Effective Contribution of Air Pollutants to Physiological and Psychological Human Diseases: A Systematic Review

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ABSTRACT

Increasing globalization, industrialization, population, and burning of fossil fuels have been adversely affecting the environment for a long time. The consequences of the effects can be seen even within a short period of time in the current scenario. The air pollutants such as SO_2, NO_2, CO, and PM are the main contributors to the adverse health effects. Long-term and short-term exposure to pollutants may cause acute and chronic effects on the human body as they can enter deep into the organ and circulate in the bloodstream. The ultimate purpose of this review is to develop a quantitative perceptive of the existing state of facts about potential health effects concerning the dose-response relationship between exposure level of air pollutants and induced diseases. We have drawn around 376 scientific research papers on high-impact factors related to air pollution and health. These publications were analyzed with consideration of experimental methods, design, observations, and reports on the exposure through inhalation which may emulate the normal direction of exposure inside the human organs. The present study suggests the effects of epidemiological studies on associations between pollutant concentrations and human health. Most of the inferences evidenced the severe adverse effects of particulate matter ($PM_{2.5}$ & PM_{10}) on the respiratory and cardiovascular systems. Our present investigation reveals the health risk due to pollutants' exposure to the vulnerable population anguishing with asthma, COPD, cardiovascular disease, diabetes, cancer (physiological diseases); dementia, depression, and stress (psychological diseases).

INTRODUCTION

Air pollutants are one of the major components of environmental pollution. The primary air pollutants released from the source such as biomass burning, vehicles, thermal power plants, etc. (Awadi 2018). NOx and VOCs react in the presence of sunlight and form highly reactive secondary pollutants. The reactions generate particulate and gaseous air pollutants and affect the health of children, the elderly, and vulnerable people adversely (Morakinyo et al. 2017). Source emission is increasing with increasing demands due to the rising population (Upadhyay et al. 2020). Increasing industrialization, urbanization, and vehicles emit injurious gases which form outdoor air pollution (Guarnieri & Balmes 2015). While biomass and fossil fuel burning generate indoor air pollution (Annesi-Maesano et al. 2013), may penetrate the respiratory tract and produce allergies. Indoor air pollution is more harmful as it covers less area and becomes more concentrated with the pollutants (Manisalidis et al. 2020). Weather conditions and geographical locations also affect the severity

and dispersal of the pollutants (Khamutian et al. 2015). Recently, pollution, unlike London smog, was noticed in Delhi, which required emergency measures thus all smog emitters were temporarily shut down to control the emissions (Singh 2016). The air quality was noticed with heterogeneous characteristics due to variation in correlations between different criteria pollutants in Delhi. This variation might have been established due to significant PM_{10} emissions from construction sites, thermal power plants, industries, and vehicles mainly during the summer season (Biswas et al. 2011, Barik et al. 2021). While a distinct seasonality was recorded in winter by observing the PM_{10} concentration level of $200\mu g/m^3$ in the residential area of Lucknow which is more than the prescribed level (60 $\mu g.m^{-3}$) of the central pollution control board. On the other hand, the concentrations of SO_2 and NO_2 were measured as 8 $\mu g.m^{-3}$ and 30 $\mu g.m^{-3}$ respectively which are in the range of permissible limits (Upadhyay et al. 2017). Cifuentes et al. observed respiratory issues in the patients due to the endotoxins formed through exposure to PM (65.5 $\mu g.m^{-3}$ $PM_{2.5}$ & 153 $\mu g.m^{-3}$ PM_{10})

(Cifuentes 2019). While particulate matter combined with CO_2 (900-1500 μg.m^{-3}) caused repeated respiratory issues enhanced by improper ventilation. Poor ventilation rapidly spreads the endotoxins due to limited area (Padhi et al. 2017) which deposits in the alveolar region of the lungs. The data showed that deteriorated air quality has become a severe issue that may cause non-communicable diseases that increase the risk to the health of 75% of the population (Schraufnagel et al. 2019). CO emission has greatly toxic effects on the sense organs which may cause numerous neuro-disorders. CO mixes with hemo-globin after entering the organism and may decrease the oxygen-carrying capacity across the system. People who had smoking habits were found as deficient in COHb (< 5-10%) levels (Schimmel 2018). The patients exposed to high levels of NO_2 concentration (30-90 μg.m^{-3}) for even short-term had severe lung damage, shortness of breath, and chest pains (Karakatsani et al. 2010). The long-term exposure to these patients may cause mortality due to asthma by 3.1% (Weinmayr et al. 2010) CVD by 69%, pulmonary diseases by 28% whilst the short-term exposure of a combination of PM_{10}, $PM_{2.5}$ & NO_2 (56.3 μg.m^{-3}, 29 μg.m^{-3}, 41.8 μg.m^{-3}) increased the respiratory hospital admissions at all age groups (Çapraz et al. 2017). Short-term combined exposure to PM, O_3, and high temperature caused diabetes mellitus, diagnosed by examining blood pressure (Hoffmann et al. 2012). High levels of $PM_{2.5}$ (10-33 μg.m^{-3}) exposure may cause an increase in blood pressure and myocardial oxygen demand. PM exposure induced more mortality due to respiratory and diabetes illness in 65-year-old persons (0.64%) than in the younger population (0.34%) (Zanobetti et al. 2014).

