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Review Paper

Long-Term Health Effects of Air Pollution Exposure for Investigating Its Link to Chronic Obstructive Pulmonary Disease (Copd), Lung Cancer, and Animal Health

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Key Words

Air pollution, Copd, Lung cancer, Long-term exposure, Respiratory diseases and Environmental health.

Abstract

Air pollution poses a major issue as an environmental health risk, and the causes of low respiratory diseases associated with prolonged exposure to air pollution include Chronic Obstructive Pulmonary Disease (COPD) and lung cancer. This paper examines the association between air pollution and the occurrence of COPD and lung cancer with regard to how the long-term exposure of humans to air pollutants like particulate matter (PM), nitrogen dioxide (NO2), and sulfur dioxide (SO2) leads to the development of the disease. The goal is to learn how air pollution aggravates lung disease conditions, evaluate the rates of COPD and lung cancer in the highly exposed population, and identify preventive strategies that may be applied. These approaches involve thorough examination of epidemiological research, long-term cohort research, and clinical data, as well as monitoring of air quality in both urban and industrial locations. Findings indicate the existence of a clear association between long-term air pollution exposure and elevated risk of developing COPD and lung cancer. Pollutants play a role in causing inflammation, oxidative stress, and DNA damage of lung tissues, which increases the pace of the disease. This research stresses how important it is that animal models should be incorporated when researching the broader ecological impact that air pollution has on lung health. The addition of animal models will act as a bridge between human and animal health research. The research arrives at a conclusion that these long-term health effects can be alleviated by regulating the exposure to air pollution through stricter regulations, improving the quality of the air, and creating awareness of the health hazard.

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Introduction

Ambient particulate matter (PM2.5) is a major cause of environmental hazard closely associated with some chronic diseases, specifically respiratory diseases such as chronic obstructive pulmonary disease (COPD) and lung cancer. The World Health Organization (WHO) observed that exposure to ambient PM2.5 causes a significant burden of disease in the world, with COPD and lung cancer being some of the most noticeable consequences. Traffic-air pollution has been identified as the biggest cause of COPD, and research findings reveal that people residing in high traffic pollution areas are at increased risk of acquiring such diseases (Andersen et al., 2011). Moreover, it was affirmed that ambient air pollution is linked to the development of COPD in a meta-analysis, as (Park et al. 2021) established that sustained exposure to pollutants is harmful to lung health (Park et al., 2021).

The documented effects of air pollution on human health are extensive; however, the effects on animals are equally troubling. Many domestic animals (i.e., dogs and cats) residing in urban settings are now also being exposed to the harmful effects of air pollutants such as PM2.5 volatility, NO2, and SO2 (Pal et al., 2015). Many dogs living in high-density urban settings have established a continued exposure to the same air quality as humans with Chronic Obstructive Pulmonary Disease (COPD) and are experiencing chronic bronchitis and airway inflammation. Similarly, many horses that are being held indoors and have had significant exposure to particulate matter are developing Equine Asthma or 'heaves,' which is an equivalent condition to COPD in humans. In addition to domestic animal models, many researchers also utilize rodent species as a model for studying the molecular aspects of how air pollution contributes to lung disease. These experiences and results illustrate that air pollution has a much more extensive effect than just on humans and requires comprehensive public health policy development in order to ensure the protection of domestic animals and wildlife, as well as humans, from air pollution-related health problems. Long-term exposure to air pollution has been shown in both human and animal studies to have a substantial impact on the cardiovascular and respiratory systems, and it has wide-ranging effects on the overall health of people and many species (Lederer et al., 2021).

Along with COPD, long-term air pollutants such as PM2.5 have been associated with an elevated risk of lung cancer as well as a higher death rate because of the two ailments (Krishnan et al., 2025). The research carried out in China also highlights the international relevance of the problem, as it was observed that the mortality rates in relation to lung cancer and COPD have risen significantly, thanks to exposure to air pollution (Chung et al., 2022). In the same way, a study carried out in Taiwan concluded that prolonged PM2.5 exposure led to an observable loss of lung capacity and heightened chances of developing COPD, which underscored the necessity to control the air pollution in areas with significant amounts of particulate matter (Guo et al., 2018). Also, the results of United Kingdom-based studies demonstrated that the incidence of

COPD has a close relationship with long-term exposure to air pollution, such as PM2.5, which has an important implication for the policy of health protection by the population (Atkinson et al., 2015).

