Chronic obstructive pulmonary disease approaches.

Aadhya Alisah*

Department of Physiotherapy, Institute for Breathing and Sleep, Victoria, Australia

Abstract

Chronic obstructive pulmonary disease, a serious global health issue, is expected to overtake heart disease as the third leading cause of mortality. Understanding the pathophysiological pathways that can result in new therapeutic approaches is necessary because, aside from the crucial preventive measures of quitting smoking, there are no other particular treatments for COPD that are as effective in reversing the condition. The creation of experimental models will aid in unravelling these pathways at the cellular and molecular levels. The hallmark of COPD is a gradual narrowing of the peripheral airways, which is linked to lung inflammation, emphysema, and mucus over secretion. Different methods for simulating COPD have been devised; however they fall short of models for allergic asthma. Commonly based on the creation of COPD-like lesions in the lungs and airways using toxic inhalants such tobacco smoke, nitrogen dioxide, or sulphur dioxide, COPD models typically do not reflect the main characteristics of human COPD. These harmful stimuli cause symptoms of persistent inflammation and airway remodelling, depending on the length and severity of exposure. Combining such exposure with the administration of tissue-degrading enzymes can result in emphysema.

Keywords: COPD-Chronic Obstructive Pulmonary Disease, Bronchodilator, Emphysema-like lesion, Vascular Endothelial cell Growth Factor Receptor-2 [VEGFR-2].

Introduction

Since no medications have yet been produced particularly for COPD, the disease is characterised by a variety of pathologies, including persistent inflammation and tissue proteolysis. One of the most crucial aspects of managing COPD is quitting smoking since it slows the loss of lung function. Beta-adrenergic and anticholinergic bronchodilators are the staple drug; adding topical corticosteroid therapy to individuals with more severe COPD may improve bronchodilator responses and lessen exacerbations. Much less research has been done on COPD compared to the extensive quantity of experimental studies that have been done on allergic asthma and the in-depth understanding that is currently available on the mediators of allergic airway inflammation. Compared to COPD, research on asthma has received more funding and attention. Research on COPD may benefit from the present understanding of the pathogenesis and pathophysiology of asthma. Many research institutions that once only looked at asthma are now also looking into the processes of COPD. An expanding number of molecules that may be responsible for the pathogenic inflammation of chronic allergic airway inflammation have been discovered using molecular and genetic methods [1].

Finding new treatments and understanding the potential roles of the many mediators and molecular mechanisms that may contribute to the pathophysiology of COPD are the current research challenges. It is also necessary to comprehend how quitting smoking affects the pathogenetic process. The use of limited in vitro experiments or morphological and molecular evaluations of lung tissues recovered during surgery are the only methods available to study the molecular pathways in human individuals. In vivo animal models are required to investigate aetiology, functional changes, and the effects of novel drugs or treatments more thoroughly.

It is important to consider that there are many disease stages within COPD, only some of them can be reproduced in animal models before characterising and reviewing the various animal models of COPD that have been established thus far. A history of exposure to noxious stimuli, primarily tobacco smoke, and abnormal lung function tests are key components in the diagnosis of COPD. A straightforward illness description has been challenging to create since COPD has a varied pathophysiology and the underlying molecular pathways are poorly understood. However, the presence of a chronic airway blockage in cigarette smoke is necessary for the diagnosis of COPD [2].

Three main experimental methods are used to imitate COPD: inhaling unpleasant stimuli; injecting tissue-degrading enzymes into the trachea to cause emphysema-like lesions; and using gene-editing methods to produce a COPD-like

***Correspondence to:** Aadhya Alisah, Department of Physiotherapy, Institute for Breathing and Sleep, Victoria, Australia, E-mail: aadhya@alisah.au

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phenotype. Additionally, these methods might be combined. Animal models of COPD should ideally exhibit several of the GOLD guidelines suggested possible indicators for the disease. Lung function assessments in experimental animals would be great because the definition of COPD still primarily relies on these findings. Invasive approaches remain the gold standard for measuring lung function in very small mammals like mice because it is not optimal to utilise the enhanced pause in conscious mice as a sign of airflow blockage. These methods should also be compared with inflammatory markers and cellular remodelling [3].

A potent method for determining the purpose and part of various genes in the control of pulmonary homeostasis in vivo is the gene-depletion and -overexpression in mice. Gene overexpression in transgenic mice, whether targeted or not, can result in gain-of-function while focused mutagenesis approaches can result in loss-of-function. These models can be a big help in pinpointing both the physiological purposes of various genes and the underlying causes of disorders like COPD. A major focus was the evaluation of matrix-related genes in the huge number of genetically altered mouse strains linked to COPD symptoms [4].

Other substances that cause airway inflammatory damage have also been identified. In this regard, the introduction of toxins like endotoxin causes the activation of macrophages and the recruitment of neutrophils, which results in the expansion of the airspace. The intravascular infusion of a vascular endothelial cell growth factor receptor-2 (VEGFR-2) blocker can also produce non-inflammatory emphysema-like lesions. Endothelial cells die when VEGF is not present, which prevents the growth of blood vessels and endothelial cell survival. Human emphysematous lungs exhibit decreased VEGF and VEGFR-2 expression as well as increased septal cell death. Additionally, airspace expansion and alveolar septal cell death are brought on by continuous VEGFR-2 blockade [5].

Conclusion

Animal models that are currently available can only imitate a small number of the distinctive features of COPD, in contrast to the diverse pathophysiology and many phases of severity in human COPD. To further our understanding of the mechanisms behind human COPD, animal models must be carefully assessed against the characteristics of human COPD. Several experimental methods to replicate the acute and chronic symptoms of COPD have been developed in recent years. These methods rely on inhalative exposure to noxious stimuli like cigarette smoke, the administration of tissue-degrading enzymes, or gene-targeting methods. They are all limited in terms of their clinical importance due to the disease's intricacy and species-specific variations. Although the generation of COPD lesions by tissue-degrading enzymes can often seem artificial, this does not indicate that these models are not useful because they can be used to examine a variety of end-stage emphysema's pulmonary pathophysiology. If the results are extended carefully, cellular mechanisms can be explored effectively, and underlying molecular mechanisms and prospective therapeutic approaches can be revealed.

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