An air quality monitoring program covering 11 fitness centers in Lisbon reported high temperature, humidity, and pollutants concentrations. The visitors to fitness centers were diagnosed with respiratory tract infections (Ramos et al.2014). High toxicity is attributed to acute infections in the lower respiratory tract (bronchi, bronchioles, and alveoli) and upper respiratory tract (nasopharynx, oro-pharynx, and hypopharynx) (Lelieveld et al. 2015). High concentration of SO_2 (65.7 μg.m^{-3}) exposure increases mortality due to the effect on FEV and FVC (He et al. 2010). Lower FEV1 (54mL) indicated respiratory illnesses in all aged groups of people. Exposure to 3.9-4.13 μg.m^{-3} level of SO_2 caused allergy and acute respiratory inflammation in the young population (Alwahaibi & Zeka 2016). The present study is conducted to understand and infer the impact of pollutant concentrations on physiological as well as psychological disorders by analyzing their methodological approach, and diagnostic pattern, and reporting the results.

MATERIALS AND METHODS

Measurement Techniques for Air Pollutants

The foremost objective of the present study is to re-evaluate air pollutants' impact on human health considering indoor and outdoor exposure for short-term and long-term duration causing acute and chronic illnesses. To achieve this aim, we have used the published data obtained through a wide range of pollutants samples measured by various techniques and health impact through diagnosis. The degree of pollution was measured by using modeling data and systemically measured data to determine the quantity and quality of the pollutants. The data for pollutants and health effects was compiled from 1960 to 2020, obtained from the research methods and results pertaining to the assessment of 24 hour-counts with respect to hours/minutes by different monitoring and diagnostic approaches. Then the averaged data was analyzed critically to find out the correlations of individual pollutants with physiological and psychological diseases. This section of this paper includes the measuring methods for criteria air pollutants ($PM_{2.5}$, PM_{10}, CO, NO_2, SO_2) and their health impacts categorized as physiological diseases (chronic obstructive pulmonary disease, asthma, cardiovascular disease, diabetes, cancer) and psychological diseases (dementia, depression, and stress).

Particulate Matter (PM_{10} & $PM_{2.5}$)

The particulate matter was first measured in 1964 by using simple tools of statistics. Afterward, the methodological technique became advanced and stimulated good quality estimations (Halonen et al. 2011). About 70% of $PM_{2.5}$ and PM_{10} concentrations from vehicular sources can be estimated from Chemical Mass Balance (CMB) model with seasonal variations in the city of Mangalore (Kalaiarasan 2018). The concentration level for 24-hour PM_{10} was measured through poison regression analysis and gravi-metric filter-based methods along with weather parameters (Achilleos 2016). The evaluation of concentration levels of particulate matter through regression analysis against hospital admissions revealed that respiratory illness was the reason for increased cases. The researchers also used another technique i.e. Lung Dose Evaluation Programme (LUPED) model to measure the concentrations of PM (PM_{10}, $PM_{2.5}$, and PM_1). The LUPED model measures the endotoxins deposition inside the cellular regions of the respiratory tract and the rate of breathing (Padhi et al. 2017). PM is also measured by low pressure three cascade Dektai impactor (www.dekati.com).

Carbon Monoxide (CO)

The CO concentration level was observed first time in neu-

ropathy analytical studies through CT and MRI during the 1970s. Presently, CO estimation has been developed and the toxicity can be diagnosed through electrocardiography by using COx multivariate proportional hazards such as COHb levels, age, exposure duration, smoking habits, etc. to examine the acute myocardial Infarction (AMI) (Kaya et al. 2016). The CO effects were examined through a simple questionnaire, CH^2OPD^2 (community, home, hobbies, occupation, personal habits, diets & drugs) to get the historical exposure data (Abelsohn et al. 2010). The capillary gas chromatography, mass spectroscopy along with HP mole sieve plot column was used to analyze the blood sample of headspace and correlated between CO exposure and COHb levels through regression models. The results revealed an elevated level of COHb concentration with the rise in CO level (Rudra et al. 2010). The CO toxicity level in blood samples of two groups of 30-40 year aged people was measured through the Mann-Whitney U test and spectrophotometer (Nair et al. 2017).

Nitrogen Dioxide (NO₂)

Earlier the health effects due to NO_2 exposure have been diagnosed in tissue through various techniques such as culture cells and regression models (Gezerman & Çorbacıoğlu 2018). Recent studies on the association between air pollutants and chronic airway disease and cardiopulmonary diseases (asthma, idiopathic pulmonary fibrosis, etc.) have established diagnostic development (Conti et al. 2018). The effect of NO_2 was diagnosed with a decrease in lung functioning parameters such as FVC and FEV1 in adults (Cole-Hunter & de Nazelle 2018).

Sulfur Dioxide (SO₂)

The simple questionnaire survey was used to examine breathing patterns of the responsiveness of SO_2 (Vahlsing & Smith 2012), the survey resulted in the need for some diagnostics in the tissues. Epithelial tissue injury due to SO_2 exposure was found through the biological marker in compound mucous cilia (Koenig 2016). The Elisa-based method was used to analyze the impact of SO_2 on health by observing inflammatory markers, fibrinogen, oxidative stress, and coagulation factors (Chuang et al. 2007). The correlation between SO_2 concentration (10 $\mu g.m^{-3}$) and Fev1 (54ml) revealed lung functioning at a lower rate. Rajeswari reported pulmonary function test, thyroid profile, and absolute eosinophil count among male petrol pump workers to determine SO_2 toxicity (Rajeswari 2019). While Russell's analysis with regression modeling was used to find out the linkage between emissions of NO_2, SO_2, PM, and meteorological factors (Russell 2018).

