

Relationship of Smoking and COVID-19: Myths and Reality

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Abstract

The alarming spread of novel corona virus, severe acute respiratory syndrome corona virus 2 (SARS-CoV-2), has resulted to an ongoing pandemic of corona virus disease since 2019. Due to this the virus is popularly known as Covid 19 virus. The world has not been able to get rid of this disease and we are into the fourth wave. There have been numerous changes in the nature of the disease but it is still causing major disruptions all over the world. Tobacco use as smoking is a major cause of mortality as well as morbidity around the world. Nobody can dispute the ill effects of smoking on health. Surprisingly, there have been few reports about low incidence of smokers among Covid 19 patients. This article aims to review available literature on the subject and try to clear the misconceptions on this topic.

Keywords: COVID-19, Smoking, corona virus disease, Tobacco.

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INTRODUCTION

COVID-19 disease was first reported in December 2019 in Wuhan, China. Soon, many other countries reported the emergence of the disease. We have already experienced the third wave of the disease. It has turned into a global pandemic and many people have succumbed to the disease. Affected individuals with pre existing chronic medical conditions such as heart disease and diabetes are considered to be at greater risk of acquiring severe form of the disease. During the Covid -19 pandemic, many health professionals are urging smokers to quit [1].

It is a common sense that all forms of tobacco are harmful and there is no safe tobacco. Millions of people are suffering from after effects of tobacco use and nearly 8 million die annually.

Smoking and disease

Smoking is one of the risk factors for the development and worsening of multiple respiratory diseases [1], including infections. It particularly worsens chronic obstructive pulmonary disease and lung cancer. It also has deleterious effects on community acquired pneumonia and Tuberculosis [2].

Smoke exposure results in infiltration of inflammatory cells into mucosa, submucosa, and glandular tissue, which in turn induces the excess production of mucus, causes epithelial –cell hyperplasia, interrupts tissue repair, thickens the small airway walls, induces emphysema, and impairs lung function including gas exchange [3]. Cigarette smoking, including active, passive and third –hand smoke exposure, is an important risk factor for upper and lower respiratory tract infection [4]. Cigarette smoke has been shown to affect the cell mediated immune response through elevated peripheral immune cell counts, CD4+/CD8+ cell ratio in the lungs, phagocytosis impairment, and Natural killer cell dysfunction [5]. Moreover, it is also known to disturb the humoral immunity, with lower immunoglobulin levels in serum but higher levels in the lungs [6].

Role of Angiotensin –Converting enzyme receptors

Angiotensin converting enzyme (ACE) II has attracted worldwide attention in relation to COVID-19 [7]. Constitutively expressed in the respiratory tract, myocardium and gastrointestinal tract, ACE-II is a type II transmembrane metalloproteinase that metabolizes angiotensin II into multiple metabolites,

such as angiotensin (1-9) [8]. In human respiration, ACE-II is expressed on the surface of type –II pneumocytes [9]. It plays an essential role in regulating blood pressure and cardiac function, but its role in the respiratory system remains more obscure [10].

Discussion of findings and relation of COVID 19 in smokers and various theories/hypothesis

Recent studies demonstrated that SARS-CoV 2 has a significantly higher affinity in binding with the ACE- II receptor; hence, it is more likely to bind and infect human cells than other coronaviruses. Importantly, studies have shown that smokers have increased expression of the ACE-II receptor, compared to non-smokers. Collectively, this information suggests that smokers may be more vulnerable to SARS-CoV 2 infection due to elevated expression in ACE-II receptors [1].

World Health Organization brief of June 2020 has stated that evidence suggests that smoking is associated with increased severity of disease and death in hospitalized COVID-19 patients. Although likely related to severity, there is no evidence to quantify the risk to smokers of hospitalization with COVID-19 [11].

Another study found from a national sample of adolescents and young adults show that e-cigarette use and dual use of e-cigarettes and cigarettes are significant underlying risk factors for COVID-19 that has previously not been shown [12]. Smoking was found to be an independent risk associated with severe progression of COVID-19, including mortality. The effects seem to be larger among younger adults. Smoking prevention and cessation was supposed to remain a priority for the public, physicians, and public health professionals during the COVID-19 pandemic [13]. In a separate study, it was reported that smokers and previous smokers aged fewer than 69 were at higher risk of COVID-19 infection, suggesting the risk is associated with increased exposure to SARS-COV2 virus [14]. Smoking was also found to be associated with negative progression and adverse outcomes in case of COVID-19 in another systemic review [15].

In contrast, a study found unusually low prevalence of smoking, approximately 1/4th the expected prevalence, among hospitalized Covid -19 patients. Any association between smoking and COVID-19 should not be generalized according to them. This showed the limitations of various studies. Also nicotine has been known to be used in therapeutics [16]. There can be numerous possibilities of interactions between smoking and COVID-19. Potential harms include sharing of cigarettes, disruption of the normal epithelial lining of respiratory system, increased expression of ACE2 leading to increased risk of attachment to mucosa and subsequent entry into cells, decreased expression of ACE2 leading to less breakdown of angiotensin 2 causing inflammation,

vasoconstriction and thrombosis and also increased prevalence of cardiac disease, stroke and COPD. Potential protective effects of smoking may include less reactive immune system, increased expression of ACE2 causing less risk of acute injury and thrombosis, decreased expression of ACE2 causing less risk of SARS cov2 attachment, increased production of NO which may potentially inhibit entry of SARS CoV 2. These present a paradox of whether smoking is harmful in case of Covid [17].

