A NEW ARTIFICIAL INTELLIGENCE MODEL FOR AIR QUALITY PREDICTION AND ANALYSIS

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ABSTRACT
Traffic and power generation are the main sources of urban air pollution. The idea that outdoor air pollution can cause exacerbations of pre-existing asthma is supported by an evidence base that has been accumulating for several decades, with several studies suggesting a contribution to new-onset asthma as well. In this Series paper, we discuss the effects of particulate matter (PM), gaseous pollutants (ozone, nitrogen dioxide, and sulphur dioxide), and mixed traffic-related air pollution. We focus on clinical studies, both epidemiological and experimental, published in the previous 5 years. From a mechanistic perspective, air pollutants probably cause oxidative injury to the airways, leading to inflammation, remodelling, and increased risk of sensitisation. Although several pollutants have been linked to new-onset asthma, the strength of the evidence is variable. We also discuss clinical implications, policy issues, and research gaps relevant to air pollution and asthma.
INDEX TERMS:air pollution,exacerbation's,particulate matter,epidemiological.

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 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.

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.

2.3TRAFFIC-RELATED AIR POLLUTION

Because the pollutants of interest, including TRAP, can cause oxidative stress, the ability of antioxidant defences 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.

MODULES

1) Upload Air Quality Dataset: Using this module we will upload dataset to application and then calculate features importance using correlation formula. The feature which is important for prediction will have high value
2) Preprocess Dataset: using this module we will analyses entire dataset and then remove empty and missing values
3) Run PM2.5 Quality Prediction: using this module we will predict PM2.5 air quality by using Light GBM, GBDT, XGBOOST and DNN (Deep Neural Network) .
4) Run PM10 Quality Prediction: using this module we will predict PM10 air quality by using Light GBM, GBDT,

XGBOOST and DNN (Deep Neural Network).

5) Comparison Graph: using this module we will compare RMSE(Root Mean Square Error) error rate between all algorithms for PM2.5 and PM10.

IMPLEMENTATION

Information about air pollutants is obtained from the sensors, analysed, and then saved as a dataset. This dataset has been pre-processed with a variety of features, which includes attribute selection and normalisation. Once it is available, the dataset is divided into a training set and a test dataset. The training dataset is then used to apply a Machine Learning algorithm. The obtained results are matched with the testing dataset and results are analysed.

Machine Learning model Machine Learning algorithm is implemented to predict the air pollution. Machine Learning (ML) is a subfield of Artificial Intelligence (AI) that enables the software applications to be accurate in predicting the outcomes without being explicitly programmed to do so. To predict the new outcomes, Machine Learning algorithms make use of existing past data as the input. With the help of Machine Learning, a user can provide a computer program huge amount of data, and the computer will only examine that data and draw conclusions from it. KNN is the Machine Learning algorithm used for the prediction of air pollution. The K-Nearest Neighbors (KNN) algorithm is one of the types of Supervised Machine Learning algorithms. KNN is incredibly simple to design but performs quite difficult classification jobs. KNN is called the lazy learning algorithm as it lacks the training phase. Instead, it classifies a fresh data point while training on the entire dataset. It does not make any assumptions, hence it is called non-parametric learning method. Steps in KNN:Determine the distance between each sample of the training data and the test data.To determine distance, we can utilise the Euclidian or Minkowski or Manhattan distance formula.Sort the estimated distances in ascending order.Vote for the classes.Output will be determined based on class having most votes.

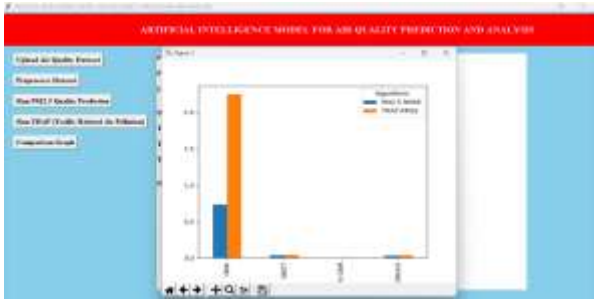
Calculate the Accuracy of the model, if required rebuild model.Another purpose to try to stationarize a time series is the capacity to obtain meaningful sample statistics, such as means, variances, and correlations with other parameters. Such statistics can only be utilized to forecast behaviour in the future if a series is stationary. The sample mean and variance, for instance, will rise with sample size and consistently undervalue the mean and variance in succeeding periods if the series is increasing continuously over time. Moreover, the series' mean and variance are not specifically articulated if the mean, variance, and correlations with other variables are not. For this reason, consider caution when extrapolating regression models fitted to nonstationary data.

RESULTS

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 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

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

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 economically disadvantaged neighbourhoods, 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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