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Impact of biomass induced black carbon particles in cascading COVID-19

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ABSTRACT

We explore the association of biomass-induced black carbon aerosolized virus with COVID-19 in one of the top-ranked polluted hot spot regions of the world, Delhi, at the time when other confounding factors were almost stable and the pandemic wave was on the declining stage. Delhi was worst affected by COVID-19. However, when it was fast returning back to normal after about 6 months with minimum fatalities, it suddenly encountered a reversal with a 10 fold increase in infection counts, coinciding with the onset of the stubble burning period in neighbouring states. We hereby report that the crop residue burning induced lethal aged Black carbon-rich particles which engulf Delhi during the post-monsoon months of October–November are strongly associated with COVID-19 and largely responsible for the sudden surge. It is found that the virus efficacy is not necessarily related to any particulates but it is more of source-based toxicity of its component where the virus is piggybacking. We conclude that the aged biomass BC particles tend to aggregate and react with other compounds to grow in size, providing temporary habitat to viruses leading to the rapid increase in COVID-19 cases which declined after the crop burning stopped.

1. Introduction

It has been reported that in 2019, air pollution caused more than 16.7 lakh deaths in India, causing economic losses of nearly $36.8 billion (Pandey et al., 2020). India ranked 2nd in terms of coronavirus infected cases, having an infection count of about ~10 million by the end of 2020. Exposure to high levels of pollution severely affects the lungs and causes inflammation to the lining of the windpipe and lungs, which reduces immunity and increases vulnerability to infection. Delhi became one of the hot spots with 0.582 million cases and 9425 Deaths by the end of 2020. By late June and mid-September, the daily infection count reached ~4500 in Delhi (https://www.mygov.in/covid-19/). Thereafter, daily infection count cases declined rapidly and reduced to ~12% of peak count with a minimum of ~500 per day signalling the closure. During this period, the pollution level was at its lowest. Tiny particulate matter penetrates deep into the respiratory system which later enters the bloodstream. During the COVID-19 outbreak, it was very hard to associate particulate parameters with the spread of Coronavirus due to several confounding factors such as; population, population density and age, number of tests performed, infected people, poverty rate, healthcare workers in the area, and other demographics. However, several studies around the world have linked air pollution to higher COVID-19 cases. A study carried out in severely affected Italy, correlated the incidence of COVID-19 cases with PM$_{2.5}$ levels (Lolli et al., 2020). The study at Harvard University (Wu et al., 2020) in the United States, found an association between air pollution over many years with an increase in mortality from COVID-19 infection. The most
recent work by Liu et al., 2020 revealed that SARS-CoV-2 may have the potential to be transmitted through aerosols. Although WHO (World Health Organization) acknowledged these findings but have largely ignored the airborne transmission route (Zhang et al., 2020) mainly due to conflicting views (Beig et al., 2020a, 2020b; Coronavirus Disease (COVID-19), 2021) and insufficient database evidence. We undertook this study, to critically examine whether the COVID-19 has any direct association with particulate pollutants or is it more of source-based toxicity of its component? PM$_{2.5}$ consists of constituents such as Black Carbon (BC) and Polycyclic Aromatic Hydrocarbons (PAH’s), etc. both of them have an adverse effect on human health (Ambade et al., 2021a). Atmospheric airborne PAH’s are mostly contributed from the combustion of fossil fuel and biomass burning (Kumar et al., 2020a). Ambade (2014) has reported the presence of heavy metals (HMs) in aerodynamic particulate matter (PM$_{2.5}$) in the hilltop of Dongargarh, India. In Jamshedpur’s urban site PAH’s are almost 0.25% of PM$_{2.5}$ concentration during winter months (Kumar et al., 2020b). The highest concentration of PAH’s is observed in urban sites of Jamshedpur than rural and semi-urban sites due to high vehicular emission, industrial, power plants and coal combustion. (Kumar et al., 2020a) The amount of BC produced during the combustion process depends on the availability of oxygen and temperature (Panicker et al., 2018). Exposure to BC may cause premature death, harmful effect on the cardiovascular system and respiratory ailments. Almost 40% of BC emissions are attributed to open biomass burning, 40% to fossil fuel burning, and the remaining 20% to biofuel burning (Ambade et al., 2021b; Ramanathan and Carmichael, 2008). Recently, Ambade et al. (2021a, 2021b, 2021c) reported the characterization, seasonal variation, source apportionment and health risk assessment of black carbon over an urban region of East India. We hereby explore the association of biomass induced black carbon aerosolized virus in one of the top-ranked polluted hot spot regions of the world, Delhi, at the time when other confounding factors were almost stable and the pandemic wave was on a declining stage. We also explored the association of COVID-19 with PM$_{2.5}$ particulate load.

