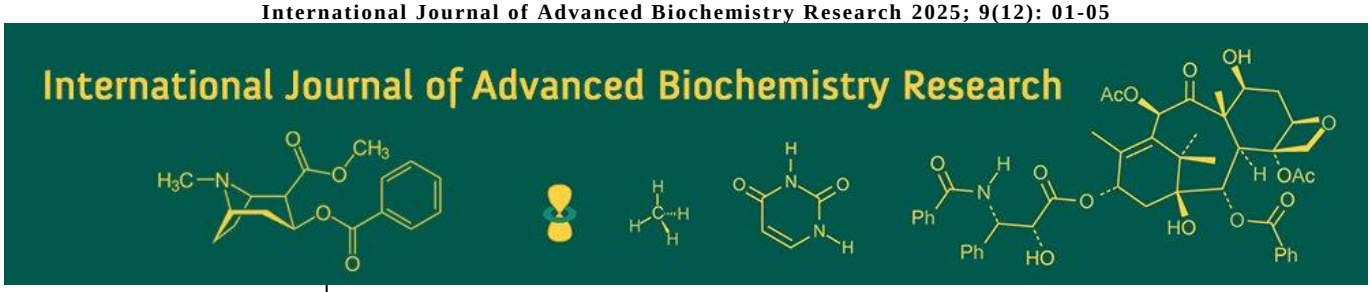

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Alka Suman
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Adesh Kumar
Veterinary Extension Officer, State Government, Madhya Pradesh, India

SK Gupta
Associate Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Yogita Pandey
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Anchal Keshri
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Dimpee Singh Gonge
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Shashi Tekam
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Jabalpur, Madhya Pradesh, India

Rashmi Kulesh
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Jabalpur, Madhya Pradesh, India

Bulbul Shivhare
Post Graduate student (Pursuing) College of Veterinary Science & A. H., NDVSU, Jabalpur, Madhya Pradesh, India

Corresponding Author:
Alka Suman
Assistant Professor, College of Veterinary Science & A. H., NDVSU, Mhow, Madhya Pradesh, India

Architecture of lungs of domestic animals with reference to pulmonary surfactant

Alka Suman, Adesh Kumar, SK Gupta, Yogita Pandey, Anchal Keshri, Dimpee Singh Gonge, Shashi Tekam, Rashmi Kulesh and Bulbul Shivhare

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Abstract
Mammalian lungs (pulmones), a paired organ that occupies the majority of the thoracic cavity, are not identical in shape or size, with the right being significantly larger than the left. The lung is very elastic, flexible, and spongy. It floats in water and crepitates when pressed between the thumb and finger. The lung's elasticity and strain from external air pressure cause it to instantly collapse to around one-third of its original size and lose its normal structure when the thoracic cavity is opened. The anatomy of the lung is covered, with a particular emphasis on lung parenchyma. The lung parenchyma mainly consists of multiple air-containing channels and intervening fine structures, such as alveolar ductal lumens and alveoli, as well as alveolar septa and small pulmonary arteries, which account for 10% of total parenchymal volume. The structural morphology and three-dimensional architecture of the alveolar ducts and alveoli are closely explored, with a focus on their functional organization within the terminal respiratory unit. This is followed by a succinct description of the bronchial circulation and pulmonary lymphatic system, both of which play critical roles in tissue perfusion, fluid balance, and lung immune defense. The pulmonary surfactant is a lipoproteinaceous secretory complex made up of lipids and particular surfactant-associated proteins that the lungs' alveolar type II (AT-II) pneumocytes produce and secrete. It is a highly surface-active substance that creates a thin layer at the alveolar air-liquid interface, reducing surface tension and maintaining alveolar stability while preventing collapse during respiration. Throughout the respiratory cycle, pulmonary surfactant is essential for preventing alveolar collapse and shielding the lungs from infections and mechanical damage brought on by inhaled pathogens and foreign particles. It is essential for maintaining the structural integrity and functional stability of the alveolar architecture as well as for the effective diffusion of breathing gases.

