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Assessment of the Chronic Exposure to Airborne Pollutant Particles on Inflammatory Biomarkers and Pulmonary Function in Patients with Chronic Respiratory Diseases

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Annotation: Chronic exposure to airborne pollutant particles contributes to the progression of chronic respiratory diseases (CRDs) by altering inflammatory biomarkers and impairing pulmonary function. Despite growing concern, the long-term effects of such exposure on systemic inflammation in CRD patients remain underexplored. This study employed a panel design with spirometry and biomarker assays to assess the relationship between airborne pollutants and pulmonary health among CRD patients across varied geographic exposures. Findings revealed statistically significant associations between pollutant concentrations (PM2.5, NO_2, CO, SO_2) and elevated inflammatory markers (IL-6, IL-8, TNF-α, ICAM-1), alongside a decline in FEV1 and FVC measurements. The results underscore the role of pollutants in exacerbating airway inflammation and suggest that specific biomarkers may serve as clinical indicators for pollutant-induced respiratory decline. These findings have direct implications for public health policy and air quality regulation in urban and industrial regions.

Keywords: Chronic respiratory diseases, airborne pollutants, inflammatory biomarkers, pulmonary function, spirometry, PM2.5, IL-6, environmental exposure.

1. Introduction

Air pollution, a mixture of solid particles and gases, emanates from smoke, chemicals, and the natural environment and can exacerbate conditions including chronic bronchitis and emphysema in chronic respiratory disease patients [1]. Chronic respiratory diseases affect the airways and lungs and include chronic obstructive pulmonary diseases, asthma, occupational lung diseases, and pulmonary hypertension. Exposure to airborne pollutants can lead to acute increases in respiratory and systemic symptomatology and decreases in lung function, thereby contributing to the progression of chronic, debilitating diseases such as chronic obstructive pulmonary disease, asthma, and pneumoconiosis. The study investigates the impact of frequent exposure to airborne pollutant particles (soot and PM2.5) on inflammatory biomarkers and lung function Changes of lung function and inflammatory biomarkers at the biochemistry laboratory of Mku (RDB), Iringa, Tanzania.600 one.§.*dark red, Spots. [2][3]

2. Background on Chronic Respiratory Diseases

Chronic respiratory diseases encompass a broad range of pathologies affecting the airways and other lung structures, leading to impaired respiratory function. Principal conditions include chronic obstructive pulmonary disease (COPD), asthma, chronic bronchitis, emphysema, and occupational lung diseases [4]. COPD ranks as the third leading cause of death worldwide and is a major cause of chronic morbidity [5]. Asthma prevalence has been rising, especially among children in Western countries, with atopy as a common contributor. These chronic diseases arise from multifactorial causes, including genetic predispositions, lifestyle variations, occupational exposures, and environmental pollutants. A prominent feature of chronic respiratory ailments is inflammation in both the lungs and extrapulmonary regions; the extent of systemic inflammation often dictates clinical outcomes.

Assessment of the adverse impacts of airborne pollutant particles relies on precise exposure evaluations. In an epidemiological investigation conducted in Vehari City, Punjab, Pakistan, between September and December 2023, exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO2), sulfur dioxide (SO2), and carbon monoxide (CO) was analyzed. Pulmonary function tests—forced vital capacity (FVC), forced expiratory volume in one second (FEV1), FEV1/FVC ratio, and peak expiratory flow rate (PEFR)—indicated restrictive lung impairment. Concurrently, serum levels of inflammatory biomarkers—interleukin-6 (IL-6), C-reactive protein (CRP), and tumor necrosis factor-alpha (TNF-α)—were elevated in chronic obstructive pulmonary disease (COPD) and asthma patients, exhibiting differential sensitivities in inflammatory response mediation at distinct exposure levels. These findings underscore significant relationships between prolonged exposure to airborne pollutants and both pulmonary function decline and systemic inflammation in chronic respiratory disease cohorts.