Myths about use of tobacco originated in some media. Tobacco industry also tried to benefit from those reports and promoted tobacco during COVID epidemic. Many of the reports appeared in non peer reviewed papers. Under reporting of smoking was also a problem. Health care professionals need to take active steps to sensitize people [18]. The role of governments is also very important as was noted during COVID-19 when only India and South Africa banned sale of tobacco products [19].

CONCLUSION

Tobacco use is a worldwide menace which cannot be denied. There have been various unsubstantiated claims from previous times when some or other beneficial aspects of smoking were propped up by somebody. All these were subsequently found to be poorly reviewed papers. Tobacco lobby and companies seem to promote these for very obvious reasons. Governments were also not very careful in forcefully denying these reports. Fuelled by social media these myths took a life of their own and became big stories. Health care professionals in particular need to be careful and promote cessation and also tell public at large about the various ill effects of tobacco. During these difficult times of the COVID-19 pandemic, a healthcare professional's advice is of great importance to promote correct thinking among the general public and dispel myths about COVID-19 and tobacco. Considering the overall ill effects of tobacco, there should be universal and strong steps to discourage use of tobacco products and smoking among people of all ages. This has also been substantiated in most of the peer reviewed journals and articles.

REFERENCES

1. Shastri, M. D., Shukla, S. D., Chong, W. C., Rajendra, K. C., Dua, K., Patel, R. P., ... & O'Toole, R. F. (2021). Smoking and COVID-19: What we know so far. *Respiratory medicine*, 176, 106237.
2. Bates, M. N., Khalakdina, A., Pai, M., Chang, L., Lessa, F., & Smith, K. R. (2007). Risk of tuberculosis from exposure to tobacco smoke: a systematic review and meta-analysis. *Archives of internal medicine*, 167(4), 335-342.
3. MacNee, W. (1994). Pathophysiology of cor pulmonale in chronic obstructive pulmonary

- disease. Part One. *American journal of respiratory and critical care medicine*, 150(3), 833-852.
4. Cohen, S., Tyrrell, D. A., Russell, M. A., Jarvis, M. J., & Smith, A. P. (1993). Smoking, alcohol consumption, and susceptibility to the common cold. *American journal of public health*, 83(9), 1277-1283.
 5. Hickman, E., Herrera, C. A., & Jaspers, I. (2019). Common e-cigarette flavoring chemicals impair neutrophil phagocytosis and oxidative burst. *Chemical research in toxicology*, 32(6), 982-985.
 6. Arcavi, L., & Benowitz, N. L. (2004). Cigarette smoking and infection. *Archives of internal medicine*, 164(20), 2206-2216.
 7. Brake, S. J., Barnsley, K., Lu, W., McAlinden, K. D., Eapen, M. S., & Sohal, S. S. (2020). Smoking upregulates angiotensin-converting enzyme-2 receptor: a potential adhesion site for novel coronavirus SARS-CoV-2 (Covid-19). *Journal of clinical medicine*, 9(3), 841.
 8. Harmer, D., Gilbert, M., Borman, R., & Clark, K. L. (2002). Quantitative mRNA expression profiling of ACE 2, a novel homologue of angiotensin converting enzyme. *FEBS letters*, 532(1-2), 107-110.
 9. Hamming, I., Timens, W., Bulthuis, M. L. C., Lely, A. T., Navis, G. V., & Van Goor, H. (2004). Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis. *The Journal of Pathology: A Journal of the Pathological Society of Great Britain and Ireland*, 203(2), 631-637.
 10. Tikellis, C., & Thomas, M. C. (2012). Angiotensin-converting enzyme 2 (ACE2) is a key modulator of the renin angiotensin system in health and disease. *International journal of peptides*.
 11. Smoking and COVID-19, scientific brief, 30 June 2020, WHO. Reference number: WHO/2019-nCoV/Sci_Brief/Smoking/2020.2
 12. Gaiha, S. M., Cheng, J., & Halpern-Felsher, B. (2020). Association between youth smoking, electronic cigarette use, and COVID-19. *Journal of adolescent health*, 67(4), 519-523.
 13. Patanavanich, R., & Glantz, S. A. (2021). Smoking is associated with worse outcomes of COVID-19 particularly among younger adults: a systematic review and meta-analysis. *BMC Public Health*, 21(1), 1-9.
 14. Prats-Urbe, A., Xie, J., Prieto-Alhambra, D., & Petersen, I. (2021). Smoking and COVID-19 infection and related mortality: A prospective cohort analysis of UK Biobank data. *Clinical Epidemiology*, 13, 357.
 15. Vardavas, C. I., & Nikitara, K. (2020). COVID-19 and smoking: A systematic review of the evidence. *Tobacco induced diseases*, 18.
 16. Farsalinos, K., Bagos, P. G., Giannouchos, T., Niaura, R., Barbouni, A., & Poulas, K. (2021). Smoking prevalence among hospitalized COVID-19 patients and its association with disease severity and mortality: an expanded re-analysis of a recent publication. *Harm Reduction Journal*, 18(1), 1-9.
 17. Usman, M. S., Siddiqi, T. J., Khan, M. S., Patel, U. K., Shahid, I., Ahmed, J., & Michos, E. D. (2021). Is there a smoker's paradox in COVID-19?. *BMJ evidence-based medicine*, 26(6), 279-284.
 18. Van Westen-Lagerweij, N. A., Meijer, E., Meeuwssen, E. G., Chavannes, N. H., Willemsen, M. C., & Croes, E. A. (2021). Are smokers protected against SARS-CoV-2 infection (COVID-19)? The origins of the myth. *NPJ Primary Care Respiratory Medicine*, 31(1), 1-3.
 19. Braillon, A. (2021). Association between Cigarette Smoking and COVID-19 Outcomes. *JAMA Internal Medicine*, 181(8), 1137-1138.