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KEYWORDS	ABSTRACT
Air pollution, Pollutants, Occupational hazards, chronic kidney diseases.	Air pollution is the major public health problem and vehicular exhaust fumes are the principal source of air pollution in urban areas. The hazardous traffic fumes contain a variety of pollutants such as particulate matter (PM), carbon monoxide (CO), carbon dioxide (CO ₂), nitrogen dioxide (NO ₂), sulfur dioxide (SO ₂), ozone etc, all of which can cause respiratory problems and other systemic diseases including cancer. Since occupation is a major determinant of health, traffic police personnel face multiple occupational hazards. They are continuously exposed to vehicular emissions, work in noisy and polluted environment. Standing for long hours in a static position makes them vulnerable to major health problems. Managing high volumes of traffic density results in physical and mental fatigue among traffic policemen making them susceptible to physical and mental stress. As per the systematic analysis of the global burden ambient air pollution is recognized as one of the leading causes of global disease. Chronic Kidney Diseases are among major problems caused by Pollution leading to kidney damage and ultimately kidney failure. This study demonstrates the toxic effects and major outcomes of pollution on renal function of traffic wardens.
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Introduction

Air Pollution

Air pollution is the major public health problem and vehicular exhaust fumes are the principle source of air pollution in urban areas. A European assessment that suggested air pollution accounts for 6% of all mortality and 50% of mortality due to air pollution is accounted for by vehicular pollution (Kunzli et al., 2000). The hazardous traffic fumes contain a variety of pollutants such as particulate matter (PM), carbon monoxide (CO), carbon dioxide (CO₂), nitrogen dioxide (NO₂), sulfur dioxide (SO₂), ozone etc, all of which can cause respiratory problems and other systemic

diseases including cancer (Anderson et al., 2004) . In addition, vehicular fumes also contain dangerous polyaromatic hydrocarbons like benzene, toluene, xylene which are classified as potentially carcinogenic and genotoxic. PAH are of particular concern because their concentrations in the urban air are 10–30 times higher than rural air concentration. The respiratory effect due to exposure to vehicular fumes could be both allergic and nonallergic in nature (Suglia et al., 2008).

Every material in the air which could affect human health or have a profound impact on the environment is defined as air pollutants. According to the World Health Organization (WHO), particle pollution, ground-level O₃, CO, sulfur oxides, nitrogen oxides, and lead (Pb) are the six major air pollutants which can harm human health and also the ecosystem. There are many pollutants of suspended materials such as dust, fumes, smokes, mists, gaseous pollutants, hydrocarbons, volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), and halogen derivatives in the air which at the high concentrations cause vulnerability to many diseases including different types of cancers (Loomis et al., 2014; Kjellstrom et al., 2006; Rodopoulou et al., 2014; Carugno et al., 2016). Heavy metals can induce kidney damage such as an initial tubular dysfunction evidenced by an increased excretion of low molecular weight proteins, which progresses to decreased glomerular filtration rate (GFR). In addition, they increase the risk of stone formation or nephrocalcinosis (Damek-Poprawa and Sawicka-Kapusta, 2003; Jarup, 2003).

Since occupation is a major determinant of health, traffic police personnel face multiple occupational hazards. They are continuously exposed to vehicular emissions, work in noisy and polluted environment. Standing for long hours in a static position makes them vulnerable to major health problems. Managing high volumes of traffic density results in physical and mental fatigue among traffic policemen making them susceptible to physical and mental stress. Since stress is not considered healthy in any case so both physical and mental health manifestations get accentuated with the increasing length of service (Satapathy et al., 2009). Outdoor occupations are generally hazardous in nature due to prolonged periods of exposure to high concentrations of vehicular pollution which puts the employees at increased risk of respiratory, renal and cardiovascular diseases (Mills et al., 2011). Traffic police personnel are at the highest risk for the adverse health effect of air pollution, compared to the general population. Occupational studies on traffic police personnel help us to understand the effects of vehicular pollution and its specific adverse effect due to the opportunity for defined exposures measurements (Brandt et al., 2003). Another very important advantage of studying the impact of vehicular pollution on traffic police is that it helps in quantification of environmental exposures and risk characterization of the health outcome.

Effect of different metals on renal function

As per the systematic analysis of the global burden ambient air pollution is recognized as one of the leading causes of global disease. There are different metal which adversely affect the traffic wardens. Cadmium is one of them. CD exposure leads to renal proximal tubular damage, and severe

renal tubular damage can lead to declined reabsorption of cadmium metallothioneine complexes so the Cd exposure can lead to initially higher and later to lower urine Cd concentrations (Orr and Bridges, 2017). For this reason, urine Cd may cease to be a marker of chronic exposure among those with renal disease. A positive association between urinary Cd levels and estimated glomerular filtration rate (eGFR) and a negative association between eGFR and blood Cd were reported by Buser et al., (2016) among US adults aged ≥ 20 years. Navas-Acien et al., (2009) reported elevated levels of blood Pb to be associated with increased odds of reduced kidney function. Both inorganic and organic compounds of Hg accumulate in the kidneys. Renal toxicity can occur following exposure to either organic or inorganic Hg, and it is the Hg^{2+} conjugates that appear to be the most nephrotoxic (Orr and Bridges, 2017).

Uranium toxicity is although relatively rare, it can also cause renal damage and indeed, following inhalation or ingestion the kidney is the primary target for uranium toxicity. Approximately 40% of plasma uranium is complexed to transferrin and the remaining 60% is complexed with carbonate or bicarbonate, which are filtered by the glomerulus (Roszell et al., 2009). Moreover, air pollution is a risk factor for upper and lower respiratory airways and air pollution may also impact other organs such as heart, vessels and kidneys. The inflammatory mediators induced by PM and other pollutants in the lungs could spill over into the circulation, resulting in systemic inflammation, oxidative stress and damage to distant organs including kidneys (Brook et al., 2010).

Ambient air pollution has recently been recognized as one of the most serious issues worldwide. According to World Health Organization (WHO) data, more than 90% of the world's population lives in places that do not meet the WHO standards for air quality. The number of deaths due to outdoor air pollution is estimated to be about 3 million a year. Exposure to air pollution increases the risk of developing various diseases, including cardiovascular disease (CVD), chronic obstructive pulmonary disease, type 2 diabetes mellitus, and autoimmune rheumatic diseases (Brook et al., 2010; Farhat et al., 2011; Hoek et al., 2013; Sunyer, 2001). Indeed, few studies have demonstrated the negative effects of air pollution on renal function indicators such as estimated glomerular filtration rate (eGFR) and CKD (Mehta et al., 2016; Yang et al., 2017).

Experimental evidence and observations from several small clinical research studies suggest that exposure to higher amounts of air pollution for longer times adversely affects kidney function. Higher kidney disease mortality in coal mining regions of the Appalachian Mountains (USA) has been ascribed to environmental exposure to air pollutants (Hendryx et al., 2009). Residential proximity to major roads, which in large part is an indirect measure of exposure to air pollutants (and other possible factors, including noise pollution), is generally associated with a reduced estimated glomerular filtration rate (eGFR) (Lue et al., 2013). In a large cohort of US veterans, 3 higher amounts of fine particulate matter were associated with increased risk of incident chronic kidney disease, eGFR decline, and end-stage renal disease (Bowe et al., 2017).

Anatomy of kidney

The kidneys are central to homeostasis (Hoenig and Zeidel 2014). Through exquisite sensory mechanisms they regulate blood pressure, water, sodium, potassium, acidity, bone minerals, and hemoglobin (Pluznick and caplan 2014; Danziger and Zeidel 2014; Palmer and Schnermann 2014; Subramanya and Ellison 2014). But their core function is the excretion of the waste products of metabolism in urine. About 22 % of cardiac output goes to the kidneys and about 20 % of the plasma is filtered, producing about 170 L of glomerular filtrate per day. Ninety-nine percent of this is reabsorbed as it flows along the nephrons so only about 1.5 L of urine is produced per day. Filtration occurs through the glomerular filtration barrier (Pollak et al., 2014). This is made up of five layers (Arkill et al., 2014).