Classification of Diseases

Air pollutants are one of the most crucial contributors of the environment to the burden of disease. It is an important health risk factor for major non-communicable diseases. The efficiency of the pollutant concentration may be modified due to increasing population and industrialization which may affect even in a short period of time. Research reveals the linkages between several serious diseases among various age groups and air pollution. Based on the efficiency of the diseases, we have classified them into two categories namely physiological diseases and psychological diseases. The health effects due to individual air pollutants are discussed here under these two categories.

Association of the Pollutants with Physiological Diseases

Diseases of the respiratory system are often evident by one or more symptoms that can be easily acknowledged. The presence of a meticulous symptom or a set of symptoms facilitates identifying an underlying disease in the respiratory system. We have reviewed several epidemiological studies in which the prevalence of respiratory symptoms has been assessed to obtain an insight into the occurrence of physiological diseases due to pollutants are CVD, COPD, asthma, diabetes, and cancer.

PM₁₀/PM₂.₅ and Associated Physiological Diseases

The PM exposure caused pulmonary inflammation and activates systemic hypercoagulability which increases the heart rate. Rising heart rate may become the sequel reason of myocardial infarction, heart attack, and mortality due to CVD (Lin et al. 2017, Chang et al. 2014). The women in the postmenopausal phase had suffered from obesity and found 76% increased mortality risk due to CVDs, they have regularly been exposed to10 $\mu g.m^{-3}$ PM exposure (Miller et al. 2012). The associations of PM_{10} (23 $\mu g.m^{-3}$) and CO (1.66 $\mu g.m^{-3}$) concentrations increased the cases of CVD by 2.8%. PM exposure causes a remarkable elevation in C-reactive protein (CRP), a protein produced by the liver that indicates inflammation, breast cancer, and CVD (Chandrasekhara 2014). The long-term air pollutants' exposure may cause hypercoagulability and thrombosis; which occur due to unfavorable effects on PT, ATPP, CRP, and tissue-type plasminogen activator (tPA), etc. (Mohammadi et al. 2016). Mölter et al. reported that long-term exposure to a combination of PM_{10} and NO_2 decreased lung volume and airway resistance in children (Mölter et al. 2013). High levels of PM_{10} (10 $\mu g.m^{-3}$) increased by 0.72% increasing the CHF admission rate, while short-term exposure of 10 $\mu g.m^{-3}$ $PM_{2.5}$ increased by 13% cases of CHF, ischemic stroke, and

cerebrovascular diseases (Wellenius et al. 2012). Fine and ultra-fine particles reach the blood by entering deep into the alveoli of the respiratory system and then into cellular regions and lymph nodes. Long-term traffic exposure to $PM_{2.5}$ (45.38 $\mu g.m^{-3}$) and PM_{10} (80.07 $\mu g.m^{-3}$) indicated the rising levels of InterLeukin-6 expression, fibrinogen, TNF-alpha, Soluble platelet selectin, t-Hcy, CIMT, PA-1, t-PA; that may be the cause of respiratory and cardiovascular diseases (Bauer et al. 2010, Wu et al. 2016). The association of PM_{10}, NO_2, and CO at low concentration levels caused rising cases of allergic rhinitis (AR), whereas short-term exposure to PM_{10} & SO_2 association revealed elevated levels of FVC and FEV1 (Chen et al. 2016). The concentration level of 10 $\mu g.m^{-3}$ $PM_{2.5}$ was found to be associated with high ERV1 (Xu et al. 2016).

Exposure to $PM_{2.5}$ has adverse effects on glucose tolerance, insulin activity, and blood lipid concentration which may cause type-2 diabetes (Chen et al. 2016). A study revealed that long-term exposure to $PM_{2.5}$ and/or in combination with NO_2 may be an attributable factor to the risk of diabetes mellitus and hypertension (Bowe et al. 2018). Consequently, blood pressure and body mass index increased and resulted in obesity, CVD, respiratory diseases, and cancer even non-smokers may also be affected (Hansen et al. 2016, Katanoda et al. 2011). Exposure to $PM_{2.5}$ and NO_2 may increase HbA1C levels which is the reason for misbalancing glucose metabolism (Qiu et al. 2018).

