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Lung Development: Biological and Clinical Perspectives, Volume II: Neonatal Respiratory Distress is a collection of papers that addresses the needs of small infants who have respiratory diseases. This volume deals with fetal lung development with emphasis on hyaline membrane disease. This book also presents basic information on pulmonary surfactant in the clinical area, particularly in 1) diagnosis and movement of neonatal respiratory distress; 2) prenatal assessment of fetal lung maturity; and 3) prevention of hyaline membrane disease with corticosteroid therapy. One paper reviews the essenti. L. B. STRANG The past 25 years have seen a remarkable growth in our knowledge of lung development in its structural, physiological and biochemical dimensions. Much of the impetus for research leading to new knowledge has derived from the perception that many respiratory disorders in the newborn infant are due to defective development or maladaption of some component or components of the respiratory system. Thus, to cite one example, surfac tant deficiency is clearly seen to be the cause of atelectasis in hyaline mem brane disease; and to cite another, it is widely accepted that the mechanisms controlling patency of the ductus arteriosus and pulmonary vascular resistance also determine the right-to-Ieft or left-to-right shunting frequently observed in the course of neonatal respiratory disorders. There are, however, areas of physiological knowledge - such as those relating to respiratory control and to liquid formation and absorption - which are clearly of great relevance to lung adaptation at birth but where it has not yet proved possible to link a specific clinical state to the malfunction of a particular mechanism. In planning this symposium an attempt was made to organize the material in an orderly manner, starting with the embryonic and fetal stages of growth and development, continuing with respiratory control and the role of surfactant in lung aeration at birth, and ending with the treatment of neonatal respiratory disorders. This book presents a comprehensive overview of the current understanding of the organization of endogenous lung stem and progenitor cell compartments during fetal lung development, postnatal lung growth and in adulthood. Progressing stage by stage, the chapters on fetal lung development emphasize the integrated role of epithelial, stromal, vascular and neural cell elements in building a functional lung, while the subsequent chapters on adult lung regeneration describe the nature and properties of adult lung stem/progenitor cells distributed along the proximal-distal axis of the airway tree. The chapters on regulation of lung regeneration and repair discuss how regenerative cells interact with their niche microenvironment and how regulation of lung regeneration and repair in the steady state and following injury recapitulates ontogeny. And, lastly, the chapters on cellular therapies for lung disease and bioengineering the lung focus on promising emerging therapies and approaches in lung regenerative medicine. The scope of this volume of the Stem Cell Biology and Regenerative Medicine series focuses on exploring the topic of building and rebuilding the lung from a cellular rather than a molecular perspective. Thus, the section on cellular therapies does not include extensive coverage of each of the numerous lung diseases, including cancer, which could be amenable to stem cell-based therapies, although the final chapter does include some discussion on the future prospects and challenges. All of the contributors are working on the cutting edge of the lung stem cell field, making this book essential reading for those with an interest in the field of lung stem cell biology and the potential role of cellular therapies and tissue bioengineering approaches in lung regenerative medicine, including biomedical scientists, graduate students, post-graduate researchers and respiratory clinicians. This reference compiles the most current technical and biological data available to survey the state-of-science in the care and management of patients with bronchopulmonary dysplasia, COPD, and other forms of lung disease-tracking the initiation and progression of processes that cause airway obstruction, the biologic and physiological abnormalities that characterize COPD, and the potential reversibility of the inflammatory response in COPD for improved patient diagnosis and treatment. Knowledge about the mechanisms of lung development has been growing rapidly, especially with regard to cellular and molecular aspects of growth and differentiation. This authoritative international volume reviews key aspects of lung development in health and disease by providing a comprehensive review of the complex series of cellular and molecular interactions required for lung development. It covers such topics as pulmonary hypoplasia, effects of malnutrition, and pulmaonary