- the glycocalyx covering the surface of the endothelial cells
- holes (fenestrations) in the glomerular endothelial cells
- the glomerular basement membrane
- the slit diaphragm between the foot-processes of the podocytes

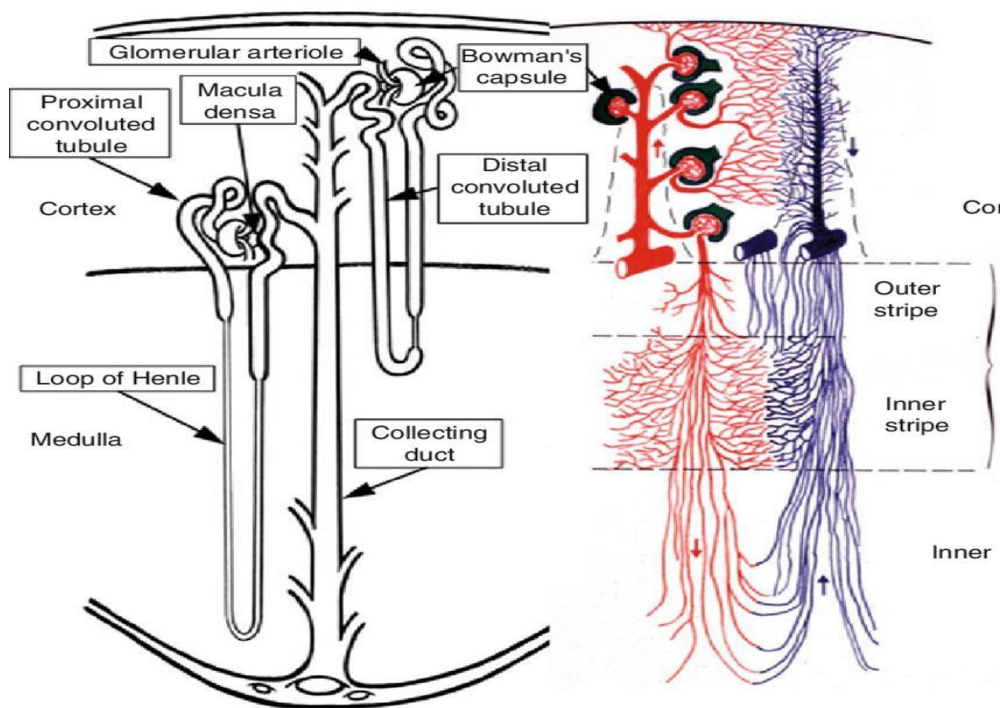


Fig.1. Nephrons and their blood supply. A short looped- and a long looped-nephron. The different vascular territories and their location in the four renal zones. (Vasudevan, D. M., and Sreekumari, S. (2013).

The composition of glomerular filtrate is determined by the structure, arrangement and electrical charge of the collagen protein molecules that form the filtration barrier. So glomerular filtration is both size-selective and charge-selective; molecules that are too large or too highly charged cannot get through. Glomerular filtration is held constant over a wide range of systemic and renal artery pressures by a process called autoregulation. Constriction and dilatation of the afferent arteriole is controlled by the macula densa, which is adjacent to the glomerulus. The macula densa senses the flow of sodium chloride through the tubule next to it. When this flow is increased, the macula densa causes constriction of the afferent arteriole to reduce the glomerular filtration rate(Schießl et al., 2015). The major functions of the kidneys are to excrete metabolic waste products as well as to maintain water, pH, electrolyte balance, production of calcitriol and erythropoietin. A decrease in kidney function is due to a reduction in the performance of nephrons. The functional unit of the kidney is the nephron, which is composed of the Bowman’s capsule with the glomerular tuft of capillaries, the proximal convoluted tubule (PCT), loop of Henle, distal convoluted tubule (DCT) and collecting tubules.(Fig. 1)

Functions of the Tubules

When the glomerular filtrate is formed, it contains almost all the crystalloids of plasma. In the proximal convoluted tubules, about 70% water, Na+ and Cl– as well as 100% glucose, amino acids and K+ are reabsorbed. Urea, phosphate and calcium are partially absorbed.

Reabsorption of Water

The osmolality of urine can vary between 60–1200 mosmol/kg , depending on the water intake and state of hydration. The GFR is about 125 mL/min. In the proximal convoluted tubules, most of this is reabsorbed. Since Na+, Cl– and HCO3– ions are absorbed, water has to move along with the solutes to maintain the osmolality. Hence, this is called obligatory reabsorption of water. By the time it reaches the loop of Henle, the filtrate is only 25 mL/min. Here sodium is again reabsorbed, but water absorption is less so that, urine, is hypotonic at this level.

Table 1.Common tests to assess renal function(Vasudevan and Sreekumari , 2013).

Constituent	Blood level or Urine excretion	Factors affecting urinary excretion
Urea	B = 15–40 mg/dL U = 15–30 g/day	Dietary proteins, protein catabolism, renal blood flow
Creatinine	B = 0.7–1.4 mg/dL (M) B = 0.4–1.3 mg/dL (F) U = 1–2 g/day	GFR, tubular secretion, age, sex, muscle mass

Uric acid	B = 3–7 mg/dL (M) B = 2–5 mg/dL (F) U = 0.5–0.8 g/day	Purine catabolism, tubular excretion
Sodium	B = 135–142 mmol/L	State of hydration, dietary sodium, renal function
Potassium	B = 3.5–5 mmol/L	Dietary potassium, acid–base balance, renal function
Calcium	B = 9–11 mg/dL	Dietary calcium, PTH, calcitonin, renal function

Renal Diseases

Chronic Kidney Disease (CKD)

GFR calculated from serum creatinine and MDRD equation are used to grade CKD. CKD is a silent killer. CKD is a growing problem that affects approximately 12

% of the adult population. Major risk factors of CKD are diabetes mellitus, hypertension, glomerular nephritis, urinary tract infection, autoimmune diseases, kidney stones and toxic effects of some drugs. Markers for CKD are serum creatinine, eGFR, microalbumin and Cystatin C

Acute Kidney Injury (AKI)

AKI is characterised by a rapid rise of serum creatinine with low urine output. Onset of AKI can be swift and often deadly. In acute kidney injury, serum creatinine (sCr), can take days to show an increased level. Novel biomarkers of tubular injury such as urinary neutrophil gelatinase-associated lipocalin (uNGAL) may enable the early detection of acute kidney injury before a change in GFR is observed. The uNGAL is very useful when sCr level is not increased (Vasudevan and Sreekumari, 2013).

Hyperuricemia is common in CKD patients, and the association between uric acid and worsening renal function is well established (Nacak et al., 2014). Animal studies have shown that oxidative stress and endothelial dysfunction due to hyperuricemia cause renal damage (Mazzali et al., 2001). Furthermore, one epidemiological study suggested that increases in serum uric acid levels precede the development of CKD (Bo et al., 2001). The specific mechanisms by which ambient air pollution may elevate or modify uric acid concentrations, leading to hyperuricemia, are yet to be elucidated. One potential hypothesis is that exposure to pollution increases systemic inflammation that leads to vascular, capillary, and eventually glomerulus damage. Renal uric acid metabolism is closely related to glomerular filtration rate (Weiner et al., 2008). A series of recent epidemiologic studies reported that ambient air pollution might lead to a decreased glomerular filtration rate, ultimately resulting in hyperuricemia and an elevated uric acid/serum creatinine ratio (Lue et al., 2013; Chen et al., 2018).

Air pollution and Diabetes

Diabetes is an ever-increasing worldwide health problem. Systematic reviews, meta-analyses and epidemiological studies confirm that exposure to air pollution may be associated with the incidence or prevalence of DM (Janghorbani et al., 2014; Sohn, 2017; Rajaagopalan and Brook, 2012; Rao et al., 2015). The association with O₃ with incident diabetes was stronger in women than in men and among those aged

<50 years (Renzi et al., 2018). Higher exposures to 3- to 7-day moving averages of black carbon and NO_x were associated with higher plasma glucose, whereas the associations for ozone were negative, but there was no association with insulin resistance as evaluated by the HOMA-index (Li et al., 2018). Describing in short term exposure to air pollution was associated with higher levels of glucose, insulin or HOMA-IR (Brook et al., 2013; Chen et al., 2016), whereas in longer term exposure, most of the positive associations were found with glucose but not with insulin or HOMA-IR (Ward et al., 2015; Wolf et al., 2016).

Residential proximity to major roads, which in large part is an indirect measure of exposure to air pollutants (and other possible factors, including noise pollution), is associated with a reduced estimated glomerular filtration rate (eGFR) (Lue et al., 2013). In a large cohort of US veterans, 3 higher amounts of fine particulate matter were associated with increased risk of incident chronic kidney disease, eGFR decline, and end-stage renal disease (Bowe et al., 2017). The prevalence of chronic kidney disease (CKD) is increasing, and it has become one of the most important health problems worldwide. Individuals with CKD have an eight- to 10-fold higher risk of CV-related mortality than those without renal dysfunction (Couser et al., 2011; Kim et al., 2019). DM, hypertension, dyslipidemia, smoking, old age, and male sex are well-established traditional risk factors for CKD (Agarwal, 2008). However, recent reports have shown that air pollution, as well as traditional risk factors, play an important role in renal disease (Bowe et al., 2018; Yang et al., 2017). Several mechanisms have been proposed to link exposure to air pollution with BP and kidney damage. Inhaled PM can trigger acute autonomic nervous system imbalance and systemic proinflammatory responses and can activate vascular endothelial dysfunction and arterial vasoconstriction (Pope and Dockery, 2006; Brook, 2008).