Keywords: Architecture, epithelium, lungs, microanatomy, pulmonary surfactant, respiratory tract

Introduction
The respiratory tract consists of the nasal cavity, pharynx, larynx, trachea, bronchi, terminal bronchioles, and alveoli. Functionally, it is separated into two major regions: conducting airways, which assist air movement to and from the lungs, and respiratory airways, which are specialized for gas exchange inside alveolar structures (Hiemstra *et al.*, 2019) [29]. These hollow tubular structures are lined with epithelia ranging from columnar to squamous cells, facilitating airflow and providing protective functions (Jeffery and Reid, 1975; Hollenhorst *et al.*, 2011) [33, 30]. The respiratory system conditions inhaled air by warming, humidifying, and filtering particulates. Additionally, the upper airways contribute to olfaction and thermoregulation, especially in panting species (Kuehn, 2013) [37]. The lungs are internal, fluid-lined, gas-filled organs that undergo periodic inflation and deflation to aid in breathing, despite anatomical and developmental differences among species. The lower respiratory tract's epithelium is generated from the endodermal layer of the embryonic foregut, whereas the nasal cavity's epithelium is produced from the ectoderm (Harmans *et al.*, 2015) [25]. Both lungs reside within their pleural sacs, connected solely at the hilum to the mediastinum, allowing mobility within the thoracic cavity (Seadler *et al.*, 2020) [48]. Alveoli, comprising the lung parenchyma, mediate gas exchange and adapt structurally to environmental variations (Maina, 2000) [41]. The alveolar interstitium contains collagen and elastic fibers, fibrocytes, pericytes, macrophages, lymphocytes, and plasma cells, contributing to tissue integrity and immune defense (Hewitt and Lloyd, 2021) [28].

Pulmonary surfactant, a lipoprotein compound released by alveolar type II cells through lamellar bodies, is required for lung function (Eurell and Frappier, 2007) [17]. It lowers alveolar surface tension by diffusing across the air-liquid interface, stabilizing alveoli, and avoiding collapse during expiration (Walther, 2019; Milad and Morissette, 2021) [51, 42]. Dipalmitoylphosphatidylcholine, phosphatidylglycerol, and cholesterol are among the most important lipid components. Surfactant proteins, particularly SP-A and SP-D, influence immunological responses by limiting pathogen infections and controlling immune cell activation (Choi *et al.*, 2020) [8].

Architecture of Lungs of Domestic Animals

Anatomy of lungs

Respiratory tract of mammal divided into extrapulmonary area i.e. outside of lungs & intrapulmonary area present inside lungs (Dyce, 2010) [14]. Generally, mammals, the right major bronchus differentiates into four lobar bronchi (to cranial, middle, caudal & accessory lobes), whereas the left gives rise to two lobar bronchi (to cranial & caudal lobes). The trachea and lungs receive cartilage and connective tissue from the thoracic splanchnic mesoderm (Akers and Denbow, 2013) [3]. The lungs and trachea receive respiratory epithelium from endoderm. Throughout the prenatal and postnatal stages, bronchial branching persists. The endoderm turns into a thin epithelium, and terminal branches become hollow, dilated, and sac-like (terminal sacs). Septa that divide the terminal sacs form alveoli (Fletcher and Weber, 2013) [20]. Due to the hypertrophy (increased size) of alveoli and air pathways, new alveoli and bronchial trees form after birth. (Table 1). Production of pulmonary surfactant start in terminal gestation i.e. neonates born prematurely may have insufficient amount of surfactant, which results in labored breathing (Islam *et al.*, 2020) [31].

Table 1: Development of lung in the gestational days

Stage of lung development	Cow	Horse	Pig	Dog
Embryonic stage: bronchial tree division	50	50	55	25-30
Pseudoglandular stage: lungs appear glandular tissue	50-120	50-190	50-80	32+
Canalicular stage: development of respiratory bronchioles, increases vascularity and pneumocytes differentiation	120-180	190-300	80-92	47+
Terminal sac stage: alveoli formation	180-240	300+	92-110	55+

Source: Textbook of Veterinary Anatomy (Dyce, 2010) [14]

Lungs and pleural membrane

The lung is encapsulated by a layer of connective tissue covered by mesothelial cells called pulmonary pleura. The reflection of the diaphragmatic pleura and the costal pleura from the sternum and vertebral column combine to generate the mediastinal pleura. The mediastinal pleura is mirrored onto the lung surface as the lung invaginates into the pleural sac, where it continues as the pulmonary (visceral) pleura. In horses, the mediastinum may become fenestrated as a result of postnatal fenestrations that permit contact between the left and right pleural cavities. Carnivores, on the other hand, have a thin and delicate mediastinal pleura; these interpleural communications are infrequent in sheep and nonexistent in oxen and goats (Evans & Miller, 1993) [18].

