medical Segmentation Decathlon, and 98.65% on LNDb [22].

B. 3D and Volumetric Models

Because CT acquisitions are inherently three-dimensional, volumetric architectures such as 3D U-Net and 3D V-Net can exploit inter-slice context that slice-by-slice 2D approaches necessarily discard, which matters most for irregularly shaped or obliquely oriented tumors. One 3D-VNet implementation reached a Dice Similarity Coefficient of 99.34% on LUNA16 nodule segmentation while holding the false-positive rate to just 0.4% [13], illustrating how far volumetric modeling can push performance on a well-curated benchmark.

C. Self-Configuring and Transformer-Based Models

nnU-Net sidesteps manual architecture search by automatically adapting its preprocessing pipeline and network topology to the dataset at hand, selecting among 2D, 3D, and cascaded configurations as appropriate; this

adaptability has made it the default comparison baseline in most recent segmentation papers [14]. Transformer-based architectures such as Swin UNETR and TransUNet take a different approach, using self-attention to model long-range spatial dependencies that convolutional receptive fields capture only indirectly, and have demonstrated performance competitive with nnU-Net on multi-subtype tumor segmentation benchmarks [15].

D. Multimodal and Multi-Lesion Pipelines

Multitask connected U-Net architectures inject PET-derived knowledge into a CT-only inference pathway through a teacher-student training framework, improving tumor localization in the common clinical scenario where only CT is available at the point of deployment [16]. Other pipelines fuse segmentation with detection and classification into a single end-to-end system, for instance pairing a 3D Res-U-Net for segmentation with YOLO-based nodule detection and a vision transformer for malignancy classification [17]. Reflecting more realistic clinical conditions, multi-lesion pipelines that localize the thoracic region, perform multi-instance segmentation, and apply a cascade classifier to suppress false positives have reported average Dice scores of 76% and 73% on internal and external multicenter test sets, respectively, across 1,081 scans containing 5,322 annotated lesions [17], figures that are notably lower than the single-lesion benchmarks reported elsewhere in this section, underscoring how much harder multi-lesion generalization remains in practice.

E. Training Objectives and Evaluation Metrics

Most segmentation networks are optimized with a Dice loss, or a combined Dice and cross-entropy loss, specifically to counter the severe class imbalance between tumor voxels and background lung tissue. Data augmentation, random rotation, scaling, elastic deformation, and intensity jitter, is standard practice given how small annotated medical imaging datasets remain relative to natural-image benchmarks. Reported evaluation metrics typically include the Dice Similarity Coefficient (DSC) and Intersection over Union (IoU) for volumetric overlap, sensitivity and specificity for detection performance, and, increasingly, the Hausdorff distance or Average Symmetric Surface Distance (ASSD), which penalize boundary errors that overlap-based metrics alone can understate [15].

F. Challenges and Limitations

Three challenges recur throughout the literature. First, models trained on single-center, curated datasets such as LUNA16 or LIDC-IDRI often lose accuracy when evaluated on external, multicenter cohorts, a consequence of differences in scanner protocol, slice thickness, and patient

population. Second, tumor morphology varies substantially across histological subtypes, and a single architecture rarely performs optimally across all of them without subtype-specific tuning [32]. Third, most published benchmarks assume a single, well-defined primary lesion, whereas real-world patients frequently present with multiple synchronous lesions, a setting in which reported Dice scores are

consistently lower than single-lesion benchmarks suggest [17]. A related, more practical constraint is that voxel-level tumor annotation requires substantial radiologist time, which caps the size of most publicly available training sets and helps explain why self-configuring and transfer-learning approaches remain so attractive.