Air pollution contributes to an estimated 6.5 million deaths per year (Landrigan et al., 2018), according to a 2017 study, and ranks fourth in a list of risk factors for all-cause deaths. It falls after high systolic blood pressure, dietary risks, and tobacco use but above factors like high cholesterol and high body mass index. Studies in people have found higher kidney disease mortality in coal mining areas and lower estimated glomerular filtration rate (eGFR) in those who live near major roadways, since pollution is more in these areas (Lue et al., 2013). And, those who live in the midwestern and southern U.S. have a higher burden of chronic kidney disease (CKD) attributable to air pollution (Bowe et al., 2018). Globally, low- and middle-income countries are disproportionately affected by kidney disease attributable to air pollution (Bowe et al., 2016).

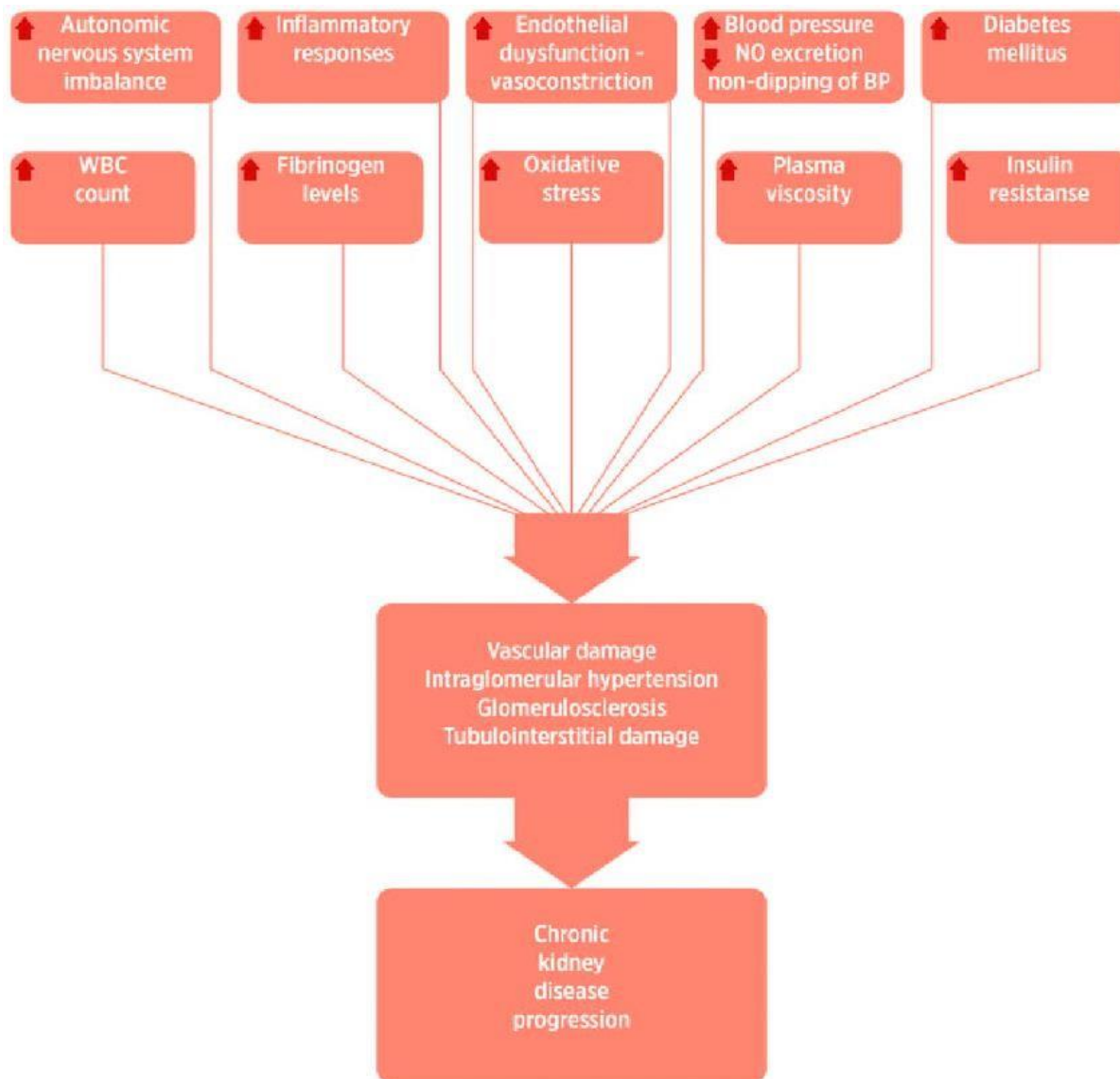


Figure 2.Flow chart of Pathogenic mechanisms of environmental pollutants leading to kidney damage

<https://www.researchgate.net/publication/331060193/figure/fig1/AS:725665598951424.png>

Aims and Objectives

The purpose of this study is to assess the health impacts of pollution on the traffic wardens. This will create awareness among people that how these traffic personnel regularly contracting to pollution develop renal and other chronic diseases. Since traffic policemen are the most exposed to

automobile exhaust, so this study will raise concerns about the possible effects of pollution on their renal functions. The aims of this study are:

- The assessment and quantification of health impacts of air pollution
- To create link between pollution and health problems
- To assess the extent of pollution and its negative outcomes
- To understand the risk that traffic wardens possess about their health conditions

Review of Literature

Al-Aly et al., (2020) worked on the air pollution and kidney disease and draw the attention of people towards the association of kidney to the pollution. In the past several decades, a lot of attention was focused on individual-level risk factors for kidney disease, and for so long the relationship between environmental exposures and kidney disease has not been seriously examined. Therefore Al-aly and his coworkers focused on how the nanoparticles ingested get filtered by the kidney and leads to its damage. Recent advances in nanomedicine enabled studies in rodents and humans showing that inhaled gold nanoparticles, when sufficiently small, permeate through the lung alveolar tissue and enter the bloodstream where they interact with extrapulmonary organs, are filtered by the kidneys in a size-dependent manner, and ultimately excreted in the urine These proof-of-principle studies demonstrated for the first time that inhaled aerosols may come in contact and potentially interact with kidney tissue . In this study they discovered the evidence that has emerged suggesting that rodents exposed by inhalation to diesel exhaust particles (to experimentally simulate exposure to fine particulate matter air pollution) exhibited increased oxidative stress, inflammation, and DNA damage in kidney tissue. After prolonged exposure, these rodents exhibited many deformities in kidney including structural chronic kidney injury manifested by vascular damage, glomerular sclerosis, mesangial expansion, and tubular atrophy .These findings provided biologic plausibility for a putative injurious effect of aerosol pollution on the kidney and powered the generation of hypotheses to test the effect of fine particulate matter air pollution on risk of kidney disease in humans.

Burrows et al., (2018) conducted a study in US and evaluated that Chronic kidney disease (CKD) has emerged as one of the major public health problems facing the US population, with a high burden of disability, disproportionate distribution, poor outcomes, and high costs. CKD collectively represents chronic kidney damage or loss of kidney function from early stages, characterized by elevated albumin excretion in the urine, to kidney failure requiring dialysis or kidney transplantation for survival. They adopted a method for comparing changes in prevalence and treatment of CKD, diabetes, and hypertension, and changes in CKD awareness from the initial to the follow-up screening among participants who completed both screenings, then they calculated the standardized difference for each variable. They observed an important benefit of screening which is that diagnosing CKD at an earlier disease stage may lead to slower disease progression and a reduction of morbidity and mortality over time. This means that the CKD is time dependent if one catches it earlier then survival is possible. Management of CKD to reduce or slow

the progression of disease can reduce the incidence of kidney failure. The results were very obvious, that if the individual is already suffering from a disease, then progression towards it is easier. Compared to adults without diabetes or hypertension, individuals with these risk factors progress more quickly through the stages of CKD to kidney failure. Burrows and his colleagues were successful in finding that the kidney disease is related to the onset and its diagnosis, including the factor if one suffered from any kind of disease earlier.

Tang et al., (2019) aimed to evaluate the association between ambient air pollution and hyperuricemia. Hyperuricemia, usually defined as serum uric acid above 420 $\mu\text{mol/L}$ in males and above 350 $\mu\text{mol/L}$ in females is a major threat to human health. Hyperuricemia is associated with an elevated incidence of several diseases, including hypertension, cerebrovascular stroke, and chronic kidney disease. Guangzhou is one of the largest cities in China, with a population of approximately 14 million. It has high motor vehicle density and also air pollution related to traffic exhaust is an important environmental issue. Traffic police officers, in particular, are continuously exposed to vehicular emissions and noise during their work. As a result, they may be particularly vulnerable to adverse health effects from airborne pollutants. Recent studies suggest that ambient air pollution has adverse impacts on the cardiovascular, respiratory, endocrine, and nervous systems. The prevalence of hyperuricemia in China has increased in recent years, alongside improvements in living standards and rapid economic development. Long-term exposure to ambient air pollution was associated with a higher incidence of hyperuricemia among traffic police officers.

