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EXPLORING THE COMPLEX RELATIONSHIP BETWEEN ASTHMA AND AIR POLLUTION IN URBAN REGIONS: AN ACCURATE AND RELIABLE PREDICTIVE MODEL

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ABSTRACT
Asthma is a chronic respiratory disease that affects millions of people worldwide. It is well-established that environmental factors, such as air pollution, can exacerbate asthma symptoms and lead to increased hospitalizations and mortality rates. Understanding the association between asthma and air pollution is crucial for public health interventions and policymaking. Traditionally, epidemiological studies have been conducted to establish the association between asthma and air pollution. These studies involve collecting data from asthma patients, monitoring air quality parameters, and then statistically analyzing the data to identify correlations. While informative, these studies often have limitations such as extensive time requirements, data collection challenges, and the inability to capture real-time associations. Recent days, the machine learning algorithms are gaining lots of attention in many fields including pollution monitoring. Thus, the supervised learning algorithms can gain valuable insights into the complex relationship between asthma and air pollution in urban regions, leading to more targeted and effective interventions for public health. Therefore, this research goal is to develop an accurate and reliable predictive model that can inform public health strategies and policies. This model should aid in proactive decision-making, allowing healthcare providers to allocate resources efficiently and enabling policymakers to implement targeted interventions to reduce air pollution and minimize the impact of asthma on vulnerable urban populations.

Keywords: Asthma and Air pollution, Urban regions, Epidemiological.

1. INTRODUCTION

Outdoor air pollution contributed more than 3% of the annual disability-adjusted life years lost in the 2010 Global Burden of Disease comparative risk assessment, a notable increase since the previous estimate was made in 2000.1 Previous assessments of global disease burden attributed to air pollution were restricted to urban areas or by coarse spatial resolution of concentration estimates.2 In a study of ten European cities, 14% of the cases of incident asthma in children and 15% of all exacerbations of childhood asthma were attributed to exposure to pollutants related to road traffic.3 Urbanisation is an important contributor to asthma and this contribution might be partly attributed to increased outdoor air pollution (figure 1).4–6 Because many urban centres in the developing world are undergoing rapid population growth accompanied by increased outdoor air pollution, the global burden of asthma is likely to increase. In this context, it is notable that the populations of China, India, and Southeast Asia are equal to the rest of the world combined. In view of the burden of asthma attributed to outdoor air pollution, a better understanding of why asthmatic individuals are susceptible to this exposure should enable the design of effective preventive strategies. The idea that air pollution can cause exacerbations of preexisting asthma is supported by an evidence base that has been accumulating for several decades,7–10 but evidence has emerged that suggests air pollution might cause new-onset asthma as well.11–21 Not all studies support a causal link between air pollution and asthma, and a recent meta-analysis22 of cross-sectional studies that compared communities with different levels of pollution showed no effect of long-term exposure to pollution on asthma prevalence. Although outdoor air pollution almost always occurs as a mixture, air quality is regulated by most jurisdictions in terms of its individual components. Such regulation has meant that experimental studies of humans and animals have been focused on individual pollutants. Because epidemiological studies inherently involve

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exposure to mixtures of pollutants, substantial efforts are usually made to try to identify the individual effects of pollutants, which often obscures the health effect of the mixture as a whole. With increasing attention to traffic-related air pollution (TRAP) as the exposure variable of interest, a shift has occurred away from a focus on individual components of the pollution mixture. In this Series paper, we will attempt to discuss the effects of several gaseous pollutants (ozone, nitrogen dioxide, and sulphur dioxide), the independent effects of various forms of PM, and then focus on the effects of TRAP as a mixture. We concentrate on studies published in the past 5 years that report results relevant to both exacerbation and onset of asthma. We focus primarily, although not exclusively, on epidemiological and experimental clinical studies. Controlled exposure studies in human beings are restricted by small sample size and an inability to study the potentially most susceptible subgroups (eg, children and adults with severe asthma) and the effects of chronic exposure. Epidemiological studies are restricted by imprecise methods of both exposure and asthma outcome assessment and often inadequate data about potentially confounding variables. Although the potential effect of indoor air pollution on asthma is an important concern, especially in developing countries where much domestic cooking is done with solid fuels, it is outside the scope of this review.

