E-Newsletter

ICFAI
School of Pharmaceutical Sciences

MEDXPLORE

Nurturing Every Breath, Sustaining Every Life

THEME - LUNGS HEALTH

Bi-Annual
E-NEWS LETTER
2026 April-September | Vol.-2 | Issue-1

About ICFAI School of Pharmaceutical Sciences (ISPS)

ICFAI School of Pharmaceutical Sciences (A Constituent School of the ICFAI University, Dehradun), a pioneer in pharmaceutical education, offers the **Bachelor of Pharmacy (B.Pharm)** Program. The ICFAI School of Pharmaceutical Sciences (ISPS) endeavors to deliver high-caliber education tailored to meet the demands of the pharmaceutical industry and address the broader healthcare needs of society, and achieve excellence in pharmaceutical education. The school embodies a student-centered educational model, fostering hands-on learning experiences and embracing a holistic approach to education.

The ICFAI School of Pharmaceutical Sciences features state-of-the-art laboratories, highly qualified, dedicated, and experienced faculty members, an enriched library, an information Centre and modular labs with high-tech instruments, meeting the standards and **approval of the Pharmacy Council of India**, New Delhi, to fulfil the practical and research needs of students.

About B. Pharm Program

The program is a four-year undergraduate program that provides students with a comprehensive understanding of the principles of Pharmacy and prepares them for a career in the pharmaceutical sciences. Its curriculum covers a wide range of subjects, including **Anatomy, Physiology, Pharmaceutical analysis, Pharmaceutical Chemistry, Pharmacology, Clinical Pharmacy, Communication Skills, remedial mathematics, remedial biology, biochemistry, pathophysiology, and Pharmaceutical Management**. This course also includes practical training in various aspects of pharmacy, such as drug design, formulation development, quality control, and regulatory affairs. Students also learn about the legal and ethical aspects of pharmacy practice.

The course equips students with the skills required to work in various sectors of the pharmaceutical industry, including research and development, manufacturing, marketing, and sales.

MESSAGE FROM THE VICE CHANCELLOR'S DESK

Prof. (Dr) R.K Singh
Vice Chancellor

Dear Students, Faculty Members, and Esteemed Stakeholders,

It gives me immense pleasure to extend my warmest congratulations to the ICFAI School of Pharmaceutical Sciences on the successful release of the 3rd edition of *MedXplore*, thoughtfully centered on the significant theme of **"Lungs Health"**. This subject is not only timely but also profoundly relevant, as respiratory disorders continue to pose a formidable challenge to global public health, affecting diverse populations across all age groups.

Lung health represents a dynamic and multifaceted domain encompassing prevention, early diagnosis, lifestyle optimization, and the continual evolution of innovative therapeutic interventions. As we engage with this crucial area, it becomes imperative to acknowledge the pivotal role of pharmaceutical sciences in advancing respiratory care and improving patient outcomes. Confronting the complexities of lung diseases necessitates sustained research efforts, interdisciplinary synergy, and an unwavering commitment to scientific innovation.

At ICFAI, we remain steadfast in fostering an intellectually stimulating ecosystem that promotes scientific curiosity, collaborative engagement, and pioneering research. *MedXplore* stands as a testament to this vision, serving as a vibrant platform for disseminating impactful knowledge, highlighting emerging trends, and encouraging critical discourse that inspires our academic fraternity to contribute meaningfully toward transformative healthcare solutions.

I take this opportunity to sincerely commend the editorial team, faculty members, and students for their exemplary dedication, creativity, and relentless pursuit of excellence in bringing out this edition. Your collective efforts continue to enhance the stature of the ICFAI School of Pharmaceutical Sciences as a hub of academic brilliance and research distinction.

With Best Wishes
Prof. (Dr.) R. K. Singh
Vice Chancellor
The ICFAI University, Dehradun.

MESSAGE FROM THE REGISTRAR

Prof. (Dr) R.C. Ramola
Registrar

Dear Students, Faculty Members, and Esteemed Stakeholders,

I am delighted to present the 3rd edition of *MedXplore*, the flagship newsletter of the ICFAI School of Pharmaceutical Sciences, thoughtfully devoted to the vital theme of **"Lungs Health"**. This subject is especially significant in the current global context, as respiratory diseases continue to rise, making awareness, prevention, and timely intervention more crucial than ever.

Lung health is an expansive and evolving domain that integrates preventive healthcare, early diagnosis, lifestyle modulation, and the advancement of sophisticated treatment modalities. This edition endeavours to holistically explore these dimensions, offering a nuanced understanding of the challenges, innovations, and opportunities that define modern respiratory care.

Through *MedXplore*, we strive to disseminate meaningful insights, spotlight recent advancements, and capture emerging trends within the realms of respiratory health and pharmaceutical sciences. It is our earnest hope that this edition not only enriches knowledge but also stimulates intellectual curiosity, deepens academic engagement, and inspires proactive contributions toward the advancement of lung health.

Let us collectively endeavour to strengthen knowledge systems, drive innovation, and contribute purposefully toward building a healthier and more resilient society. May this edition of *MedXplore* serve as a catalyst for insightful dialogue, fresh perspectives, and impactful progress in the domain of lung health.

With Best Wishes
Prof. (Dr.) R.C. Ramola
Registrar
The ICFAI University, Dehradun.

MESSAGE FROM THE PRINCIPAL

Prof. (Dr) Alka N Choudhary
Principal

Dear Students, Faculty, and Valued Readers

It gives me immense pleasure to present 3rd edition of *MedXplore*, dedicated to the vital theme of **"Lungs Health"** - a subject that has gained unprecedented global importance in recent years.

The lungs, delicate yet powerful, are central to sustaining life, enabling every breath that fuels our existence. In an era marked by rising air pollution, respiratory disorders, and post-pandemic challenges, safeguarding lung health has become not only a medical priority but also a societal responsibility. As future pharmacists, researchers, and healthcare professionals, our role extends beyond dispensing medicines; we are custodians of public health, advocates of prevention, and contributors to innovative therapeutic solutions.

This edition of *MedXplore* reflects the intellectual curiosity and scientific commitment of our students and faculty. It highlights a comprehensive overview of various respiratory diseases, emphasizing rational drug therapy, emerging research trends, and impactful awareness initiatives, while underscoring the critical importance of early diagnosis and lifestyle modifications in promoting and sustaining respiratory wellness.

At the ICFAI School of Pharmaceutical Sciences, we are committed to fostering a culture of excellence, innovation, and empathy. We continuously strive to equip our students with the knowledge and skills required to address contemporary healthcare challenges, including those related to pulmonary health.

I am confident that this newsletter will inspire readers to reflect, learn, and actively contribute toward building a healthier society.

Let us continue to breathe life into knowledge and transform it into meaningful action.

Wishing you all good health and a future filled with purpose and innovation.

Warm Regards
Prof. (Dr.) Alka N. Choudhary
Principal
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun.

MESSAGE FROM THE EDITORIAL BOARD

Dear Students, Faculty Members, and Esteemed Readers,

This newsletter examines the lungs' critical role in respiration and systemic homeostasis, integrating anatomy, disease pathogenesis, pharmacological innovations, and non-pharmacological therapies, while presenting the foundational science with clinical relevance, and reinforcing a commitment to academic excellence and impactful scientific engagement.

With Best Wishes
Ms. Anzla Shirin
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

I am pleased to present this edition of the newsletter, which highlights the importance of lung health and the impact of pollution, smoking, and respiratory diseases on millions of lives, while promoting awareness, healthy lifestyles, and scientific advancements.

With Best Wishes
Mrs. Myrnal Chamoli
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

I am pleased to present this edition, which reflects a spirit of resilience and growth, bringing insights into knowledge, research, and lung health to inspire continuous learning and self-improvement. As we move forward with clarity and determination, we remain committed to fostering progress and meaningful engagement together.

With Best Wishes
Mr. Naveen Sharma
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

I am honored to present this edition of the newsletter, which is for every student curious about pulmonary pharmacology, every healthcare professional looking to strengthen patient conversations, and every reader who simply wants to breathe better and live fuller. Whether you are a first-time reader or a long-time member of our *Medxplore* family, I encourage you to read with curiosity and share with care, because good health information, when it travels, saves lives.

With Best Wishes
Mrs. Santoshi Shah
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

MESSAGE FROM THE EDITORIAL BOARD

Dear Students, Faculty Members, and Esteemed Readers,

Our newsletter highlights the role of pharmaceutical sciences in advancing research, awareness, and holistic care for improved respiratory outcomes. Overall, the newsletter inspires scientific engagement and collective responsibility toward building a healthier society.

With Best Wishes
Dr. Subhajit Hazra
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

Breathing is often taken for granted until compromised, yet lung health remains fundamental to overall well-being as respiratory diseases increasingly affect populations worldwide. Rising pollution, lifestyle changes, and harmful exposures have led to a surge in conditions such as asthma, COPD, and infections, highlighting the need for awareness and preventive care.

This edition of the newsletter aims to provide meaningful insights and practical knowledge to promote respiratory health, emphasizing that informed choices can make every breath healthier.

With Best Wishes
Mr. Manish Nawani
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

This edition of *MedXplore* brings a comprehensive perspective on lung health, connecting fundamental science with clinical understanding and emerging treatment approaches. It underscores the growing impact of respiratory diseases while highlighting the importance of innovation, awareness, and interdisciplinary efforts in healthcare.

With Best Wishes
Mrs. Mansi Sharma
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

Dear Students, Faculty Members, and Esteemed Readers,

I am privileged to present *MedXplore* centered on lung health, offering a comprehensive blend of foundational insights, disease understanding, and evolving approaches in respiratory care. This edition reflects our continued commitment to bridging medical knowledge with public awareness, making complex concepts accessible and meaningful to a wide readership. We hope it inspires curiosity, informed thinking, and collective efforts toward advancing respiratory health and academic excellence.

With Best Wishes
Mr. Tushar Negi
Assistant Professor
ICFAI School of Pharmaceutical Sciences
The ICFAI University, Dehradun

LUNGS HEALTH

I. ANATOMY AND HISTOLOGY OF LUNGS

The lungs are a pair of highly elastic, spongy respiratory organs situated in the thoracic cavity. They are the principal organs of respiration, facilitating the exchange of oxygen and carbon dioxide between inspired air and blood. They occupy almost the entire thoracic cavity except the mediastinum and are separated from each other by this central compartment.