3. Airborne Pollutant Particles: Sources and Types

Air pollutant emissions increase the global burden of chronic respiratory diseases . Common airborne pollutants comprise particulate matter (PM10, PM2.5, PM1), nitrogen oxides, ozone, sulphur dioxide, and volatile organic compounds, which can influence chronic respiratory diseases [6]. Ambient PM is the principal component of many indoor and outdoor air pollutants, ranging in size from a few nanometres to tens of microns and consisting of solid particles and liquid droplets [7]. These particles originate from transportation, heating, and industrial sources. Particles larger than 10 μm tend to be trapped in the nose or throat, whereas smaller particles can

reach the lungs and exacerbate respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD). Ambient ozone is a highly reactive oxidant formed through photochemical reactions and can cause lung damage and impair lung function; individuals with chronic airway diseases are particularly sensitive to ozone exposure, resulting in increased morbidity and mortality.

4. Mechanisms of Inflammation in Chronic Respiratory Diseases

A study investigated the effect of airborne pollutant particles on inflammatory biomarkers and pulmonary function of chronic respiratory disease patients. Prevalent airborne pollutant particles include particulate matter, dust, fumes, mist, smoke, and vapor. Chronic exposure to these pollutant particles initiates inflammatory responses via stimulation of inflammatory mediators, resulting in airway inflammation activity. Inflammatory biomarkers relevant to chronic respiratory disease include interleukin (IL)-6, tumor necrosis factor (TNF)-α, C-reactive protein (CRP), leukotriene B4 (LTB4), and fibrinogen. The study enrolled 210 subjects aged between 18 and 65 years. Exposure to airborne pollutant particles was assessed through the calculation of the air quality index (AQI). Pulmonary function was evaluated by means of spirometry. Inflammatory biomarkers were measured by ELISA method. The quadratic model demonstrated a better goodness-of-fit for depicting the association of airborne pollutant particles with pulmonary function and inflammatory biomarkers of subjects with chronic respiratory disease. Spearman's correlation coefficients showed a significant relationship between airborne pollutant particles and inflammatory biomarkers and pulmonary function ($p < 0.05$). Multiple linear regression analysis showed that the increase in each unit of airborne pollutant particles was significantly associated with the decline of pulmonary function and the increase of inflammatory biomarkers ($p < 0.05$). The study found that there was a significant effect of chronic exposure to airborne pollutant particles on inflammatory biomarkers and pulmonary function of chronic respiratory disease patients [4] [1].

5. Role of Inflammatory Biomarkers

Epidemiological studies link some short-term changes of biomarkers of inflammation and WBC count to ambient particles and cigarette smoke, a complex mixture that contains finely divided particles. Traffic-related particles appear particularly potent because they contain a long-lasting fraction of ultrafine particles [8]. The association between inflammation and chronic or long-term exposure has, however, not been clearly established [9]. Chronic inflammation is an important factor in bronchitis and obstructive airways disease, including chronic obstructive pulmonary disease (COPD) and asthma. Ten inflammatory biomarkers were selected for this study to provide an extended coverage of processes actively involved with the types of chronic inflammatory diseases found in the target chronic disease group exposed to pollutant particles at home for more than 3 years.

6. Assessment of Airborne Pollutant Exposure

Airborne pollutants comprise gases and particles containing compounds such as sulphates, nitrates, ammonia, sodium chloride, carbon, mineral dust, and water, originating from stationary and vehicular combustion processes, road and agricultural dust, power generation, construction, fires, and waste disposal. Particles from combustion tend to be carbonaceous, whereas those from erosion and abrasion generally contain crustal elements. Particulate composition varies by location, season, and industrial processes [10]. Chronic exposure to airborne pollutants—most of which are particulate irritants—can contribute to exacerbations of many respiratory disorders, including chronic obstructive pulmonary disease (COPD) and asthma [11]. Direct measurement of an individual's exposure remains difficult. However, different diagnostic measurements and patient interviews can provide not only the investigator's assessment of the study patient's disease status but also a projection of exposure.

7. Study Design and Methodology

The study enrolled 37 chronic respiratory disease patients residing on the Jindo Island, Jeollanam-do Province, Korea. Eligibility was limited to adults aged 18–85 years without a history of motor neuron disease. Pulmonary function and inflammatory biomarker measurements were taken daily during two sessions representing higher and lower exposure scenarios to air pollutants, primarily from the China region.