2. LITERATURE SURVEY

2.1 Asthma So Affected by Exposure to Air Pollution

Why are individuals with asthma so affected by exposure to air pollution? At high concentrations, such as those noted in megacities in India and China, air pollutants might have direct irritant and inflammatory effects on airway neuroreceptors and epithelium, but such levels of exposure rarely occur in North America or Europe. At the lower concentrations that are more typical in high-income countries, other mechanisms are probably in operation. Specific pollutants can induce airway inflammation (eg, ozone, nitrogen dioxide, and PM <2·5 μm in diameter [PM2·5])23–28 and airway hyper-responsiveness (ozone and nitrogen dioxide),23,29 two characteristic features of asthma. In addition, oxidative stress (a feature of severe asthma) has been associated with pollutant exposures (ozone, nitrogen dioxide, and PM2·5).30–32 Therefore, exposure to these pollutants is unsurprisingly associated with exacerbations and possibly even the onset of asthma. The mechanisms by which pollutants induce these effects are not completely clear.

2.2 Air Pollution Might Contribute

A framework for how air pollution might contribute to the development and exacerbation of asthma proposed by the UK's Committee on the Medical Effects of Air Pollutants identified four main mechanisms: oxidative stress and damage, airway remodelling, inflammatory pathways and immunological responses, and enhancement of respiratory sensitisation to aeroallergens (figure 2).33 Variation in the genes that regulate these mechanisms could confer increased susceptibility to development of new-onset asthma or exacerbations of existing disease with exposure to air pollution.

2.3 Traffic-Related Air Pollution

Because the pollutants of interest, including TRAP, can cause oxidative stress, the ability of antioxidant defenses to handle the increased load of reactive oxygen species generated in the lungs after exposure is an important determinant of risk for subsequent adverse effects. Specific polymorphisms in antioxidant enzyme genes, such as glutathione S-transferase genes, GSTM1 and GSTP1, can modify risk of asthmatic responses to pollutants34,35 and these variants (GSTM1 null and GSTP1 Ile105Val) might also interact with a tumour necrosis factor (TNF) promoter variant (G-308A) that affects expression of TNF and hence the early inflammatory response.36 Additionally, neonatal rats are more prone to oxidative stress from PM exposure at least in part due to relative deficiency of nuclear factor-like 2 (Nrf2).37 Proinflammatory effects of oxidative stress are mediated by the redox-sensitive MAP

kinase and nuclear factor-κB cascades that are responsible for the expression of cytokines, chemokines, and adhesion molecules, and reduced antioxidant capacity in the airways can result in altered expression after pollutant exposure.38 Other pathways through which oxidising pollutants might affect severity of asthma involve control of immune responses. TRAP, specifically ambient polycyclic aromatic hydrocarbons and diesel-exhaust particles, affect regulatory T cell (Treg) function through an epigenetic mechanism.39,40 Hypermethylation of CpG islands in Foxp3 associated with chronic exposure to polycyclic aromatic hydrocarbons39 or diesel-exhaust particles40 leads to suppression of Treg function and increased asthma severity as assessed by symptoms and lung function. Hypermethylation of interferon γ in effector T cells, contributing to a shift towards a Th2 response, has also been associated with exposure to air pollution.41Studies in animals and in vitro42,43 suggest that exposure to PM results in allergic inflammation with Th2 and Th17 phenotypic differentiation, with a specific role for environmentally persistent free radicals and polycyclic aromatic hydrocarbon fractions of PM in this differentiation. In addition, exposure to diesel-exhaust particles is associated with increased serum interleukin 17 and increased symptoms in children with allergic asthma; a parallel study44 that used a murine model of allergic airway inflammation showed that combined exposure to diesel-exhaust particles and antigen from a house dust mite induced a mixed Th2/Th17 response.

2.4 A Potential Enhancing Effect of Pollutant Exposure On Responses

A potential enhancing effect of pollutant exposure on responses to inhaled allergen has been studied in both animals and man, with evidence for such an effect on lung function and inflammatory responses to ozone, nitrogen dioxide, sulphur dioxide, and diesel-exhaust particles.45–48 Several mechanisms through which air pollutants could enhance sensitisation to aeroallergens have been proposed and include increased deposition of allergen in the airways due to carriage by particles, increased epithelial permeability due to oxidative injury, increased antigenicity of proteins from chemical modification, and a direct adjuvant effect (including for diesel-exhaust particles in human beings).49In summary, air pollutants might cause oxidative injury to the airways that leads to inflammation and remodelling, which in a genetically predisposed individual could result in clinical asthma. One predisposing factor might be atopy, and air pollutants could increase the risk of sensitisation and the responses to inhaled allergen in individuals with asthma.