Fig. 1. A schematic depicting the proposed mechanism (right panel) that when additional black carbon transported from the external biomass burning sources that are active only during October and November in the surrounding regions of megacity Delhi, combine with available moisture and other compounds (aged BC) under cooler conditions acts as vectors for the spread and survival of virus thereby enhances the COVID-19 fury. The regular process of urban fresh black carbon generated only from local sources of emissions like industrial, fossil fuel, etc. is shown in the left side panel.
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2. Material and methodology

Delhi, the capital of India, has a population of ~18 million and is located in the Northern part of India, at 28.61° N, 77.23° E as shown in the top left corner of Fig. 1. It is a landlocked city that experiences very hot summers and severe winters and is located at an elevation of 216 m above mean sea level. The monsoon starts in late June and lasts until mid-September. The study is carried out using the wide monitoring network of System of Air Quality and Weather Forecasting And Research (SAFAR), a research project of the Ministry of Earth Science, Government of India and a pilot study of the World Meteorological Organization (Beig et al., 2015). The impact of fire smoke exposure to infect the upper and lower respiratory system is generally not paid much attention (Yao et al., 2020). It is pertinent to mention here that, during the initial period there were many confounding factors and the pandemic wave was on the upward swing. However, when there was a decline in cases and other influencing factors were fairly uniform and stable, except pollution level, it provided us with the much-needed opportunity to better understand the association of COVID-19 with variable factors like BC and PM$_{2.5}$. Fig. 1 shows a schematic of the mechanism to explain how stubble fire-induced black carbon particulate pollution likely due to local emissions when combined with externally generated biomass induced BC contributed to the exacerbation of COVID-19 cases. The major component of soot, black carbon (BC), causes premature human mortality and disability (Goto, 2014). Furthermore, changes in the chemical composition of soot BC are accomplished due to heterogeneous oxidation reactions in the environment (Browne et al., 2015; Ivleva et al., 2013). The left side panel in Fig. 1 shows the normal scenario where due to various local sources, emissions of BC lead to urban fresh BC without any significant additional emissions from transported stubble burning. The right side panel of Fig. 1 depicts the processes when additional BC produced due to emissions from stubble burning regions get transported to Delhi under favourable meteorological conditions. Fine particulates (particulates <2.5 μm) have a longer lifetime, due to which their chances of transportation and accumulation in the atmosphere increase (Chen et al., 2016). The locally generated BC and the additional pyrogenic BC increases the abundance of total BC in Delhi’s environment which is discussed in the next section. However, the additionally transported BC gets transformed into aged BC particles becomes more toxic as they grow in size and may combine with other molecules (OC, sulphates, etc.) under the cold condition as shown in Fig. 1. These pyrogenic BC provide temporary habitat for air microbes aerosolized. The larger numbers of Virus get adsorbed on the surface and piggyback on BC. The BC particles also tend to accumulate and react with other compounds in the ambient air, such as organic carbon and sulphates. The presence of secondary pollutants such as nitrates and sulphates increase the reproduction rate of the SARS-CoV-2 virus (Liu et al., 2020). When we inhale a large number of these particles, the virus particle also goes deep down into the respiratory system, thereby increasing the total dose delivered and total duration for which the virus stays there. Oxidative stress produced by soot is linked with inflammation in the lungs, which results in the development of asthma and other diseases (Sarnat et al., 2012). The inflammation causes serious damage to lung functions. Hence, the carbonised virus will make it a double whammy for the lungs and is expected to aggravate the COVID-19 response as symbolically depicted with increasing width between 2 arrows in Fig. 1. Evidence supporting the above mechanism is discussed in the next section.