According to Sebastiampillai et al., (2015) traffic policeman are identified to be at a higher risk of exposure to air pollution and its contaminants such as lead. A study done prior to the introduction of unleaded petroleum in Srilanka revealed mean blood lead level of 53.07 $\mu\text{g/dL}$, which was well above the Centre for Disease Control defined acceptable safe levels. This study aimed to determine whether unleading of fuel has made an impact on the blood lead levels of traffic police working in an urban area with high traffic density. Thirty eight percent had detectable levels of lead in blood and 24.4

% of the study samples had blood levels above Centre for Disease Control defined safe limits. Sample mean was 4.82 $\mu\text{g/dL}$ (95% CI 3.58-6.04), and this is a 91% overall reduction when compared to data prior to unleading. Lead poisoning though still present in the high risk traffic warden population shows a considerable reduction following unleading.

Liu et al., (2020) has demonstrated that Metals are widespread pollutants in the environment which have been reported to be associated with kidney dysfunction in many existing epidemiological studies. However, most of the studies are cross-sectional design and mainly focus on several toxic metals including arsenic, lead and cadmium. Therefore, they conducted this prospective study within the Dongfeng-Tongji cohort to evaluate the associations of plasma multiple metals with the decline in kidney function among Chinese middle-aged and elderly. Chronic kidney disease (CKD) has become an important public health issue. It may progress into end-stage renal disease (ESRD) requiring dialysis and transplantation and may increase the risk of

a series of disorders including metabolic and endocrine functions decline, cardiovascular disease, frailty and cognitive impairment. They found that plasma concentrations of aluminum, arsenic, barium, lead, molybdenum, rubidium, strontium, vanadium and zinc were significantly associated with the decline in kidney function measured by annual eGFR decline, rapid renal function. The findings suggest that exposure to multiple metals contribute to the decline in kidney function among the middle-aged and elderly. Co-exposure to multiple metals may have synergetic effect on the kidney function.

Johri et al., (2010) has studied that the heavy metal cadmium (Cd) is known to be a widespread environmental contaminant and a potential toxin that adversely affects human health. The exposure is mainly via the respiratory or gastrointestinal tracts; important non-industrial sources of exposure are cigarette smoke and food (from contaminated soil and water). The kidney is the main organ affected by chronic Cd exposure and toxicity. The preferential uptake by receptor-mediated endocytosis of freely filtered and metallothionein bound Cd (Cd-MT) in the renal proximal tubule take place when Cd accumulates in the kidney. Internalized Cd-MT is degraded in endosomes and lysosomes, releasing free Cd²⁺ into the cytosol, where it can generate reactive oxygen species (ROS) and activate cell death pathways. An early and sensitive manifestation of chronic Cd renal toxicity, which can be useful in individual and population screening, is impaired reabsorption of low molecular weight proteins (LMWP) (also a receptor-mediated process in the proximal tubule) such as retinol binding protein (RBP). This so-called 'tubular proteinuria' is a good index of proximal tubular damage, but it is not usually detected by routine clinical dipstick testing for proteinuria. Continued and heavy Cd exposure can progress to the clinical renal Fanconi syndrome, and ultimately to renal failure. Therefore, environmental Cd exposure can prove to be a significant contributory factor to the development of chronic kidney disease, especially if the person is suffering from a disease such as diabetes or hypertension; therefore, more attention should be paid to limit the exposure of cadmium.

Xu et al., (2016) demonstrated that the Glomerular disease remains the leading cause of ESRD in Asia. Published studies have shown geographical and racial variations in the patterns of glomerular diseases. IgA nephropathy (IgAN) is the most common primary glomerulopathy in Asia, Europe, Australia, and some regions of the United States. However, recent studies, mostly from Europe and Australia, have suggested a temporal change in the pattern of glomerular disease. In these studies, FSGS is increasing in incidence and has emerged as the most common primary glomerulopathy in some countries. The temporal change in the pattern of primary glomerulopathy, especially within comparable ethnic groups in different countries, suggests the presence of unidentified environmental factors that have clinically significant effects on primary glomerular disease. A study conducted in China reveals that China comprises 20% of the world population. With rapid developments in its economy and urbanization, especially during the past decade, air pollution has become a public health problem in some cities. Exposure to air pollution, especially particulate matter of <2.5 µm (PM_{2.5}), has been associated with increased death and incidence of cardiovascular events. Different animal studies have shown that exposure to fine particulate promotes the production of autoantibodies and immune complexes and results in immune

dysregulation, which is implicated in the pathogenesis of some glomerulopathies. These findings led us to hypothesize that long-term exposure to air pollution may cause temporal changes in the profile of glomerulopathy in China. There are several single-center biopsy series have reported the frequency of glomerular disease in China.

Weitekamp et al., (2020) has stated that diesel exhaust is a complex mixture comprised of gases and particulate matter and is a contributor to ambient air pollution. To reduce health risks, recent changes in diesel engine technology have significantly altered the composition of diesel exhaust, primarily by lowering emissions of particulate matter. To gain an understanding of the role of both particle-filtered and whole diesel exhaust on specific health outcomes, they conducted a systematic review in which they examined animal toxicological and controlled human exposure studies that included a comparison between inhalation of particle-filtered and whole diesel exhaust on any health endpoint. There were also several health endpoints for which significant effects were associated with exposure to either particle-filtered or whole diesel exhaust, but not to both. Overall, the results from this systematic review demonstrate that exposure to different components in diesel exhaust can have distinct and independent health effects.

Cadmium, and lead are amongst the most toxic metals. Exposures to one or more of these metals have been documented to be associated with a variety of adverse health effects including nephrotoxicity. Cross-sectional data (N = 25427) from the National Health and Nutrition Examination Survey for 2003–2014 for US adults were used to estimate the impact of co-exposure to high levels of cadmium, lead, and mercury on the unhealthy kidney function. If observed concentrations of cadmium, lead, and total mercury were above the 75th percentile of their respective distributions, the exposure to the corresponding metal was considered to be high. High exposure to lead along with any level of exposure to cadmium and total mercury was found to adversely affect the health of kidney function. High exposure to mercury does not affect unhealthy kidney function (Jain, 2019).

Zaza et al., (2013) studied the exposure to inorganic mercury released into the air from industrial activity. This study was conducted in Europe and highlighted as a possible cause of the adverse renal effects in the area, because of the renal toxicity of this metal at relatively low exposure levels and current health concerns about the release of this substance in Europe. Air dispersion modeling was used to assess ambient mercury levels and to determine exposures. Mercury is well absorbed by the body following inhalation. It is obvious that industries either flow their waste in water or blow them in air through chimneys, these industries release significant quantities of nephrotoxic heavy metals and solvents, exposing the local population to a mixture of toxic substances, the combined effects of which are largely unknown. Low-level exposures to heavy metals and solvents have been shown to have an adverse effect on the kidneys (often evidenced by an increased prevalence of tubular proteinuria/enzymuria. While the kidneys are able to compensate for these preclinical changes, relatively minor alterations in renal function can lead to more major changes, which beyond a certain point will be irreversible and ultimately require medical intervention.

Traffic policemen are the most exposed population to lead (Pb) from automobile exhaust. There has been increasing concern about the possible harmful effects of Pb from automobile exhaust on health of traffic policemen. However, no such study was concerned with the impact of Pb exposure on renal function among them. Therefore, they aimed to study the effect of Pb exposure from automobile exhaust on renal integrity among traffic policemen. Pb levels in blood, urine, hair and nails were determined in the two groups as exposure indices of Pb. The results obtained show that the Pb-exposed group had higher Pb levels in blood, urine, hair and nails than the controls. Traffic policemen are liable to Pb toxicity, and the determination of Pb in blood, hair and nails are good markers of such toxicity. In these exposure conditions, kidney damage is possible. Such damage is both tubular and glomerular in nature and can be documented by determination of the urinary excretion of NAG and albumin (Mortada et al., 2001).

Jagannathan and Patzer in 2017 focused in his research about the most basic reasons of chronic kidney disease (CKD) which is the growing urbanization in almost considerable parts of the globe. The rate of urbanization is increasing day by day in low- and middle-income countries (LMIC) as well. This has effect almost all the people living. The other related diseases such as obesity, hypertension, dyslipidemia, and diabetes etc. According to survey around more than 500 million people have been diagnosed with the CKD out of which more than 85% of the population belongs to the LMIC. The United Nations also recommended in the survey study that the likelihood of the developing urban population will increase from 3.5-5.0 billion globally at the end of 2030. Therefore, the swift and spontaneous increase in the urbanization is ultimately rising the risk of various non-communicable diseases along with the urge of CKD.

The study basically involves the data that has been evaluated from the China Health and National Survey that had investigated the association of urbanization on the function of kidneys or more precisely on renal function (Inoue et al., 2017). The study is therefore related to the population living in urban areas have more decreased function of kidney, which is independent of other factors and cardiometabolic measures. The effect of urbanization on renal function decline was dose-dependent, where mean eGFR (ml/min/1.73m²) declined from 84.8 in the lowest urbanization quartile to 82.8, 80.9, and then 77.8 in the highest urbanization quartile. The main causes of the chronic kidney failure is growing urbanization. The increasing rate of urbanization which is entirely without any considerable reasons and planning resulted into other noticeable problems such as unavailability of food and other essential requirements, medical aid, sanitization system, malnutrition, generic disposition, environmental toxins and intrauterine fetal exposures all plays an essential role in prevalence of chronic kidney diseases.