2.5 Software Environment

Python is a high-level, interpreted scripting language developed in the late 1980s by Guido van Rossum at the National Research Institute for Mathematics and Computer Science in the Netherlands. The initial version was published at the alt. Sources newsgroup in 1991, and version 1.0 was released in 1994.

Python 2.0 was released in 2000, and the 2.x versions were the prevalent releases until December 2008. At that time, the development team made the decision to release version 3.0, which contained a few relatively small but significant changes that were not backward compatible with the 2.x versions. Pythons 2 and 3 are very similar, and some features of Python 3 have been back ported to Python 2. But in general, they remain not quite compatible.

Both Python 2 and 3 have continued to be maintained and developed, with periodic release updates for both. As of this writing, the most recent versions available are 2.7.15 and 3.6.5. However, an official End of Life date of January 1, 2020 has been established for Python 2, after which time it will no longer be maintained. If you are a newcomer to Python, it is recommended that you focus on Python 3, as this tutorial will do.

Python is still maintained by a core development team at the Institute, and Guido is still in charge, having been given the title of BDFL (Benevolent Dictator For Life) by the Python community. The name Python, by the way, derives not from the snake, but from the British comedy troupe Monty Python's Flying Circus, of which Guido was, and presumably still is, a fan. It is common to find references to Monty Python sketches and movies scattered throughout the Python documentation.

4. PROPOSED SYSTEM

Ambient PM is a ubiquitous atmospheric aerosol with both anthropogenic and natural sources that has been associated with various health effects.50 PM is categorized on the basis of its aerodynamic diameter, with implications for its typical site of deposition when inhaled (figure 3). Coarse PM, with an aerodynamic diameter of 2·5–10 μm, deposits mainly in the head and large conducting airways. Fine PM or PM2·5 deposits throughout the respiratory tract, particularly in small airways and alveoli. Ultrafine PM (Some evidence suggests PM is a cause of incident asthma (aside from the literature on TRAP). Independent associations between exposure to PM10 in utero and during infancy with asthma diagnosed by a doctor were identified in a nested case-control study within a large birth cohort.18 Although several studies have identified associations between asthma prevalence and exposure to outdoor PM,11,64,65 this finding has not always been consistent.22 Furthermore, PM is frequently strongly correlated with ozone, nitrogen oxides, and sulphur oxides, serving to confound these associations.

Advantages

In summary, substantial evidence supports the idea that ambient levels of PM exacerbate existing asthma, particularly by contributing to oxidative stress and allergic inflammation, and some evidence exists in support of PM as a cause of new cases of asthma.

