HISTOLOGICAL CHARACTERIZATION OF PNEUMOCYTES TYPE I AND PNEUMOCYTE TYPE II: A SUBJECT REVIEW

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ABSTRACT

The alveoli regards as a specialized region of the distal parts of the lung, in conductive morphology to an efficient gas exchange. There were two types of epithelial cells that lined the alveoli. Type I pneumocyte (T1Pn) cells exhibited a broad, more flattened morphology and covered near 95% of the surface areas, while the cuboidal alveolar type II pneumocyte (T2Pn) lined the remainders of the alveolus. T1Pn cells provided gaseous exchanges interfaced with the endothelium, while T2Pn cells served as both progenitor of T1Pn cells and played an important role to maintain the homeostasis of the alveoli, also T2Pn secrete a surfactant proteins by specialized organelles inside it called lamellar bodies to the alveolar spaces, in order to maintains surface tensions and prevent alveolar collapses.

INTRODUCTION

Type I pneumocytes (T1Pn) was the larger of the two cells type; it was a thin to flattened epithelial cell that formed the structures of the alveolus. It characterized by a squamous morphology and had a long cytoplasmic extension which covered nearly 95% of the alveoli, besides the T1Pn cells were involved the process of gaseous exchanges of the alveoli and blood

circulation (figure 1). Their nuclei may occupied a large area of the cytoplasm. In addition, it was unable to replicate and was susceptible to toxics insult. Therefore, when it damaged, it cannot proliferated and differentiated in order to compensate.

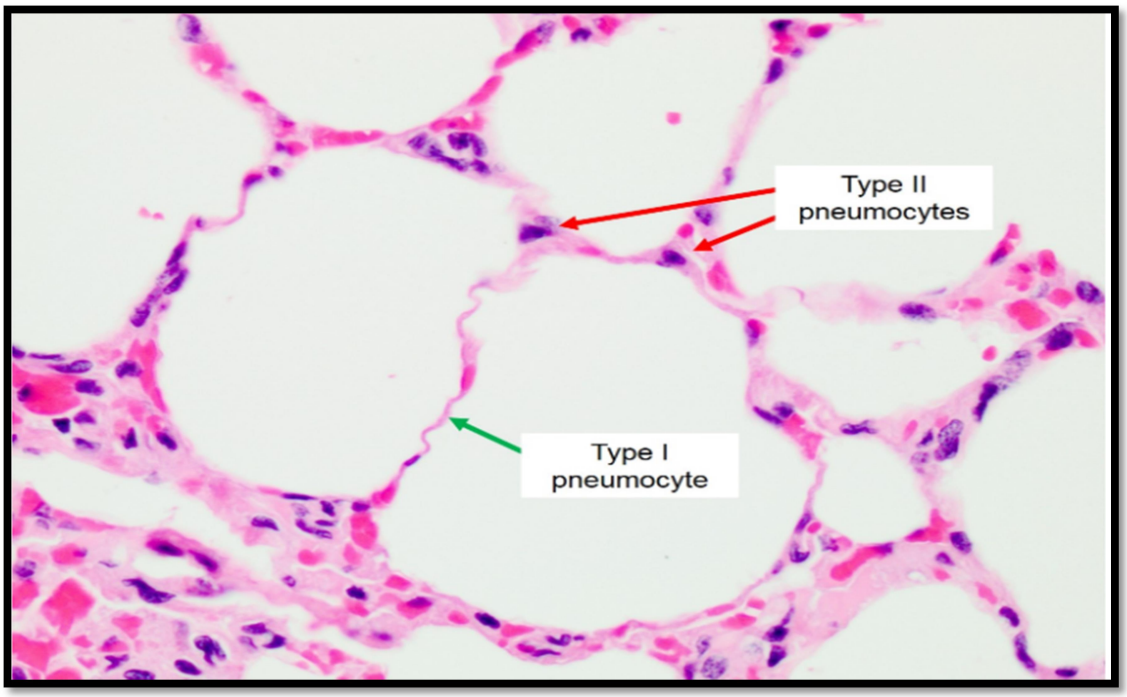

Figure (1): The role of the T1Pn and T2Pn cells in gas exchange cited from (Jennings and Premanandan, 2017).

The pulmonary alveoli lined in two types of cells, the type I pneumocytes (T1Pn) and type II pneumocytes (T2Pn), a roughly cuboidal cell that is usually found at the alveolar septal junctions. T1Pn cells cover near to 95% of the all surface area of the lung alveoli, while T2Pn cells had a squamous morphology and a vulnerable covered near to 5% of remained total area.

The T2Pn cells acts as the progenitors cell for T1Pn cells, moreover the T1Pn cells when damaged, the T2Pn cells proliferated, migrated, and spread to the denuded basal membranes of the surfaces, in order to reform the epithelial layer and thus will differentiated into T1Pn cells. Therefore, it was suggested to play very important roles in repairing processes of the alveolar epithelial barriers.

