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# Alcohol consumption and obesity: The hidden scare with COVID-19 severity

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A B S T R A C T

Obese individuals seem to be at the highest risk of contracting COVID-19 infection. Furthermore, severity of morbidity and mortality rates are higher in the developed world as compared to the developing world. One probable reason for this difference could be the difference in living conditions and exposure to other infections. Secondly, the difference in food especially, alcohol use may have deteriorating effects superimposed with obesity. Our hypothesis suggests that a combination of alcohol consumption and obesity causes low immunity and makes the individual prone to develop 'cytokine storm' and 'acute respiratory distress syndrome'; the hallmark of COVID-19 mortality and morbidity. Thus, we propose that reducing any one trigger can have a beneficial effect in combating the disease severity.

## Background

In December 2019, several patients represented with the symptoms of acute respiratory illness in Wuhan, Hubei province, China, labelled as the novel COVID-19 infection. The causal agent of COVID-19 was identified as a newly discovered member of Corona virus family named as SARS-COV-2 [1]. SARS-COV-2 possesses the propensity to be released from the infected individual in to the environment through respiratory droplets, thus it was able to rapidly transmit and infect people within a short span of time converting into a pandemic affecting ~11,756,093 individuals and ~541,079 deaths around the globe as of 7th July 2020 globally (www.covidvisualizer.com). Interestingly, symptoms of COVID-19 infection are non-specific, thus speculation regarding the total number of actual cases may be underestimated because people suffering from mild and asymptomatic cases go largely undetected.

## Hypothesis

A balance nutrition is required to maintain good immunity which is essential for prevention and management of this viral infection [2]. This is in concordance with a good organ function especially liver and spleen that not only get rid of the toxins but also provide safety against common infections. One plausible cellular/immunological disruption that can effect the immune cells is the combination of obesity and alcohol consumption, which might be involved in severity of COVID-19 pathogenesis [Fig. 1].

## Hypothesis evaluation

### *Relationship between obesity, alcohol consumption and COVID19 pathogenesis*

Alcohol consumption has been characterized as one of the leading causes of acquired immunity disorders [3]. Approximately 2.4 billion people (1.5 billion males; 0.9 billion females) consume alcohol around the globe with regular intervals [4]. Alcohol consumption has been reported to affect the immune-related health responses and might just put an individual at risk for acute respiratory distress syndrome [5]. Acute respiratory distress syndrome (ARDS) is related with an over production of chemokines, termed as "cytokine storm"; a common occurrence with COVID-19 infections. Recent studies on immune-compromised models have also emphasized that heavy alcohol consumption has been associated with overproduction of inflammatory cytokines by hepatic cells, leading to elevated inflammatory markers [6,7]. More specifically, it has been observed that the levels of Interleukin-8 and TNF-α increase by significant proportion in response to even an acute alcohol consumption [8]. Studies by Pilotto et al, 2020 and Casillo et al, 2020 have also provided similar trends in increment of IL-8 and TNF-α in COVID-19 effected patients [9,10].

Another aspect related to pathogenesis of COVID-19 is related to over-expression of angiotensin II type I receptors (ACE2). It has been characterized that SARS-COV-2 interacts with these receptors through its spike protein to proliferate into the cell. Studies by Bechara et al, 2003 and Tan et al, 2012 have proved the over expression of ACE2 receptors in response to alcohol consumption, thus providing a possible pathway through which alcohol consumption can lead to facilitated proliferation of SARS-COV-2 into the cells [11,12]. Furthermore, the