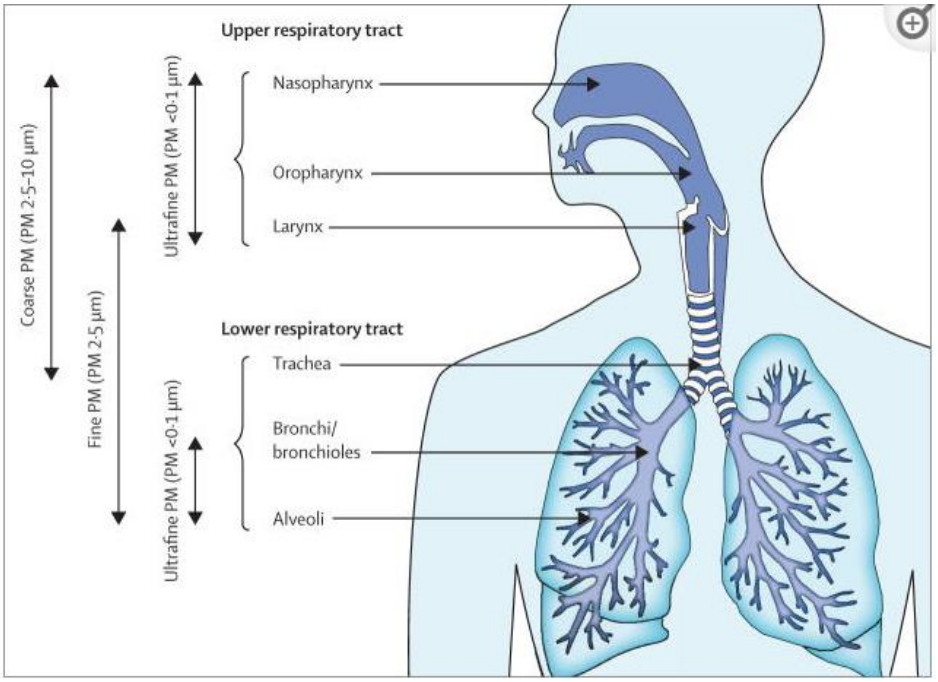
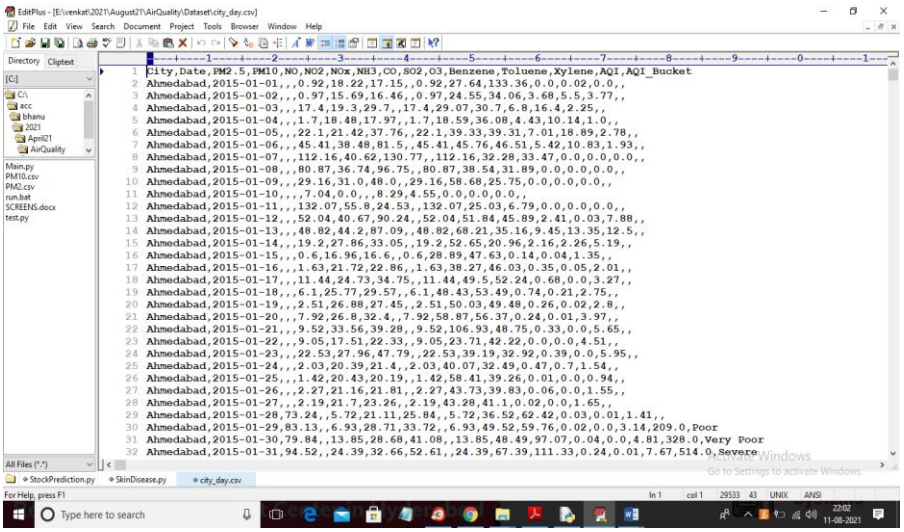


Figure. 1: Compartmental deposition of particulate matter.

4. RESULTS

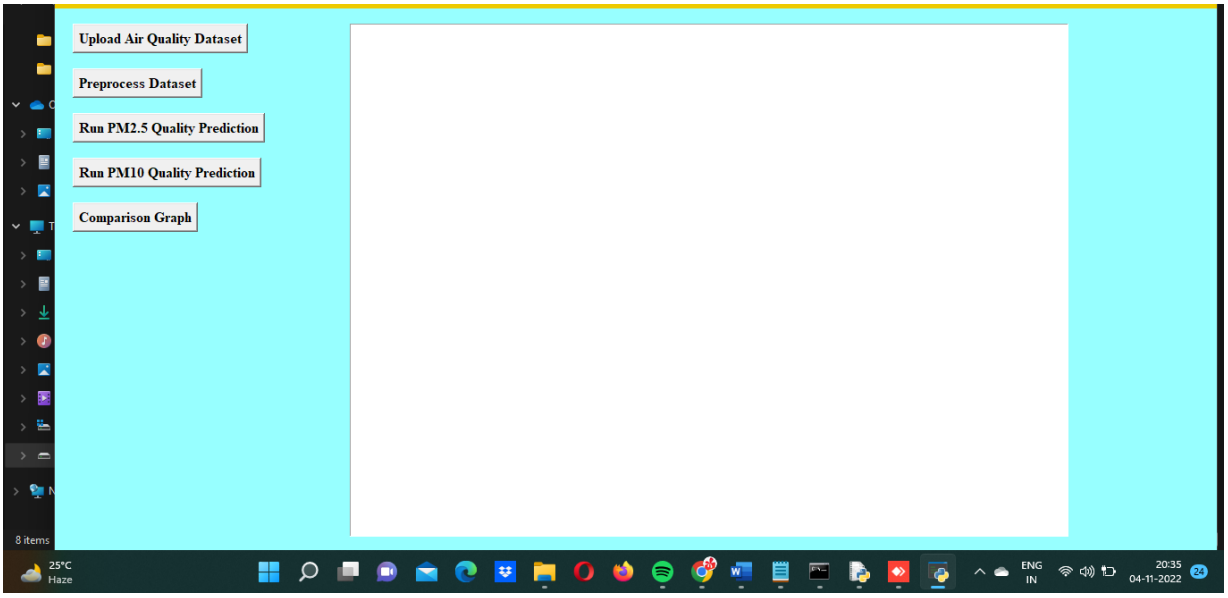
In this paper author is using various machine learning algorithms to predict air quality as this air quality put heavy effect on human health. In this paper author has propose LIGHT GBM algorithm which can predict air quality efficiently from high-dimensional large-scale data. In proposed paper author is comparing RMSE (Root Mean Square Error) error rate with other algorithms such as ADABOOST, GBDT (Gradient Boosting Decision Tree), XGBoost and DNN. The lower the RMSE error rate the better is the prediction model and in all algorithms Light GBM is giving less error rate.