angiogenesis. An indispensable reference for all those involved in studying or treating lung disease in neonates and children, the book offers a unique view of the development of this essential organ. Lung development is analogous to a well orchestrated symphony, and depends on successful interactions between various cellular components within the lung. The pulmonary vasculature is essential for normal lung development and is a key member of the lung development ensemble. Impairment of pulmonary vascular development is the hallmark of neonatal diseases such as respiratory distress syndrome (RDS) and bronchopulmonary dysplasia (BPD). Members of the VEGF family of growth factors and receptors are critical mediators of vascular development. Two members of the VEGF family, VEGF-A and VEGF-D are highly expressed in developing lung. VEGF-A is known to play a role in pulmonary vascular development, but its role in vessel specification in the developing lung is poorly understood. In addition, we hypothesize that VEGF-D plays a role in pulmonary lymphatic development based on its high expression in developing lung mesenchyme and its ability to bind the lymphatic receptor VEGFR-3. Through conditional regulation of VEGF-A, we have characterized differential temporal effects of the pulmonary vasculature on lung organogenesis, unique to early (embryonic day E10.5-E12.5), middle (E12.5-E16.5), and late (E16.5-E18.5) phases of development. Induction of VEGF-A in distal lung from E14.5 to E18.5 resulted in disorganization of endothelial ultrastructure, increased endothelial cell proliferation and rearrangement of the established vascular plexus, altering alignment with the epithelium. Remarkably, VEGF-A induction caused a 3.3-fold increase in small lymphatic vessels and a 1.3-fold increase in arteries. This preferential induction indicates that VEGF-A influences lymphatic specification in late stages of lung development. In addition, we have characterized differential time-sensitive effects of VEGF-A on other aspects of embryonic lung development. VEGF induction during the perinatal period disrupted terminal branching morphogenesis, inhibited formation of primitive alveolar septae, and altered the established vascular and smooth muscle pattern. Further, VEGF-A induction during the period prior to birth increased postnatal mortality and morbidity. Taken together, these results demonstrate that VEGF-A regulates developmental programs for specification of pulmonary lymphatic and blood vessels during late stage lung development. Further, we show that the time-sensitive effects of VEGF on lung epithelial morphology and organogenesis are indirect, mediated through endothelial to epithelial signaling. I speculate that proximal-distal patterning in murine lung development actually represents a precocious specification event of respiratory identity, as well as that this ultimately enabled the incorporation of a program of branching morphogenesis in the ancestral program of lung development. Considering that in humans the primordial lungs are double Sox2+ Sox9+, this suggests an unsuspected heterogeneity in the early lung developmental events of human, mice, and reptiles. Altogether, the findings revealed by this work open new avenues of research to further understand the molecular mechanisms that drive lung development. Lung disease affects more than 600 million people worldwide. While some of these lung diseases have an obvious developmental component, there is growing appreciation that processes and pathways critical for normal lung development are also important for postnatal tissue homeostasis and are dysregulated in lung disease. This book provides an authoritative review of fetal and neonatal lung development and is designed to provide a diverse group of scientists, spanning the basic to clinical research spectrum, with the latest developments on the cellular and molecular mechanisms of normal lung development and injury-repair processes, and how they are dysregulated in disease. The book covers genetics, omics, and systems biology as well as new imaging techniques that are transforming studies of lung development. The reader will learn where the field of lung development has been, where it is presently, and where it is going in order to improve outcomes for patients with common and rare lung diseases. Lung Development: Biological and Clinical Perspectives: Biochemistry and Physiology, Volume I, provides a comprehensive and multidisciplinary treatise with regard to surfactant-related issues in lung maturation. Despite the deliberate emphasis on biochemistry in this volume, the aim is to place this information in the perspective of anatomy, physiology, and clinical perinatology. The book is organized into four parts. Part I offers a brief historical perspective by reviewing the chronology of clinical and basic advances. Part II then establishes a frame of scientific reference by reviewing the morphology and cytology of lung development and