CO and Associated Physiological Diseases

The poisonous gas, carbon monoxide mainly emitted from transportation means. Its exposure may cause an adverse impact on health such as severe headache, dizziness, CVD, premature births, low birth weight in infants, etc. The CO binds with hemoglobin (Hb) through the cell affinity of an oxygen molecule called hypoxemia. Hypoxemia restrains the oxygen binding capacity which may stimulate gasometrical and respiratory problems (Anand et al. 2017). A positive association was observed between Serum Ischemia modified albumin (diagnostic biomarker in blood COHb) and CO poisoning (Veronesi et al. 2017). The CO mixed with a low concentration of blood plasma was reported to have a high rate of morbidity and mortality due to a misbalance of the bilirubin which increases the risk of ischemic heart diseases, CAD-related diseases, and arteriosclerosis (Chi et al. 2013). The COex is a simple surrogate biomarker related to wealth consequences. Exposure to CO concentration increased the level of COex and increased the risk of myocardial infarction and respiratory infections in smokers (Zhang et al. 2013). A combination of exposure to CO, NO_2 and PM causes inflammation, COPD, exhaled CO may increase the risk of stroke, cardiovascular issues, hypertension, anemia, and

metal concentration, etc. (Nayor et al. 2016). The association of CO (0.9ppm for 8hrs; 6.0%) and NO_2 (5.1 ppb for 24 h, 6.9%) enhances the risk of cardiac issues whereas the low concentration of CO showed severe cases of COPD (Yang et al. 2011). Exposure to CO and O_3 also observed a rise in COPD and asthma cases in hospitals (Khamutian et al. 2015). The roadside workers were reported with some issues such as depression, forgetfulness, tidiness, fatigue, respiratory illness, high pulse rate, etc. while exposed to higher concentrations of CO (Mathur et al. 2017). Meteorological changes with CO exposure may cause coronary issues and heat stress which indicate the risk of CVD (Almendra et al. 2017). CO exposure during long and short-term reported inflammation due to rising WBC counts and ferritin (Lee et al. 2018).

NO_2 deeply penetrates the peripheral bronchi and causes mild and moderate asthma (Greenberg et al. 2017). Exhaled nitric oxide (eNO) increase neutrophilic inflammation in asthmatic person by reducing airway acidification & resistance and lung function (Zhang et al. 2013). Increased asthma visits were reported in the cold season while high annual mean temperatures were reported to enhance the risk of asthma and rhinitis due to NO_2 exposure (Abe et al. 2009, Weaver & Gauderman 2017). An association between NO_2 exposure and pollen was found with elevated emergency admissions of asthma (Galán & Simón 2015). The two-pollutant model concluded the CVD effects and inflammation, diagnosed through the measurement of the bow-like biomarkers and hsCRP (high sensitivity C - reactive protein) (Wu et al. 2016). Short-term vehicular NO_2 and other pollutant emissions promote mortality due to cardiovascular and respiratory issues, and lung cancer (Atkinson et al. 2016). The association of NO_2 and CO exposure was linked with a 2-3% increase in COPD hospitalizations (Peel et al. 2005). The short-term exposure to NO_2 associated with PM_{10}/ O_3 caused acute lower lung function indicated by Fev1 measurements (Nkosi et al. 2016). Exposure to vehicular NO_2 emission caused diabetes mellitus even in healthy non-smokers (Coogan et al. 2012). Short-term exposure to NO_2 (2.67%) indicated increased CVD and diabetes (3.5%) (Goldberg et al. 2013). NO_2 exposure with a concentration of 5 $\mu g.m^{-3}$ in Taiwan caused rising levels of FBG and HbA1C (Honda et al. 2017). While the combination of PM and NO_2 exposure caused the risk of increased diabetes through the C3c marker used in the inflammation process (Krämer et al. 2010). The NO_2 with PM long-term exposure may cause mortality due to chronic diseases such as CVD and diabetes (Bowe et al. 2018).

NO_2 and Associated Physiological Diseases

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SO_2 and Associated Physiological Diseases

The meteorological conditions alter the ambient SO_2 concentration. A high concentration of SO_2 exposure may cause adverse health effects such as breathing problems, airway inflammation, asthma, cardiac issues, psychiatric alterations, etc. Mild optic asthma, high plasma viscosity, and low CBU (Cumulative breath units), Fev1, and FVC levels were reported in people exposed to SO_2 (200 ppb) and NO_2 (400 ppb) under meteorological conditions (Greenberg et al. 2017). The association of SO_2 with PM caused elevated blood pressure and lipid peroxidation, which altered the antioxidant enzyme activities (Kocamaz et al. 2012) and the risk of morbidity due to neuronal dysfunction and COPD (Tingting et al. 2017). The mortality due to lung cancer and respiratory issue was reported in Japan and Stockholm with high annual mean concentration levels of SO_2 (Katanoda et al. 2011). SO_2 exposure also stimulates the action of endogenous 2-AG which suppresses the inflammation associated with microvasculature dysfunction and brain inflammation (Greenberg et al. 2017). NO_x and SO_2 exposed women

having early pregnancy and preconception may be affected by gestational diabetes mellitus (Robledo et al. 2015). The association of PM_{10}, SO_2 & NO_2 exposure causes elevated FBG levels and lipid levels including low-density lipoprotein cholesterol, total cholesterol, triglycerides, etc. which indicate diabetes risk (Wang et al. 2014).

Association of the Pollutants with Psychological Diseases

The efficiency of psychological behavior is measured in terms of stress or depression. Psychological behavior influences humans in twin folds: unhappiness, self-suffering, awful confrontations, depressing feelings, and various other sentiments that cause depression. Mainly adults are suffering from the symptoms of depression which are linked with hypotension and low lipid level. The study on the effect of air pollution on human behavior began in 1970. Further research on the relationship between air pollutants and human behavior may vary conceptually. Exposure to the high concentration level of particulate and gaseous air pollutants affects the process of psychology adversely which stimulate stress. This stress may proceed as a disruptive environmental pollutant which may create psychological issues in humans. This issue has been focused very less therefore; we decided to observe the air pollutants' effect on psychological as well as physiological diseases. This section includes the effect of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO on stress, dementia, and depression.