To conclude the study it is highly recommended and required to alarm the citizens living in developing urban areas to know the current situation of their surrounding environment along with their vulnerability to those harmful conditions which can ultimately lead them to the chronic kidney diseases including degradation in renal function and other non-communicable diseases. In order to make this effort into action. We have must need to held conferences along with the national and international efforts to warn and alarm people about the severity of the causes and the diseases

that will bring with them. Moreover, we need interventions that gives good better understanding of epidemiology along with the strong public awareness that address the unconventional and conventional risks of CKD. Furthermore, we need more surveillance data to enhance more accurate results about the real threat of prevailing CKD.

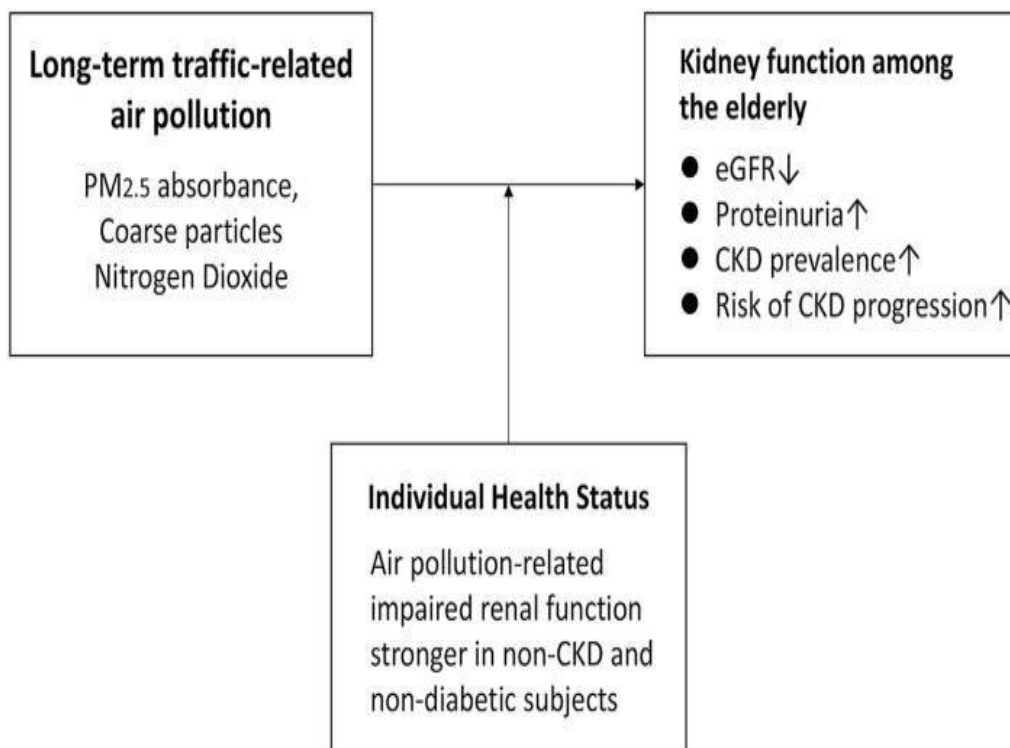


Fig.3 Traffic-related air pollution associated with chronic kidney disease https://ars.els-cdn.com/content/image/1-s2.0-S0269749117306802-41_lrg.jpg

As per the study (ElHafeez et al., 2018) Africa is the second largest continent in the world, with a population of over 1 billion; 961.5 million people live in sub-Saharan Africa and 195 million in Northern Africa. Africa now faces the dual challenge of infectious illnesses and chronic diseases. Unfortunately, CKD problem remains underestimated on the entire continent due to lack of epidemiological information from different African countries. There exists only a single systematic review conducted in sub-Saharan Africa, which concluded that CKD is a prevalent and potentially escalating disease across sub-Saharan Africa, with both communicable and non-communicable risk factors. Strategies aimed at managing CKD epidemics in Africa critically depend on a reliable assessment of the burden of the problem and the establishment of affordable early detection programs. Previous studies reported the prevalence of CKD among the general population or the specific prevalence of this condition in diseases that are recognised as drivers of renal damage (eg, diabetes mellitus). These estimates have varied across studies due to differences in the methods of glomerular filtration rate (GFR) measurement, background risk (general population vs high-

risk groups) or demographic characteristics (eg, age, gender). Age and gender are well-known determinants of the risk of CKD development, progression and complication. While the prevalence of CKD tends to be higher in women, the disease is more severe in men, who also have a higher risk of all-cause and cardiovascular disease (CVD) mortality across different levels of renal function. However, the risk relationships of reduced eGFR and higher albuminuria with mortality were steeper in women than in men.

Shakya et al., (2019) published their research they have mentioned the clear harmful effects of pollution on people among them more precisely the traffic police. As traffic police are the most susceptible individuals that are highly affected by the pollution on daily basis. Six busiest sites were selected in the city, where is the highest-level air pollution exposure. The study was mainly concerned with exposure levels of air pollution and notice the regulations of biomarkers in the blood samples of the participating individuals in the study. Essential results are that all the inflammatory biomarker levels were higher in the summer season than in the spring season, while particulate levels were higher in the spring season than in the summer season. They did not find significant association between 24-hour average PM_{2.5} or black carbon (BC) exposure levels with most of analyzed biomarkers for the traffic volunteers working and residing near busy roads in Kathmandu, Nepal, during 2014. Inflammation and vascular injury marker concentrations were generally higher in females, suggesting the important role of gender in inflammation biomarkers. Because of the small sample size of female subjects, further investigation with a larger sample size is required to confirm the role of gender in inflammation biomarkers.

Traffic pollution is harmful in various aspects such as noise pollution and most importantly in air pollution. Hsu et al., (2019) briefly suggested the notorious effects of air pollution relating to traffic. The small sized particulate matter (PM) of air pollution is most significant exposure in traffic air pollution. The air that is densely polluted with the exhaust gases of the vehicles has various effect on a healthy person. The PM is basically aggravating causing agent of kidney diseases. It is in long run is further related to chronic kidney disease. The fundamental processes related to kidney failure due to the exposure of PM has not been discussed before. However, in their study they

have adequately discussed the procedure of molecular level damage in renal function by apoptosis and autophagy due to the long-term exposures in PM.

The air pollution mainly consists of two types of particulates i.e. particulate matter (PM) and diesel particulate matter (DPM). The particulate matter that is enriched in air pollution is responsible for various harmful diseases such as asthma, lung cancer and cardiovascular diseases. Whereas DPM is held accountable for maximum small sized particulate matter in air pollution of urban areas. The DPM is mainly comprised from fine sized particles to ultrafine sized particles. According to the previous studies the particulate matter which constitutes of most fine particles they would have the more capacity for toxicity. The studies have also shown that the single and repeatedly exposure to the DPM ultimately infuriate the cisplatin-induced acute renal failure (ARF). The complete pathway that has affected the cells of kidney due to the prolonged exposure of PM (Fig. 4). PM increases mitochondrial ROS and reduces mitochondrial membrane potential. Furthermore, PM

induces ER stress and then causes apoptosis and autophagy. PM-induced autophagy may be mediated by the inhibition of the Akt/mTOR signaling pathway. Inhibition of autophagy by Beclin 1 siRNA increases the cytotoxicity, indicating a protective mechanism of autophagy. In addition, autophagy can suppress apoptosis. PM activates the caspase pathway and then induces apoptosis, eventually leading to nephrotoxicity.

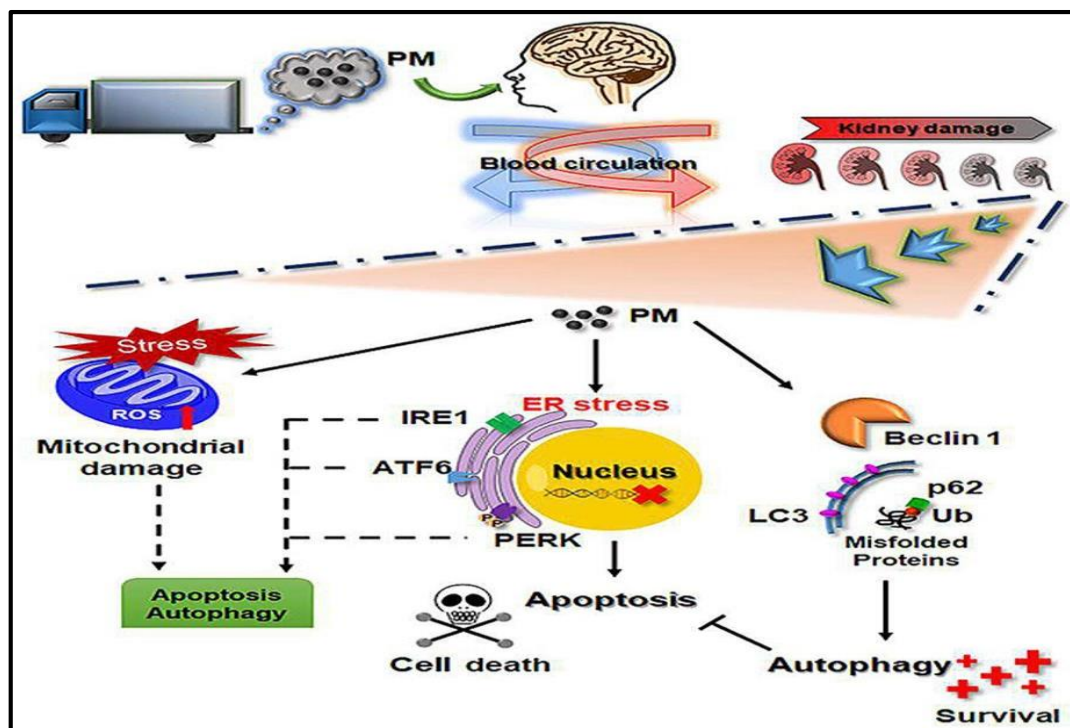


Fig.4 Schematic procedure of PM ultimately affecting the kidney tissues https://ars.elsa-cdn.com/content/image/1-s2.0-S0891584918326340-tx1_lrg.jpg