3. Proposed mechanism

Stubble burning is a source of bio-aerosols that differ in composition and concentration from those found under background conditions, and most of these microbes in smoke are capable of surviving in varying environmental conditions (Moore et al., 2020). The effect of fire smoke exposure to infect the upper and lower respiratory system is generally not paid much attention (Yao et al., 2020). It is pertinent to mention here that, during the initial period there were many confounding factors and the pandemic wave was on the upward swing. However, when there was a decline in cases and other influencing factors were fairly uniform and stable, except pollution level, it provided us with the much-needed opportunity to better understand the association of COVID-19 with variable factors like BC and PM$_{2.5}$. Fig. 1 shows a schematic of the mechanism to explain how stubble fire-induced black carbon particulate pollution likely due to local emissions when combined with externally generated biomass induced BC contributed to the exacerbation of COVID-19 cases. The major component of soot, black carbon (BC), causes premature human mortality and disability (Goto, 2014). Furthermore, changes in the chemical composition of soot BC are accomplished due to heterogeneous oxidation reactions in the environment (Browne et al., 2015; Ivleva et al., 2013). The left side panel in Fig. 1 shows the normal scenario where due to various local sources, emissions of BC lead to urban fresh BC without any significant additional emissions from transported stubble burning. The right side panel of Fig. 1 depicts the processes when additional BC produced due to emissions from stubble burning regions get transported to Delhi under favourable meteorological conditions. Fine particulates (particulates <2.5 μm) have a longer lifetime, due to which their chances of transportation and accumulation in the atmosphere increase (Chen et al., 2016). The locally generated BC and the additional pyrogenic BC increases the abundance of total BC in Delhi’s environment which is discussed in the next section. However, the additionally transported BC gets transformed into aged BC particles becomes more toxic as they grow in size and may combine with other molecules (OC, sulphates, etc.) under the cold condition as shown in Fig. 1. These pyrogenic BC provide temporary habitat for air microbes aerosolized. The larger numbers of Virus get adsorbed on the surface and piggyback on BC. The BC particles also tend to accumulate and react with other compounds in the ambient air, such as organic carbon and sulphates. The presence of secondary pollutants such as nitrates and sulphates increase the reproduction rate of the SARS-CoV-2 virus (Liu et al., 2020). When we inhale a large number of these particles, the virus particle also goes deep down into the respiratory system, thereby increasing the total dose delivered and total duration for which the virus stays there. Oxidative stress produced by soot is linked with inflammation in the lungs, which results in the development of asthma and other diseases (Sarnat et al., 2012). The inflammation causes serious damage to lung functions. Hence, the carbonised virus will make it a double whammy for the lungs and is expected to aggravate the COVID-19 response as symbolically depicted with increasing width between 2 arrows in Fig. 1. Evidence supporting the above mechanism is discussed in the next section.

