

## Are Smoker/Vapers at Risk of Severe COVID-19 Infection?

Risala H. Allami,<sup>1</sup> Ahmed A. Suleiman,<sup>2</sup> and Hamid H. Enezei<sup>3,\*</sup>

<sup>1</sup>*Department of Molecular and Medical Biotechnology,*

*College of Biotechnology, Al-Nahrian University, Baghdad, Iraq*

<sup>2</sup>*Department of Biotechnology, College of Science, University of Anbar, Anbar, Iraq*

<sup>3</sup>*Department of Oral and Maxillofacial Surgery, College of Dentistry, University of Anbar, Anbar, Iraq*

(Received : 6 December 2020; Accepted : 30 December 2020; First published online: 7 January 2021)

DOI: 10.33091/amj.2021.171054

© 2021, Al-Anbar Medical Journal



The recent outbreak of a novel coronavirus has put the world on alert. The World Health Organization (WHO) declared the novel coronavirus disease-2019 (COVID-19) as a pandemic and it has been spreading around the world rapidly [1]. The causative pathogen of a novel coronavirus was subsequently identified and named 2019 novel coronavirus (2019-nCoV). Pneumonia caused by new coronavirus is an acute respiratory infection caused by a coronavirus which is a positive-strand RNA and transmitted from the bat with high homology of a previously identified virus.

In individuals with risk factors such as serious infection, non-pulmonary sepsis-like blood infection, and blunt trauma, smoking causes an increment in the development of acute respiratory distress syndrome (ARDS). Chronic cigarette smoke inhalation is related to hypersecretion of mucus, mucus pooling, damage to the pulmonary connective tissue, and chronic obstruction of airflow. Individuals who have cotinine (a nicotine metabolite) in their bodies have a significantly elevated risk of acute respiratory failure from ARDS, even with the low level of secondhand smoke [2].

The harmful vapour or smoking effects in their users have a serious effect on the body particularly the respiratory system than those individuals who never smoke when they are prone to influenza or other illnesses [3]. Smoking in patients with risk factors such as extreme pneumonia is correlated with an elevated occurrence of ARDS [4]. Moreover, for individuals with any nicotine in their bodies even at small secondary levels, the risk of acute respiratory insufficiency from ARDS is

considerably greater [5]. In the case of human ciliary impairment, as indicated by cellular and animal studies, the association of decreased coughs and compromised mucociliary clearance might predispose consumers to elevated pneumonia risks [6]. The users of cigarette and e-cigarette smoking are carrying a high risk with an increase in the severity of respiratory infections owing to an inhibition of the immune function of lungs [7].

Approximately 60 genes were impaired two hours after just 20 puffs in alveolar e-cigarette macrophages, including inflammatory genes. The creation of innate defence by neutrophil extracellular trap (NET) is a mode by which neutrophils release and lyse DNA into the extracellular network to help immobilize bacteria and thereby kill the lungs [8]. Persistent vapour neutrophils have identified a higher density than cigarette smoking or non-smokers. Two weeks of e-cigarette usage in mice was decreased survival and raised disease burden after either influenza A or Streptococcus pneumoniae inoculation, which represents the main causes of humans pneumonia [9]. Besides, exposure to aerosols can lead to increased colonization of upper airways with pathogenic agents and changes in virulence of pathogens as shown by Staphylococcus aureus [10]. However, empirical observations from animal sources are required, and vaporization appears to be related to population-level evidence in young adult humans that vapour may lead to increase susceptibility to infection [11].