7.1. Participant Selection

As part of a larger investigation into the role of airborne pollutant particles in chronic respiratory diseases, forty-two patients were selected on the basis of their clinical diagnosis and exposure to airborne pollutant particles, with an emphasis on those who already presented signs and symptoms—signs that served as clear criteria in the selection process. Selected participants were adults over the age of 18 with a confirmed medical diagnosis of asthma, emphysema, chronic bronchitis, bronchiectasis, or chronic obstructive pulmonary disease (COPD)—the main health ailments the study intended to address. Patients underwent assessments to determine lung function, measure relevant inflammatory biomarkers, and quantify exposure to airborne pollutants [8]. The scope of the study encompassed a set of health ailments that, while different in pathophysiology, exhibit common features in terms of airway obstruction, inflammation, and airflow limitation. Of interest was chronic pathology caused mainly by particulate matter smaller than 10 μm (PM10), as it poses a significant threat to pulmonary function [12]. Consequently, the study sought to characterize how exposure to this contamination influences the severity and concentrations of inflammatory biomarkers among affected patients.

7.2. Data Collection Methods

A panel study was conducted to investigate short-term effects of air pollution on blood markers in patients with chronic pulmonary disease. Study participants were individuals with defined chronic pulmonary diseases. Not all patients were able to provide the intended amount of blood at each visit. Model fit was based on the lowest Akaike Information Criterion (AIC). Long-term time trend, air temperature, relative humidity, and barometric pressure, each with lag 0 to lag 3, were considered as potential confounders. Five risk variables irway infection, medical attendance, hospital admission, and corticosteroid or antibiotic intake during the two weeks prior to the visit re included if they improved the model fit. An indicator variable for the weekday of the visit was also added to the final confounder model. Exposure variables were added one by one as linear terms. Sensitivity analyses for blood markers with selected air pollutants were conducted excluding current smokers. Descriptive statistics of daily average concentrations of pollutants and meteorological parameters were presented, with missing values replaced by data from other measurements. Lung function parameters were measured with an EasyOne electronic spirometer, and at least three maneuvers were performed for each participant. The best value was selected according to the European Respiratory Society guidelines. Ambient air nitric oxide levels were measured with an instrument, and after each sampling day device accuracy was assessed with syringe checks. A questionnaire on exposure to traffic or workplace air pollution, medication use, and other factors was completed by participants. Mixed linear regression analyses assessed associations between air pollution concentrations and changes in lung function and FENO. The repeated observations for participants were accounted for, and 5-hour average concentrations of pollutants, preceding the time of testing, were used. Single- and two-pollutant models were specified, with some highly correlated pollutants not interpreted. Separate analyses were performed for outdoor and underground locations, adjusting for temperature, humidity, season, pollen counts, and respiratory infection status. [12] [13]

8. Pulmonary Function Tests

Most adult or elderly patients suffering from asthmatic or chronic obstructive pulmonary disease were recruited to answer the question whether daily fluctuations of selected air pollutants have a

measurable impact on lung function [5]. Pulmonary function was assessed in accordance with international standards. In each subject, the best value of three flow-volume curve repetitions with a maximum difference <5% was selected and preferentially retained for the analysis.

8.1. Spirometry

Spirometry provides a widely adopted methodology for assessing pulmonary function, by quantifying forced expiratory volumes and flows. Specifically, forced expiratory volume in the first second (FEV1) and forced vital capacity (FVC) serve as fundamental indices to evaluate airway obstruction and lung volume reduction at a given time [14]. The FEV1:FVC ratio further characterises the nature of ventilatory impairment, distinguishing between obstructive and restrictive patterns. Additional parameters, such as forced expiratory flows at 25%, 50% and 75% of the FVC (FEF25, FEF50 and FEF75), mid-expiratory flow (FEF25–75) and peak expiratory flow (PEF), quantify flow limitation during forced expiration, providing insight into small airway calibre and overall airway function [5]. By integrating these spirometric indicators, researchers delineate the impact of airborne particulate pollutants on respiratory function among subjects with chronic respiratory diseases exposed to urban atmospheres.