PM_{10} & $PM_{2.5}$ and Associated Psychological Diseases

Exposure to SO_2, NO_2, $PM_{2.5}$, PM_{10}, and CO has been reported as a factor to enhance depression with the risk of asthma, CVD, diabetes, etc. (Hazlehurst et al. 2018). Individuals suffering from CVD were tending to have a 10.1% increase in stressful suicidal threats and 6.7% in dementia, and depression at 0-45 depression scales due to $PM_{2.5}$ exposure (Pun et al. 2017, Kim et al. 2010). A recent study revealed the strong correlation of $PM_{2.5}$ exposure with stress-induced toxicants based on gender, age, demographic, and socioeconomic status. The lung structure with its function and its immune function can be adversely impacted by stress (Sass et al. 2017). An erectile dysfunction, a sign of psychological stress was observed in people due to air pollutants exposure. The measurement of some blood markers such as IgE, interleukin-5, and interaction beta indicated chronic stress in asthmatic children (Cheng et al. 2018). Long-term association of PM_{10} and O_3 exposure pollution caused dementia, mild cognitive impairment and Alzheimer's indication, and other psychological disorders (Oudin et al. 2016). However, emission from traffic exposure with APOE decreases cognitive impairment (Schikowski et al. 2015). The psychosocial

stress may be caused due to the secretion of plasma and adrenocorticotropic hormone (ACTH) which is enhanced by air pollution exposure (Cordella & Poiani 2003). The ambient pollutants may influence the risk of suicidal tendencies in people who are maintaining low social-economic status (Lee et al. 2018).

CO and Associated Psychological Diseases

High levels of pollutants emitted from diesel revealed the symptoms of acute psychological stress (Laumbach et al. 2011). The study on vehicular emission (NO_2 & CO) exposure reported a significant increase in the cases of dementia in Taiwan (Chang et al. 2014). An impaired cognitive behavior was observed due to the combination of HBO_2 (Hyperbaric Oxygen) and CO concentration (Weaver et al. 2007). The number of emergency hospital visits was also reported as a strong positive correlation between CO level and meteorological parameters (Szyszkowicz et al. 2016). The rural women were observed with increased depressive symptoms due to the CO emission from kerosene stoves resulting in higher cognitive impairment (Banerjee et al. 2012). This study also corroborates with cognitive impairment diagnosed in 43 years aged women due to exposure to high CO concentration (Mayer 2000).

NO_2 and Associated Psychological Diseases

Exposure to elevated concentration levels of air pollutants may cause stress due to neurological imbalance. The stress may disturb the parasympathetic activity which may cause inflammation. A questionnaire survey revealed that exposure to NO_2, $PM_{2.5}$ & PM_{10} caused depression in people (Zijlema et al. 2016). The women were diagnosed with more severe depression than men due to the concentration level of 10 $\mu g.m^{-3}$ NO_2 (Vert et al. 2017). Chang et al. conducted a population-based survey and found that the people were suffering from dementia due to NO_2 and CO exposure (Chang et al. 2014). The hereditary effect was noticed for decreasing lung function due to NO_2 exposure through the analysis of non-smokers (Shankardass et al. 2015). Elevated temperature with rising NO_x concentration caused the risk of suicidal tendencies. A low level of gaseous air pollutant exposure may also report increased cases of psychiatric disorders along with the increased risk of suicidal distress (Oudin et al., 2018). The infants exposed to NO_2 were reported with inverse effects on mental development during prenatal and postnatal periods (Lertxundi et al. 2015).

SO_2 and Associated Psychological Diseases

The record revealed the impact of air pollutants exposure on physiological coordination and resulted in mood distortions and stress. Short-term SO_2 exposure was reported with a high risk of depressive mood followed by chronic stress (Wang et al. 2014). Exposure to elevated concentrations of PM_{10}, SO_2 & NO_2 affected pregnant women in the winter season and diagnosed with PSS 75-100 scores which indicate severe depression. The people exposed to high concentrations of $PM_{2.5}$, BC, and SO_4^{2-} were caused a high perceived stress scale (PSS) score (Mehta et al. 2015). The linkages of mechanisms between air pollutant levels and suicidal loss increase the depression probability (Lin et al. 2017). The air pollution exposure was measured and analyzed between prenatal and postnatal periods and the results indicated parental stress may cause the probability of childhood asthma (Deng et al. 2018). A recent study reported post-surgery cognitive disorder, which is an indication of dementia (Che et al. 2017).

RESULTS AND DISCUSSION

Description of Study Material

We have accessed a total of 369 high-impact factor research articles to carry out this review analysis. The articles having incomplete information, repetitive and outdated observations (n=82), and unknown time duration (n=02) were excluded. The remaining research papers for considering the study were 285. Further 60 papers out of 285 were excluded based on mislaid data for morbidity, mortality, sex, and age, related to diseases categorized as physiological and psychological diseases. Thus a total of 225 research articles were sorted out for this study. Regarding the number of patients, out of the records of 23 years (1997-2020) international publications which reported insufficient patients were excluded (n=22). The number of remaining patients (7781054) was integrated into the analysis. A primary cohort of 2754056 patients includes a record considering the effect of air pollution concentration levels on different age group populations. The methodology and results about the impact of air pollution in terms of physiological diseases (2088092 patients) and psychological diseases (665975 patients) were incorporated into the study for an efficient outcome. Figure 1 shows the detailed flow diagram for the description of the study material.