II. GROSS ANATOMY OF LUNGS

1. Shape, Size, and Position

The lungs are **cone-shaped**, with:

(A) Apex: Projects - 2–3 cm above the medial one-third of the clavicle into the root of the neck

(B) Base (Diaphragmatic surface): Concave and rests on the diaphragm

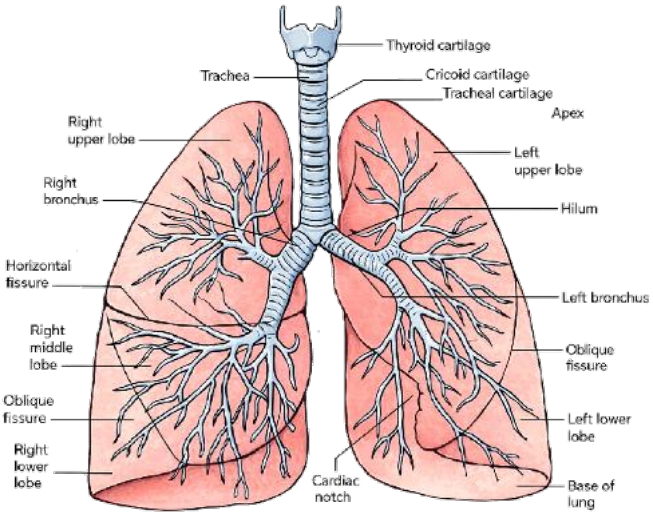
- **Right lung**: Larger, heavier, shorter, and wider
- **Left lung**: Smaller due to cardiac impression

The two lungs are separated by the **mediastinum**, which contains the heart, great vessels, trachea, oesophagus, and other vital structures. This anatomical separation ensures that the collapse of one lung (e.g., pneumothorax) does not necessarily impair the function of the other lung.

2.Surfaces of Lungs

Costal Surface: It is convex and is in direct contact with ribs and intercostal muscles

Mediastinal Surface: It is concave and contains hilum and shows impressions of heart, aorta and oesophagus. Left lung shows cardiac notch.



Diaphragmatic Surface (Base): It is concave and rests on diaphragm

3. Borders of Lung

- **Anterior Border**: Thin and shows cardiac notch (left lung)
- **Posterior Border**: Rounded and lies along vertebral column
- **Inferior Border**: Sharp and separates base from costal surface

4. Pleura and Pleural Cavity

Pleura: It is a serous membrane forming a closed sac around each lung. The layers of Pleura include a double-layered serous membrane known as the pleura:

1. **Parietal Pleura**: Lines the inner surface of the thoracic cavity. Subdivided into costal, diaphragmatic, mediastinal, and cervical parts
2. **Visceral Pleura** - Closely adheres to the lung surface. Dips into fissures and is inseparable from lung tissue

Pleural Cavity: The potential space between the

two pleural layers contains a thin film of 10–20 mL serous (pleural) fluid, which:

- Reduces friction during respiration
- Maintains surface tension, helping lungs remain expanded

Pleural Recesses

Costodiaphragmatic Recess: It is a potential space within the pleural cavity formed at the junction where the costal pleura reflects onto the diaphragmatic pleura**.**

Location:

- Situated inferiorly and posteriorly in the thoracic cavity
- Lies between: Ribs (costal pleura) and Diaphragm (diaphragmatic pleura)
- Extends from :
- Approximately the 8th to 10th ribs (mid-axillary line)
- More inferior posteriorly (up to 12th rib)

Costomediastinal Recess: It is a slit-like space located along the anterior border of the pleural cavity**,** created by the reflection of pleura from the inner surface of the thoracic wall (costal pleura) to the mediastinum (mediastinal pleura).

Location:

- Found anteriorly, behind the sternum
- More prominent on the left side due to the presence of the cardiac notch of the left lung
- Extends roughly from the 4th to 6th costal cartilage

5. Histology of Lungs

(I) LOBES, FISSURES, AND SEGMENTS

(A) FISSURES AND LOBES

a. Right Lung

Has 3 lobes**:**Superior, Middle, and Inferior lobes

Divided by 2 fissures**:**

● **Oblique fissure**: separates inferior lobe from superior and middle lobes

● **Horizontal fissure**: separates superior lobe from middle lobe

This makes the right lung larger and more subdivided**.**

b. Left Lung

Has 2 lobes**:** Superior and Inferior lobes

Divided by 1 fissure:

Oblique fissure: separates the two lobes

Special Features of Left Lung

● **Cardiac notch**:
A concave indentation on the anterior border to accommodate the heart.

● **Lingula**:
A tongue-like projection of the superior lobe. It is functionally equivalent (homologous) to the middle lobe of the right lung

(B) BRONCHOPULMONARY SEGMENTS

- Structural and functional units of lung
- Pyramidal in shape with apex toward hilum
- Each segment has: Segmental bronchus and Segmental artery
- Veins are intersegmental

Number of segments:

- Right lung: 10
- Left lung: 8–10

(C) HILUM AND ROOT OF LUNG

Hilum: It is the depression on mediastinal surface. It acts as the entry and exit point for structures such as bronchi, blood vessels, nerves, and lymphatics.

Root of Lung

- Main bronchus
- Pulmonary artery
- Pulmonary veins
- Bronchial vessels

- Lymphatics
- Autonomic nerves

(II) BRONCHIOLES AND BRONCHI

The bronchi and bronchioles are components of the conducting part of the respiratory system, responsible for carrying air from the trachea to the alveoli.

A. BRONCHI

The trachea divides into right and left primary bronchi

These further divide into:

Secondary (lobar) bronchi: There is one for each lobe

Tertiary (segmental) bronchi: It supply bronchopulmonary segments

Features:

- Walls contain cartilage plates to maintain airway potency
- Lined by pseudostratified ciliated columnar epithelium
- Presence of goblet cells for mucus secretion

B. BRONCHIOLES

- Smaller airways formed by repeated branching of bronchi
- Include:
- **Terminal bronchioles** – It is the end of conducting part
- **Respiratory bronchioles** – It is beginning of respiratory part

Features:

- No cartilage
- Lined by simple cuboidal epithelium
- Few or no goblet cells
- Contain smooth muscle that regulates airflow

C. BRONCHIAL TREE (TRACHEOBRONCHIAL TREE)

(III) ALVEOLI AND ALVEOLAR STRUCTURE

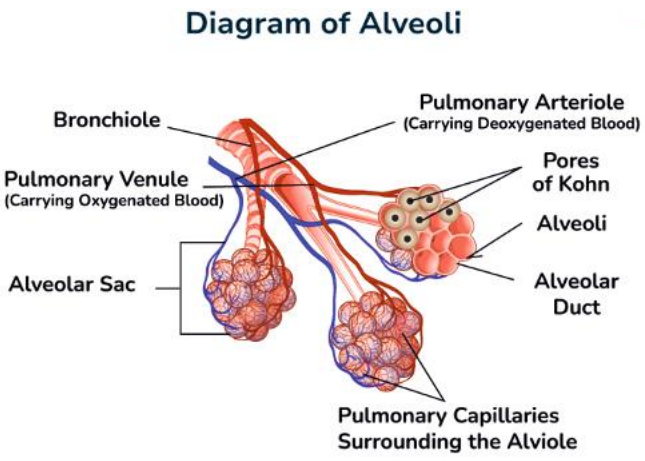
These structures form the terminal part of the respiratory system and are directly involved in gas exchange.

- Alveolar ducts terminate in clusters called alveolar saccules (alveolar sacs)
- These resemble a bunch of grapes
- Each alveolar sac contains 20–30 alveoli
- Diameter of each alveolus: 200–300 µm (0.2–0.3 mm)

The lungs contain approximately 300 million alveoli, providing a large surface area (~70–100 m^2) for gas exchange.

Differences between Bronchi and Bronchioles

Features	Bronchi	Bronchioles
Size	Large airway	Smaller airways
Position	Arise directly from trachea	Formed by branching of bronchi
Cartilage	Present	Absent
Epithelium	Pseudostratified ciliated columnar	Simple cuboidal
Goblet cells	Present	Absent
Smooth Muscles	Less prominent	Well developed
Lumen	Wider	Narrow
Function	Conduct air	Conduct air and regulate airflow
Role in respiration	Only conducting part	Terminal bronchioles – conducting; Respiratory bronchioles – start gas exchange



CELLS OF ALVEOLI

1. TYPE I PNEUMOCYTES

They are imple squamous epithelial cells that cover about 95% of alveolar surface area. They are extremely thin that allows rapid diffusion of O_2 and CO_2.

Main Function: Gaseous exchange

2. TYPE II PNEUMOCYTES (SEPTAL CELLS)

They are Cuboidal cells with microvilli and are fewer in number .They secrete pulmonary surfactant

Functions:

- Reduces surface tension → prevents alveolar collapse
- Helps in lung expansion
- Can regenerate Type I cells

3. ALVEOLAR MACROPHAGES (DUST CELLS)

These are free cells present in alveoli that perform phagocytosis

Function:

- Remove dust, microorganisms, and debris
- Provide immunity.

4. RESPIRATORY MEMBRANE (BLOOD-AIR BARRIER)

The respiratory membrane is the site of gas exchange and consists of:

- Alveolar epithelium (Type I cells)
- Fused basement membrane
- Capillary endothelium

Thickness: 0.5 µm**,** which is approximately $1/6^{th}$ the diameter of a red blood corpuscle, facilitating rapid diffusion of gases.

5. PARTS OF ALVEOLI

A. Alveolar Wall (Interalveolar Septum)

These are thin partition between adjacent alveoli

It contains**:** Type I and Type II pneumocytes, Capillaries, Elastic and collagen fibers. It

provides structure and facilitates gas exchange

B. Alveolar Lumen

It is the central air-filled space of the alveolus. This is the site where air is present for gas exchange

C. Respiratory (Alveolo-Capillary) Membrane

Formed by: Type I pneumocytes, Basement membrane, Capillary endothelium. It is the main site of O_2 and CO_2 diffusion

D. Pulmonary Capillaries

It is a dense network surrounding alveoli that bring deoxygenated blood and carry oxygenated blood away

E. Pores of Kohn

These are small openings between adjacent alveoli that allows collateral ventilation and equalize pressure

F. Surfactant Layer

It is a thin fluid layer lining alveoli. It is secreted by Type II cells. Helps in reducing surface tension and prevents collapse.

III. DISEASES OF LUNGS

1. OBSTRUCTIVE LUNG DISEASES

A. ASTHMA

Asthma is a chronic inflammatory disease of the airways characterized by recurrent episodes of wheezing, shortness of breath, chest tightness, and cough, associated with variable, usually reversible, airflow obstruction and bronchial hyper-responsiveness to various stimuli. It results from complex interactions between genetic susceptibility and environmental factors such as allergens, respiratory infections, air pollution, and occupational exposures.

Asthma is highly prevalent worldwide, affects both children and adults, and imposes a substantial burden due to impaired quality of life, frequent hospital visits, and, in severe cases, risk of respiratory failure and death. Early diagnosis and appropriate long-term management are essential to prevent exacerbations and airway remodelling.

