BioMed Research International

Special Issue on
Epithelial Mesenchymal Transition in Chronic Lung Disease

CALL FOR PAPERS

Epithelial mesenchymal transition (EMT) is a biological process in which epithelial cells undergo extensive molecular reprogramming allowing these cells to undergo numerous biochemical changes and acquire a mesenchymal phenotype. EMT is a vital process during embryogenesis (Type I EMT) but can also be induced as a result of persistent insult and tissue inflammation in adult stage. There are then two subsequent outcome possibilities with active EMT: severe and even complete organ fibrosis (Type II EMT), or development of a premalignant stroma when associated with angiogenesis (Type III EMT). In the last decade, a number of studies have examined the role of EMT in different organs and various contexts of physiology and disease. EMT has convincingly been proven to play an important role in both normal development and disease. EMT is not completely understood however. This is illustrated by the fact that the exact role of EMT in the respiratory tract's response to aeropollutant injury and development of tissue remodelling, fibrosis, and/or cancer is not clearly understood. A few studies have reported active EMT in chronic obstructive pulmonary disease (COPD), asthma, idiopathic pulmonary fibrosis (IPF), and bronchiolitis obliterans syndrome (BOS) which may be related to the core pathophysiology of small airway fibrosis and/or its relationship with lung cancer. Better understanding may lead to new markers for lung tissue remodelling and incipient neoplasia and better preventive management of patients. Specifically, there is serious need to understand key components of airway EMT in smokers with and without COPD and/or lung cancer and to demarcate novel drug targets for the prevention of lung cancer and airway fibrosis, as well as better secondary management of COPD.

Since over 90% of human cancer arises in epithelia and the involvement of EMT in all of these may be a central paradigm, insights gained in lung disease may have important generalizable value. For instance, in human epidemiological studies, it is suggested that COPD patients on inhaled corticosteroids (ICS) have an appreciable (50%) reduction in the risk of lung cancer. EMT might be the process by which this effect of ICS occurs. Furthermore, it has been suggested that statins also decrease the risk of lung cancer in COPD patients; again, EMT may be playing a central role. This warrants further studies on EMT in lung disease. We believe that detailed understanding of this process will have huge implications for therapeutic/clinical and public health policy. Therapeutic approaches to EMT are still in their infancy and we do not know which protein or pathways we should target to block this profound epithelial plasticity. Given the fact that disease-associated EMT is of two types, it becomes more complicated to tease out what proteins/pathways are contributing independently to fibrosis and cancer and what therapeutic modalities may be required for new treatments. This complexity is shown by the fact that types II and III EMT share a common proteome but empirically seem to differ in the neoangiogenesis component.

In this call for papers, we invite investigators to contribute original research articles as well as review articles on recent advances made in evidence of active processes of EMT in chronic lung disease, its clinical significance, mechanisms, and EMT as a therapeutic target.

Potential topics include, but are not limited to:

- Evidence for EMT in very early stages of lung disease
- Investigations into evidence for EMT in chronic lung disease
- Particulate matter, biomass fuel, and EMT in lung disease
- EMT and lung fibrosis
- EMT in small airway disease which is seen before the development of classic COPD
- EMT and lung tumour metastasis
- COPD and lung cancer, EMT as a potential link
- Lung inflammation and EMT
- Drivers of EMT in lung disease
- EMT as a therapeutic target in lung disease

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/bmri/pulmonology/emt/.

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First Round of Reviews
Friday, 8 April 2016

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