Authors' Interpretation of Systematic Evidence

We have downloaded more than 200 peer-reviewed international research articles for interpretation based on analysis (all the considered articles are not included in the reference section of this study). Out of those, 50 research articles about each pollutant ($PM_{2.5}$, PM_{10}, CO, NO_2 & SO_2) and associated diseases were taken into account. We have simplified the complex health effects due to air pollution exposure by classifying them into two groups of diseases-physiological
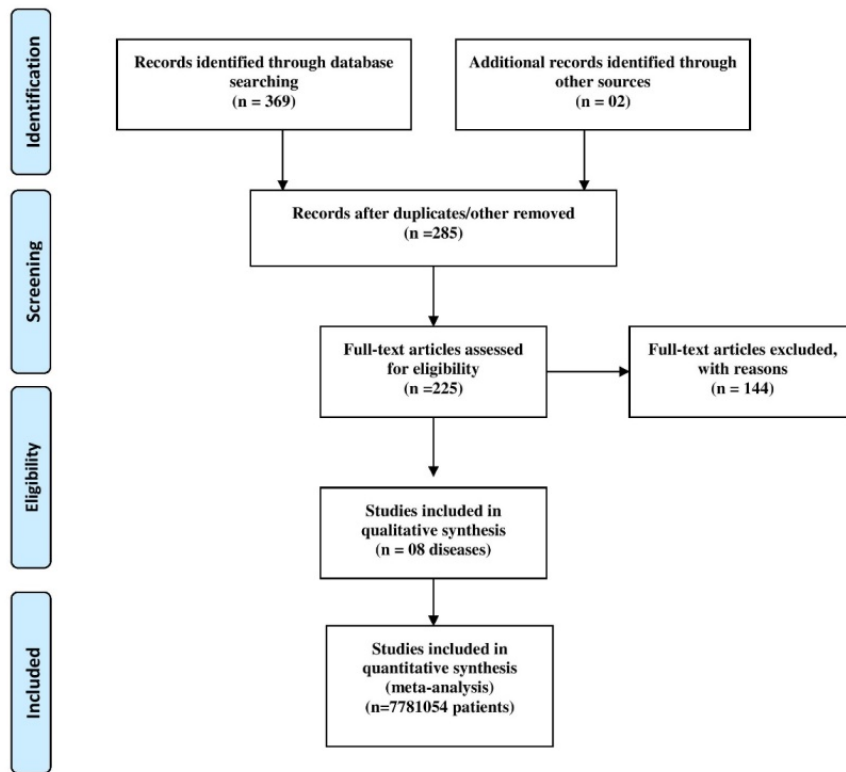
Fig. 1: Flow diagram representing the description of study material.

diseases and psychological diseases. Physiological diseases refer to CVD, COPD, asthma, diabetes, and Cancer, whereas psychological diseases include dementia, stress, and depression. Direct or combined exposure to air pollutants may cause physiological diseases. While psychological diseases occurred as a consequence of physiological disorders. The air pollutants cause direct or indirect effects on endocrine glands, deteriorate hormonal coordination, and may cause neuropsychological diseases. The mortality and morbidity due to short-term and long-term exposure to air pollutants and their acute and chronic effects have also been examined. Over the years trends for concentration levels of air pollutants, and associated age-wise effects on diseases were analyzed for critical assessment. The patterns helped us to analyze and realize the crucial interpretation for this study.

An overview of the diseases across the globe is shown in Fig. 2a. The figure depicts the hospital admissions for the physiological diseases that occurred due to air pollutants' direct and indirect exposure. The highest hospitalization was more than 50% for COPD cases associated with 40 $\mu g.m^{-3}$ PM_{10} exposure and more than 15% of COPD cases were associated with 50 $\mu g.m^{-3}$ $PM_{2.5}$ exposure. About 50% of cases of cancer were associated with 35 $\mu g.m^{-3}$ $PM_{10,}$ 20

$\mu g.m^{-3}$ $PM_{2.5}$ & 40 $\mu g.m^{-3}$ SO_2 exposure. More than 30% of cases of diabetes were associated with 30 $\mu g.m^{-3}$ CO exposure and found as

the fourth highest disease as per our analysis. PM_{10} is again the key contributor to 15% of cases of asthma, however, more or less all five air pollutants were found to be responsible for asthma. CVD hospitalizations with more than 20% cases were associated with 20 $\mu g.m^{-3}$ SO_2 concentrations.