To implement this paper author has used Beijing dataset but this dataset is not available on internet so we are using Air Quality dataset from KAGGLE website and below screen showing details of dataset. In this paper we are using PM2.5 and PM10 quality values to predict air quality.



In above dataset screen we have city name, date and other air quality values and we will use above dataset with above mention algorithms to predict air quality and compare RMSE error rate.

To run project double, click on 'run.bat' file to get below screen



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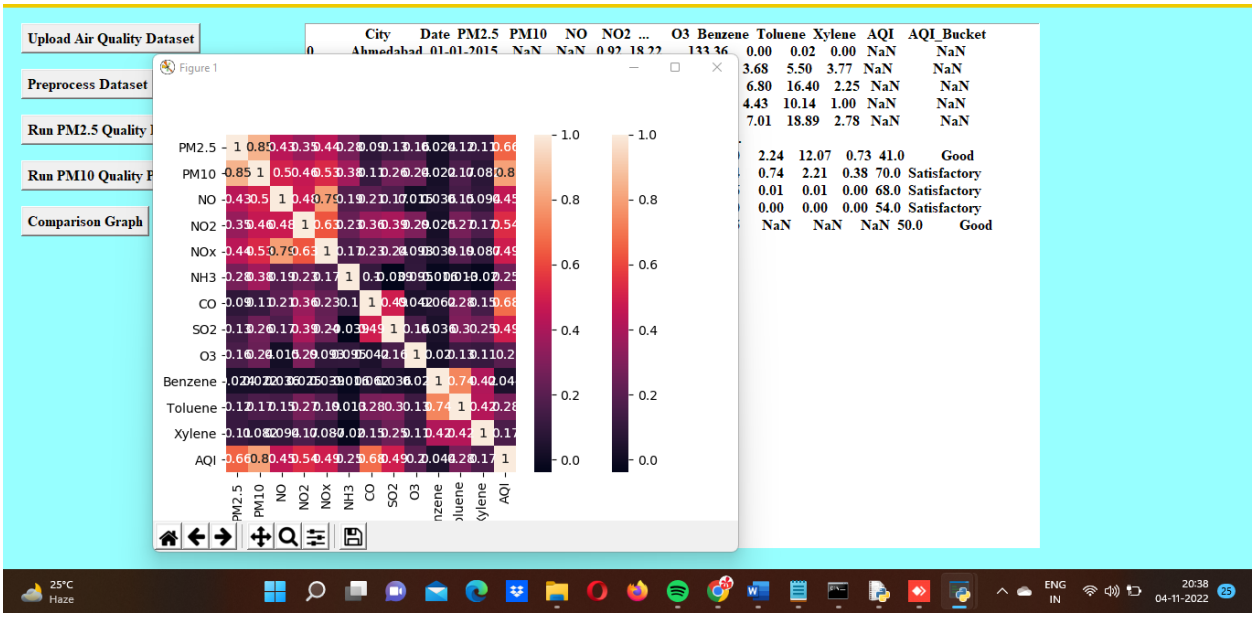
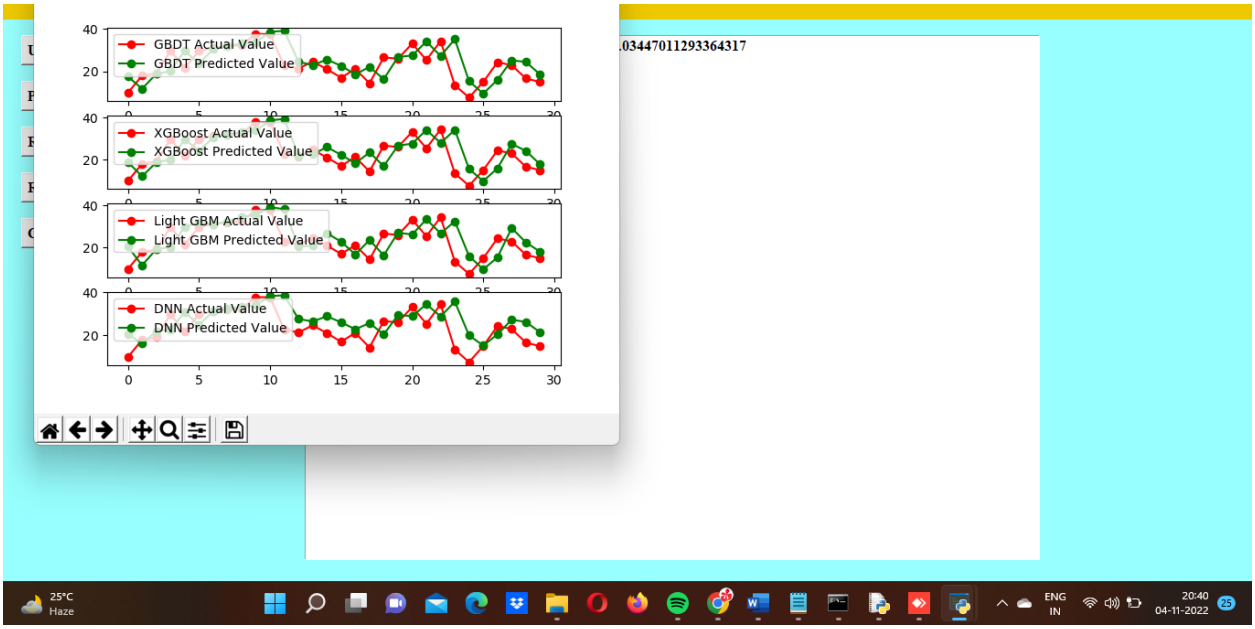
In above screen click on 'Upload Air Quality Dataset' button to upload dataset and to get below screen



In above screen dataset loaded and we can see dataset values in text area and in graph we can see column name from dataset and its correlation feature importance values in graph boxes. Now close above graph and click on 'Preprocess Dataset' button to remove missing values and then calculate air pollution rate date wise in dataset like below screen



In above screen we can see RMSE error rate for all 4 algorithms and in all algorithms Light GBM got less RMSE error rate. Below is the predicted and original values from all 4 algorithms