Broncheal tree

The bronchial tree is divided into two parts based on function: the intrapulmonary area with two airways (bronchi and bronchioles) comprises approximately 6% of the lung, and the respiratory airways for gas exchange consist of respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli comprise approximately 85% of the lung; the remaining 9-10% of the lung comprises of pleura, intrapulmonary nerves, pulmonary veins, arteries, and bronchial arteries (Konig *et al.*, 2009) [36].

Conducting airways: (Transport of air)

- Principle bronchi (primary)
- Lobar bronchi (secondary)
- Segmental bronchi (tertiary)
- Sub segmental bronchi
- True bronchiole
- Terminal bronchiole

Respiratory airways: (Gaseous exchange)

- Respiratory bronchiole
- Alveolar ducts
- Atria
- Alveolar sacs
- Pulmonary alveolus

Air passages in the lungs

Club cell/bronchiolar exocrine cells/Clara cells are dome-shaped cells without cilia present in bronchioles. Similar to the lung surfactant component, these cells may release glycosaminoglycans to shield the bronchiole lining. They are also in charge of cleaning the lungs of dangerous compounds that are breathed. Evans and Miller (1993) [18].

Microscopic Details of Lungs			
Region	Support	Epithelium	Cell types
Trachea and primary bronchi	C-rings of hyaline cartilage, smooth muscles and connective tissue	Ciliated pseudostratified columnar	Ciliated, goblet and basal
Secondary and tertiary bronchi	Plates of cartilage with smooth muscles helically oriented and dense connective tissue	Ciliated pseudostratified columnar	Ciliated, goblet, and basal cells
Primary bronchioles	Smooth muscle helically oriented and dense connective tissue	Simple columnar to simple cuboidal	Ciliated and non-ciliated, Clara cells
Terminal bronchioles	Smooth muscle and dense connective tissue with strong elastic fiber	Simple cuboidal	Ciliated and non-ciliated and Clara cells
Respiratory bronchioles	Smooth muscle bundles and dense connective tissue with strong elastic fiber	Simple cuboidal and simple squamous	Clara cells, type I and type II alveolar cells
Alveolar ducts	Smooth muscle bundles and dense connective tissue with strong elastic fiber	Simple squamous	Type I, type II alveolar cells and macrophages
Alveolar sacs and Alveoli	Loose connective tissue with strong elastic fiber	Simple squamous	Type I, type II alveolar cells and macrophages

Source: Sistema respiratorio. In: Tratado de Histologia Veterinária (Samuelson, 2007) [46]

3. Microanatomy of Alveoli

Alveoli constitute the lung parenchyma for gaseous exchange. These open into the alveolar sac, alveolar duct or respiratory bronchiole. Inter alveolar septa separate the adjacent alveoli containing a capillary plexus. Alveolar epithelium comprises type I, which is squamous cells and constitute 97% whereas type II alveolar epithelial cells that are cuboidal, constitute 3% of the alveolar septal surface. Septal or alveolar pores are lined by squamous epithelial cells and permit air & macrophages to pass from one alveolus to another. **(**Eurell and Frappier, 2007) [17]. Type II alveolar epithelial cells have more cell organelles and are metabolically active, while type I alveolar epithelial cells have fewer cell organelles and are less metabolically active (Maina, 2000 & Ridge *et al.*, 2003) [41, 45]. At the alveolar level, AT-I cells lower tissue resistance and permit free gas passage (Maina, 2000) [41]. The lamellar bodies found in AT-II cells are where the pulmonary surfactant is synthesized, stored, and secreted (Jeffery & Reid, 1975) [33].

Function AT-I and AT-II cells

Alveolar type I (AT-I) cells actively regulate ion, water, and macromolecule transport across the alveolar epithelium, allowing the alveolar lining fluid to maintain its composition and volume. The alveolar type II (AT-II) cells act as progenitor (stem) cells for the regeneration of AT-I cells (Kauffman *et al.*, 1947) [35] and are essential for alveolar fluid balance, epithelial repair, apoptotic cell clearance, immunological control, and host defense. These cells also connect with other alveolar cells via direct intercellular contacts and indirect signaling processes that use soluble mediators (Borok *et al.*, 2002) [6].