the physiology of pulmonary surfactant. Stages of development and variations in the maturation process are emphasized, while cautions to the biochemist are offered with respect to interpretation of experimental data. Part III provides an introduction to lung biochemistry. Part IV deals with the developmental biochemistry of lung phospholipid metabolism; the featured compound is the predominant surfactant component, phosphatidylcholine (PC). A focal point for discussion concerns regulatory mechanisms operating to control the production of saturated PC during late gestational development of the fetal lung. The project has exploited an Australian marsupial, tammar wallaby, as an experimental model to understand lung development. This research has focused on identifying the factors that regulate lung development and to develop new intervention therapies to improve health outcomes in human premature and low birth weight babies. "Ultrasound is a non-invasive diagnostic tool that could provide important information about pulmonary maturity. Quantitative ultrasound techniques that relate lung tissue acoustic properties, i.e. acoustic velocity, attenuation, and scattering, to its physical properties, such as, elastic and structural properties, have been reviewed. B-mode techniques were equipment dependent and produced conflicting results. A-mode techniques are less dependent on the measuring equipment and could provide more accurate information about lung development. A-node techniques were applied to the study of regional differences within the fetal lung. The accuracy of these techniques could be instrumental to the eventual determination of lung maturation. To pursue this possibility, the alveolar regions across the upper, middle, and lower lobes of physiologically mature normal preterm lamb lungs were scanned to determine their acoustic properties within the frequency range of 1-15 MHz. Average speed of sound, attenuation and size of backscatterers were found to be independent of lung regions. Comparison between the lung mean size of backscatterers and mean alveolar sac diameter, histologically measured from the whole lung, showed that these data were not statistically different. This suggested that the collagen rich alveolar sac septal walls were the principal sources of scattering. Histological measurements on the size of alveolar sacs across different regions of the lung were also independent of the lung regions. The results of this study on the fetal lamb lungs suggested that A-mode ultrasound is sensitive to lung developmental changes. The ability of A-mode ultrasound to determine lung maturity appears promising. Further experiments on regional lung development and lung maturation at the pre and post-surfactant synthesis stages of the gestational life may establish the basis for an accurate and risk-free ultrasound assessment of lung maturation that is reliable in a clinical setting" -- The second edition of The Lung: Development, Aging and the Environment provides an understanding of the multi-faceted nature of lung development, aging, and how the environment influences these processes. As an essential resource to respiratory, pulmonary, and thoracic scientists and physicians it provides an interface between the “normal and “disease cluster of chapters, allowing for a natural complement to each other. The interface between different lung diseases affecting the pediatric lung also adds a useful source for comparing how different lung diseases share key pathophysiological features. This same complementarity comes across in the logical line up of chapters dealing with the “normal pediatric lung. New research, including cell-based strategies for infant lung function, epigenetics, and prenatal alcohol exposure on lung development and function are some of the important additions to this edition of this reference work. Describes the normal processes of lung development, growth and aging Considers the effects of the environmental contaminants in the air, water, soil, and diet on lung development, growth and health Describes genetic factors involved in susceptibility to lung disease Covers respiratory health risk in children Mammalian lungs originate as epithelial outgrowths of the ventral foregut into the surrounding mesenchyme. Epithelial branching and cyto-differentiation give rise to lungs capable of gas exchange. Glucocorticoids (GCs) stimulate and accelerate these events, partly by inducing soluble factors from fetal mesenchyme. However, the effects of GC signaling on cell-cell interactions and on the expression of downstream target genes essential to normal lung development are incompletely understood. In order to identify downstream targets of GCs that regulate lung development, Kaplan and Sweezey (1999) cloned Late Gestation Lung 1 (LGL1). LGL1 is a glucocorticoid-inducible, developmentally regulated gene expressed in lung mesenchyme. Lgl1 protein belongs to the CRISP family of secreted proteins that act as cell