4. Results

Fig. 2 strengthen our hypothesis that is explained in Fig. 1 where we find that BC concentration directly corresponds to the speed at which the infections due to novel coronavirus disease spread after the onset of winter and stubble burning period and then reduced
with a declining trend in BC related with reduction in stubble fire counts as evident from Fig. 2 (a, b). The surge in BC is directly related to the additional contribution of stubble burning induced PM$_{2.5}$ concentration transported externally from stubble burning regions (Fig. 2 (c)). The share of PM$_{2.5}$ due to biomass burning is simulated from the SAFAR-WRF-Chem model (Beig et al., 2020a, 2020b) (Supplementary material – 1. Model Methodology). It is noted from Fig. 2(b) that the peak in death is noticed after a lag time of about 10 days of peak infection which implies that probable death of around 10–12% of the infectious patients occurs after about 10 days of infection. A highly significant positive correlation ($r = 0.74$, $P_{value} = 8.8*10^{-16}$ at 95%, CI = [0.62,0.82]) of BC with infection counts and with Death considering 10 days lag ($r = 0.84$, $P_{value} = 2.02*10^{-13}$ at 95%, CI = [0.73,0.91]) for the entire period, supports the above interpretation (Fig. 2(a, b)). Fig. 2(c, d) shows the correlation of total PM$_{2.5}$ with infection and death, respectively and the correlation is listed in Table 1. The correlation of PM$_{2.5}$ with COVID-19 infection counts remains significant ($r = 0.76$, $P_{value} = 4.11*10^{-10}$ at 95%, CI = [0.61,0.86]) only during the increasing phase of BC [25th Sept-10th Nov] but thereafter it starts to weaken and become insignificant if we consider the whole study period. This implies that the correlation holds good only until BC abundance is sufficiently high which contains a major fraction from the transported aged BC. After the peak, the level of PM$_{2.5}$ continued to remain elevated due to unfavourable local meteorological condition but the contribution of BC started to decline with the reduction in fire counts and its contribution in PM$_{2.5}$ touched the pre-biomass burning levels conforming to the declining trend in COVID-19 cases. Table 1 shows the summary of the correlation coefficient as obtained in this work which is stronger as long as BC from fire counts are contributing to PM$_{2.5}$ but becomes insignificant when other normal periods are included. The potential for fire’s microbial content to affect humans who breathe in smoke-laden polluted environments, especially from high-emission prolonged fires like Kharif harvesting in India extended during the study period, leads to a severity trend. Microbial cells have been found to associate positively with particulate matter (Moore et al., 2020). It is reported (Kobziar and Thompson, 2020) that smoke-related immunologic deficits and inflammatory responses may exacerbate the effects of inhalation of airborne microbial particulates and toxicants in smoke. All these reported findings support our hypothesis and are likely to be one of the major reasons for exacerbated COVID-19 cases.

5. Conclusion

We examined the association of COVID-19 with particulate pollutant, particularly the black carbon in this paper in one of the largest smoggy megacities of the world during the post-monsoon season when farmers burn the stubble aggressively to ready the field for the next crop in a tight window period of two months. The BC is a major constituent of soot and extremely harmful to human health and hence makes this study very useful for the common population. The study concludes that SARS-CoV-2 has the potential to be transmitted aggressively through an aged Black carbon-rich environment with fire’s microbial content rather than any particulate pollution. This implies that the association of COVID-19 with particulate pollutants depends on the source-based toxicity of its components. The mechanism is supported by a strong correlation of relevant parameters obtained in this work. Although we have not established the infectivity of the carbonised virus clinically, we propose that SARS-CoV-2 may have the potential to be more lethal when piggybacking on aged BC whose oxidative effect itself leads to serious damaging effects to lungs. Atmospheric and public health sciences need to expand their perspectives to include the potential impact of smoke’s black carbon and microbial cargo on human populations. The conjunction of epidemiological trends and smoke microbial content need to be addressed in public health and atmospheric sciences.

Consent for publication

Both authors agree to the publication of results.

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Availability of data and materials

Available on request from authors.

| S.N. | Parameters          | Correlation (R) (Full Period) | Correlation (R) (Until Infection Peak: 10th Nov) |
|------|---------------------|-------------------------------|-----------------------------------------------|
| 1.   | Black Carbon v/s Infection (10 days lag) | 0.74* | – |
| 2.   | Black Carbon v/s Death (10 days lag) | 0.83* | – |
| 3.   | PM$_{2.5}$ v/s Infection (10 days lag) | 0.43 | 0.76* |
| 4.   | PM$_{2.5}$ v/s Death (10 days lag) | 0.56 | 0.84* |

* Significant at 95% level with $p$-value $<$0.05.
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