The increasing air pollution has affected the half of the environment including its inhabitants. All are suffering from severe diseases as the intensity of the pollution is rising from mid of nineteenth century. Afsar et al., (2019) suggested the main and basic numbers of pollutants that has been rising from nineteenth century to up till now. As the world has been growing advance in technology it has also growing in the prevalence of pollution as well. Around the start of mid twentieth century the effects of pollution had been started to feel globally. At the beginning of the effects we have received respiratory diseases and cardiovascular diseases, however, as the time passed by and technology around has been advanced too we came to understand that the effects have been received on every single organ of the body and kidney had not any exception.

Now, there is particular evidence that supports the fact that kidney damage has been done by the air pollution. Baris Afsar with his colleagues suggested the brief and summarized epidemiology,

disease states and mechanisms of air pollution and kidney damage. The adverse effects of air pollution include various factors. Firstly, smog was detected in London in 1952. The smog is basically a mixture of fog and smoke emitted by the heavy traffic. After this, people started to notice the ambient effects of air pollution. Therefore, it was concluded that the air pollution is predominantly consist of gaseous particles (solid and liquid components) which are then inhale by the individuals. These gaseous particles hence comprised of different chemical components that obviously varies from place to place and city to city depending on the pollution in surrounding environment.

The particulate matter (PM) is mainly formed due to the combustion of gasoline and fuel which are the typical abundant waste product of industrial areas. The chimneys emitting smoke form the furnaces that has been operating enormously in industries had adversely affected our environment specially the air that we breathe. The major sources that has been affecting our environment is given in table 2. As every pollutant has an origin, from where the journey had started and how it gets harmful over the time and it is easy to understand the mechanism of their growth and how it gets harmful on exposure. In the past decade, a growing body of research has suggested a causal relationship between ambient air pollution exposure and adverse cardiovascular health.

Table 2 . The table of main pollutants and their source.(Afsar et al., 2008)

Pollutant	Source
PM (Particulate Matter)	Mostly traffic-related air pollution (mainly local emissions). Other sources include domestic heating, industries, etc.
NO _x (Nitrogen Oxides)	Mainly derived from road traffic and the industrial burning of fuels. Strongly related to diesel motor vehicles.
SO _x (Sulphur Oxides)	Industrial production of sulphur-based products.
O ₃ (Ozone)	Industrial combustion and processes.
CO (Carbon Monoxide)	Road traffic and industrial fuel burning.
Cadmium	Diet in non-smokers. Tobacco in smokers.
Lead (Pb)	Gasoline, batteries, pipes and ammunition. In the past, paints and ceramic glazes. Occupational exposure to the inorganic form of Pb (Pb ²⁺), including welding, manufacture of Pb-containing batteries, Pb melting and refining, and production of pottery. Children ingesting Pb ²⁺ -contaminated soil.
Mercury	Occupational, environmental and dietary sources. Mainly through ingestion of food contaminated with CH ₃ Hg ⁺ .
Arsenic	Primary route of human exposure is ingestion of contaminated drinking water. Methylated forms of As (MAs and DMAs) from pesticides used in cotton crops.
Uranium	Mining and milling.

Heavy metals are among the best-known environmental pollutants (Fig. 5) causing kidney disease. It is less well known that they are also air pollutants, particularly Cd, Pb and Hg, but also arsenic (As) and uranium (U). Every pollutant has its own diverse effects to health-related diseases. Each one of the above-mentioned pollutants badly affects the kidney on their prolong exposure and causes chronic kidney disease (CKD).

Arsenic (As) and Chronic Kidney Disease (CKD)

The intake of arsenic via any source such as in drinking water. The contaminated water that has been used for drinking purposes does have fair amount of arsenic in it. It is quit held accountable for the damage of renal tissues and development of hypertension. The patients in Taiwan have shown the relatable coincidence between the arsenic of urinary and incidence chronic kidney disease. Therefore, it has been deduced from the results that the increasing amount of arsenic can increase the chances chronic kidney diseases by four times. Acute As-induced renal intoxication may lead to tubulointerstitial nephritis and acute tubular necrosis manifested by hypercalciuria, albuminuria, nephrocalcinosis and necrosis of the renal papillae.

Cadmium and Chronic Kidney Disease (CKD)

The source of cadmium is the most common source of all the environmental pollutants. The persons to its exposure are basically divided into two main groups i.e. smokers and non-smokers. The smoker's intake of cadmium is through tobacco in cigarettes and other tobacco related products, whereas, the non-smokers exposure for this pollutant is from their diet and the level of cadmium increases from children going school to teenagers as they were exposed to more air pollution rich in cadmium. As the age increase the concentration of cadmium also increases but on the other hand the concentration of cadmium is at its peak in teenager smokers as they used tobacco related products which have greater levels of cadmium in it. The cadmium is basically known as nephrotoxic and it is highly capable of inducing severe damage to renal tubule (polyuria, generalized tubular dysfunction, i.e. Fanconi syndrome) and progressive loss of glomerular filtration rate (GFR). Long-term Cd exposure is thought to accelerate the CKD-related decline in GFR. Cross-sectional studies have shown that Cd exposure is a risk factor for the development of CKD. The most frequent long-term consequence of Cd exposure is proteinuria, and it may precede a slowly progressive and irreversible renal tubular dysfunction. The incidence of kidney stones also increases in individuals exposed chronically to or to larger Cd dose, possibly due to the increased concentration of calcium in tubular fluid and urine. Last but not least, Cd exposure has been associated with the severity of diabetes and hypertension, which themselves are risk factors for CKD.

Lead and Chronic Kidney Disease (CKD)

The main source of lead exposure is both kidney and the bone. As the lead in the common excretion of kidneys and hence its accumulation is due to the induction of mitochondrial oxidation stress and inflammation. The glomerular hypertrophy is caused by the minimum level of lead exposure which

may have started in growing age whereas the act level exposure of lead may cause proximal tubular dysfunction and the severe and maximum exposure of lead may leads to progressive tubulointerstitial nephritis.

Uranium and Chronic Kidney Disease (CKD)

Approximately 40% of plasma uranium is complexed to transferrin and the remaining 60% is complexed with carbonate or bicarbonate, which are filtered by the glomerulus. In proximal tubules. Uranium toxicity, although relatively rare, may also cause renal damage and indeed, the kidney is the primary target for uranium toxicity following inhalation or ingestion. The complexed uranium dissociates with decreasing pH, releasing the reactive uranyl ion, which can interact with the proximal tubule membrane.

Mercury and Chronic Kidney Disease (CKD)

All forms of Hg are nephrotoxic through various mechanisms. The pars recta of the proximal tubule appear to be most sensitive to the Hg toxicity. Hg has been associated with CKD progression. Acute exposure causes altered mitochondrial structure, endoplasmic reticulum dilation and nuclear pyknosis. After 12 h, microvilli are lost, and cell death is associated with plasma membrane rupture and cell detachment from the basement membrane. Chronic exposure to mercuric compounds can lead to glomerular injury. Hg is well absorbed following inhalation, and air levels correlate with exposure estimated by urinary Hg excretion.

The ambient air pollution also has adverse effects on human health. The persons subjecting to the exposure of this pollution for a long time are diagnosed with renal injuries which can ultimately leads to the chronic kidney diseases. Wang et al., 2020 suggested the cross-sectional studies on hospital patients that have been subjected to the exposure of ambient air pollution in one way or another. The main objective of their study is to understand that the patients that have been living in the hospitals of urban areas, having dense air pollution in Shanghai, China. They aimed to study and determine that these patients having long-term exposure in ambient air pollution has directly affected their glomerular filtration rate and its extent of damage to the kidneys.