SYMPTOMS

Typical symptoms of asthma include:

- Wheezing: High-pitched whistling sounds, especially during expiration.
- Dyspnea (shortness of breath): Often episodic, worse at night or early morning.
- Chest tightness or pressure: Sensation of constriction in the chest.
- Cough: May be dry or productive; often nocturnal or triggered by exercise, laughter, cold air, or allergen exposure.

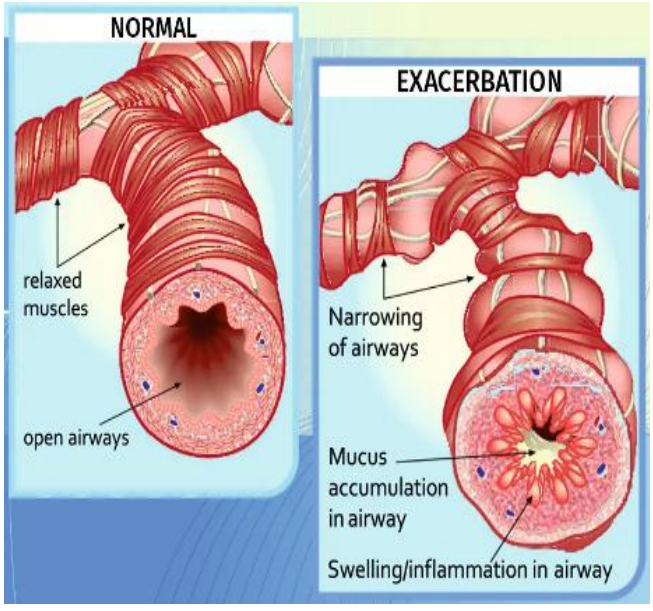
PATHOGENESIS

- **Chronic airway inflammation:** In many patients, type 2 (T2) inflammation predominates, involving Th2 lymphocytes, type 2 innate lymphoid cells (ILC2), eosinophils, mast cells, and production of cytokines such as IL-4, IL-5, and IL-13. These mediators promote IgE class switching, eosinophil recruitment/survival, mucus hypersecretion, and airway hyper-responsiveness.
- **Airway hyper-responsiveness (AHR)**: The inflamed airway smooth muscle contracts excessively in response to nonspecific stimuli (cold air, exercise, irritant gases, and allergens), leading to episodic bronchoconstriction and variable airflow limitation.
- **Structural changes (airway remodelling)**: Persistent inflammation can cause thickening of the basement membrane, sub-epithelial fibrosis, smooth muscle hypertrophy and hyperplasia, goblet cell hyperplasia, and increased mucus gland size. These changes contribute to fixed or less reversible airflow obstruction in long-standing disease.
- **Genetic and environmental interactions**: Multiple susceptibility genes (e.g., involved in immune regulation and

epithelial function) interact with environmental exposures such as allergens, tobacco smoke, viral infections, and air pollutants to initiate and perpetuate disease.

B. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable, and treatable chronic lung disease characterized by persistent respiratory symptoms and airflow limitation that is not fully reversible. The airflow obstruction is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, most commonly cigarette smoke. Pathologically, COPD encompasses two main components: chronic bronchitis (chronic productive cough with airway inflammation and mucus hypersecretion) and emphysema (abnormal, permanent enlargement of airspaces distal to the terminal bronchioles with destruction of alveolar walls without obvious fibrosis). Environmental exposures (e.g., biomass fuel smoke, occupational dusts and chemicals) and genetic factors such as alpha-1 antitrypsin deficiency also contribute. COPD is a major cause of morbidity, mortality, and healthcare burden worldwide, making early recognition and management essential.



SYMPTOMS

- **Dyspnea (shortness of breath):** Initially occurs on exertion and progressively worsens, eventually limiting daily activities. Patients often describe "heavy," "tight," or "air-hungry" breathing.
- **Chronic cough:** Often the earliest symptom; may be intermittent at first and then daily.
- **Sputum production:** Classically associated with chronic bronchitis; sputum may be mucoid or purulent, especially during exacerbations.
- **Wheezing and chest tightness:** Reflect airflow limitation and bronchial hyper-responsiveness.
- **Exercise intolerance and fatigue**: Due to ventilatory limitation, dynamic hyperinflation, and impaired gas exchange.
- **Exacerbations:** Acute worsening of respiratory symptoms (increased dyspnea, cough, sputum volume or purulence), frequently triggered by respiratory infections or environmental pollutants; they accelerate lung function decline and increase mortality.

PATHOGENESIS

The pathogenesis of COPD involves chronic inhalational injury leading to persistent inflammation and structural remodelling in both airways and lung parenchyma:

- **Chronic inhalational exposure:** Long-term exposure to tobacco smoke, biomass fuel combustion, or occupational pollutants causes epithelial injury and activation of innate and adaptive immune responses.
- **Airway inflammation and remodeling:** Activated epithelial cells, neutrophils, macrophages, and CD8+ T lymphocytes release cytokines, chemokines, proteases, and growth factors. These mediators drive goblet cell hyperplasia, mucus gland

enlargement, smooth muscle hypertrophy, and fibrosis of small airways, resulting in narrowed, obstructed bronchioles.

- **Protease–antiprotease imbalance and parenchymal destruction**: Excess proteolytic enzymes (e.g., neutrophil elastase, matrix metalloproteinases) overpower endogenous antiproteases (such as alpha-1 antitrypsin), leading to degradation of elastin and other extracellular matrix components. This causes destruction of alveolar walls (emphysema), loss of alveolar attachments, and reduced elastic recoil.
- **Oxidative stress:** Cigarette smoke and inflammatory cells generate reactive oxygen species that directly injure lung tissue, amplify inflammation, and inactivate antiproteases. Oxidative stress also impairs repair mechanisms and enhances mucus production.
- **Small airway collapse and gas trapping:** Loss of elastic recoil and structural support, combined with airway narrowing, promote expiratory airway collapse, air trapping, and hyperinflation, increasing the work of breathing and impairing gas exchange.
- **Systemic effects:** Chronic inflammation and hypoxemia can contribute to systemic manifestations such as skeletal muscle dysfunction, weight loss, osteoporosis, cardiovascular disease, and metabolic disturbances.

C. CHRONIC BRONCHITIS

Chronic bronchitis is a common clinical phenotype of chronic obstructive pulmonary disease (COPD), defined by chronic productive cough due to inflammation of the bronchi. It is most frequently caused by long-term cigarette smoking, but exposure to biomass fuel smoke, air pollution, occupational dusts/chemicals, and recurrent respiratory infections also contribute. Chronic bronchitis is associated with mucus hypersecretion, impaired mucociliary clearance, and airflow limitation, leading to recurrent infections, dyspnea, and reduced quality of life. Its high prevalence, association with COPD, and contribution to morbidity, mortality, and healthcare costs make it a major global health problem.

SYMPTOMS

- **Chronic productive cough:** Daily cough with sputum production, especially in the morning. Persists for ≥3 months per year for at least 2 consecutive years.
- **Sputum production:** Mucoid, mucopurulent, or purulent; volume often increases during exacerbations.
- **Dyspnea (shortness of breath):** Initially on exertion; may progress to dyspnea at rest in advanced disease or when COPD is established.
- **Wheezing and chest tightness:** Due to airway narrowing and mucus plugging.

PATHOGENESIS

- **Airway Inflammation:** Long-term exposure to cigarette smoke or other irritants activates airway epithelial cells and resident macrophages. Pro-inflammatory cytokines and chemokines (e.g., IL-8, TNF-α) are released. Neutrophils, macrophages, and CD8+ T lymphocytes are recruited into the bronchial mucosa. This chronic inflammatory response leads to structural changes in the airway walls.
- **Mucus Hypersecretion:** Hypertrophy and hyperplasia of submucosal mucous glands in the larger bronchi increase mucus production. Goblet cell metaplasia and hyperplasia extend into smaller bronchi and bronchioles. The Reid index (ratio of mucous gland thickness to total bronchial wall thickness) is increased, reflecting gland enlargement. Excess, thick mucus accumulates in the airways, contributing to cough and sputum production and predisposing to infection.

- **Impaired Mucociliary Clearance:** Chronic exposure to irritants causes damage and loss of ciliated epithelial cells. Ciliary dysfunction reduces the clearance of mucus and inhaled particles. Stagnant mucus provides a medium for bacterial colonization, leading to recurrent infections and further inflammation.
- **Airflow Limitation and Airway Remodeling:** Edema of the bronchial wall, smooth muscle hypertrophy, peribronchial fibrosis, and mucus plugging narrow the airway lumen. Small airways (bronchioles) become particularly narrowed and obstructed, causing expiratory airflow limitation. Over time, this contributes to the development of COPD with reduced FEV_1 and FEV_1/FVC on spirometry.

D. EMPHYSEMA

Emphysema is a chronic obstructive lung disease characterized by abnormal, permanent enlargement of airspaces distal to the terminal bronchioles, accompanied by destruction of alveolar walls, without obvious fibrosis. This structural damage leads to loss of elastic recoil, air trapping, and impaired gas exchange. Cigarette smoking is the principal cause, but occupational/environmental exposures and genetic factors (especially alpha-1 antitrypsin deficiency) are also important.

SYMPTOMS

TYPICAL SYMPTOMS

Progressive Dyspnoea: Initially on exertion, later at rest.

- Chronic cough: often dry or with scant sputum (less productive than in chronic bronchitis).
- Wheezing and chest tightness due to airflow limitation.
- Reduced exercise tolerance and easy fatigability.

Physical signs:

- Hyper inflated "barrel-shaped" chest.
- Use of accessory muscles, pursed-lip breathing, prolonged expiration.
- Decreased breath sounds, hyper resonant percussion.
- Low body weight and muscle wasting in advanced disease ("pink puffer" phenotype).
- Cyanosis and signs of right heart failure may appear in late stages or when combined with chronic bronchitis.

PATHOGENESIS

- **Protease–antiprotease imbalance**: Inhaled irritants (mainly cigarette smoke) recruit neutrophils and macrophages to the lungs.

  These cells release proteolytic enzymes (e.g., neutrophil elastase, matrix metalloproteinases) that digest elastin and other components of alveolar walls.

  Normally, antiproteases (especially alpha-1 antitrypsin) neutralize these enzymes. In smokers and in alpha-1 antitrypsin deficiency, this balance is tipped toward excess protease activity, causing progressive alveolar destruction.
- **Oxidative stress:** Cigarette smoke and activated inflammatory cells generate reactive oxygen species (ROS).