Fluid Alveolar Lining

A thin layer of fluid covers the respiratory epithelium, acting as an interface between the surface of the epithelial cells and the air in the respiratory tract. The underlying cells receive both physical and immunological protection from this fluid layer, which is made up of mucus, immunoprotective proteins, phospholipids, and pulmonary surfactant (Seadler *et al.*, 2020) [48]. The alveolar lining fluid (ALF) is the part of this fluid that covers the alveolar epithelium. Changes in this fluid's quantity or composition can reduce the effectiveness of gas exchange, resulting in hypoxia and hypoxia-induced cellular stress—phenomena that are frequently linked to a number of pulmonary conditions. While the alveolar lining fluid of the distal airways is rich in surfactant, which is essential for preserving alveolar stability and lowering surface tension, the airway surface liquid of the upper respiratory tract has higher concentrations of mucus and immune-protective proteins (Coppens *et al.*, 2007) [12]. The alveolar lining fluid protects the underlying cells from desiccation, infections, and tissue damage while also facilitating gas transport. Ion and water channels in diverse cells regulate the fluid content of the alveolar lining. A decreased surfactant with a lower volume of fluid lining causes lung atelactasis, whereas a decreased surfactant with an increased fluid causes pulmonary edema (Hollenhorst *et al.*, 2011) [30].

4. The Surfactants and Surface Tension

Surface-active agents, sometimes known as surfactants, are substances that lower surface tension. Surface tension occurs at the interface of solid-liquid, liquid-liquid, or liquid-gas phases when uneven intermolecular forces operate on molecules at the boundary (Harwood, 1987) [27]. This effect causes molecules at the interface to be pulled inward, lowering surface area and hence decreasing interactions between the two phases. Surfactant molecules are amphipathic, meaning they have both a hydrophilic (water-attracting) and a hydrophobic (water-repelling) area, allowing them to position themselves at interfaces and effectively reduce surface tension (Bzdek *et al.*, 2020) [7]. "stick or stay together "cohesive force is the action of like molecules sticking together, being mutually attractive. Surface tension is defined as the cohesive force of attraction that exists between molecules at the interface of two distinct mediums. Surfactants, being amphipathic molecules, orient themselves at these interfaces to make a thermodynamically stable film, thereby minimizing intermolecular attraction and reducing surface tension (Glasser & Mallampalli, 2012) [23]. In the lungs, a reduction in alveolar surface tension by pulmonary surfactant facilitates the expansion of alveoli during inspiration and supports efficient gaseous exchange by preventing alveolar collapse and keeping structural stability (Seadler *et al.*, 2020) [48].

Pulmonary Surfactant

Pulmonary surfactant increases *lung compliance* facilitating proper ventilation. Maintain the volume of fluid lining the alveoli and size of the alveoli in different phases of the respiratory cycle

Laplace law, $P = 2T/r$

P-deflating pressure, T-surface tension, r-radius of alveoli

Surface tension increases as the radius of the alveoli increases, and vice versa. The surfactant in the lungs changes cyclically to maintain low surface tension during inspiration and prevent alveolar collapse during expiration (Creuwels *et al.*, 1997) [13]. Cyclical alterations, such as structural shape and composition. During breathing, the lung capacity constantly expands and contracts (Akella and Deshpande, 2013) [2]. According to Agassandian and Mallampalli (2013) [1], pulmonary surfactant creates a layer on the alveolar epithelium that lowers surface tension at the air-fluid contact on the alveolar surface. Alveolar expansion and gas exchange are made possible by this decreased alveolar surface tension (Seadler *et al.*, 2020) [48].

Functions of pulmonary surfactant

In neonates, expand the lungs against hydrostatic forces when they take their first breath. Surfactant contributes to decreased airway potency and improved mucociliary clearance. Surface tension forces also draw fluid from capillaries into alveolar gaps. Surfactants prevent fluid collection and maintain the airways dry by lowering these forces (Lazarov *et al.*, 2001) [39]. Maintaining structural integrity (alveolar size), lung compliance, the elasticity of lung tissue after each breath, preventing atelactasis, balancing hydrostatic pressure in neonates at the moment of first breath, keeping the airways dry, and host defense, etc. (Glasser and Mallampalli, 2012) [23].

Surfactant Composition

According to the animal species, level of lung development, and physiological requirements, pulmonary surfactant

composition (table 03) and function differ (Han and Mallampalli, 2015) [24].

Surfactant = 90% lipid +10% protein

Phospholipid Composition

The phospholipids' molecular structure PL molecules have an amphipathic structure: a three-carbon glycerol backbone is joined to two hydrophobic, nonpolar fatty acid (FA) chains and a hydrophilic, polar head group. The primary constituents that give surfactants their capacity to reduce surface tension are phospholipids. The most prevalent phospholipid, phospholipid choline, is primarily composed of saturated molecular species. At least 50% of PC molecular species are dipalmitoyl-PC (DPPC), which is essential for attaining the greatest possible drop in surface tension (Shelley *et al.*, 1984; Notter, 2000) [49, 43].