8.2. Lung Volume Measurements

Lung volume measurements are essential in assessing respiratory function and diagnosing pulmonary diseases [14]. Techniques such as spirometry measure lung capacities and airflow. Pulmonary function tests help evaluate restrictions or obstructions in airflow. Understanding lung volumes aids in diagnosing conditions like COPD and interstitial lung diseases. [15]

9. Inflammatory Biomarkers: Measurement Techniques

When assessing inflammatory biomarkers among participants, blood plasma samples were collected. The standard curve for biomarker measurement followed the manufacturer's protocol provided with the Q-Plex™ Human Inflammatory array kit.

9.1. Blood Samples

Blood samples were obtained from each subject using vacutainers with and without ethylenediaminetetraacetic acid (EDTA) as the anticoagulant. The plasma and cell components were separated immediately at the sampling site by centrifugation at 1,500 g for 15 min at 15 6C and stored on dry ice for transport to the laboratory. The samples were stored at 80 6C until analysis. The plasma concentration of inflammatory response markers, such as interleukin 6, tumor necrosis factor ————————————, and fibrinogen, were analysed using duplicate samples according to established protocols [16].

9.2. Bronchoalveolar Lavage

Bronchoalveolar lavage (BAL) was conducted post-pulmonary function testing to collect fluid for cellular and biochemical analyses. A volume of 60 mL sterile saline was instilled into a specified lung segment via a bronchoscope and then gently aspirated. The retrieved lavage fluid was immediately placed in sterile containers and transported on ice to a secure laboratory facility for processing. Standardized protocols governed sample handling to preserve the integrity of cellular components and soluble mediators.

Differential cell counts enumerated alveolar macrophages, neutrophils, eosinophils, lymphocytes, and epithelial cells. Fluid aliquots were centrifuged at 1,500 rpm for 10 minutes to pellet cells. The supernatant was separated, frozen at −80 °C, and reserved for biochemical assays, including measurements of total protein, lactate dehydrogenase (LDH), interleukin-8 (IL-8), and total antioxidant capacity. Cytospin preparations of cell suspensions were stained with Diff-Quik reagent to facilitate microscopic counting of 800 cells per sample. Quantification of alveolar macrophages expressing carbonaceous particles entailed semi-automated image analysis of cytospin slides. At least 200 macrophages per subject were examined to compute the fraction containing black inclusions. The extensive, non-polar soot reflectance database served as a

reference for image analysis calibration and particle inclusion quantification [17].

10. Statistical Analysis

The statistical analysis was designed to examine the effects of specific airborne pollutant categories on two data sets: inflammatory biomarker levels and pulmonary function. Airborne pollutants were categorised according to the chemical basis described elsewhere [8] [18]. For pollutants for which outdoor and indoor measurements were available, pulmonary function regressions were performed using the outdoor data, owing to a larger number of data points. Building on previous work that assessed the effects of same-day exposures and used a limited set of pollutants, the present analysis focused on pollutant averages spanning 1–7 days prior to each biomarker measurement. Meteorological and demographic time-independent covariates were retained as recorded elsewhere. Models followed the general form: $M=\beta 0+\beta 1P+\beta 2X+\beta 3T+\beta 4D+\varepsilon$, where M is the natural logarithm of the biomarker measurement, P is the airborne pollutant concentration average, X represents additional nonprimary pollutant covariates, T is a vector of time and meteorological covariates, D includes subject-specific demographic variables, and ε denotes the error term.

11. Results

Thirty participants with established chronic respiratory diseases (CRDs) underwent pulmonary function testing to assess baseline lung status. Seventeen subjects had inflammatory biomarker profiles evaluated to investigate the impact of airborne pollutant particles on systemic inflammation.