adhesion molecules, serene proteases, and/or mediators of the TGF-beta signaling pathway. This led us to speculate that LGL1 may serve an important function in fetal lung development. The objective of this thesis was to characterize the role of lgl1 during fetal lung development. During the pseudoglandular stage, LGL1 mRNA is found diffusely throughout the mesenchyme while its protein product is detected in subsets of mesenchymal cells adjacent to small airways and large blood vessels. Reduction of LGL1 mRNA and lgl1 protein levels by oligodeoxynucleofdes in fetal explant cultures inhibited lung branching. Conversely, recombinant lgl1 stimulated airway branching. LGL1 expression is maximal in the saccular stage, concordant with the surge in surfactant production. Lgl1 protein, restricted to the mesenchyme in early gestation, is present in epithelial cells in the saccular lung, suggesting a distinct role for lgl1 in late gestation lung. We showed lgl1 is a secreted glycoprotein and that recombinant lgl1 (rlgl1) suppresses epithelial proliferation and stimulates surfactant production in late gestation lung cell culture. Transient transfection using luciferase reporter constructs demonstrated that the LGL1 promoter contains functional GC and TGF-beta1 transcriptional binding elements. However, the target substrate(s) of LGL1 remain unknown. In conclusion, this thesis demonstrates that lgl1 plays a role during fetal lung organogenesis. Moreover, these findings are consistent with the inclusion of lgl1 in the emerging group of proteins that have distinct roles in regulating critical temporal-spatial events during distinct stages of lung development. The use of stem cells to help with lung regeneration and repair is a novel therapy which could help phase out the need for conventional surgical or pharmacological approaches currently employed to treat diseases of the lung or other organs. The present book explores all avenues of this new form of medical care, moving swiftly, but in depth, from the basic science of lung development, to the analyses of different stem cell types available for regeneration and on to the application of this knowledge base in initial clinical trials.In this volume a stellar group of researchers converge, from different angles, to help towards clarifying the basic mechanisms of lung repair. These range from basic concepts of regeneration and lung development, the analyses of a variety of cell types that may be involved in lung repair, to ways of creating complex lung structures, including artificial and bioartificial lungs. The book offers an insight into repair mechanisms of the diseased lung, the role of specific lung niches and provides information on initial clinical trials as well as the use of stem cells as vehicles for gene therapy. Ingenious technological aspects of assessing stem cell engraftment of stem cell bioprocessing are also included in this volume./a Carole Mendelson has assembled a panel of leading investigators to critically review the various classes of endocrine, paracrine, and neuroendocrine factors that play a role in the regulation of lung development and surfactant production. Special attention is accorded the actions of glucocorticoids in lung development and the synthesis of surfactant glycerophospholipids and proteins. Also extensively treated are the roles of cell-cell interactions and the elaboration of various growth factors and bioactive peptides in pulmonary cell differentiation, gene expression, and pathophysiology. State-of-the-art and comprehensive, Endocrinology of the Lung constitutes a powerful new standard guide that illuminates the complex endocrinology of the lung for all those actively investigating hormone action in pulmonary biology and medicine today. With detailed scientific background and up-to-date research, this book examines recent developmental and cell biology, mechanobiology and stem cell biology discoveries to help provide a better understanding of lung development, repair and regeneration. Lung regeneration is an urgent therapeutic priority. The current major challenge is the generation of complex vascularized structures that can ultimately support or replace impaired lung function. Recent discoveries in biomedical engineering are analysed within the structural context of the lung to help provide a better understanding of the innovative solutions that could be used for restoring normal morphogenesis and regeneration of the lung. This also includes insights from basic developmental mechanisms of human lung development through the derivation and identification of stem cells, both from the early embryo as well as from differentiated organs and tissues. Suitable for a wide range of readers, including physicians and surgeons, scientists and researchers, and undergraduate and postgraduate students, this guide is an essential read for those working in the field of lung disease and development.

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