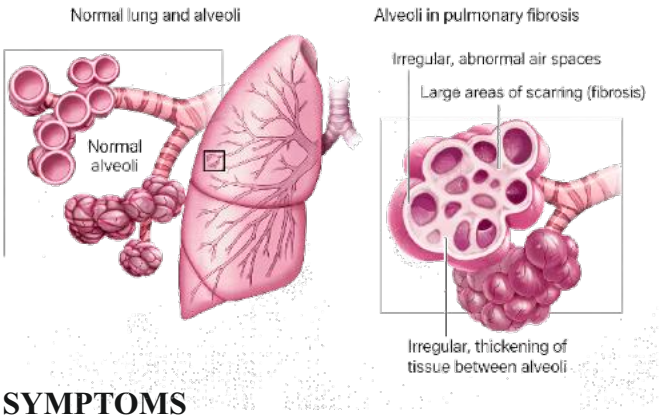
  ROS directly damage epithelial and endothelial cells and inactivate antiproteases (e.g., oxidizing alpha-1 antitrypsin), further enhancing proteolysis.
- **Chronic inflammation and remodeling:** Persistent low-grade inflammation leads to apoptosis and loss of alveolar epithelial and endothelial cells.

  The capillary bed is reduced, decreasing the surface area available for gas exchange.
- **Genetic and environmental factors:** Alpha-1 antitrypsin deficiency: causes early-onset, panacinar emphysema (especially in lower lobes).

Environmental exposures (biomass fuel, occupational dusts, air pollution) and host susceptibility genes modulate risk and severity.

2. PULMONARY FIBROSIS

Pulmonary fibrosis is a group of chronic, progressive interstitial lung diseases in which scarring of the lung interstitium distorts normal architecture, stiffens the lungs, and impairs gas exchange Idiopathic pulmonary fibrosis (IPF) is the prototype, defined as a fibrosing ILD of unknown cause, with a usual interstitial pneumonia (UIP) pattern and poor prognosis.



SYMPTOMS

- Progressive exertional dyspnea (shortness of breath on walking, then at rest).
- Persistent dry, hacking cough.
- Fatigue, reduced exercise tolerance, weight loss or poor appetite.
- On exam: bibasilar "velcro-like" inspiratory crackles and frequent digital clubbing in IPF.

PATHOGENESIS

- **Alveolar epithelial injury:** Repeated damage to alveolar epithelial cells in susceptible, often older individuals as the key trigger**.**
- **Aberrant wound healing:** Instead of normal repair, there is self-sustaining, dysregulated repair with myofibroblast proliferation and formation of fibroblastic foci**.**
- **Excess matrix and scarring:** Overproduction and disordered deposition of collagen and extracellular matrix cause thickened, fibrotic interstitium, honeycombing, and architectural distortion.
- **Progressive fibrosing phenotype**: Similar mechanisms occur in other ILDs (CTD-ILD, fibrotic hypersensitivity pneumonitis, NSIP), leading to a progressive-fibrosing ILD (PF-ILD) picture with shared behavior and treatment targets.

3. OCCUPATIONAL LUNG DISEASES

Occupational lung diseases are a group of respiratory conditions caused or made worse by inhaled exposures at work (dusts, fumes, gases, vapors, allergens, fibers). They can mimic common lung diseases seen in the general population, so they are often missed unless a work history is taken. Many are serious but almost all are preventable with proper control of workplace exposures.

SYMPTOMS

- Persistent cough, dyspnea, wheeze, chest tightness, sometimes sputum.
- Worse during work shifts and improve on weekends/holidays (especially asthma, HP).
- Delayed by years to decades after first exposure (silicosis, asbestosis, pneumoconiosis, cancers)

PATHOGENESIS

- **Inhaled workplace agents** (coal/silica dust, asbestos, metals, organic dusts, fumes, gases) deposit in airways/alveoli and overwhelm defenses.
- This triggers oxidative stress, inflammation, and immune responses, with cytokine release, macrophage and neutrophil activation, and sometimes specific immune sensitization (e.g., IgE in occupational asthma, granulomatous response in chronic beryllium disease or hypersensitivity pneumonitis).
- Chronic exposure leads to airway

remodeling (COPD/asthma), fibrosis and nodules (pneumoconioses, occupational ILD), or malignant transformation (lung cancer, mesothelioma).

4. INFECTIOUS LUNG DISEASES

A. PNEUMONIA

Pneumonia is an acute infection and inflammation of the lung parenchyma, mainly involving the alveoli and distal airways, leading to impaired gas exchange and respiratory symptoms. It is a leading cause of morbidity and mortality worldwide in all age groups, especially young children, older adults, and people with comorbidities.

SYMPTOMS

- Cough (often with sputum), fever, dyspnea, pleuritic chest pain.
- May have chills, fatigue, loss of appetite; in children and elderly, symptoms can be atypical (headache, abdominal pain, confusion, or absence of fever).
- Exam and imaging: tachypnea, crackles, bronchial breath sounds; chest X-ray with new infiltrate/consolidation is key for diagnosis.

PATHOGENESIS

- **Entry of pathogen**: Bacteria, viruses, fungi (and rarely parasites) reach the lower respiratory tract and are not effectively cleared.
- **Alveolar inflammation:** Pathogens multiply in the alveoli → local immune response with cytokine release, white blood cell influx, and exudate filling the air spaces, causing consolidation and impaired oxygen exchange.
- **Host response & severity**: Disease severity depends strongly on host immune status, age, and comorbidities, more than on pathogen alone.
- **Types by acquisition:**

  A. Community-acquired pneumonia (CAP): Acquired outside hospital.

  B. Hospital-acquired pneumonia (HAP) and **ventilator-associated pneumonia (VAP)**: Occur ≥48 h after admission or intubation, with more resistant pathogens.

B. TUBERCULOSIS

Tuberculosis (TB) is a chronic infectious disease caused by Mycobacterium tuberculosis, mainly affecting the lungs but potentially any organ. It remains one of the leading infectious killers worldwide, especially in low- and middle-income countries and in people with weakened immunity.

SYMPTOMS

- Cough >2 weeks, sometimes with blood (hemoptysis).
- Chest pain, breathlessness.
- Systemic:fever, night sweats, weight loss, fatigue.
- Extrapulmonary TB shows organ-specific features (e.g., abdominal pain in GI TB, neurological signs in TB meningitis)

PATHOGENESIS

- Spread by airborne droplets from coughing, sneezing or talking; bacilli reach alveoli and are taken up by macrophages.
- M. tuberculosis survives in macrophages by blocking phagosome–lysosome fusion, leading to intracellular replication and granuloma formation.
- Most infections become latent, but breakdown of immune control (HIV, diabetes, malnutrition, and immunosuppressive therapy) allows reactivation, cavitary lung disease and transmission.

5. LUNG CANCER

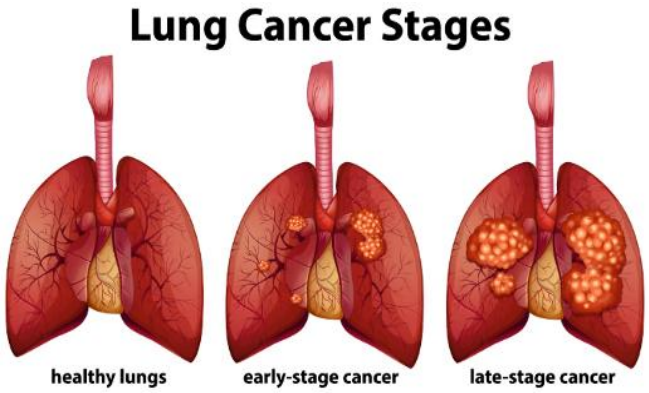
Lung cancer (lung carcinoma) is a malignant tumor originating from epithelial cells of the

tracheobronchial tree and distal lung parenchyma, characterized by uncontrolled proliferation, ability to invade surrounding structures, and to metastasize via lymphatic and blood vessels to distant organs.

It is the most frequently diagnosed cancer worldwide and the leading cause of cancer-related death in both men and women, accounting for ~13–15% of all new cancers but nearly 20% of all cancer deaths 453. About 85% of cases are non-small cell lung cancer (NSCLC) and ~15% are small cell lung cancer (SCLC), which differ in histology, biology and treatment.

In most cases, lung cancer develops as a primary bronchogenic carcinoma, arising from the bronchial/bronchiolar epithelium; primary tumors are almost always carcinomas (not sarcomas or lymphomas). Tumor growth begins locally within the lung (central or peripheral nodules/masses), then may extend to pleura, chest wall, mediastinum, and regional lymph nodes, and finally spread to brain, bone, liver and adrenal glands by metastasis if untreated.

Lung cancer is strongly linked to long-term exposure to carcinogens, especially tobacco smoke (responsible for ~75–90% of cases), but also radon, asbestos, other occupational and environmental pollutants, and genetic susceptibility. Because most patients present at an advanced stage, lung cancer is not only a localized lung disease but a systemic malignant syndrome with major impact on survival and quality of life.



SYMPTOMS

- **Respiratory:** Persistent cough or change in chronic cough, hemoptysis, dyspnea, wheeze, chest pain.
- **Systemic:** Weight loss, anorexia, fatigue, low-grade fever.
- **Local invasion:** Hoarseness (recurrent laryngeal nerve), dysphagia, superior vena cava (SVC) obstruction, pleural effusion.
- **Metastatic:** Bone pain, pathological fractures, neurological deficits, seizures, jaundice.
- **Paraneoplastic:** Hypercalcemia, SIADH, Cushing-like features, neuromuscular syndromes (especially SCLC).

PATHOGENESIS

- Major risk factor is tobacco smoke; others include radon, asbestos, air pollution and genetic susceptibility.
- Carcinogens induce DNA damage in bronchial epithelial cells, causing mutations (e.g., TP53, KRAS, EGFR, ALK) and disruption of cell-cycle control.
- Accumulation of genetic/epigenetic alterations leads to progression from metaplasia → dysplasia → carcinoma in situ → invasive carcinoma.
- Tumor cells acquire abilities for angiogenesis, invasion and metastasis through lymphatic and hematogenous spread.

6. VASCULAR LUNG DISEASES

Vascular lung diseases are disorders that primarily affect the blood vessels of the lungs (arteries, capillaries, veins), leading to abnormal pulmonary blood flow, gas exchange disturbance, and often pulmonary hypertension. These conditions can occur alone or as complications of chronic lung disease, heart disease, or systemic disorders, and are major causes of dyspnea, right heart failure, and death.

SYMPTOMS

Common: Exertional dyspnea, fatigue, reduced exercise capacity, sometimes syncope.

Right heart strain: Peripheral edema, jugular venous distension, hepatomegaly, signs of right heart failure in advanced PH.

Gas-exchange abnormalities: Hypoxemia, hypocapnia due to V/Q mismatch, dead space increase, or shunt (especially in AVMs).