The chronic kidney disease is the most spreading disease globally. As the source of this disease is the pollution of air that surrounds all of us. The pollutants in it did not miss any chance for our internal damage to health. In 2012, according to the study 10.8% people were diagnosed with sever chronic kidney disease and the percentage of the patients hence rise with the passage of time and also along with increase in the particulate matter of particle having 2.5 microns in size along with other chemicals in the air that we breather and get exposure to. After this the prevalence of this disease then followed its path to Norway and United states etc.

One-year PM₁₀ exposure was associated with lower eGFR; each standard deviation (SD) increase in PM₁₀ was significantly associated with the increased prevalence of CKD. However, the alliance of increased in the rate of exposure of PM₁₀ in one standard deviation, along with the spread of

chronic kidney disease and the potentially modified glomerular filtration rate (eGFR) is summarized in the table (Fig. 6) along with the variables of age sex gender and others applied to every individual that has been diagnosed. Therefore, according to the research and the methods that have been efficiently adopted supported that exposure to ambient PM10 in ambient air pollution has increased the chance of having Chronic Kidney Disease in individuals and had negatively affected the renal function of the kidneys among Chinese adults.

As the considerable amount of persons have been diagnosed with chronic kidney disease since the last decade and amount of the patients have been drastically increasing as they were came into the exposure of dense air pollution having the most common particulate matter of around 2.5 microns size (PM 2.5). Therefore, Bragg-Gresham et al., 2018 published their study regarding the spread of chronic kidney disease (CKD) in United States as they have got a lot more population surviving in the dense traffic and country level pollution. Hence, there is a wide range of variation in the spread of chronic kidney disease across all over the United States. However, the variation also lies in patients as well and they can be explained by differences in patient level risk factors, substantial variability still exists. They then hypothesize the reason behind this that probably because of the understudied environmental exposures such as air pollution.

They preferably used the particular method for their study. They have used the method of cross-sectional study which is entirely anonymous, and the data is collected from 1.1 million persons that are greater than and equivalent to the age of sixty-five years old. All these individuals were enrolled in a program of US Medicare in 2010 (5% Medicare sample). Further, included that the persons should be involved in this Medicare program for whole year in two parts that are A and B. Also, they would not have and coverage of health management organizations (HMO). CKD was defined using a large set of ICD-9-CM diagnosis codes indicating CKD, which are identical to the codes utilized by the United States Renal Data System.

Adjusted Prevalence Ratio

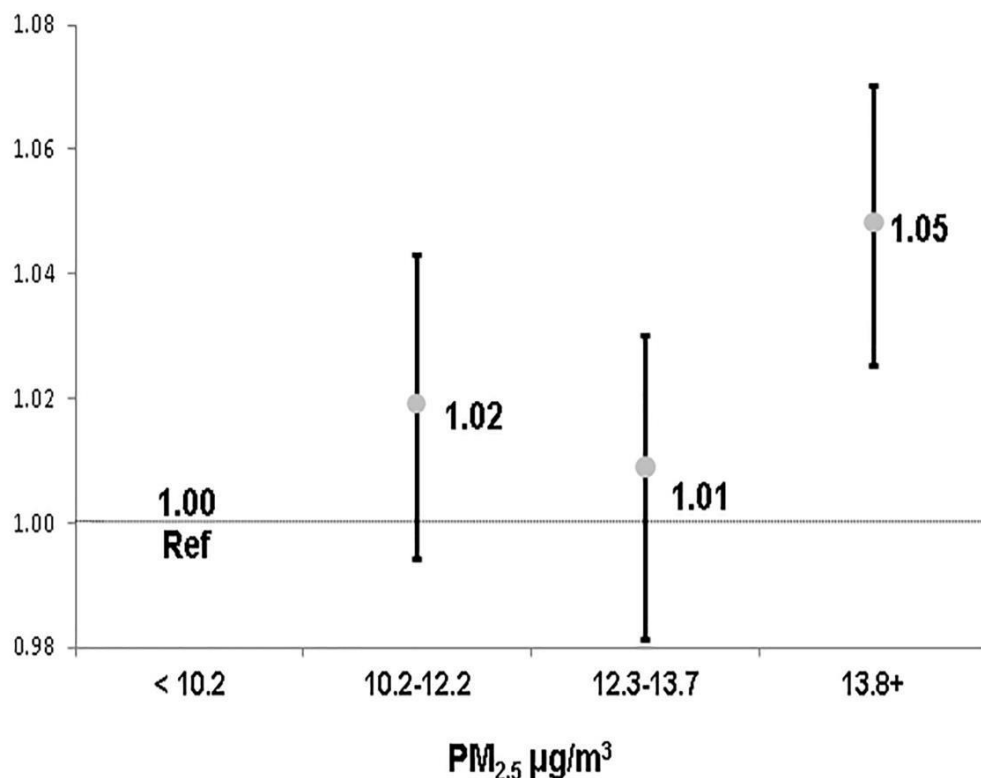


Figure.5 Graph between county level PM_{2.5} and the prevalence of chronic kidney disease

<https://journals.plos.org/plosone/article/figure/image?size=large&id=info:doi/10.1371/journal.pone.0200612.g003>

So, in order to conclude their work, that involved the cross-sectional study of a very large population enrolled in Medicare program and were 65 years or more aged people in United States. They have been proving positive association, that was critically analyzed and observed between county-level PM_{2.5} concentration and diagnosed Chronic Kidney Disease. The reliance on CKD diagnostic codes likely identified associations with the most severe CKD cases. These results can be strengthened by exploring laboratory-based diagnosis of CKD, individual measures of exposure to multiple pollutants, and more control of confounding.

This study was also limited to the use of administrative healthcare claims data for identification of CKD. It is likely that individuals with early stages of CKD do not have a diagnosis and are therefore classified as non-cases. We chose to use the list of ICD-9 codes utilized by the United States Renal Data System, which includes all diagnoses of CKD, because although some diagnoses, such as

posterior urethral valves or pyelonephritis, are not likely associated with air pollution, we cannot exclude this possibility based on our study. Also, a systematic review of coding for CKD and related conditions has shown the sensitivity of using only diagnostic codes to be low, typically under 50%. Moreover, these conditions would be extremely rare in the population under study. The reliance on claims data also excluded an examination of these associations by stage of CKD. Future work would benefit by focusing on cohorts that include laboratory data for use in classifying individuals into appropriate CKD categories. The authors also acknowledge that there may be air pollution data quality limitations and refer the reader to the CDC Wonder website for details.

Although this study included over one million individuals, the cross-sectional design and lack of individual exposure data severely limit causal inference. It does, however, support further research in this area, using more detailed air pollution exposure data mapped to the patient- or zip-code level rather than the cruder averaged, county-level estimates utilized in this study. If this association is borne out by future studies, it would have clinical and public health implications for reducing air pollution exposure for those with CKD and also for those at risk for the condition. The potential public health significance of this finding is even greater for regions and countries with much higher levels of air pollution than the United States.

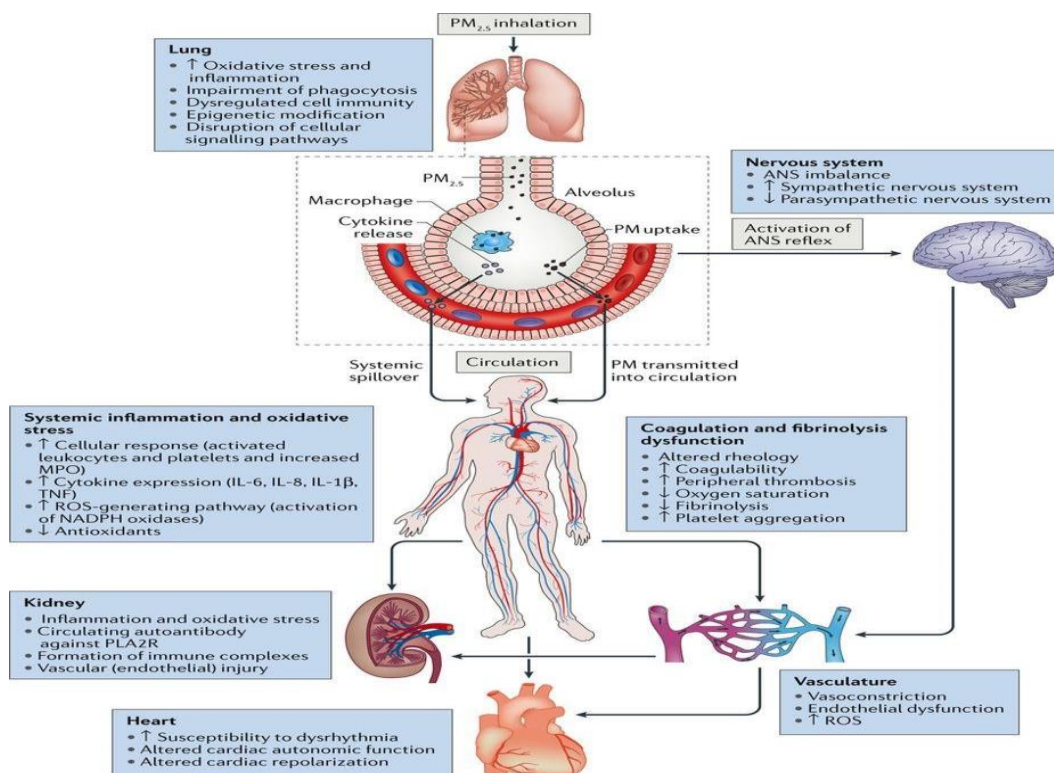


Fig.6 Potential mechanisms linking inhaled particles to pulmonary, cardiovascular, cerebral and kidney diseases.