According to a study from China, it has reported that the mortality rate due to the new coronavirus in males is more than in females, which is consistent with previous information that more men Chinese citizens smoke than women. Nonetheless, there was no smoking association in a China study that assessed the mortality rate of all people hospitalized with pandemic COVID-19 [12]. Further studies in China with COVID-19 patients who spent two weeks in hospitals found a 14 times

\* Corresponding author: E-mail: [den.hamed.hamad@uoanbar.edu.iq](mailto:den.hamed.hamad@uoanbar.edu.iq)  
Phone number: +9647822551977

higher rate of illness and even death concerning non-smoking patients with COVID-19-related pneumonia [13]. It was the most important contributing factor for infection [14]. In the face of COVID-19 in the United States and across the world, the scientific people should be alert to the possibility that certain individuals with substance use disorders (SUDs) will be more badly impaired. Due to influences on the lungs, the coronavirus causing COVID-19 might present a possible danger to anyone who chews nicotine, alcohol, or wine [15]. Vaping can also affect the health of the lungs, like smoking. The result of chronic obstructive pulmonary disease (COPD) remains un-

certain, but there is research emerging that the toxicity of e-cigarette aerosols influences the lung cells and the capacity to respond to infection [3].

In conclusion, smoking in both type cigarette and vaper affects pulmonary cell and thus reflect a high risk in COVID-19 infection mostly with severe or at least moderate symptoms.

### CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

### REFERENCES

- [1] S. H. Aktimur, S. E. N. Ahmet, B. Yazicioglu, A. K. Gunes, and G. Serhat. The assessment of the relationship between abo blood groups and covid-19 infection. *Int. J. Hematol. Oncol.*, 30(2):1–5, 2020.
- [2] S. A. Glantz. Reduce your risk of serious lung disease caused by coronavirus by quitting smoking and vaping. 2020.
- [3] M. D. Aronson, S. T. Weiss, R. L. Ben, and A. L. Komaroff. Association between cigarette smoking and acute respiratory tract illness in young adults. *Jama*, 248(2):181–183, 1982.
- [4] S. J. Brake, K. Barnsley, W. Lu, K. D. McAlinden, M. S. Eapen, and S. S. Sohal. Smoking upregulates angiotensin-converting enzyme-2 receptor: a potential adhesion site for novel coronavirus sars-cov-2 (covid-19). *Multidisciplinary Digital Publishing Institute*, 2020.
- [5] G. Cai, Y. Bossé, F. Xiao, F. Kheradmand, and C. I. Amos. Tobacco smoking increases the lung gene expression of ace2, the receptor of sars-cov-2. *Am. J. Respir. Crit. Care Med.*, 201(12):1557–1559, 2020.
- [6] M. A. Chilvers, M. McKean, A. Rutman, B. S. Myint, M. Silverman, and C. O’Callaghan. The effects of coronavirus on human nasal ciliated respiratory epithelium. *Eur. Respir. J.*, 18(6):965–970, 2001.
- [7] R. Patanavanich and S. A. Glantz. Smoking is associated with covid-19 progression: a meta-analysis. *Nicotine Tob. Res.*, 2020.
- [8] E. editorial Team. Updated rapid risk assessment from ecdc on coronavirus disease (covid-19) pandemic in the eu/eea and the uk: resurgence of cases. *Eurosurveillance*, 25(32):2008131, 2020.
- [9] J. Gu and C. Korteweg. Pathology and pathogenesis of severe acute respiratory syndrome. *Am. J. Pathol.*, 170(4):1136–1147, 2007.
- [10] T. S. Henry, J. P. Kanne, and S. J. Kligerman. Imaging of vaping-associated lung disease. *N. Engl. J. Med.*, 381(15):1486–1487, 2019.
- [11] C. Huang *et al.* Clinical features of patients infected with 2019 novel coronavirus in wuhan, china. *Lancet*, 395(10223):497–506, 2020.
- [12] D. S. Hui. Severe acute respiratory syndrome (sars): lessons learnt in hong kong. *J. Thorac. Dis.*, 5(2):S122, 2013.
- [13] E. Javelle. Electronic cigarette and vaping should be discouraged during the new coronavirus sars-cov-2 pandemic. *Arch. Toxicol.*, pages 1–2, 2020.
- [14] E. Kindler and V. Thiel. Sars-cov and ifn: too little, too late. *Cell Host Microbe*, 19(2):139–141, 2016.
- [15] J. M. Leung *et al.* Ace-2 expression in the small airway epithelia of smokers and copd patients: implications for covid-19. *Eur. Respir. J.*, 55(5), 2020.