TABLE I. Comparative Results of Representative Segmentation Models

Model	Dataset	Task	Reported Results
2D U-Net (baseline)	LUNA16	Lung parenchyma segmentation	Dice ~ 0.94-0.97
U-Net + ResNet50	Private CT (416/416)	Cancerous lung segmentation	Dice 0.9495 +/- 0.109
U-Net + VGG16	Private CT cohort	Non-cancerous lung segmentation	Dice 0.9532 +/- 0.099
R2U-Net	LUNA16	Lung parenchyma segmentation	Dice 98.80%
3D U-Net	AAPM LCTSC 2017	Left/right lung segmentation	Dice 97.9% / 97.3%
3D-VNet	LUNA16	Pulmonary nodule segmentation	DSC 99.34%, FP 0.4%
Wavelet U-Net++	LIDC-IDRI (1018 CT)	Nodule/tumor segmentation	Dice 0.936, IoU 0.878
nnU-Net (2D+3D+cascade)	899 NSCLC patients	Lung lesion segmentation	Best DICE 0.78 +/- 0.12
Hybrid Seg. Network (HSN)	134 CT (SCLC)	SCLC tumor segmentation	Sens. 96.05%, Spec. 96.39%
Multitask Connected U-Net	4 public datasets	CT+PET tumor segmentation	Avg Dice 0.56
Swin UNETR / nnU-Net / TransUNet	1530 CT, 5 centers	Subtype tumor segmentation	DSC, IoU, Hausdorff (comparative)
Multi-instance pipeline	1081 CT, 5322 lesions	Multi-lesion segmentation	Avg Dice 76%/73%, Sens. 85%
CSEA-Net (attention)	LUNA16 / MSD / LNDb	Nodule/tumor segmentation	DSC 92.7% / 97.2% / 98.7%

VI. OBSERVATIONS

- Lung parenchyma and lobe segmentation is essentially solved, with most architectures reporting Dice scores above 0.95 thanks to the sharp, high-contrast boundaries of aerated lung tissue against the chest wall and mediastinum.
- Tumor and lesion segmentation is a different story: reported Dice scores span roughly 0.56 to over 0.99 depending heavily on lesion size, image quality, and how curated the evaluation dataset is.
- Attention-augmented CNNs are closing part of the gap with more complex architectures on nodule-level tasks, at times matching or exceeding nnU-Net and Attention
- nnU-Net remains the default baseline against which newer architectures are benchmarked, owing largely to its automated configuration rather than any single architectural innovation.
- Transformer-based and multimodal models narrow the gap with CNN-based approaches on harder, multi-subtype tasks, but typically need larger training sets to do so.

Performance consistently drops when models trained on single-center, curated data are evaluated on external, multicenter test sets, reinforcing that external validation deserves at least as much attention as architectural novelty.

VII. DISCUSSION

The clinical and computational threads covered in this review are becoming more tightly intertwined rather than less. Earlier detection through LDCT screening only translates into better survival if downstream diagnostic and staging workflows can keep pace with the resulting flood of incidentally detected nodules, precisely the bottleneck that automated segmentation and detection pipelines are best positioned to relieve. The growing role of liquid biopsy in tracking treatment response and resistance mutations adds a further computational dependency: as ctDNA monitoring becomes more routine, correlating longitudinal molecular changes with longitudinal imaging changes will require segmentation that is not just accurate on a single scan but consistent across a patient's entire treatment course. Likewise, the expansion of molecularly targeted therapies raises the clinical stakes of accurate, reproducible tumor

delineation, since radiomic features extracted from segmented tumor volumes are increasingly used to gauge treatment response and, in research settings, to predict which patients are likely to benefit from a given drug class. On the surgical side, the shift toward robotic and video-assisted resection places a premium on precise preoperative volumetric planning, since minimally invasive approaches leave less room for intraoperative correction than open surgery. Meanwhile, the persistent gap between curated benchmark performance and real-world, multicenter generalization suggests that segmentation research should weight external validation more heavily than incremental gains on saturated single-center benchmarks such as LUNA16, where multiple architectures already exceed 0.95 Dice. Future work that jointly optimizes for clinical workflow integration, such as reduced manual correction time, alongside standard overlap metrics is likely to matter more in practice than further architectural refinement alone.

VIII. CONCLUSION

Lung cancer remains the deadliest cancer worldwide, but the picture is no longer as bleak as it once was: declining smoking rates, expanding LDCT screening, minimally invasive and robotic surgery, and a transformed drug landscape built on targeted therapy, immunotherapy, antibody-drug conjugates, and liquid-biopsy-guided monitoring have together lifted five-year survival from roughly 18% to nearly 30% in recent years. Computationally, segmentation models have progressed just as quickly, moving from simple 2D U-Net baselines to attention-augmented CNNs, self-configuring frameworks such as nnU-Net, and transformer-based pipelines such as Swin UNETR. Whole-lung and lobe segmentation can now be considered largely solved, with Dice scores routinely above 0.95, while tumor and multi-lesion segmentation remain active research problems, particularly with respect to generalizing across heterogeneous, real-world multicenter data. Continued progress on both fronts will depend on expanding equitable access to screening, precision therapies, and molecular testing, and on improving segmentation robustness across the diverse clinical settings and tumor subtypes that curated public benchmarks do not fully represent.

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