<https://pbs.twimg.com/media/D-OghsKX4AIQvv0?format=jpg&name=small>

The effects of ambient air pollution have a negative and sparse effect on many diseases, such as cardiovascular and respiratory diseases. Kim et al., (2018) suggested in their paper that they have generally reported a relationship between air pollution having many particulate matters of different sizes which has different harmful effects and renal function, but the results of their research were limited to exposure to particulate matter (PM). Recent studies have focused on understanding the relationship between renal function and air pollution because a rapid decline in renal function or chronic kidney disease (CKD) is directly related to cardiovascular events and the cardiovascular mortality among peoples that were subjected to long term exposures of air pollution. Several researchers have hypothesized that exposure to air pollution in the form of particulate matter (PM) influences renal function via mechanisms similar to those proposed for Cardio-Vascular Diseases, such as inflammation or oxidative stress, provided that the cause of renal dysfunction is a severe risk factor for Cardio-Vascular Diseases. This study was generally proposed to recognize the associations between various air pollutants and renal function among the adults, living in Korea. Although, there are several other studies that shows the harmful effects on the function of renal indicators including failure in glomerular filtrate rate and chronic kidney disease. The associations between ambient air pollution and CKD were determined by multiple logistic regression analyses, estimating the odds ratios (ORs) and 95% CIs of each air pollutant for CKD. Previous epidemiological studies have identified significant associations between exposure to air pollutants and renal function.

Kataria et al., (2015) stated the adverse effects of environmental contaminants on renal function. According to him Melamine is an organic nitrogenous compound used in the industrial production of plastics, dyes, fertilizers and fabrics that was considered safe on the basis of standard animal studies. This molecule was deliberately added to diluted raw milk at milk-collecting stations in China, with the presumed aim of falsely elevating assay results for protein content. Melamine was later detected in numerous food and milk-containing products that were exported from China to many countries worldwide. More than 300,000 infants and children exposed to milk-based formulas contaminated with melamine developed radiolucent stones, as well as impaired renal function and renal growth. The long-term consequences of melamine exposure in infancy and early childhood remain unknown, but exposure to melamine during adulthood might increase the risk of urolithiasis. The melamine story, therefore, provides a striking cautionary note regarding the potential serious adverse consequences of exposure to environmental organic chemicals during normal consumer activity. Their study revealed that clinical effects of organic compounds on measures of cardiorenal function, including changes in blood pressure, albuminuria, glomerular filtration rate (GFR), and uric acid concentration.

In 2013, Lue et al., (2013) investigated the association of eGFR level with traffic-related air pollution exposure using residential proximity to major roadways in 1103 patients hospitalized for acute ischemic stroke. This showed significantly lower eGFR levels in patients living within 50 m of the nearest major roadway than in those living at least 1000 m away from the nearest major

roadway. More recent studies reported a relationship between ambient PM exposure and renal function. A longitudinal study in 669 older American men found that chronic exposure to ambient PM_{2.5} negatively influenced their renal function and was also associated with an increase in reduction in age-related eGFR level.

In addition, Yang et al., (2016) evaluated the association between renal function and PM including PM₁₀, coarse particles (PM Coarse), PM_{2.5} and PM_{2.5}Absorbance among Taiwanese adults, reporting significant associations of PM₁₀ and PM Coarse with renal function indicators including glomerular filtration rate (eGFR) and Chronic Kidney Disease. These previous findings regarding PM exposure and eGFR were in line with our results, although we did not evaluate PM_{2.5} due to absence of data. On the contrary, urinary albumin excretion, one of the indicators of renal function, showed no significant association with long-term exposure to PM_{2.5} or PM₁₀ in the longitudinal analysis of the Multi-Ethnic Study of Atherosclerosis. No significant association between PM₁₀ concentration and Chronic Kidney Disease was observed in their study. The discrepancy in these results may be explained by differences in ethnicity, the indicators of renal function, the prevalence of Chronic Kidney Disease, the study region, the exposure assessment method, and covariates. They conclude from their findings that the exposures to ambient air pollution and pollutants in which more specifically among them their exposure to PM₁₀ and NO₂ were significantly associated with decreases in glomerular filtration rate (eGFR) levels, but not Chronic Kidney Diseases, diagnosed in the adults of Korea. Their study thoroughly investigated the relationship between the air pollutants PM₁₀, NO₂, SO₂, and CO and the kidney function indicators i.e. glomerular filtration rate (eGFR) and chronic kidney disease (CKD) in Korean adults. This clearly demonstrated that exposure to PM₁₀ and NO₂ in ambient air pollution were significantly associated with decreased glomerular filtration rate (eGFR) levels, but not with CKD. Their estimated findings provide evidence that exposure to the air pollutants PM₁₀ and NO₂ in ambient air pollution is closely associated with decreased glomerular filtration rate (eGFR) level before the onset or diagnose of renal disease.

The article about the relation between land contaminated and renal function written by Jarup, 2013 describe the association between them. Land contaminated by current or historical industrial activity is common in many countries, and the influence of soil pollutants on the environment often causes concern. It has been recognized that not only may the quality of the environment suffer as a consequence of the pollution, but there may also be specific problems for human health associated with contaminated land, as well as a consequent economic burden. During recent decades it has become increasingly obvious that the kidney is adversely affected by an array of chemicals. Human exposure to (nephrotoxic) chemicals may result from industrial emissions and other environmental pollution, from several naturally occurring substances as well as from medicines. Some of these chemicals may cause acute injury to the kidneys, whereas others may result in chronic renal damage and eventually end stage renal failure. For example, a recent Swedish study showed excess risk of end stage renal disease (ESRD) in a population living in the vicinity of cadmium emitting battery plants.

Renal tubular dysfunction may progress to glomerular dysfunction and ESRD, which is a major public health problem due to the long period exposures to air pollution, causing both suffering to the affected individuals and a severe burden on health services providing dialysis and kidney transplantation. Data on human health effects associated with exposure to most renal toxicants which is more densely lies in the air pollution, are sparse. Considering the potentially large effects on public health, there is a significant need for further research about this, in particular on low level exposure to persistent chemicals and the potential progress of tubular dysfunction to more severe kidney disease, to warn and enlightened the people so that there will be more precautions and less patient in coming future. There is also a need to investigate effects associated with combined exposure to two or more renal toxicants, a research area that is virtually unexplored.

Burrows et al., (2018) conducted a study in US and evaluated that Chronic kidney disease (CKD) has emerged as one of the major public health problems facing the US population, with a high burden of disability, disproportionate distribution, poor outcomes, and high costs. CKD collectively represents chronic kidney damage or loss of kidney function from early stages, characterized by elevated albumin excretion in the urine, to kidney failure requiring dialysis or kidney transplantation for survival. They adopted a method for comparing changes in prevalence and treatment of CKD, diabetes, and hypertension, and changes in CKD awareness from the initial to the follow-up screening among participants who completed both screenings, then they calculated the standardized difference for each variable. They observed an important benefit of screening which is that diagnosing CKD at an earlier disease stage may lead to slower disease progression and a reduction of morbidity and mortality over time. This means that the CKD is time dependent if one catches it earlier then survival is possible. Management of CKD to reduce or slow the progression of disease can reduce the incidence of kidney failure. The results were very obvious, that if the individual is already suffering from a disease, then progression towards it is easier. Compared to adults without diabetes or hypertension, individuals with these risk factors progress more quickly through the stages of CKD to kidney failure. Burrows and his colleagues were successful in finding that the kidney disease is related to the onset and its diagnosis, including the factor if one suffered from any kind of disease earlier.

Conclusion

Present study shows that pollution can adversely effect an individual. It can lead to chronic diseases and renal dysfunction is one of them. Traffic wardens are most susceptible to the risky effects of pollution. To stand in static position for long hour and managing heavy traffic increase mental and physical stress among them. The major metals affecting the renal function of the traffic wardens are cadmium, arsenic, lead, mercury and chromium. These can have lethal effect on the body of an organism. Hyperuricemia, hypercalciuria, albuminuria, nephrocalcinosis, and diabetes are some of renal diseases mainly caused by the Pollution. The major effect is on the glomerular filtration rate of the kidney (GFR). Hence concluded that Pollution is mainly responsible for the renal deformities and other associated diseases.

Recommendations

- More detailed studies and experimental designs are needed to demonstrate combined relationships between air pollutants and kidney damage as well.
- Further steps should be taken as serious regarding the unfavorable results of Pollution.
- People must create awareness regarding the rising issues of pollution and renal diseases in number of different ways.

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