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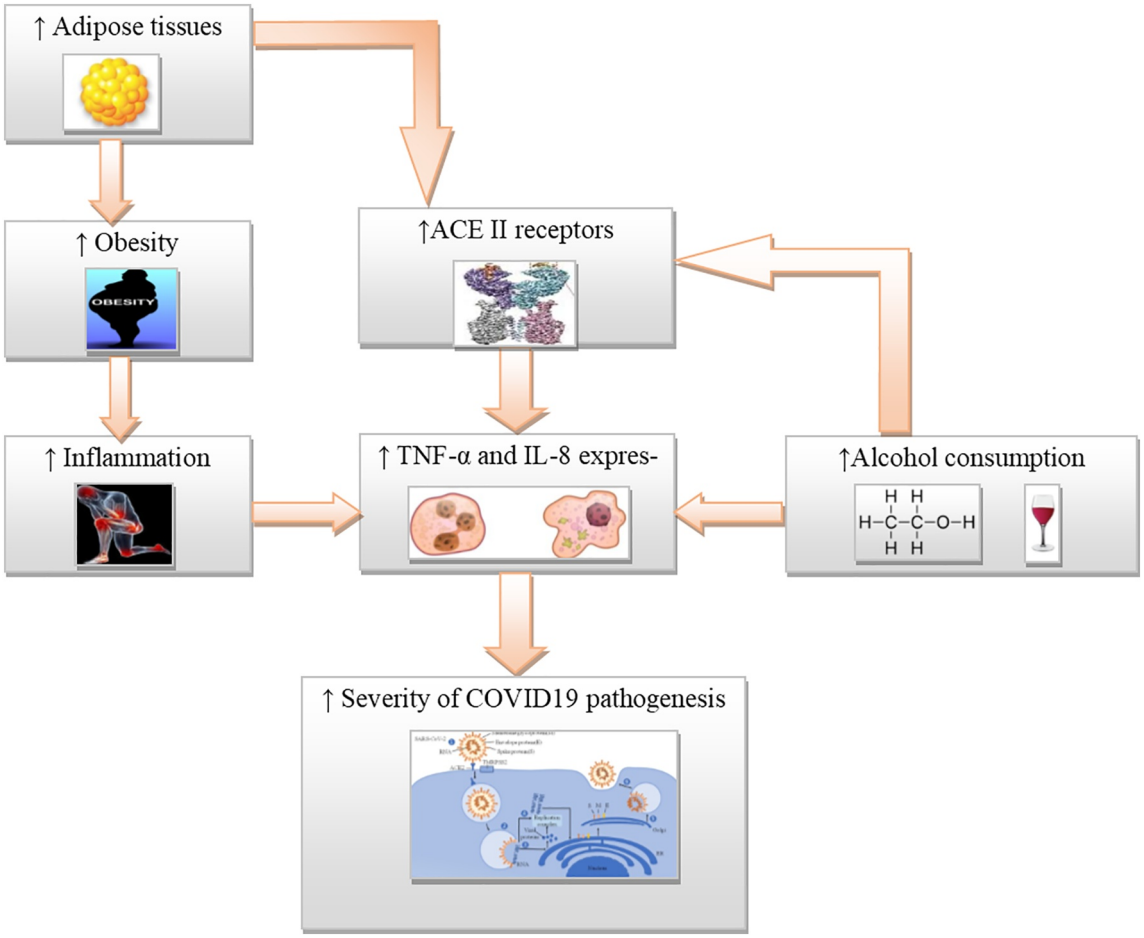


**Fig. 1.** Flow path-showing relationship between alcohol consumption/obesity and COVID19 pathogenesis. In brief, an increased production of adipose tissues can lead to onset of inflammatory responses that might result in higher production of inflammatory cytokines of such as TNF-α, IL-8 and ACE II receptors. High alcohol consumption on the other hand can exert the same effect on the regulation of TNF-α, IL-8 and ACE II receptors. Both of these perturbed dietary habits lead to potential increase in severity of COVID-19 pathogenesis.

increase expression of ACE2 receptors also lead to enhance TNF-α cytokine cytotoxicity [11], which relates both to the molecular (SARS-COV-2 receptor interaction) as well as immunological (Cytokine storm) aspects of COVID-19 pathogenesis. These molecular and immunological clues can be a suggestive of how acute respiratory distress syndrome (ARDS) prone environment is generated during COVID-19 pathogenesis in alcohol consumers.

Moreover, since last decade obesity and respiratory dysfunction has been a point of interest for the scientific community and it has been implied that even without any ongoing or prior respiratory illnesses, people suffering from obesity demonstrate breathlessness and dyspnea patterns which might become more severe in case of respiratory pathogenesis [13]. For instance, obese individuals have a higher mortality rate for a H1N1 infection compared to normal weight individual [14], in addition, time required for virus shedding is increased by 42% in obese H1N1 infected patients versus non obese counterparts [15]. Hence, obese individuals may retain respiratory viral infection for a longer duration of time and in severe cases are more prone to life threatening chronic illnesses. A recent study has characterized obesity as the major factor for increased mortality rate in SARS-COV-2 infected individuals, it was observed that the patients having a BMI and age of $\geq 34$ and $\geq 30$, respectively were more prone to severe COVID-19 symptoms [16]. Possible explanation regarding such a trend could lie in metabolic/immunological and bleeding discrepancies associated with SARS-COV-2 infection. As discussed in previous section both these immunological markers have also been concomitant with alcohol ingestion and COVID-19 pathogenesis. Approximately 31% patients suffering from COVID-19 develop thrombotic complications [17]. More specifically, an increase in D-dimer protein fragment and enhanced prothrombin time was observed in 46% of critically ill COVID-19 infected individuals [18]. Roughly more than % of total observed patients demonstrated 0.5 mg/L increase in D-dimer, while they also demonstrated an increase of approximately 15–16 seconds in prothrombin time [19–21]. Such changes might be related to increase levels of interleukin 8 (IL-8) and Tumor necrosis factor alpha (TNF-α) in obese individuals [22].

## Conclusion

Increase levels of interleukin-8 (IL-8), Tumor Necrosis Factor Alpha (TNF- α) seems to be a common factor in alcohol ingestion/obesity complications and COVID-19 pathogenesis. Although, several studies have implied such changes but none of those have been able to correlate these trends together in terms of COVID-19 pathogenesis. Obesity cannot be modified on urgent basis, but a negligible alcohol consumption might reduce the severity of the COVID-19 infections observed in the world.

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## Author contribution

SSF and BB conceptualized the project. All authors wrote the manuscript and approved the final version before publication.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.mehy.2020.110272.

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