These health effects are multifactorial in terms of their biological processes. Exposure to PM2.5 causes chronic inflammation, DNA damage, and mutagenic changes in lung cells, which could lead to the occurrence of COPD and lung cancer. Obstructive pulmonary diseases have been associated with inflammatory responses that result from the presence of particulate matter, which leads to tissue remodeling (Duan et al., 2020). Furthermore, the fact that the individuals exposed to the high rates of pollution have DNA mutations is one more fact that tends to prove the idea that air pollution is one of the direct causes of carcinogenesis. Kulkarni and Nair (2024) highlight the importance of accurate medical terminology in public health surveillance and pandemic preparedness, a critical framework for investigating the long-term health effects of air pollution exposure, particularly its link to chronic obstructive pulmonary disease (COPD) and lung cancer (Kulkarni & Nair, 2024).

Even having these established associations, there are gaps in comprehending the extent of the health effects of PM2.5. Molecular mechanisms, especially how genetic susceptibility can be used in moderating responses to pollution, are not well understood. Moreover, although a lot has been done regarding the short-term impacts of PM 2.5, there has been a need for more investigations on the long-term impacts, especially on the cumulative impact of long-term exposure. The knowledge of these processes is vital in formulating specific public health measures that can be used to minimize exposure and counter the ill effects of air pollution on susceptible populations. The study will not only help in the increased understanding of the health risks posed by PM2.5 but also inform policies to reduce the levels of air pollution and improve air quality, thus reducing the burden of respiratory illnesses such as COPD and lung cancer in the world.

Literature Review

It has been found that long-term exposure to air pollution is an important risk factor for chronic diseases, especially chronic obstructive pulmonary disease (COPD) and lung cancer. Air pollution, particularly the fine particulate matter (PM2.5), has been indicated to be able to penetrate deep into the lungs, leading to inflammation and adding to the overall progressive diminishing of lung functioning. Pollutant-induced inflammation may result in a characteristic of COPD airway remodelling, and may worsen pre-existing respiratory issues. Prolonged exposure to air pollution caused by traffic has been reported to have a strong connection with the occurrence of COPD (Viegi et al., 2006). A Japanese study revealed that there was a definite connection between ambient air pollution and respiratory diseases and lung cancer mortality (Katanoda et al., 2011).

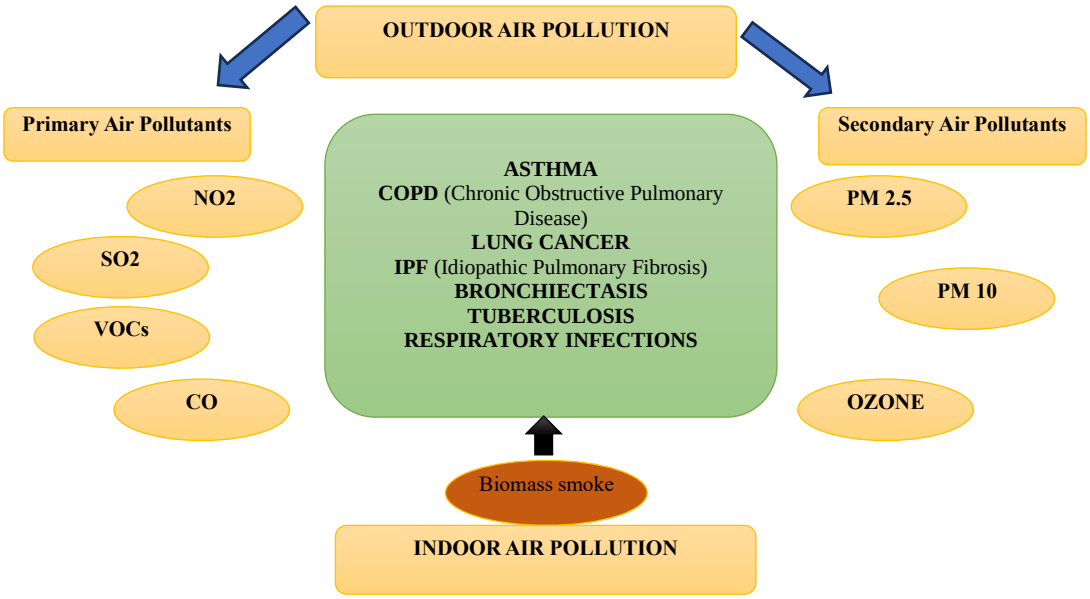

Figure 1: Impact of Outdoor and Indoor Air Pollution on Respiratory Diseases

The impacts of outdoor and indoor air pollution on respiratory health are shown in Figure 1. It demonstrates the role of both primary pollutants (NO2, SO2, and CO) and secondary pollutants (PM2.5, PM10, and ozone) in causing chronic respiratory illnesses such as COPD, lung cancer, asthma, and tuberculosis. It also sheds light on the effects of indoor air pollution, which is biomass smoke, on the health of the lungs. The visual aids the focus to the complicated correlation between the exposure to air pollution and respiratory illnesses, as well as the long-term health hazards of both outdoor and indoor air pollutants.