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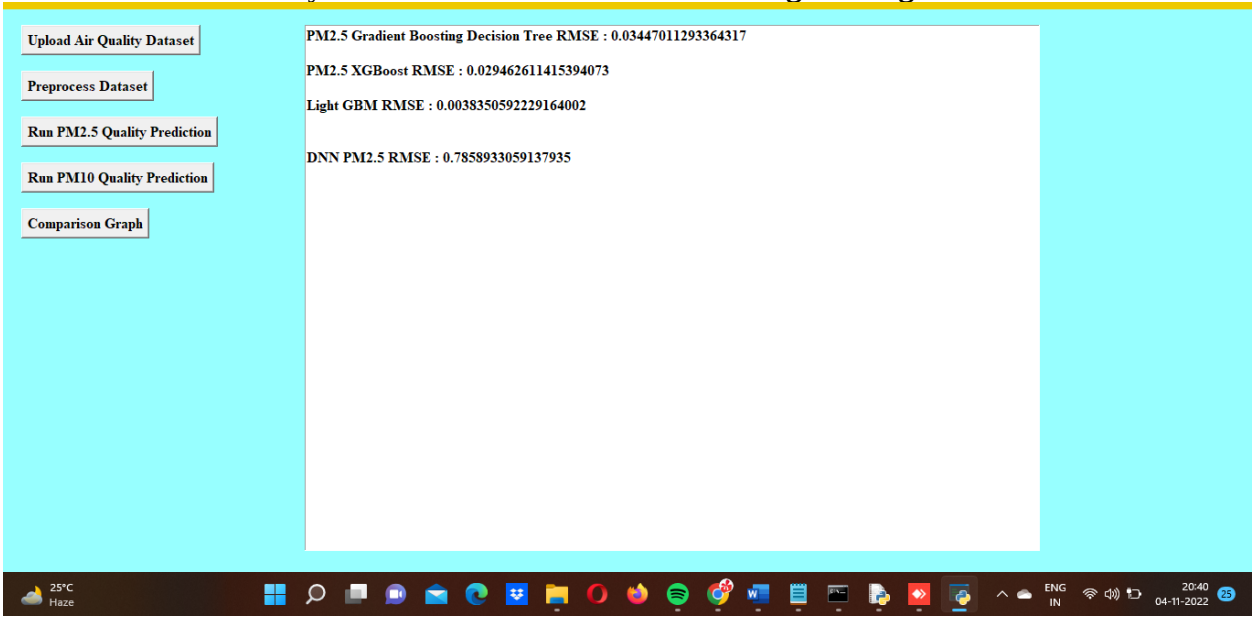

In above screen we can see RMSE error rate for all 4 algorithms and in all algorithms Light GBM got less RMSE error rate. Below is the predicted and original values from all 4 algorithms

In above graph red line represents original air quality and green line represents predicted air quality and I am displaying prediction graph for all 4 algorithms and from all 4 Light GBM is closed and now closed above graph and then click on 'Run PM10 Quality Prediction' button to predict air quality for PM10 using all 4 algorithms