T2Pn cells synthesized, stored, and secreted pulmonary surfactants, which reduced the alveolar surfaces tensions and stabilizes alveolar units for efficiently gaseous exchanges.

Moreover, it secretes a vary types of cytokines and proteins can modified the inflammatory responses and oxidative stresses responses and inhibited fibroblasts proliferations and collagens synthesizes which was an implicate in the pathogenesis of chronic obstructive pulmonary disease (COPD).

T2Pn cells pulmonary surfactant was a surface activated materials composed of phospholipids and proteins near to 10% and two types of the hydrophilic surfactant-associated proteins, surfactant protein – A (SP-A) and surfactant protein – D (SP-D), in order to modulated hosts innate and adaptive immunity.

The other two hydrophobic proteins, surfactant protein – B (SP-B) and surfactant protein – C (SP-C), it may plays an essential roles in reduce the surface tensions, the after synthesis, the pulmonary surfactant was packed in lamellar bodies in order to secrete it as a thin liquid hypophase which covered the alveolar epithelial layers, also the surfactant recover the lung lavages that separated into two types by centrifugation, the highly surfaces activated sediment fractions that named the large aggregates and the poorly surface activated called non-sedimenting or termed as a small aggregates.

The pulmonary surfactant didn't maintains only the alveolar and airways stability, but may had a role in the regulation of the airways liquid balances and the bronchi clearance rates, it was recognized as an important and crucial components in the host immunity, particularly SP-A and SP-D. A variety of the mechanisms was involved in processes of surfactants modifications, like a genetic mutations or absences of the synthetic substances, therefore the activities of neutrophil elastase enzymes as well as metalo-myeloperoxidases (MMPs) and proteolytic enzymes mainly augment via the constituents of nitrites and oxidants subsequently affected the surfactant functions.

The oxidative stresses by the oxidant and / or antioxidant imbalances led to the surfactant suffered from lipid peroxidation processes and directly damaged the surfactant homeostasis, it was evident of the increased oxidative stresses in the airway of patients in COPD. The body defenses impaired by the oxidative damages to the functional capacities of SP-D to agglutinated bacteria (Fig. 2 and 3). Moreover, the biochemical and biophysical dysfunctions of the surfactant had been reported in a variety of diseases, like in acute respiratory distress syndrome (ARDS), the asthmas, the pneumonias, the mechanical ventilated lung injury and COPD.

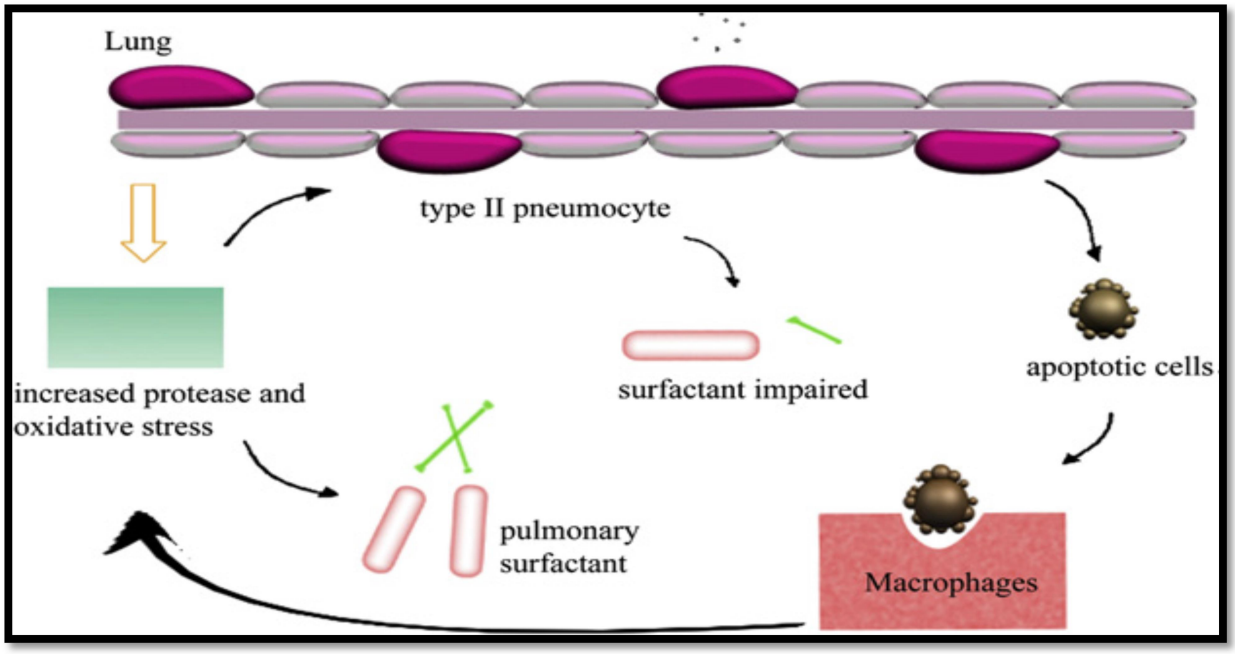

Figure (2): the noxious particles increase the rates of apoptosis and then causes an injuries of T2Pn cells, the activating and enhancing macrophages in order to phagocytosis of the apoptotic cells. The impaired T2Pn may decreased the main production and the function of pulmonary surfactant that lead to increase activities of proteases enzymes and then the oxidative stress (Zhao, *et al.,* 2010).