Figure 2b shows the effect of pollutants' exposure on hospitalizations due to the cases of psychological diseases pertaining to the direct and/or consequences of other ailments. CO (30 $\mu g.m^{-3}$) and SO_2 (35 $\mu g.m^{-3}$) were observed as the key contributors to more than 50% of cases of dementia. Even a low concentration of CO (5 $\mu g.m^{-3}$) was associated with more than 50% of cases of depression but it is the major contributor to depression. $PM_{2.5}$ (15 $\mu g.m^{-3}$) and SO_2 (15 $\mu g.m^{-3}$) are the second leading contributors associated with 25% of cases of depression. NO_2 is also a notable contributor to depression as the stress was allied with 40% of cases due to PM_{10} (35 $\mu g.m^{-3}$), 30% NO_2 (13 $\mu g.m^{-3}$), and 20% of cases due to SO_2 (15 $\mu g.m^{-3}$) concentrations. PM_{10} is the major air pollutant that was alone associated with 40 -50% of cases of all three types of psychological diseases. However,
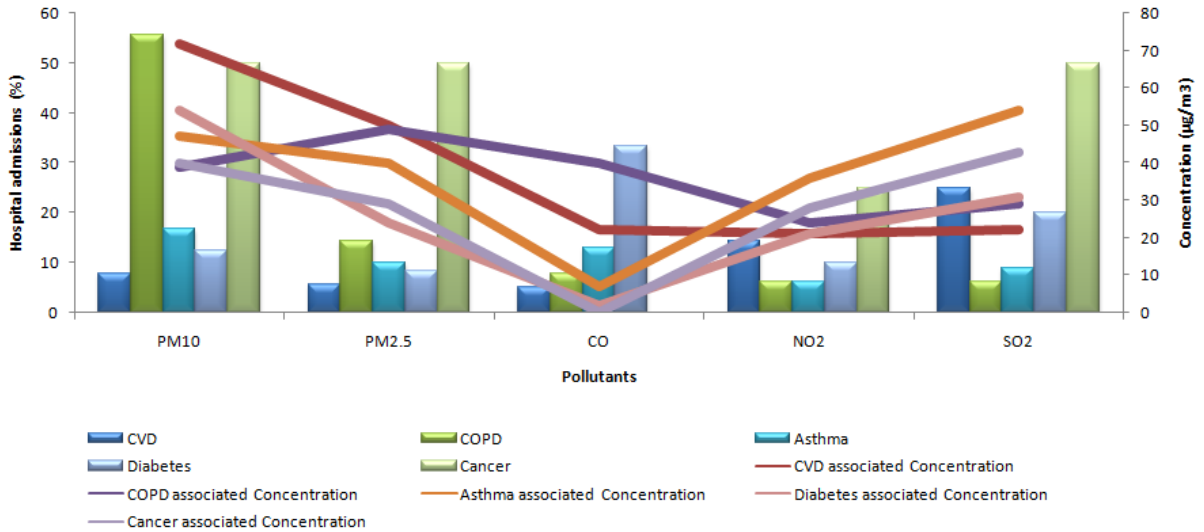
Fig. 2a: Overall outline of hospital admissions due to physiological diseases and associated concentration levels of $PM_{2.5}$, PM_{10}, CO, NO_2 & SO_2.
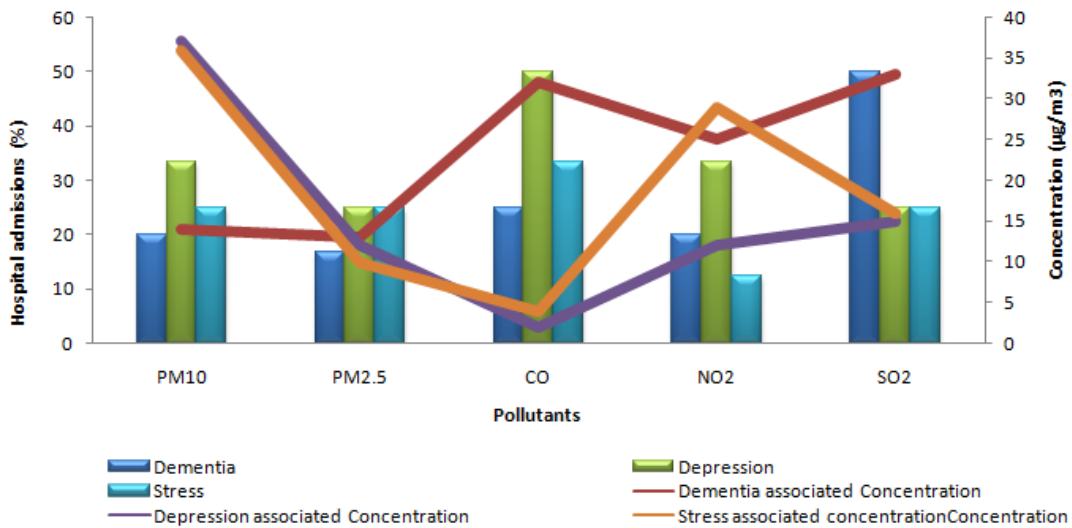

Fig. 2b: Overall outline of hospital admissions due to psychological diseases and associated concentration levels of $PM_{2.5}$, PM_{10}, CO, NO_2 & SO_2.

$PM_{2.5}$ is the least contributor associated with 15% of cases of psychological diseases.