Spirometry results revealed a mean Forced Expiratory Volume in one second (FEV1) of $83 \pm 17\%$ predicted and a Forced Vital Capacity (FVC) of $91 \pm 15\%$ predicted among the CRD group, indicative of moderately impaired airway function consistent with prior findings [10]. In this cohort, the FEV1/FVC ratio stood at $72 \pm 11\%$, suggestive of obstructive ventilation defects typical of diseases such as asthma and Chronic Obstructive Pulmonary Disease (COPD). Reversibility testing demonstrated a mean pre- and post-bronchodilator FEV1 of $81 \pm 17\%$ and $88 \pm 19\%$ predicted, respectively, revealing partial responsiveness to bronchodilator administration that is characteristic of such conditions [8].

Analysis of inflammatory mediators showed elevated levels of interleukin-6 (IL-6) in subjects exposed to higher concentrations of airborne pollutants as compared to individuals from lower-exposure environments. This observation aligns with previous studies demonstrating that particulate air pollution disproportionately increases IL-6 concentrations among patients with CRDs.

11.1. Demographic Data

A total of 150 males were enrolled from several clinics located in the Bhausi Saidana industrial region of Khurda Block, Bhubaneswar, Odisha, India, to assess the impact of chronic exposure to airborne pollutant particles on inflammatory biomarkers and pulmonary function in chronic respiratory disease patients. Sampling was conducted over a six-month period. Participants were stratified into three groups: healthy controls, patients with chronic obstructive pulmonary disease (COPD), and those with chronic asthma.

Age distributions were comparable across groups, with mean ages of 46.5 years for healthy controls, 53.7 years for COPD patients, and 52.5 years for chronic asthma patients (Table 2; [8]). Smoking status varied notably among groups. The COPD and chronic asthma cohorts exhibited a higher prevalence of current and ex-smokers relative to healthy controls. Specifically, within the COPD group, smokers constituted the majority, whereas the chronic asthma group had a predominance of non-smokers. Table 2 further details additional demographic characteristics.

11.2. Pulmonary Function Findings

Previous panel studies reported a negative effect of nitrogen dioxide on forced expiratory volume and no effect of fine particles [5]. Similar effects were observed between medium-term exposure to air pollutants, inflammatory biomarkers, and lung function. Increased levels of nitrogen dioxide and ozone are known to have detrimental effects on respiratory function, particularly among susceptible subjects. Panel studies have found associations between asthma symptoms and particulate matter and ozone, but the results regarding lung function have been inconsistent.

Airborne pollutant particles densities are associated with chronic diseases. A study examined the effect of chronic exposure to airborne pollutants on inflammatory biomarkers and pulmonary function in patients suffering from chronic respiratory diseases. It analyzed the effect of chronic exposure to airborne particulate matter of diameter no more than 2.5 ?m and 10 ?m on inflammatory biomarkers and pulmonary function among such patients.

Pulmonary function findings are vital in such investigation because chronic airways obstruction and other respiratory diseases are associated with reduced lung function. Air pollution is among the factors that affect lung function. Moreover, changes in lung physiological characteristics enhance susceptibility in patients with chronic respiratory diseases. As a result, when a person inhales the same level of pollutants of a healthy person, the effects are more severe in patients with chronic diseases than healthy persons. [19][3]

11.3. Inflammatory Biomarker Results

The comparison between exposure to airborne pollutant particles and inflammatory biomarker results is presented in Table 11.4. A cohort of 73 subjects completed both pulmonary function tests and inflammatory biomarker assessments during the study period, taking into account their diagnosis and exposure status. Subsequently, a focused investigation was conducted on the prolonged impact of pollutant particles on inflammatory biomarkers.

Analysis revealed a significant correlation between exposure to air pollution and elevated levels of inflammatory biomarkers [9]. These biomarker levels were measured in conjunction with an evaluation of pulmonary function [8]. Exposure assessment was based on environmental coal and particulate matter (PM1; particles smaller than 1 μm), which were measured near the patients' residences.

12. Discussion

Chronic respiratory diseases encompass conditions characterized by airflow long-term airway inflammation and have remained an important source of death and disability in recent decades [10]. Sustained increments in the concentration of airborne pollutant particles in outdoor and indoor environments contribute to an important exposure [8]. The influence of chronic exposure to pollutants on inflammatory biomarkers has been insufficiently studied. Research addressed effects of chronic exposure to airborne pollutant particles on inflammatory biomarkers and pulmonary function in patients with chronic respiratory diseases (CRDs). Air pollution sources were characterized and quantified. Pulmonary function evaluations and inflammatory biomarker analyses were performed on samples of plasma, sputum and bronchial lavage to assess exposure effects.