PATHOGENESIS

- **Vascular remodeling:** Hypertrophy and fibrosis of arterial, capillary, and/or venous walls; loss or obliteration of small vessels →increased pulmonary vascular resistance.
- **Cellular and molecular changes in PAH**: Cancer-like proliferation, apoptosis resistance, mitochondrial/metabolic shift (Warburg state), inflammation, and BMPR2/EIF2AK4 and other genetic mutations.
- **Role of hypoxia and lung parenchymal disease**: Hypoxic vasoconstriction, destruction of vascular bed by fibrosis/ emphysema, and primary vasculopathy in ILD and sarcoidosis contribute to PH.
- **Thromboembolic and congenital mechanisms:** Organized thrombi, congenital heart disease, vein stenosis, and AVMs alter flow and pressures.

Pleural Diseases (Outer Lung Covering):

Pleural diseases affect the pleura, the thin double layer (visceral and parietal pleura) plus a small amount of fluid that separates lung from chest wall and allows smooth breathing motion. They are common, ranging from simple effusions to pneumothorax and malignant involvement, and create a major global health and economic burden.

SYMPTOMS

- Dyspnea (most frequent), worse with larger effusions or pneumothorax
- Pleuritic chest pain (sharp, worse on inspiration/cough)
- Cough (often dry)
- Reduced breath sounds, dullness (effusion) or hyperresonance (pneumothorax) on exam.

PATHOGENESIS

- Systemic or local factors disturb normal balance of pleural fluid formation and drainage, causing transudative (e.g., heart failure, cirrhosis) or exudative effusions (infection, malignancy, TB, PE).
- Infection (parapneumonic effusion, empyema) and malignancy trigger intense pleural inflammation, low pH/glucose, fibrin deposition and septation; can lead to unexpandable/trapped lung.
- Air entry into pleural space from lung, chest wall or procedures causes pneumothorax and lung collapse.
- Chronic inflammation, asbestos or autoimmune disease may produce pleural thickening and fibrosis, restricting lung expansion.

IV. PHARMACOLOGICAL TREATMENT FOR LUNG RELATED DISEASES

A. OBSTRUCTIVE LUNG DISEASES

Asthma, Chronic Bronchitis, Emphysema					
Disease	**Drug Class**	**Examples**	**Mode of Action**	**Effect**	**Limitations**
Asthma	β2-agonists (SABA/LABA)	Salbutamol, Formoterol	Stimulate β2 receptors → bronchodilation	Rapid relief of bronchospasm	Overuse → tolerance, tachycardia
	Corticosteroids (Inhaled)	Budesonide, Fluticasone	Anti-inflammatory, reduce airway hyperresponsiveness	Prevents exacerbations	Oral thrush, long-term effects
	Leukotriene modifiers	Montelukast	Block leukotriene receptors → ↓ inflammation	Useful in allergic asthma	Less effective than steroids
	Anticholinergics	Ipratropium	Block muscarinic receptors → bronchodilation	Add-on therapy	Less potent than β2 agonists
Chronic Bronchitis	Bronchodilators	Salmeterol, Tiotropium	Relax airway smooth muscles	Improve airflow	Symptomatic only
	Corticosteroids	Prednisolone	Reduce airway inflammation	Decrease exacerbations	Long-term side effects
Emphysema	Anticholinergics (LAMA)	Tiotropium	Block vagal tone → bronchodilation	Improve lung function	Does not reverse damage
	PDE-4 inhibitors	Roflumilast	Reduce inflammation by inhibiting PDE-4	Fewer exacerbations	GI side effects, costly

B. RESTRICTIVE LUNG DISEASES

Pulmonary Fibrosis, Occupational Lung Disease					
Disease	**Drug Class**	**Examples**	**Mode of Action**	**Effect**	**Limitations**
Pulmonary Fibrosis	Antifibrotic agents	Pirfenidone, Nintedanib	Inhibit fibroblast proliferation & collagen formation	Slow disease progression	Expensive, not curative
	Corticosteroids	Prednisolone	Anti-inflammatory	Temporary symptom relief	Limited long-term benefit
Occupational Lung	Bronchodilators	Salbutamol	Relax airway smooth muscle	Symptom relief	No effect on fibrosis
	Corticosteroids	Prednisone	Reduce inflammation	Useful in early stages	Ineffective in advanced fibrosis
	Oxygen therapy		Improves oxygen delivery	Relieves hypoxia	Does not treat cause

C. INFECTIOUS LUNG DISEASES

Pneumonia, Tuberculosis					
Disease	**Drug Class**	**Examples**	**Mode of Action**	**Effect**	**Limitations**
Pneumonia	β-lactam	Amoxicillin	Cell wall synthesis inhibition	First line therapy	Resistance & allergy
	Macrolides	Azithromycin	Protein synthesis inhibition	Atypical coverage	QT prolongation & may upset G.I.
	Fluoroquinolones	Levofloxacin	DNA gyrase Inhibition	Bactericidal activity against respiratory pathogens leading to resolution of infection	Resistance risk
Tuberculosis(TB)	Hydrazides	Isoniazid	Inhibits the synthesis of mycolic acids, which are essential components of the mycobacterial cell wall	Bactericidal against actively dividing Mycobacteriu m tuberculosis organisms	Resistance develops if used as monotherapy; requires combination therapy; risk of hepatotoxicity and peripheral neuropathy
	Rifamycins	Rifampicin	Inhibits DNA-dependent RNA polymerase	Bactericidal; sterilizing effect	Strong enzyme inducer → drug interactions
	Nicotinamide analogues	Pyrazinamide	Disrupts membrane energetics and fatty acid synthesis	Active in acidic environments (intracellular organisms)	Hepatotoxic; limited duration use
	Ethylenediamines	Ethambutol	Inhibits arabinosyl transferase → impaired cell wall synthesis	Prevents resistance development	Optic neuritis (vision impairment)
	Aminoglycosides(secor line)	Streptomycin	Inhibits protein synthesis (30S ribosome)	Bactericidal against extracellular mycobacteria	Ototoxicity, nephrotoxicity
	Diarylquinolines(MDR-TB)	Bedaquiline	Inhibits mycobacterial ATP synthase	Effective in MDR/XDR-TB	QT prolongation risk; restricted use Cardiotoxicity, hepatotoxicity
	Nitroimidazoles	Delamanid	Inhibits mycolic acid synthesis	MDR-TB treatment	QT prolongation
	Nitroimidazooxazines(Novel)	Pretomanid	Disrupts cell wall synthesis & respiratory poisoning	Used in combination regimens for XDR-TB	Hepatotoxicity, neuropathy

D. Neoplastic and Other Lung Conditions

Lung Cancer, Vascular Lung Diseases, Pleural Diseases					
Disease	**Drug Class**	**Examples**	**Mode of Action**	**Effect**	**Limitations**
Lung Cancer	Chemotherapy	Cisplatin	DNA damage	Cytotoxic effect resulting in reduction of tumor burden	High toxicity, nephrotoxicity
	Targeted therapy	Erlotinib	EGFR inhibition	Mutation specific effect	Resistance and rashes
	Immunotherapy	Pembrolizumab	PD-1 blockade	Enhances immunity	Expensive, autoimmune reactions
Vascular Lung Diseases	Anticoagulants	Heparin	Inhibits clotting factors	Prevents clot extension	Bleeding risks, requires monitoring
	Thrombolytics	Alteplase	Clot dissolution by converting plasminogen to plasmin, resulting in degradation pf fibrin clots	Rapid dissolution of thrombus	Bleeding risks
Pleural Diseases	NSAIDs	Ibuprofen	COX inhibition	Pain relief of pleuritic chest pain and inflammation	G.I. toxicity
	Diuretics	Furosemide	Fluid removal	Effusion management	Electrolyte imbalance

V. NON-PHARMACOLOGICAL TREATMENT OF LUNG DISEASES

Lung diseases are a major global health burden, affecting millions of individuals across all age groups. While pharmacological therapies play a crucial role, non-pharmacological interventions are equally important in improving respiratory function, reducing symptoms, and enhancing quality of life. These approaches focus on lifestyle modification, rehabilitation, environmental control, and supportive therapies.

A. ASTHMA

Non-pharmacological strategies aim to reduce triggers and improve breathing control:

- Avoidance of allergens (dust, pollen, smoke, pet dander)
- Breathing exercises (e.g., diaphragmatic breathing, pursed-lip breathing)
- Regular physical activity (improves lung capacity)
- Patient education on trigger identification and inhaler technique
- Weight management to reduce airway inflammation

B. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD), CHRONIC BRONCHITIS & EMPHYSEMA

These chronic conditions require long-term supportive care:

- Smoking cessation (most critical intervention)
- Pulmonary rehabilitation programs (exercise training + education)
- Nutritional support (high-protein diet to prevent muscle wasting)
- Oxygen therapy (in severe cases)
- Chest physiotherapy (to clear mucus)
- Energy conservation techniques for daily activities

C. PULMONARY FIBROSIS

- Pulmonary rehabilitation to improve endurance
- Breathing techniques to reduce breathlessness

- Oxygen therapy (when required)
- Psychological support (for anxiety and depression)

D. OCCUPATIONAL LUNG DISEASES

(i.e., Silicosis, Asbestosis)

- Avoidance of exposure (dust, chemicals, fibers)
- Use of personal protective equipment (PPE) (masks, respirators)
- Workplace safety measures and ventilation
- Regular health screening and monitoring

E. INFECTIOUS LUNG DISEASES

1. PNEUMONIA

- Adequate rest and hydration
- Chest physiotherapy (for secretion clearance)
- Nutritional support to boost immunity
- Breathing exercises to improve lung expansion

2. TUBERCULOSIS (TB)

- Isolation (in active cases) to prevent transmission
- Proper nutrition (protein-rich diet)
- Ventilation and sunlight exposure (reduces bacterial survival)
- Patient counseling and adherence support

F. NEOPLASTIC AND OTHER LUNG CONDITIONS

1. LUNG CANCER

- Smoking cessation
- Nutritional therapy
- Psychological and palliative care
- Pulmonary rehabilitation (post-surgery or during therapy)

2. VASCULAR LUNG DISEASES

(e.g., pulmonary hypertension)

- Exercise training under supervision
- Oxygen therapy
- Lifestyle modifications (low-salt diet, fluid balance)

3. PLEURAL DISEASES (OUTER LUNG COVERING)

(e.g., pleural effusion, pleuritis)

Positioning techniques to ease breathing

- Breathing exercises
- Thoracic physiotherapy
- Drainage procedures (non-drug supportive care)

Non-pharmacological management plays a vital complementary role in treating lung diseases. Interventions such as smoking cessation, pulmonary rehabilitation, environmental control, nutrition, and patient education significantly improve outcomes and quality of life. A multidisciplinary approach combining both pharmacological and non-pharmacological therapies ensures optimal respiratory health.

