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Multiple Approaches for COPD Prevention and Treatment via Redox Mechanism

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Chronic obstructive pulmonary disease (COPD) is one of the leading causes of death in the United States. Although the exact pathophysiological mechanism of COPD is not clear, the increased reactive oxygen species (ROS) play important role in the pathogenesis of COPD. Numerous studies have shown that cigarette smoking is the most significant risk factor to induce COPD. However, early smoking cessation does not effectively alleviate the symptoms of COPD. Impaired immune function activated via long-term exposure to cigarette smoke elicits detrimental effect on the lungs. An extensive literature research demonstrated the strong relationship among chronic tobacco exposure, oxidative stress, and immune response in COPD. We aim to develop potential therapeutic approaches targeting restoring immune homeostasis and host defenses via regulating the disrupted balance of oxidant production and antioxidant defense. The combination of pharmacological therapy and non-pharmacological approaches are more effective in halting COPD exacerbation. In particular, development of new drugs with high selectivity and bioavailability targeting epigenetic regulation of redox status and inflammatory response at the different stages of COPD will be the future challenge. Moreover, moderate intensity structured exercise not only reduces the risk of the development of COPD but also improves respiratory muscle function in COPD patients.

Biography

Feng has completed her PhD from Purdue University. Currently, she is a tenure-track Assistant Professor at the Department of Kinesiology at California State University-Chico. She has published 10 papers in reputed journals and has been serving in review board member of reputed journal, *Frontiers in Physiology*.

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