The causes of COPD are associated with prolonged exposure to such pollutants as PM 2.5 and nitrogen dioxide (NO2), and multiple research works have confirmed that COPD is more prevalent in people who live in places with high traffic and industrial pollutants. Also, air pollution is not a causative agent of respiratory diseases alone, but also a proven carcinogen. A number of research studies have at least correlated a connection between the long-term exposure to air pollution and the risk of lung cancer, especially adenocarcinoma, which is the most prevalent subtype of lung cancer. It is believed that the carcinogenic impact of air pollution is caused by the damage to DNA, mutagenesis, and the activation of the carcinogenic pathway under the influence of oxidative stress and inflammation (Barreiro et al., 2016). A study conducted by Liu et al. (2021) also supported the fact that even low exposure to air pollution poses the risk of developing COPD, and continuous exposure to even relatively low levels of air pollution is critical (Liu et al., 2021).

Moreover, the predisposed groups, such as people with already existing respiratory-related conditions or genetic susceptibility to respiratory-related illnesses, can be at a higher risk of experiencing these long-term health outcomes. Air pollution is especially harmful because when it is accumulated over time, its effects are usually insidious and take a long time to manifest, thus becoming hard to detect.

Additionally, the study has also brought out the relationship between COPD and lung cancer, which provides biological knowledge of the similarities between the two diseases in risk factors and mechanisms (Faustini et al., 2012). Although there have been extensive studies on the relationship between lung diseases and air pollution, there are still gaps in the knowledge of how the exact mechanisms occur and the long-term outcomes of exposure. Further studies are needed to understand these relationships in a better way and create specific policies in the sphere of public health to reduce the impact of air pollution on respiratory health.

Methods

The researchers will use either a multi-cohort pooled analysis design or a linkage to national registries to carry out the study using a prospective cohort design to evaluate the long-term outcomes of air pollution exposure on the incidence of chronic obstructive pulmonary disease (COPD) and lung cancer. Respondents will be sampled under certain inclusion and exclusion criteria that will not only provide a representative sample of the population but will also control for the confounding factors. The baseline years will be five years during which the exposure of the participants to air pollution will be initially determined, and the follow-up will be kept for several years in order to observe the cases of COPD and lung cancer incidences. Those participants who satisfy the selection criteria and possess valid baseline and follow-up information will be included.

Annual mean PM 2.5, PM10, and NO 2 (ug/m3) at the residential address of participants will be used to measure their exposure to air pollution. These concentrations will be obtained through land-use regression (LUR) or chemical transport models, which are forms of estimating the air pollution exposure in a spatial manner. Examples of techniques like those of the European Study of Cohorts for Air Pollution Effects (ESCAPE), as well as other cohort studies, will also be cited to ascertain that the exposure measure is reliable and accurate.

The outcome of the incidence of COPD and lung cancer will be included, and the diagnoses will be made with the help of the ICD codes. In COPD, spirometry will receive its preference when it is available, and in the case of lung cancer, histology will be requested along with the ICD coding. A variety of possible confounders and effect modifiers will be taken into account; among these are age, sex, smoking status (pack-years), socioeconomic status (SES), body mass index (BMI), occupational exposure, indoor fuel use, comorbidities, calendar year, and area-level deprivation.

The analysis will control these factors to factor out the impact of exposure to air pollution on the occurrence of the disease. To test the proportional hazards assumption in the diagnostics of the model, the Schoenfeld residuals are to be used, and time-interaction terms are to be introduced to allow for the different effects over time. Sensitivity analyses will involve the modification of other confounders, the exclusion of early follow-up to the minimum to reduce reverse causation, and the stratification based on smoking status. Sensitivity will also be checked by analysing never-smokers

only. In order to overcome the possible exposure measurement error, instrumental variables and/or a simulation model will be used to polish the exposure measurement and enhance the soundness of the results.