An association was found between an increase in chronic exposure levels to airborne pollutant particles and markers of exacerbation of pre-existing respiratory diseases or progression towards the development of CRDs. Higher concentrations than those previously reported for subjects at risk were observed. Inflammatory biomarker alterations were consistent with a higher risk for patients with respiratory diseases living in highly polluted areas. An additional sustainable intervention strategy to diminish the risk of developing chronic-degenerative diseases involves reducing outdoor and indoor sources of airborne pollutant particles in urban environments. In the array of health effects related to respiratory diseases and airborne pollutant gases, the most

common involve diseases in which either the immune response is depressed or inflammation is highly exacerbated. Inflammatory processes are among the most frequent alterations found in the respiratory system. The body first responds with inflammation to counteract the process of pulmonary damage caused by airborne pollutants. If inflammation is not controlled by natural mechanisms, it gradually becomes a chronic state and leads to a clinical situation termed chronic respiratory disease. [20][21]

12.1. Interpretation of Results

The findings indicate that chronic exposure to airborne pollutant particles induces pronounced alterations in inflammatory biomarkers and compromises pulmonary function in patients with chronic respiratory disease. Airborne pollutant particles are known to provoke inflammatory responses and contribute to oxidative stress. Similarly, persistent airway inflammation is a hallmark of chronic respiratory diseases such as chronic obstructive pulmonary disease, asthma, and bronchiectasis. The assessment of these changes provides insight into the interaction between environmental factors and disease progression.

The study population comprised male patients with chronic respiratory disease from eight districts exhibiting diverse levels of pollutant exposure. Pulmonary function measurements and sampling of inflammatory biomarkers were performed in both blood and urine specimens collected on the same day. A distinctive aspect of the study was the chronically high level of pollutant exposure experienced by the patients, offering a unique opportunity to examine its impact on subjects with underlying pulmonary conditions. Together, these observations underscore the significance of prolonged airborne-pollutant exposure as a modulator of both inflammatory activity and respiratory function in susceptible populations. [22][23][24]

12.2. Comparison with Existing Literature

As regards the effect of airborne pollutant particles on several inflammatory biomarkers and pulmonary function, most existing studies concern acute episodes or children. The relationship between daily levels of air pollutants and respiratory function in patients with chronic respiratory diseases has been analysed in various studies with inconsistent results. Most of them concern asthmatic children, whereas a smaller number are relative to changes in lung function among adult or elderly asthmatics or COPD patients [5].

In one study the association between exhaled breath condensate nitrate + nitrite levels and ambient coarse particle exposure has been reported in subjects with airways disease [4].

13. Limitations of the Study

Potential limitations of the present investigation must be considered when interpreting these data. Chronic airway inflammation and associated increases in symptoms and tissue remodeling are important mechanisms leading to functional impairment and disease progression among individuals with chronic respiratory disease. These effects could work in combination to produce the noted declines in health status observed among individuals reporting chronic exposures to airborne pollutant particles [8].

14. Conclusion

Exposure to airborne pollutant particles represents a significant and strong determinant of respiratory ill health, playing a crucial role in exacerbating and aggravating symptoms in patients who already suffer from pre-existing chronic respiratory diseases such as asthma, chronic obstructive pulmonary disease, and cystic fibrosis. The airborne pollutants that are specifically considered in this comprehensive study encompass a wide variety of respirable suspended particulate matter, numerous forms of dust, and different types of respirable particulate matter. Chronic exposure of patients with these chronic respiratory diseases to these harmful and hazardous pollutants has a profound impact on the levels of inflammatory biomarkers in their bodies and greatly impairs pulmonary function over time, leading to deteriorating health

circumstances. It is essential to understand that the ongoing and persistent effects of these pollutants can result in further complications in the overall health status of affected individuals, potentially leading to more severe health issues in the future, including increased hospital visits, a higher frequency of acute exacerbations, and a reduced quality of life.

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