We have accessed a total of 369 high-impact factor research articles to carry out this review analysis. The articles having incomplete information, repetitive and outdated observations (n=82), and unknown time duration (n=02) were excluded. The remaining research papers for considering the study were 285. Further 60 papers out of 285 were excluded based on mislaid data for morbidity, mortality, sex, and age, related to diseases categorized as physiological and psychological diseases. Thus a total of 225 research articles

were sorted out for this study. Regarding the number of patients, out of the records of 23 years (1997-2020) international publications which reported insufficient patients were excluded (n=22). The number of remaining patients (7781054) was integrated into the analysis. A primary cohort of 2754056 patients includes a record considering the effect of air pollution concentration levels on different age group populations. The methodology and results about the impact of air pollution in terms of physiological diseases (2088092 patients) and psychological diseases (665975 patients) were incorporated into the study for an efficient outcome. Figure 1

shows the detailed flow diagram for a description of the study material.

Age is one of the important factors for measuring the degree of severity of the disease. The severity of the disease for diverse groups based on age versus hospital admissions due to physiological and psychological diseases is shown in figure 3. We have classified 6 major age groups (0-15, 15-30, 30-45, 45-55, 55-65 & >65). There were 60% cases of asthma, 30% COPD, and 10% cancer cases found to be associated with the age group of ≤15 years which is very sensitive mainly for children. The adult set of age (15-30 years) was associated with all diseases prominently except diabetes. However, 35% of cases of diabetes in the age group of 55-65, 45% of cases of CVD with >65 years, and 35% of cases of cancer were found in the≥ 65 age group.

As per our study, psychological diseases prevail in the age group of ≥ 65. This age group is mostly suffered from dementia (≤ 50%). The age group of 30-45 is the main age group that is burdened with stress, while the> 65 age group was found with high depression. The age group of 30-45 was more affected by stress levels that decreased as an increase in age was observed (45<55<65< above 65 years).

CONCLUSION

The inference of this study from the data analysis of five air pollutants' concentrations versus five physiological and three psychological diseases will be an indication for further studies on diverse characteristics related to possible causes of impact. The study also provides an overview to categorize the strength of diseases for vulnerable population groups. It was observed that particulate matter (PM_{10} & $PM_{2.5}$) is the main contributor to all types of respiratory diseases. There is more than enough work done on the impact of PM_{10} and $PM_{2.5}$ concentrations on physiological diseases, thus psychological diseases require more consideration. PM_{10} alone is the main pollutant that causes approx. 40%-50% cases of psychological diseases, whereas surprisingly $PM_{2.5}$ with only 15% cases is the least contributor for psychological diseases. Among all five air pollutants, CO evidenced minimum effectiveness for cancer and CVD cases but highly effective for depression followed by $PM_{2.5}$ and SO_2. NO_2 was found to be a prominent cause of hospital admissions due to COPD and the second largest contributor to depression also. Adults are suffered from stress and increasing order of PM_{10}, NO_2, and SO_2 concentrations are responsible for stress. There are less data available for the effects of air pollutants on psychological diseases thus more rigorous research work is required for decisive health effects.

ABBREVIATIONS

APOE: Apolipo protein E

CB1 &CB2 Receptors: Cannabinoid receptors

COAD: Chronic Obstructive Airway Disease

Coex: Exhaled Carbon monoxide

CoHb: Carboxy Hemoglobin

ERV_1: Expiratory reserve volume 1

FBG Levels: Fasting Blood glucose levels

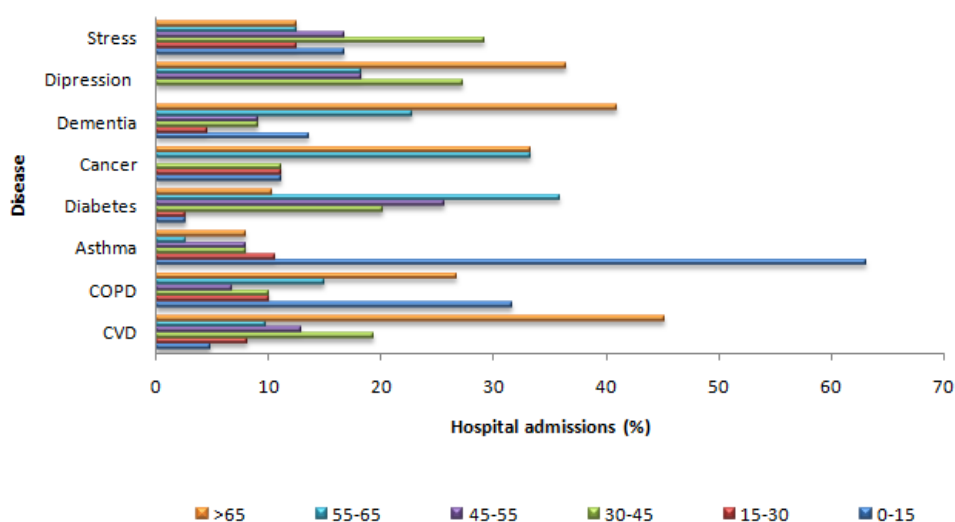FEV: Forced Expiratory Volume

Fig. 3: The overall outline of hospital admissions due to physiological and psychological diseases as per age groups.

FVC: Forced Vital Capacity

HbA1C: Glycosylated Haemoglobin A1C

HBO$_2$: Hyperbaric oxygen

IHD: Ischemic Heart Disease

LIDAR: Light detecting and ranging systems

PEF: Peak expiratory flow

SRAW: specific airway resistance z

TNF-alpha: Tumour necrosis factor-alpha

t-PA: tissue Plasminogen Ac

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