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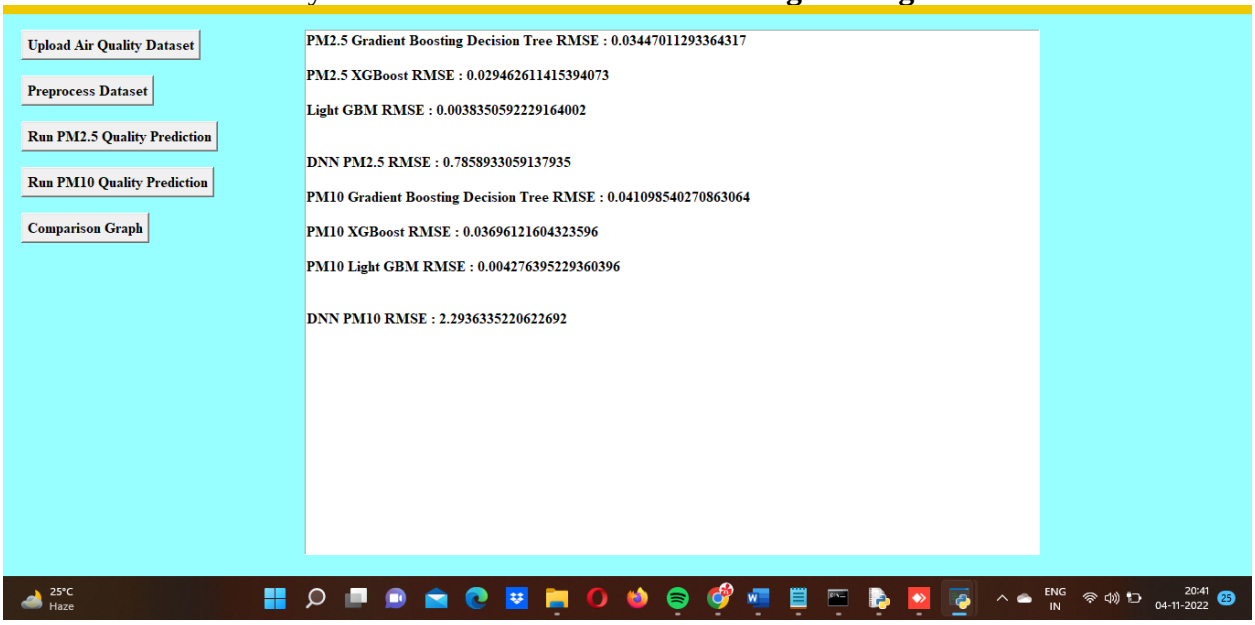
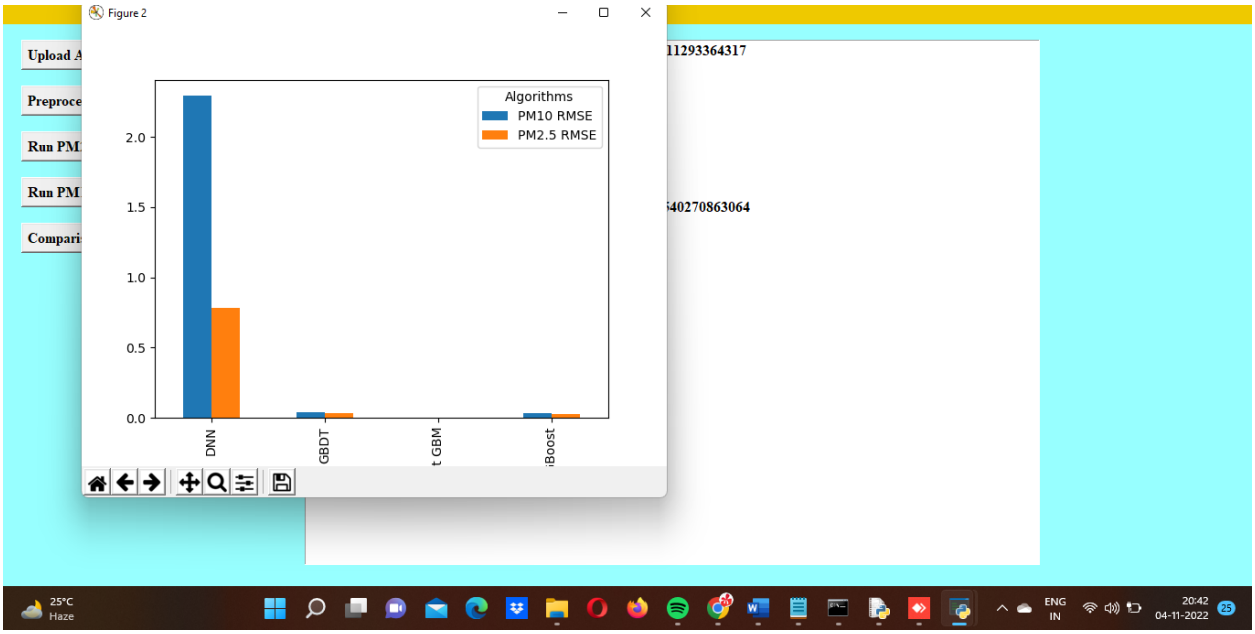


In above screen all 4 algorithms run for air quality PM 10 and in all algorithms Light GBM got less RMSE error and below is the prediction graph for all 4 algorithms



In above graph x-axis represents algorithm names and y-axis represents RMSE error rate and for both PM10 and PM2.5 air quality Light GBM got less RMSE error

5. CONCLUSION

A substantial body of research on the effects of air pollution on asthma has been published in the past 5 years, adding to the body of knowledge that has accumulated over several decades. Presently, short-term exposures to ozone, nitrogen dioxide, sulphur dioxide, PM2·5, and TRAP is thought to increase the risk of exacerbations of asthma symptoms. Increasing amounts of evidence also suggest that long-term exposures to air pollution, especially TRAP and its surrogate, nitrogen dioxide, can contribute to new-onset asthma in both children and adults. Much more about the mechanisms that are involved with

exacerbations induced by pollution and onset of asthma needs to be understood, but oxidative stress and immune dysregulation are probably both involved. Young children with asthma, especially those growing up in poor neighborhoods, are at increased risk of adverse effects from exposures to air pollution. Unravelling which components of the traffic pollution mixture are responsible for asthma exacerbations and onset is a substantial challenge. Improved air quality to prevent exacerbations and new cases of asthma will require strong governmental efforts to move economies in both developed and developing countries away from combustion of fossil fuels for transportation and energy production; this approach is also needed to mitigate climate change.

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