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Editorial

PEDX6 Antioxidative Enzyme at Oxidative Stress in Lung Biology

Yan Wang*

American Physiology Society, USA

Oxidative stress is widely recognized as a major pathophysiologic mechanism in various injuries that lead to either acute or chronic organ failure as lung is particularly susceptible to oxidative damage [1]. This stress is characterized by the increased production of reactive oxygen species (ROS) as superoxide radical, H_2O_2 or hydroxyl radical, which can result in oxidation in organs also including nucleic acids, lipidsand proteins, to protect against the harmful consequences, several antioxidant enzymes and non-enzymatic antioxidants in the body defend the damage from oxidative stress.

Prdx6 is a 1-cys enzyme that utilizes GSH as the physiological reductant to reduce H_2O_2 and organic hydroperoxides which was identified in recent over 10 years. Prdx6 is highly enriched in the lung compared with other organs and is especially expressed in lung alveolar epithelial type II cells [1-3]. These lung type II cells have role to produce surfactant, maintain cell surface area, restore damaged epithelium and host defense. The role of Prdx6 and other anti-oxidant enzymes in protecting the lung against oxidant injury has been evaluated in cell and animal models by manipulation of their expression levels. These studies have given ambiguous results for their role in anti-oxidant defense for SOD1 and 2, GPx1, and catalase in lung biology, then similar observation have been carried out with more effective in Prdx6 antioxidative function as reported. For Overexpression of Prdx6 gene mediated by adenoviral transfection or through gene transfer engineer could decrease oxidative stress and increased survival curve, while loss of Prdx6 gene resulted in more serious lung injury under hyperoxia or H_2O_2 and paraquat exposure, these results indicated that Prdx6 played functional role in lung biology to oxidative stress [3,4].

Lung Biology have the functions in air exchange, liquids permeability, surfactant product and others cardiological function. The functions in disordermay result in respiratory difficult, edemaPulmonary aortic hypertension, inflammation reaction orfunction failure. The theory mechanism could take responsibilityon it by oxidative and antioxidative states imbalance existed in lung biology through that antioxidative state as with Prdx6 antioxidative enzyme can enhance the lung function presentation at the same time observation, becausethe damage effects from inner or outside situation can cause in large part of increased formation of reactive oxygen species (ROS) which results in an imbalance between ROS generation and antioxidant defenses pathways, the lung injury has remarkable seen as acute lung injury or adult respiratory distress syndrome [1].

Considering of oxidative and antioxidative states imbalance to medical biology, lung is the selective organ to explain the mechanism of antioxidant enzymes to defense oxidative stress in biology, Prdx6 antioxidant enzyme in protection from oxidative stress is my projects which I have studied since 2002 when I worked at University of Pennsylvania Medical School. The projects first were from searching gene therapy vector to detect the Prdx6 antioxidant function, and going to directions in molecular, cell or disease models to confirm the function, currently more extend in development, currently important results on the studies have published at Journals [1,3,4].

As usual knowledge, oxidative stress is one of the main reason to result in degradation to human life span, studying in antioxidant enzymes would have activated goal to increase life value and qualify. For the future studying on the direction, more antioxidant defend need grow, and the oxidative-deduction state balance need maintain, then who is worthy the value of longer life younger span, it also requires personal real perfect character and behavioral.

***Corresponding Author:** Yan Wang, The American Physiological Society, USA, E-mail: drwangi@hotmail.com

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