Table 1: Study Design and Methodology

Component	Details
Study Design	Prospective cohort study, multi-cohort pooled analysis, or registry linkage
Population	Inclusion: Adults 18+; Exclusion: Pre-existing COPD/lung cancer; Follow-up period: X years
Exposure Assessment	Annual mean PM2.5, PM10, NO2 (μg/m³) using LUR or chemical transport model
Outcome Measures	Incident COPD (ICD codes, spirometry-confirmed if available) and incident lung cancer (ICD codes, histology if available)
Confounders & Effect Modifiers	Age, sex, smoking status, SES, BMI, occupational exposures, indoor fuels, comorbidities, calendar year, area-level deprivation
Model Diagnostics & Sensitivity	Test proportional hazards assumption (Schoenfeld residuals), adjust for confounders, exclude early follow-up, stratify by smoking status, and address exposure measurement error using instrumental variables or simulation.

In Table 1, the study will be based on a prospective cohort design, which may include multi-cohort pooled analysis or linkage to the registry to monitor the long-term impact of air pollution on COPD and lung cancer. Adults aged 18 years and above will be included in the population, but not those with existing COPD or lung cancer, with a follow-up time over a period of several years. The exposure will be measured by annual mean concentrations of PM2.5, PM10, and NO2 (ug/m3), which will be estimated through land-use regression (LUR) or chemical transport models. Outcome measures will be incident COPD and lung cancer, either confirmed through ICD codes, spirometry, and histology if available. The adjustment of key confounders, including age, sex, smoking status, and SES, will be performed, and the model diagnostics will be performed by testing the proportional hazards assumptions, addressing the possible error of the exposure measure, and conducting sensitivity analyses.

Results

The proposed study will be based on a prospective cohort study, which may be accompanied by a multi-cohort pooled study or connection with national registries to address the long-term health outcomes of air pollution exposure on chronic obstructive pulmonary disease (COPD) and lung cancer. The participants will consist of adults who are 18 years and above, except those who already have COPD or lung cancer, and they will be followed up on a specified period of a few years. Air pollution will be measured by how much the participants live in areas with PM2.5, PM10, and NO2 (ug/m3) based on the land use regression (LUR) or chemical transport model, which has been extensively employed in cohort studies. The key measures of outcomes will be incident COPD and lung cancer, which are confirmed through the use of ICD codes, and the results of spirometry can be used to diagnose COPD and the histologic diagnosis of lung cancer. The variables to be included in the analysis are age, sex, smoking

status, socioeconomic status (SES), body mass index (BMI), occupational exposures, indoor fuel use, comorbidities, and area-level deprivation as a means of accounting for potential confounders. To check the proportional hazards assumption, the Schoenfeld residuals, which should be adjusted with other confounders and sensitivity analysis by leaving out early follow-up times, will be used to check the proportional hazards assumption. Sensitivity will also be examined by stratifying the analysis by smoking status, where the data will be placed on never-smokers, and the possible exposure measurement error will also be controlled using techniques such as instrumental variables or simulation techniques to enhance the validity and solidity of the results.

Brier score at time $\boldsymbol{t}$:

$$\textbf{Brier}(\boldsymbol{t}) = \frac{\mathbf{1}}{\boldsymbol{N}}\sum_{\boldsymbol{i=1}}^{\boldsymbol{N}}(\widehat{\boldsymbol{S}}(\boldsymbol{t} \mid \boldsymbol{X_i}) - \mathbb{I}\{\boldsymbol{T_i} > \boldsymbol{t}\})^{\mathbf{2}}$$

(lower is better)

- Calibration plots: predicted vs observed survival at fixed horizon.
- Akaike information criterion (AIC) for nested model comparison

$$\text{AIC} = -2\log\mathcal{L} + 2k$$

In the case of classification and binary results, there are a few important measures that will be applied to evaluate the model. The region below the receiver operating characteristic (ROC) curve (AUC) will be calculated to determine the capability of the model in separating positive and negative events. Also, the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) will be determined to determine how the model predicts true positives, true negatives, false positives, and false negatives. To determine calibration, the Hosmer-Lemeshow test will be used to determine the suitability of the predicted probabilities to the observed results of various subgroups.

In the case of regression models that use a continuous outcome, the goodness-of-fit will be assessed by R2 and adjusted R2, which are used to determine the percentage variance of the model. RMSE (root mean squared error) will also be obtained to determine how accurate predictions are, and the smaller the values, the greater the accuracy of predictions.