Key Intervention	Benefit
Smoking cessation	Slows disease progression
Pulmonary rehabilitation	Improves lung function & endurance
Breathing exercises	Reduces breathlessness
Nutrition	Enhances immunity & strength
Environmental control	Prevents disease exacerbation

VI. RECENT ADVANCES IN THERAPEUTICS FOR LUNG HEALTH

A. KEY PULMONARY TRIALS IN 2025-26

Trial Name (Phases of Trial)	**Therapeutic Area/Focus**	**Key Question/Objective**
FIBRONEER-ILD (Phase III) *[NCT05321082]*	Progressive Pulmonary Fibrosis (PPF)	Role of Nerandomilast in Patients with Progressive Pulmonary Fibrosis
TETON (Phase III) *[NCT05255991]*	IPF	Whether inhaled Treprostinil provides antifibrotic benefit and slows disease progression.
MATINEE (Phase III) *[*NCT04133909 *]*	Chronic Obstructive Pulmonary Disease (COPD)	Role of Mepolizumab to Prevent Exacerbations of COPD with an Eosinophilic Phenotype
BOREAS and NOTUS (Phase III) *[NCT03930732, NCT044566673]*	COPD	Does add-on Dupilumab treatment improve health-related quality of life and respiratory symptoms in patients with COPD and type 2 inflammation?
BATURA (Phase IIIb) *[NCT05505734]*	Asthma	Is as-needed Albuterol–budesonide therapy effective against mils asthma?
PATHWAY (NCT02054130, Phase IIb); **NAVIGATOR:** (NCT03347279, Phase III)	Asthma	Ability of tezepelumab treatment to restore normal lung function in patients with severe, uncontrolled asthma with abnormal lung function
NAVIGATOR: (NCT03347279, Phase III)	Pulmonary Arterial Hypertension (PAH)	Effect of Tezepelumab on Sino-Nasal Outcome Test (SNOT)-22 Domain and Symptom-Specific Scores in Patients with Severe, Uncontrolled Asthma and a History of Chronic Rhinosinusitis with Nasal Polyps.
ZENITH (Phase III)	Advanced PAH	Can add-on therapies improve outcomes in high-risk PAH populations?
EndTB / BEAT-TB (Phase II/III) *[]*	Drug-resistant Tuberculosis	Are shorter, all-oral regimens safer and more effective than conventional TB therapy?
ALIENTO (Phase IIb) *[NCT05037929]*	COPD	Evaluation of efficacy and safety of Astegolimab (anti-ST2) in COPD
Phase IIa proof-of-concept trial *[NCT04968574]*	IPF	Assessing the safety and efficacy of Taladegib, in IPF in a phase 2a, proof-of-concept trial

B. KEY APPROVALS IN PULMONARY MEDICINE OR THERAPY IN 2025

Recently FDA-Approved Drugs

Drug (Generic Name)	Company	Indication / Use
Jascayd (nerandomilast)	Imported (IQVIA / Relevant Sponsor)	Idiopathic Pulmonary Fibrosis (IPF) & Progressive Pulmonary Fibrosis (PPF)
Brinsuprii (brensocatib)	Insmed	Non-cystic fibrosis bronchiectasis (first disease-modifying therapy)
Exdensur (depemokimab)	GSK	Severe eosinophilic asthma (ultra-long acting)
Nucala (mepolizumab)	GSK	Add-on maintenance for eosinophilic COPD
Hernexeos (zonterglitinib)	Boehringer Ingelheim	HER2-mutant advanced non-small cell lung cancer
Ibrotzi (taletrectinib)	Innovent /AnHeart	ROS1-positive locally advanced or metastatic NSCLC
Emrelis (telisotuzumab vedotin)	AbbVie	MET-overexpressing non-squamous NSCLC
Papizmeos (zoapoagene imadenovec) —	--	Recurrent respiratory papillomatosis
Omlyclo (omalizumab-igec)	IQVIA RDS (India)	Interchangeable biosimilar for asthma / CRSwNP

Recent Drug Approvals by CDSCO - SEC

CDSCO

SEC Pulmonary

Drug Name / Combination	Company	Indication
Sotatercept (45mg, 60mg)	Imported (IQVIA/ Relevant Sponsor)	Add-on therapy for PAH (Group 1) to reduce mortality / hospitalization
Indacaterol + Glycopyrronium + Mometasone Furoate	M/s Lupin Limited	COPD Maintenance
Glycopyrronium + Vilanterol + Fluticasone Furoate	M/s Glennark Pharmaceuticals	COPD Maintenance therapy
Benralizumab	AstraZeneca Pharma India	Add-on for Relapsing/ Refragiabic Gramopnits with Polyanglits (EEPA)
Acebrophylline + Erdosteine	M/s Macleods Pharmaceuticals	COPD / Chronic Rhronhtits (Mucolytic / Anti-inflanmatary)
Bilastine + Montelukast	M/s Ravenbhel Heaithcane	Allergic rhinitis & asthma
BI 1015550	IQVIA RDS (india)	Progressive Fihrosing Interstitial Long Besase (PF-LD)

Recent pulmonary drug approvals from the FDA (top panel) and CDSCO–SEC Pulmonary, India (bottom panel). The FDA section includes 9 therapies for conditions such as IPF, COPD, severe asthma, NSCLC, and rare respiratory diseases. The CDSCO section highlights 7 approved drugs and combinations for indications including PAH, COPD, EGPA, allergic rhinitis, and PF-ILD. **Abbreviations:** FDA: Food and Drug Administration; CDSCO: Central Drugs Standard Control Organization; SEC Pulmonary: Subject Expert Committee; GSK: GlaxoSmithKline; RDS: Respiratory Distress Syndrome; COPD: Chronic Obstructive Pulmonary Disease; NSCLC: Non-Small Cell Lung Cancer; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; EGPA: Eosinophilic Granulomatosis with Polyangiitis; PAH: Pulmonary Arterial Hypertension

FUTURE DIRECTIONS OF PULMONARY MEDICINE & LUNG HEALTH

Artificial Intelligence & Digital Health

- AI enables early diagnosis via imaging (CT, X-ray, EHR analysis)
- Supports personalized treatment plans & predictive analytics
- Remote monitoring using wearables improves outcomes

Precision & Personalized Medicine

- Tailored therapies for COPD, asthma, and lung cancer
- Predictive models for disease progression & drug response

90%+ Higher accuracy in treatment via AI precision medicine

Regenerative Medicine & Stem Cell Therapy

- **Stem cell-based** regeneration of damaged lung tissue
- Potential alternatives to lung transplantation

Artificial & Bioengineered Lungs

$2.47B → $7.63B market growth from 2024 –2035

AI-Driven Drug Discovery

- AI identifies new drug targets & accelerates clinical trials
- Promising candidates (e.g, IPF therapies) entering trials

Advanced Diagnostics & Imaging

87.6% Diagnostic accuracy for lung cancer using deep learing models

Preventive & Public Health Strategies

- Focus on air-pollution control & climate-health
- Digital screening programs & community-based intervenitions

Integrated & Remote Care Models

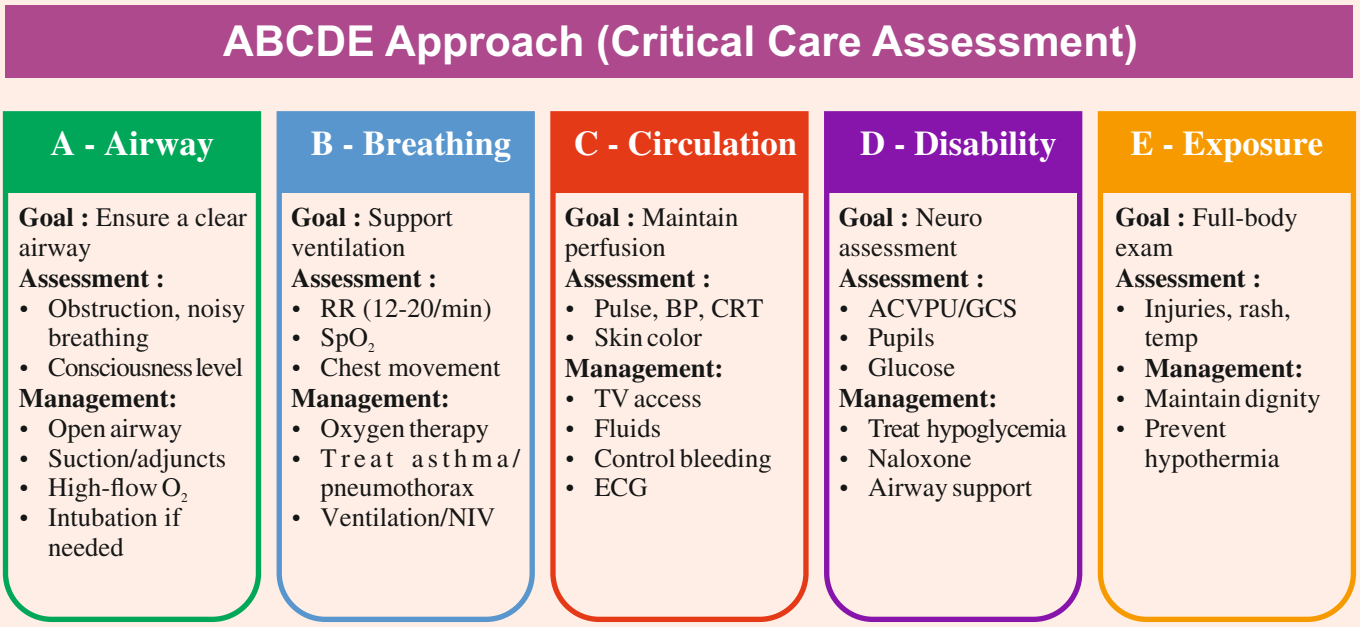
~60% Chronic lung patients benefit from remote monitoring

KEY TAKEAWAY

The future of pulmonary medicine is shifting towards AI-driven, persondized, and regenerative care-transforming lungs from reactive treatment to proactive, precision health.

D. THE ABCDE OF PULMONARY HEALTH

The management of all deteriorating or critically ill patients follows a standardized and systematic approach based on the Airway, Breathing, Circulation, Disability, and Exposure (ABCDE), framework, ensuring simultaneous assessment and treatment. A complete initial evaluation should always be performed, followed by continuous reassessment to monitor patient status and response to interventions. Life-threatening conditions must be identified and treated immediately before progressing to subsequent steps. It is essential to evaluate the effectiveness of each intervention and modify management accordingly. Clinicians should recognize the need for additional support early and promptly call for appropriate help. Effective care relies on a coordinated team-based approach, allowing multiple interventions, such as monitoring and intravenous access—to occur simultaneously. Clear and structured communication, using tools like SBAR or RSVP, is crucial for optimal patient management. The primary goal of initial treatment is to stabilize the patient and achieve early clinical improvement, thereby providing time for definitive diagnosis and further management. It is also important to remember that interventions may take a few minutes to show effect, and reassessment should be performed after a brief interval.