Protein Composition

Alveolar type II cells produce all four surfactant proteins. Airway cells such as Clara and submucosal cells also produce SP-A, SP-B, and SP-D (Wang *et al.*, 2020) [52]. Furthermore, SP-A and SP-D have been found in various extrapulmonary sites, including the brain, salivary glands, lacrimal glands, heart, kidney, pancreas, and male and female reproductive tracts (Baatz *et al.* 2001) [32]. There are four types of surfactant-associated proteins: SP-A, SP-B, SP-C, and SP-D. The most prevalent protein is SP-A, followed by SP-B, SP-C, and SP-D (Johansson and Curstedt, 1997) [34]. SP-A and SP-D are hydrophilic proteins whose primary role is to assist in innate pulmonary immune responses (Wright, 2005) [54]. SP-B and SP-C are hydrophobic proteins that regulate surfactant metabolism. SP-B and SP-C interact substantially with surfactant PLs, enhancing their ability to effectively reduce surface tension (Kuroki and Voelker, 1994) [38]. SP-B exists in the alveoli as a dimer with amphipathic characteristics, allowing it to interact with the surfactant PL. Sano and Kuroki (2005) [47] describe SP-C as the smallest and most hydrophobic surfactant protein. It has a transmembrane a-helix and two palmitoyl groups that interact with the PL side chains. (Weaver, Conkright, 2001) [53]. A schematic illustration of surfactant host defense functions, mostly provided by SP-A and SP-D. (A) Immunisation against bacteria, viruses, fungi, and allergies. (B) Increased pathogen phagocytosis, apoptotic cell phagocytosis, and control of inflammatory mediator synthesis (Bernhard, 2016) [4].

Pulmonary Surfactant Studies in Animals

Surfactant alterations have been reported in neonates and adults of several large animal species. Surfactant deficiency (quantitative deficit of surfactant) in premature animals causes neonatal respiratory distress syndrome/NRDS (Christmann *et al.*, 2009) [9]. Surfactant dysfunction (qualitative changes in surfactant) has been implicated in the pathophysiology of acute respiratory distress syndrome (ARDS) **(**Hartog *et al.*, 1995) [26]. Lung surfactant maturation occurs close to the last 2 weeks of gestation in the bovine fetus. Calves delivered one week before the estimated calving date are still considered at risk for the development of respiratory distress syndrome/RDS because of surfactant deficiency (Eigenmann *et al.*, 1984) [16]. In preterm neonates, respiratory distress syndrome (RDS) is the main cause of respiratory failure and its incidence differs depending on gestational age and birth weight (Boghossian, *et al.*, 2018) [5]. Preterm lambs delivered between 120 and 125 days of gestation are severely surfactant deficient and are likely to survive with exogenous surfactant treatment and intensive care (Notter, 2000) [43]. Neonatal pig surfactant is characterized by higher levels of PL, SP-B and SP-C. These compositional changes are believed to be responsible for an enhanced ability to lower surface tension found in neonatal pig surfactants (Rau *et al.*, 2004) [44]. Fatal RDS also called ‘‘*barker syndrome*’’ occurs frequently in neonatal large white pigs (Gibson *et al.*, 1976) [21]. Lung surfactant in neonatal foals differs from that in adult horses, neonatal foals have higher surface tension, higher levels of phospholipid inositol and lower levels of phospholipid glycerol compared with adult horses (Christmann *et al.*, 2006) [10]. The beneficial use of surfactant protein as a treatment in neonates with RDS has been a breakthrough and has been studied in-depth for neonatal medicine in the past 3 decades (Speer *et al.*, 2013) [50]. Thus, it is logical to hypothesize that restoration of Pulmonary Surfactant does improve the lung function (Echaide *et al.*, 2017) [15] and circumvent the symptoms of NRDS in infants and ARDS in adults.

Conclusion

Pulmonary surfactant is a lipid-protein combination that plays a vital role in lung ventilation and host defense. Pulmonary surfactant reduces surface tension by producing a film at the alveolar interphase, preventing alveoli from collapsing during expiration. Adult and baby respiratory distress syndromes are caused by pulmonary surfactant deficit. Preterm infants can benefit from pulmonary surfactant therapy in addition to respiratory distress syndrome treatment.

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