

Letter to Editor

Copyright © Jia Ping Wu

The Effect of Secondhand Smoke Exposure on Bronchus Airway Inflammation in COPD

Jia Ping Wu*

Department of Medical Technology, Shaoguan University Medical College, China

*Corresponding author: Jia Ping Wu, Department of Medical Technology, Shaoguan University, Shaoguan city, Guangdong Province, China, China, P.R.C.

To Cite This Article: Jia Ping Wu*. The Effect of Secondhand Smoke Exposure on Bronchus Airway Inflammation in COPD. Am J Biomed Sci & Res. 2023 20(2) AJBSR.MS.ID.002682, DOI: 10.34297/AJBSR.2023.20.002682

Dear Editor

 $(\mathbf{\hat{i}})$

Secondhand smoke exposure contributes to the development of Chronic Obstructive Pulmonary Disease (COPD) with occupational exposure and pollution from indoor fires being significant causes in some countries, and there is convincing evidence that bronchus inflammation may alter COPD individual susceptibility [1]. The primary cause of COPD is tobacco smoke, and the secondary is secondhand smoking exposure. The primary risk factor for COPD of those who smoke, about 20% will get COPD, and of those who are lifelong smokers, about half will get COPD [2]. According to estimates by the World Health Organization, the prevalence of the global population is around 5%. morbidity and mortality are currently [3]. Chronic inflammation is both a causal factor due to secondhand smoke exposure and a consequence of most chronic diseases of asthma and COPD.

However, this chronic inflammation process can persist over an extended period of weeks to months and even years [4]. Secondhand smoke exposure is a major cause of human Chronic Obstructive Pulmonary Disease (COPD). COPD has pathophysiological features of diseases [5]. Physiological COPD is individuals who cause poor lung development or growth for a variety of reasons during pregnancy, neonatal, infancy, or childhood [6]. Exercise is a good way to increase the rate of metabolism. Exercise improves heart rate and increases the rate of metabolism and burning of heat. For people who have many years of smoking, it is important to start exercising. Make sure to drink plenty of water because nicotine is soluble in water, so drinking water helps to excrete the substance through the urine [7]. Vitamin A is also helpful in removing nicotine from the body because it also has the effect of speeding up the metabolism [8]. Physiological COPD is an airway responsiveness. Physiological COPD is controlled by inherited genes. However, pathological COPD is an environmental factor. Secondhand smoke exposure is the risk of pathological COPD [9]. Therefore, chronic inflammation plays a critical role in COPD disease processes.

Acknowledgment

None.

Conflicts of Interest

None.

References

- 1. Carbone D (1992) Smoking and cancer. Am J Med 93(1A): 13S-17S.
- Ceylan E, Kocyigit A, Gencer M, Aksoy N, Selek S (2006) Increased DNA damage in patients with chronic obstructive pulmonary disease who had once smoked or been exposed to biomass. Respir Med 100(7): 1270-1276.
- Caramori G, Adcock IM, Casolari P, Ito K, Jazrawi E, et al. (2011) Unbalanced oxidant-induced DNA damage and repair in COPD: a link towards lung cancer. Thorax 66(6): 521-527.
- Borges Rodrigo C, Carvalho Celso R (2014) Impact of Resistance Training in Chronic Obstructive Pulmonary Disease Patients During Periods of Acute Exacerbation. Arch Phys Med Rehabil 95(9): 1638-1645.
- Balkan A, Bulut Y, Fuhrman CR, Fisher SN, Wilson DO, et al. (2016) COPD phenotypes in a lung cancer screening population. Clin Respir J 10(1): 48-53.
- Aoshiba K, Zhou F, Tsuji T, Nagai A (2012) DNA damage as a molecular link in the pathogenesis of COPD in smokers. Eur Respir J 39(6): 1368-1376.
- 7. Chen Q, Deeb RS, Ma Y, Staudt MR, Crystal RG, et al. (2015) Serum

Metabolite Biomarkers Discriminate Healthy Smokers from COPD Smokers. PLoS One 10(12): e0143937.

- 8. Wei J, Qin S, Li W, Chen Y, Feng T, et al. (2023) Analysis of clinical characteristics of 617 patients with benign airway stenosis. Front Med (Lausanne) 10: 1202309.
- 9. Liu J, Lu H, Hu S, Wang F, Tang X, et al. (2023) Transcriptomic profiles of age-related genes in female trachea and bronchus. Front Genet 14: 1120350.