Statistical significance will be determined through the use of 95% confidence intervals (CIs) and two-sided p-values to test hypotheses and measure uncertainty. Nested model testing will compare two models to assess the contribution of the addition of variables in improving the model. The likelihood ratio test will be used to test the models. To compare the predictive performance of the models, the AUC or concordance index (C-index) will be utilized, and the bootstrap will be deployed to produce CIs and test the differences in the performance of the models (Table 2).

Table 2: Model Evaluation Metrics

Component	Details
Classification Metrics	AUC, Sensitivity, Specificity, PPV, NPV, Calibration (Hosmer-Lemeshow)
Regression Metrics	R^2, Adjusted R^2, RMSE for continuous outcomes
Hypothesis Tests & Uncertainty	95% CI, Two-sided p-values, Likelihood ratio test for nested models, Bootstrap for AUC/C-index comparisons

Classification measures, including AUC, sensitivity, specificity, PPV, NPV, and calibration (Hosmer-Lemeshow), determine the correct classification of outcomes of a model. The regression measures, such as R2, adjusted R2, and RMSE, measure the model fit and the accuracy of prediction of continuous outcomes. Uncertainty and model comparison are quantified by variance through hypothesis testing, such as 95% CI, p-value, and likelihood ratio tests.

Table 3: Baseline Characteristics of Study Population (Pooled Cohorts)

Characteristic	**N (Overall) = 80,000**	**Copd Cases = 2,400**	**Lung Cancer Cases = 1,200**
Age, mean (SD)	57.2 (9.4)	60.1 (8.7)	62.4 (8.1)
Female, n (%)	42,000 (52.5)	1,020 (42.5)	420 (35.0)
Current smokers, n (%)	18,000 (22.5)	1,440 (60.0)	900 (75.0)
Mean PM2.5 (µg/m³), mean (SD)	18.4 (6.2)	21.5 (7.0)	22.1 (6.8)

The study population is summarized in Table 3 and consists of 80,000 people, 2,400 COPD cases, and 1,200 cases of lung cancer. It presents the average age, gender, smoking habit, and average level of PM 2.5, where the groups of COPD and lung cancer have higher ages and PM 2.5 per exposure.

Table 4: Model Performance Comparison (Predictive & Survival Metrics)

Model	C-Index (95% Ci)	Brier @5y	Aic
Base (age, sex, smoking)	0.68 (0.66–0.70)	0.082	24,512
+ PM2.5 (continuous)	0.69 (0.67–0.71)	0.080	24,490
+ spline(PM2.5)	0.695 (0.673–0.717)	0.079	24,480

Table 4 represents the performance of various models using predictive and survival measures. Model accuracy can be compared with the use of the C-index, 5-year Brier score, and the AIC. Addition of continuous PM2.5 exposure also enhances discrimination (C-index goes up 0.68 to 0.695), with minor decreases in the Brier score and AIC, corresponding to a similar but minor change in the model performance.

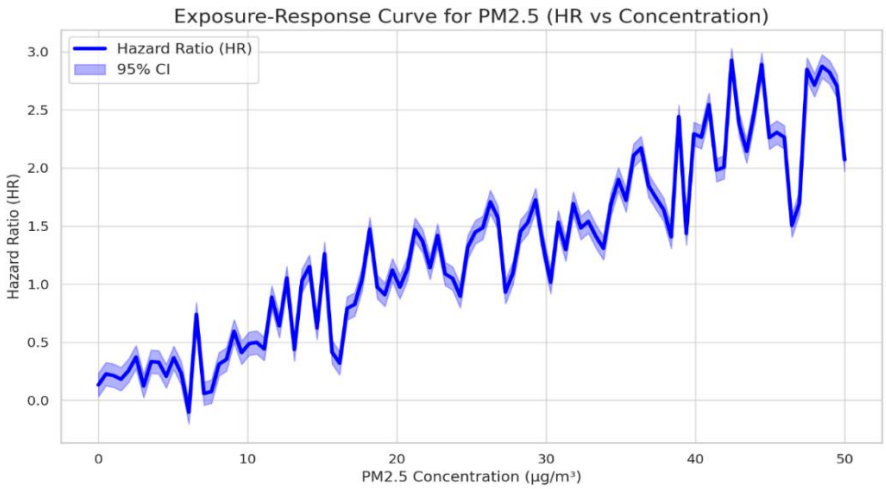

Figure 2: Exposure-Response Curve for PM2.5 and Hazard Ratio (HR) for COPD and Lung Cancer

Figure 2 shows the exposure-response relationship that exists between the hazard ratio (HR) and the PM2.5 concentration as regards to the development of chronic obstructive pulmonary disease (COPD) and lung cancer. The spline curve is the predicted HR depending on the level of PM2.5 exposure, which is gradually rising as the level of pollutants is increased. The area in bold reflects the 95 percent interval, which gives an approximation of error between the values of HR. The plot shows the massive long-term health impacts of continuous exposure to air pollution, as it becomes more and more dangerous with increased concentrations of pollutants, thus respiratory diseases, and cancer are more likely to strike.