Systematic assessment of critically ill patients using the ABCDE framework:

A: Airway (green) focusing on airway patency and emergency management;

B: Breathing (blue) outlining respiratory assessment and oxygen therapy;

C: Circulation (red) highlighting perfusion, vital signs, and fluid resuscitation;

D: Disability (purple) addressing neurological evaluation using ACVPU/GCS and glucose monitoring; and

E: Exposure (orange) emphasizing full-body examination and prevention of hypothermia. Each section includes key goals, assessment parameters, and management steps. A footer reinforces core principles: continuous reassessment, prioritization of life-threatening conditions, and early call for assistance

E. EAT RIGHT FOR HEALTHY PULMONARY HEALTH

Eating well for healthy lungs

HEALTHY LUNGS FOR LIFE

Eating well is important for lung health and overall health. Food provides fuel to all parts of the body so that they can work properly, including the lungs.

How can we eat well?

Just like a car, if we put the wrong type of fuel into our body, then it will not work as well.

Top tips for eating well for healthy lungs:

1 Eat a variety of food

We need to eat a mixture of foods as they provide different nutrients that our body needs. Some food groups, we need more of, such as fruit and vegetables.

2 Eat the colours of the rainbow

Eating a variety of colourful fruit and vegetables as part of a balanced diet.

3 Limit foods high in fat, salt and sugar

Try to limit these types of foods to no more than once per day and not every day. These foods may increase the risk of asthma and wheezing and reduce your body's chance of fighting off infections.

Too much salt has also been linked with high blood pressure. You can lower salt in your diet by choosing low-salt or no-added salt options and use herbs and spices to add flavours to food.

4 Eat more fibre

When you eat a lot of fibre, you are less likely to develop lung conditions over the course of your life.

- 2 slices of wholemeal bread **(6.6g of fibre)**
- A jacket potato **(4.7g fibre)**
- Handful of almonds **(3.8g fibre)**
- 2-3 tablespoons of wholegrain rice **(2.7g fibre)**
- 1 apple **(2.4g fibre)**

European dietary guidelines recommendations

5 Find the right balance

Your breathing system has to work harder and you increase your chance of developing some lung conditions.

Eating more than your body needs can lead to your body storing excess weight.

You are not getting enough energy and you are more at risk of infections and breathing difficulties.

Eating less than your body needs can lead to being underweight.

Dietary recommendations for maintaining healthy lungs emphasizing balanced nutrition, variety in food intake, increased consumption of fruits, vegetables, and fibre, while limiting fats, salt, and sugar. Practical tips such as "eating the rainbow," portion balance, and maintaining optimal energy intake to support respiratory health and overall well-being are highlighted. However, dietary recommendations alone are not sufficient for maintaining healthy lungs, and emphasis should be given on eating according to the existing lung condition, for nutritional intervention to have its maximum efficacy. Consult a doctor to understand which dietary patterns or regimens are suitable for your lung condition.

Building on the basics

Support breastfeeding

What we eat as a baby is important for lung development. Breastfeeding can help reduce lung infections and support healthy lung growth in childhood and into the teenage years.

Higher levels of vitamin D have been linked with better lung health.

People with lung conditions like asthma and COPD often have low vitamin D. Our bodies make vitamin D from sunlight, and it is also found in foods like oily fish and eggs. In places with little sunlight, especially in winter, it is a good idea to take a vitamin D supplement and choose foods that are high in vitamin D.

Keeping your gut healthy

We can keep our gut healthy by eating lots of fibre, along with probiotics (found in yoghurt and sauerkraut) and prebiotics (found in wholegrains, bananas, garlic). When we have a balance of healthy bacteria in the gut and the lungs, it can reduce swelling (inflammation) in the lungs, which is often the cause of wheezing, shortness of breath, chest pain and coughing.

Eating well with a lung condition

There is not one set diet plan that will be helpful for all and it is important to follow the advice of your healthcare professional, or a dietician, before making any specific changes.

Boost your immune system to lower your chance of infection. You can do this through a balanced diet, maintaining a healthy weight and getting a mixture of vitamins and minerals including vitamins A, C, D and E and zinc.

Drink plenty of water to help thin the mucus in the lungs and keep it moving, lowering your chance of infection. Sipping water in small amounts, can also help with dry mouth if you experience this.

Get the right amount of energy from food if you live with a lung condition, like COPD. This is important to avoid being underweight. Try eating small amounts often, and avoiding foods that trigger bloating, if this feeling prevents you from eating well.

Your healthcare provider or dietician can recommend specific diet information for you to ensure you are getting the energy and nutrients you need from food.

This document was produced with the aim of teaching the public about a healthy diet to maintain healthy lungs and to help healthcare professionals explain the benefits of good nutrition. It was produced by the European Lung Foundation (ELF) as part of the Healthy Lungs for Life campaign, with the support of the ELF patient network, the ERS Health and Environment Committee and healthcare professionals Megan Jensen and Neil Williams.

For further reading and to access references for this information, please visit:
www.europeanlung.org/en/information-hub/keeping-lungs-healthy/nutrition/

HEALTHY LUNGS FOR LIFE
ERS ELF

F. TAKE HOME MESSAGE

Breathe better today, live healthier tomorrow!

Every breath you take shapes your health and future. By choosing clean air, staying smoke-free, keeping your body active, and prioritizing regular health check-ups, you empower your lungs to function at their best. Minimize exposure to pollutants, adopt simple breathing exercises, and never ignore persistent respiratory symptoms. When you care for your lungs today, you invest in a stronger, healthier tomorrow—because better breathing is the foundation of better living.

SALIENT FEATURES

ICFAI School of Pharmaceutical Sciences

- Modem and state-of-the-art infrastructure
- Modular labs with hi-tech instruments
- Highly qualified, dedicated, and experienced faculty members.
- Enriched library and information center
- Research-driven education.
- Novel pedagogy methods
- Wi-Fi enabled campus.
- Career counselling for higher studies & competitive examinations
- Assistance for bank loans to students in need

BEST PRACTICES

- Leaner's centric approach in teaching
- Peer Tutoring
- Peer-review of teaching
- Student empowerment through student council
- Quality Circles to inculcate team work & positive attitude.

WHY ICFAI?

- Center of excellence m Technical & Professional Education
- Attractive Scholarships
- Nurture an attitude of problem-solving
- Imbibe skills of creativity

Program Offered	B.Pharm
Full Form	Bachelor of Pharmacy
Duration	4 Years
Course Level	Undergraduate
Examination Type	Semester
Eligibility Criteria	Passed 10+2 examination conducted by the respective state/central government, with English as one of the subjects and Physics, Chemistry, Mathematics (P.C.M) and or Biology (P.C.B / P.C.M.B.) as optional subjects individually.
Admission Process	Merit basis

CELEBRATING OUR RECENT SUCCESS

SPONSORSHIP BY UTTARAKHAND STATE COUNCIL FOR SCEINCE & TECHNOLOGY (UCOST)

SPONSOR

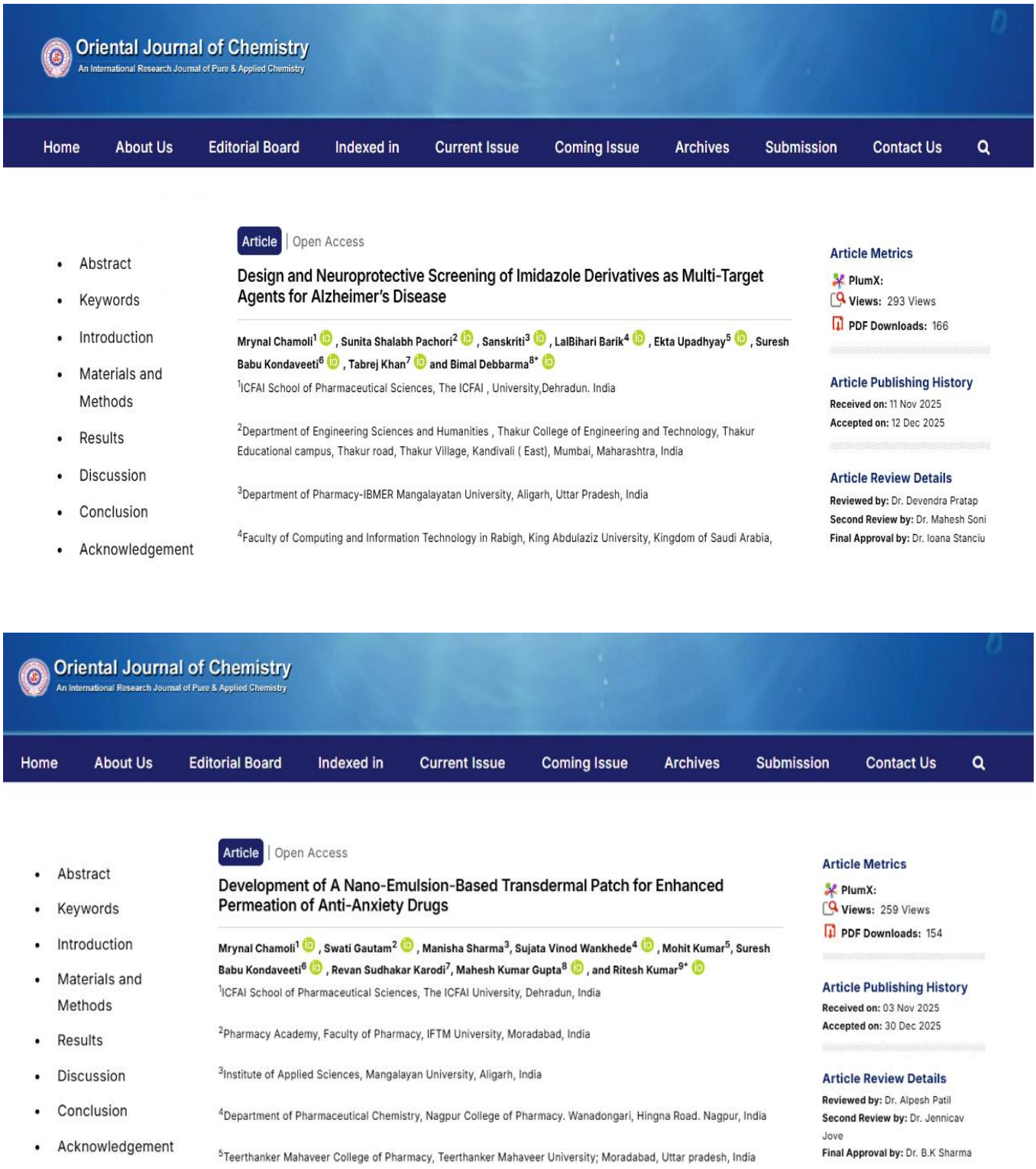
A generous sponsorship of ₹1, 00,000 was sanctioned by Uttarakhand State Council for Science and Technology (UCOST) to ICFAI School of Pharmaceutical Sciences for the successful conduct of a National Seminar. This prestigious support reflects the Council's confidence in the institution's academic excellence and research capabilities. It stands as a recognition of the university's consistent dedication to innovation and quality in pharmaceutical sciences. The sponsorship further strengthens our commitment to fostering scientific exchange and collaborative learning. This achievement marks a proud milestone for the institution and its academic community.