It was found an increases in the ratios of phosphatidylglycerol : cardiolipin as well to a decreases in the concentration of the total phospholipids in bronchial associated lymphoid tissue (BALT) of the smoking, the non-asthmatic COPD patient when compared to those non-smoking healthy. In addition, it was documented total phospholipid and the surface activities decreased in the BALT fluids in the smokers. The T2Pn cells may exposed to smokes in the cultures that decreased it secretions of pneumocytes, besides the analysis among SP-A, SP-B and SP-D genes in patients with COPD showed a correlations in the severity of the disease.

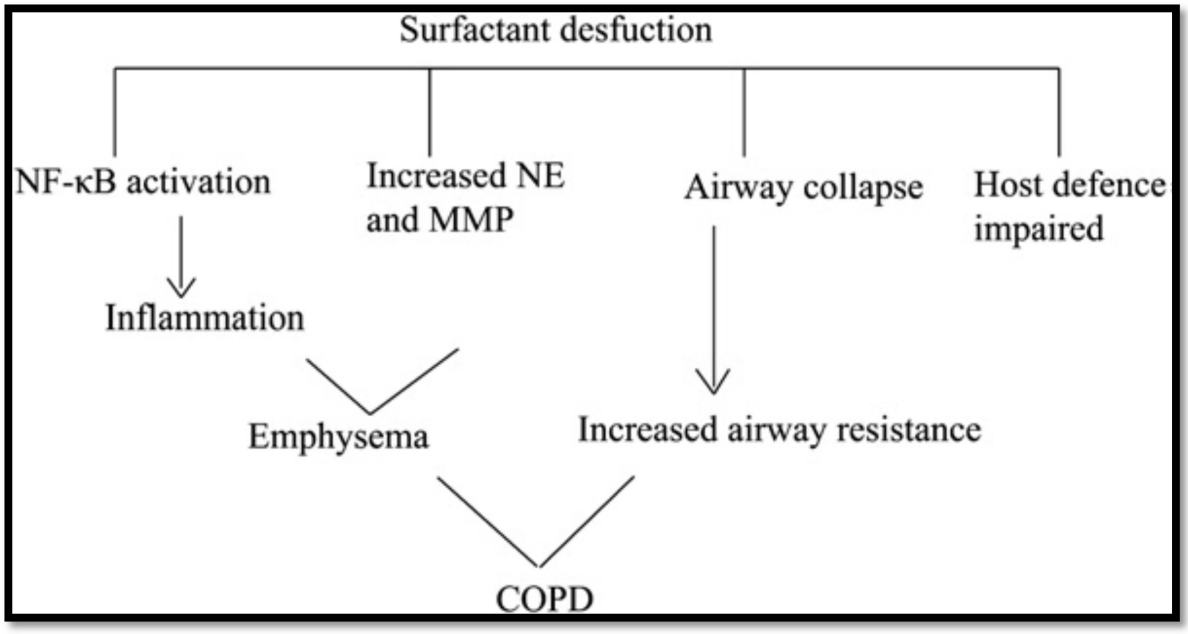

Figure (3): the role of the pulmonary surfactants dysfunctions in pathogenesis of COPD (Zhao, *et al.,* 2010).

The smokers with COPD had significantly declined SP-D values compared to the healthy histologically; also, the experimental study of the lung injured showed that the susceptibilities to ozone that induced airways inflammation were associated with significant decreases in their values of SP-D.

التوصيف النسيجي للخلايا الرئوية من النوع الأول والنوع الثاني: مراجعة علمية

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الخلاصة

تعتبر الحويصلات الهوائية منطقة متخصصة من الأجزاء القاصية من الرئة، وهي مسؤوله عن التبادل الغازي وبكفاءة لذلك هناك نوعان من الخلايا الظهارية التي تبطن الحويصلات الهوائية وهي الخلايا الرئوية نوع ١ والنوع ٢، ان الخلايا

الرئوية من النوع الأول شكلا رقيقا واكثر تسطحا حيث تغطي ما يقارب ٩٥% من كل المساحة السطحية للحويصلات الهوائية، في حين ان الخلايا الرئوية من النوع الثاني تظهر بشكل مكعب وتصطف في ٥% من ما تبقى من مساحة الحويصلات الهوائية، كذلك تلعب هذا النوع من الخلايا (النوع الثاني) دورا مهما في الحفاظ على التوازن الحيوي في الحويصلات الهوائية من خلال افرازها لبروتينات خافضة للتوتر السطحي عن طريق عضيات متخصصة بداخلها تسمى الاجسام الصفائحية لغرض الحفاظ على التوتر السطحي منع الانهيار السنخي alveolar collapse .

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