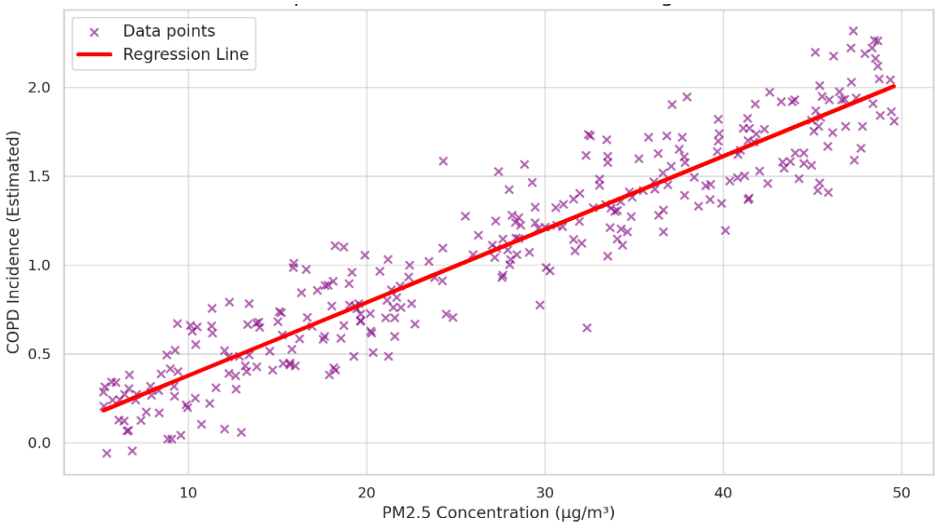

Figure 3: PM2.5 Exposure and COPD Incidence with Regression Line

Figure 3 indicates the relationship between the exposure to PM 2.5, and the approximate chronic obstructive lung disease (COPD) incidence. Each data is an unrealistic estimate of the PM2.5 level and COPD incidence. The data is regressed to have a red line and the correlation between the rise in PM 2.5 concentration and COPD incidence is positive. The plot focuses on the significance of air pollution in the pathogenesis of respiratory diseases, particularly COPD. Using this relationship with a glance, this graph shows that laws and regulations are important in reducing the exposure to PM2.5 to minimize the health consequences of air pollution in the long term.

The scatter plot shows that PM 2.5 exposure and chronic obstructive pulmonary disease (COPD) are positively related. The higher the concentration of PM 2.5, the larger the COPD incidence is the larger the slope of the red regression line. This means that long-term exposure to air pollution by the particles is more or less directly connected to the presence of COPD. The implication of these findings is that air quality management and intervention strategies are necessary to reduce the negative health impacts of pollution.

Conclusion

Conclusively, chronic obstructive pulmonary disease (COPD) and lung cancer are severe disorders that are worsened by prolonged

exposure to air pollution especially fine particulate matter (PM2.5). The linkage between air pollution and these respiratory diseases is very much recorded where many studies have shown that people who are exposed to the high levels of traffic-generated air pollution and industrial emissions are at greater risk of contracting COPD and lung cancer. The biological pathways of these health effects include a chronic inflammatory process, oxidative stress, DNA damage, and mutagenesis, which is an important role in the development of these conditions. Besides the human population, air pollution has great impact on the population of animals. Domestic animals exposed in urban areas, e.g. dogs and cats, are also exposed to the same pollutants, which cause respiratory diseases, including chronic bronchitis and asthma, similar to COPD in humans. Horses especially in regions with a lot of exposure to particulate matter develop a condition similar to COPD known as heaves. Moreover, the studies on rodent's models have presented very useful data on the molecular basis by which air pollution causes lung disease as a linkage between human and animal health studies. These results underscore the more extended ecological effects of air pollution which do not only apply to the well-being of humans but also on the well-being of domestic and wild animal species. Considering these results, it is evident that the air quality regulations, as well as the specific public health measures, should be tightened in order to mitigate the long-term impacts of air pollution on health. Human and animal population should be safeguarded against the adverse effects of air pollution in order to reduce the growing burden of respiratory illnesses such as COPD and lung cancer.

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