A research grant of ₹6, 00,000 lakhs has been sanctioned by the Uttarakhand State Council for Science and Technology (UCOST), Dehradun, to the ICFAI School of Pharmaceutical Sciences, The ICFAI University Dehradun, for a research project. This prestigious grant is a testament to the University's expertise, dedication, and commitment to high-quality research.

COLLABORATION WITH PHARMACEUTICAL INDUSTRIES

Collaborations with pharmaceutical industries, in the form of MoUs (i.e., with East India African overseas, Ishaanav Nutraceuticals) are being strengthened to enhance the student internship program. These partnerships will offer valuable industry exposure and significantly expand job opportunities for students.

RECENT PUBLICATIONS, ICFAI School of Pharmaceutical Sciences (ISPS)



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Design and Neuroprotective Screening of Imidazole Derivatives as Multi-Target Agents for Alzheimer's Disease

Mrynal Chamoli[1], Sunita Shalabh Pachori[2], Sanskriti[3], LalBihari Barik[4], Ekta Upadhyay[5], Suresh Babu Kondaveeti[6], Tabrej Khan[7] and Bimal Debbarma[8*]

[1]ICFAI School of Pharmaceutical Sciences, The ICFAI , University,Dehradun. India

[2]Department of Engineering Sciences and Humanities , Thakur College of Engineering and Technology, Thakur Educational campus, Thakur road, Thakur Village, Kandivali (East), Mumbai, Maharashtra, India

[3]Department of Pharmacy-IBMER Mangalayatan University, Aligarh, Uttar Pradesh, India

[4]Faculty of Computing and Information Technology in Rabigh, King Abdulaziz University, Kingdom of Saudi Arabia,

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Development of A Nano-Emulsion-Based Transdermal Patch for Enhanced Permeation of Anti-Anxiety Drugs

Mrynal Chamoli[1], Swati Gautam[2], Manisha Sharma[3], Sujata Vinod Wankhede[4], Mohit Kumar[5], Suresh Babu Kondaveeti[6], Revan Sudhakar Karodi[7], Mahesh Kumar Gupta[8], and Ritesh Kumar[9*]

[1]ICFAI School of Pharmaceutical Sciences, The ICFAI University, Dehradun, India

[2]Pharmacy Academy, Faculty of Pharmacy, IFTM University, Moradabad, India

[3]Institute of Applied Sciences, Mangalayan University, Aligarh, India

[4]Department of Pharmaceutical Chemistry, Nagpur College of Pharmacy. Wanadongari, Hingna Road. Nagpur, India

[5]Teerthanker Mahaveer College of Pharmacy, Teerthanker Mahaveer University; Moradabad, Uttar pradesh, India

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PERSPECTIVE article

Front. Space Technol., 06 January 2026
Sec. Space Exploration
Volume 6 - 2025 | https://doi.org/10.3389/frspt.2025.1725575

Enabling living in space through modern innovations in space medicine: a perspective on tissue-on-a-chip technology

Subhajit Hazra [1,2] Sibsankar Palit [1,3,4*] Thais Russomano [3,4,5]
G Gaurab Ghosh [6] P Polash Sannigrahi [7]

1. LIFE-To & Beyond Foundation®, Barasat/Kolkata, West Bengal, India
2. ICFAI School of Pharmaceutical Sciences, ICFAI University, Dehradun, Uttarakhand, India

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Research article

Development and evaluation of eugenol-loaded transethosomal gel as a potential topical therapy for onychomycosis: *In Vitro* dose determination, drug release profiling, and antifungal activity against *Trichophyton rubrum*

Ishu Garg [a,*], Shreya Rawat [b,*], Urmi Chaurasia [b], Tushar Negi [c], Shivani Rawat [d], Madhu Verma [c], Iti Chauhan [e]

Review Article

Decoding the Skin Micro-Immune Milieu, Homeostasis, and Keratinocyte Trafficking in Psoriatic Disease

Ishu Garg , Neelam Singh , Shivani Rawat , Tushar Negi , Urmi Chaurasia & Punnet Gupta

Published online: 08 Apr 2026

Cite this article https://doi.org/10.1080/08820139.2026.2651752 Check for updates

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Biomass-Derived Biochar Materials as Sustainable Energy Resources and Their Assessment

Biomass-Derived Biochar Materials as Sustainable Energy Resources and Their Assessment • Book Chapter • 2026 •
DOI: 10.2174/9798898811891125010l_3

Thapliyal, Sanskriti [a]; Kothari, Deepshikha [a]; Choudhary, Alka N [b]; Sharma, Neetu [c]; Bhawana [c]; +2 authors

[a] Department of Biotechnology, Graphic Era (Deemed to be) University, Uttarakhand, Dehradun, India

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Abstract

The rise in global warming, environmental pollution, and population growth has significantly increased energy consumption, leading to greater dependence on fossil fuels. Biomass and its derivatives offer substantial potential as renewable energy sources, making them promising alternatives for sustainable energy conversion, storage, and production. Through pyrolysis, biomass produces a porous, carbon-rich solid material called biochar. A green and sustainable platform for preparing various functional carbon materials is provided by biochar materials derived from biomass. The various types of biomass exhibit distinct physical and chemical properties. In this chapter, a summary of various methods used for preparing different types of biomass derived biochar materials is provided. An investigation into the numerous potential applications of biomass in environmental reduction and its mechanisms has been conducted. Further assessment of these materials, including their properties and the challenges involved, is also discussed.

(12) PATENT APPLICATION PUBLICATION

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(43) Publication Date : 23/01/2026

(54) Title of the invention : NOVEL BIOACTIVE PHYTO-SYNTHETIC FORMULATIONS AND THEIR PHARMACOLOGICAL APPLICATIONS IN DRUG DELIVERY SYSTEMS

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(71)**Name of Applicant :**
1)Dr. Pawan Singh
Address of Applicant :Associate Professor, Pharmacy Academy, IFTM University, Moradabad, Uttar Pradesh, India, Pin Code- 244001 Uttar Pradesh India
(72)**Name of Inventor :**
1)Dr. Pawan Singh
2)Ms. Rachana Belwal
3)Ms. Priya Pandey
4)Dr. Vipin Kumar Sharma
5)Dr. Hemendra Gautam
6)Dr. Pragya Prashant Gupta
7)Dr. Jatin Jaiswal
8)Dr. Gyanendra Kumar Sharma
9)Ms. Malavika P.S
10)Ms. Santoshi Shah
11)Mr. Shobhit Sharma
12)Dr. Javed Siddiqui

(57) Abstract :
The present invention relates to novel bioactive phyto-synthetic formulations comprising plant-derived bioactive compounds integrated with synthetic polymeric carriers for enhanced drug delivery systems. The formulations utilize phytochemicals extracted from medicinal plants combined with biodegradable synthetic polymers to create hybrid nano-particulate systems exhibiting superior bioavailability, controlled release kinetics, and targeted therapeutic action. The invention encompasses methods of preparing these formulations through green synthesis techniques, resulting in stable colloidal dispersions with particle sizes ranging from 50 to 500 nanometers. The phyto-synthetic composites demonstrate enhanced solubility of hydrophobic drugs, improved cellular uptake, reduced toxicity, and prolonged circulation time in biological systems. The formulations find applications in treating various diseases including cancer, diabetes, inflammatory disorders, and microbial infections. The invention provides pharmaceutical compositions with improved therapeutic efficacy compared to conventional drug delivery systems while maintaining biocompatibility and environmental sustainability through the use of natural plant-based components and biodegradable synthetic materials.

No. of Pages : 20 No. of Claims : 10

Event Organized by ICFAI School of Pharmaceutical Sciences (ISPS)

I. Inauguration of SDG Calendar

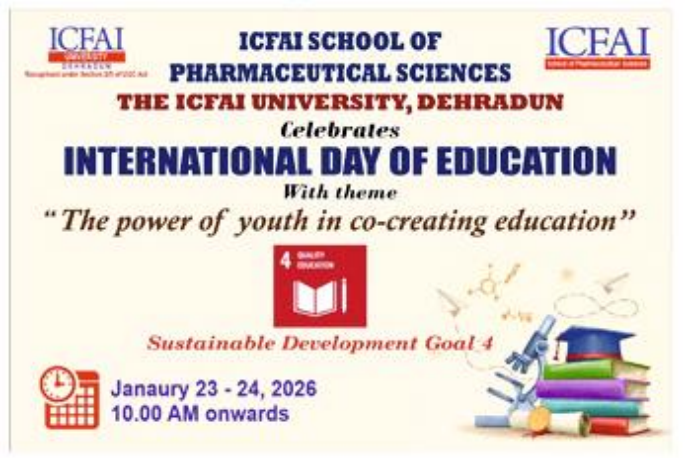
The inauguration of the e-Calendar on the Sustainable Development Goals (SDGs) was also conducted by the ICFAI School of Pharmaceutical Sciences, The ICFAI University, Dehradun. The initiative highlights the institution's commitment towards sustainability and social responsibility. The calendar showcases all 17 SDGs, emphasizing the importance of awareness and collective action in achieving these global goals. It reflects a continued effort to integrate sustainable practices within the academic community.

"Be the Reason the World Gets Better"

Inaugural ceremony of e- Calendar 2026

II. International Day of Education

International Day of Education
"Education for All, Empowering Every Mind"



Celebration of
International Day of Education

Plantation Drive

School Visits: Awareness Program

Visit to Slum Area

III. Celebration of National Pharmacy Education Day

National Pharmacy Education Day
"Legacy that inspires, Innovation that transforms"

National Pharmacy Education Day

Felicitation of Guest Lecturer
Mr. Pankaj Tiwari

Guest Lecture Delivered by an eminent speaker
Mr. Pankaj Tiwari

Idea Pitching Competition
'Rx-Thon' & 'Debate Competition'

Prize Distribution Ceremony

Collaboration with East African (India)
Overseas: MOU Signing

IV. 3 Days Hands on Training Program on Pharmaceutical Techniques

"Equipping Future Pharmacists with Advanced Laboratory Skills"

Inaugural ceremony on 3 days hands-on Training

Interactive Sessions by faculty Members

Hands-on Training by faculty Members

Valedictory Session

V. International Day of Sport for development and peace.

Students Participation in Sports Event
"Play Beyond Limits, Unite Beyond Differences"

Inaugural ceremony on the International Day of Sport for Development and peace

Sports & Fun Activities

Prize Distribution Ceremony

The ICFAI University
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