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Urban city transportation mode and respiratory health effect of air pollution: a cross-sectional study among transit and non-transit workers in Nigeria

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ABSTRACT

Objectives: To assess the respiratory health effect of city ambient air pollutants on transit and non-transit workers and compare such effects by transportation mode, occupational exposure and sociodemographic characteristics of participants.

Design: Cross-sectional, randomised survey.

Setting: A two primary healthcare centre survey in 2009/2010 in Uyo metropolis, South-South Nigeria.

Participants: Of the 245 male participants recruited, 168 (50 taxi drivers, 60 motorcyclists and 58 civil servants) met the inclusion criteria. These include age 18–35 years, a male transit worker or civil servant who had worked within Uyo metropolis for at least a year prior to the study, and had no history of respiratory disorders/impairment or any other debilitating illness.

Main outcome measure: The adjusted ORs for respiratory function impairment (force vital capacity (FVC) and/or FEV_1<80% predicted or FEV_1/FVC<70% predicted) using Global Initiative for Chronic Obstructive Lung Diseases (GOLD) and National Institute for Health and Clinical Excellence (NICE) criteria were calculated. In order to investigate specific occupation-dependent respiratory function impairment, a comparison was made between the ORs for respiratory impairment in the three occupations. Adjustments were made for some demographic variables such as age, BMI, area of residence, etc.

Results: Exposure to ambient air pollution by occupation and transportation mode was independently associated with respiratory functions impairment and incident respiratory symptoms among participants. Motorcyclists had the highest effect, with adjusted OR 3.10, 95% CI 0.402 to 16.207 for FVC<80% predicted and OR 1.71, 95% CI 0.61 to 4.76 for FEV_1/FVC<70% predicted using GOLD and NICE criteria. In addition, uneducated, currently smoking transit workers who had worked for more than 1 year, with three trips per day and more than 1 h transit time per trip were significantly associated with higher odds for respiratory function impairment at p<0.001, respectively.

Conclusions: Findings of this study lend weights to the existing literature on the adverse respiratory health effect of ambient air pollution on city transit workers globally. The role of other confounders acting synergistically to cause a more deleterious effect is obvious. In all, the effect depends on the mode and duration of exposure.

ARTICLE SUMMARY

Article focus

- Intracity transporters and commuters: Are they facing any adverse respiratory health effect due to outdoor ambient air pollution?
- Does this effect vary according to the mode of transportation and sociodemographic characteristics of the exposed and unexposed?
- Can changes in life style, environmental policy and other confounders modify this effect?

Key messages

- Air pollution is a major threat to human life and most people receive their daily air pollution dose while commuting to work irrespective of the mode of transportation.
- Adverse respiratory health effects of ambient air pollution is dose and exposure time dependent; however, multiple factors exposure can produce a more deleterious effect irrespective of the dose and exposure time.
- Improved air quality could serve as a single health promotion strategy that could be beneficial to all, since everybody commute and breathe air and air pollution is ubiquitous and widespread.

Strengths and limitations of this study

- The strength and precision of this study came from the fact that both the quantitative and qualitative variables were considered in assessing the adverse respiratory health effect of ambient air pollution on exposed participants.
- Also, the control and the case groups existed in the same environment, sharing similar environmental, cultural and ethnic factors effect.
- However, the study was limited by few confounding factors such as the effect of other psychosocial factors peculiar to their respective work environments.

INTRODUCTION

Outdoor ambient air pollution is a major threat to human health in most West African big cities including Nigeria and other parts of the world.[1–7] It reduces the life expectancy of people who are constantly exposed to it.[8]

According to the global estimate made by the United Nations Environment Programme, 1.1 billion people breathe unhealthy air.[9] This increases daily deaths and hospital admissions throughout the world,[10] because of its wide range of effects on human health, especially the cardiopulmonary system.[11–13] It is also estimated that, urban air pollution is responsible for approximately 800 000 deaths and 4.6 million loss of lives each year around the globe.[5] Global daily death from diseases related to air pollution is put at 8000 and a yearly death toll due to air pollution about 2.4 million.[14]

In Nigeria, various studies have indicated a high level of ambient air pollution in most urban cities especially the Niger Delta region, of which the Uyo metropolis, the capital city of Akwa Ibom State, is an integral part.[8 15 16] A typical air quality assessment of this region showed that the levels of volatile oxides of carbon, nitrogen, sulphur and total particulate matter exceed the existing Federal Agency Standards.[16] Common sources of air pollution in this area include: bush burning, automobile emissions, generators emission, pipeline explosion, industrial emissions and gas flaring.[17]

Many recent studies have shown that, people are more at risk of occupational exposure to ambient air pollutants than other means,[2 3 6 18–21] and evidences have also shown that urban city transit operation (eg, commercial motorcycling and taxi driving) are examples of such occupations that expose workers to unusual large amount of outdoor ambient air pollutants.[22] The work entails conveying passengers to and from their different destinations within and even to the outskirts of the city for commercial gains. This type of duty may take them from a less busy to a more congested and industrialised parts of the city. The city of Uyo in recent years has witnessed a tremendous infrastructural upgrading. These result in a massive and rapid urbanisation with an increased number of people and concomitant increase in demands for transportation.[23] In addition, increase in heavy-duty tractors, vehicles and motor cycles follows the growing trend.[23] According to statistics from the city transport authority, there is an average of 150 vehicles and 180 motor cycles to every kilometre within the metropolis. The situation is made worse as a great number of these vehicles are old and poorly maintained,[24] and worse still in an environment with ineffective or no transport regulating laws.[18] The scenario creates a high level of traffic-related ambient air pollutants, which have been shown to constitute up to 90–95% of ambient carbon monoxide (CO) level, 80–90% of nitrogen dioxide (NO_2), sulphur dioxide (SO_2), hydrocarbon and particulate matter in similar studies in other developing countries of sub-Saharan Africa and other parts of the world.[25]

The commercial motorcyclists (CMCs) and taxi drivers (TDs) during the course of their duties inhaled these harmful particles, mist, dust, black smoke, volatile organic compounds especially when passing through highly air-polluted corridors such as traffic hold-ups, checkpoints and industrialised areas of the city. Research has shown that, most people receive a significant proportion of their daily air pollution dose while commuting to work, through walking, cycling, travelling by car or public transport.[26] For those commuting by motorbike, or walking, the high levels of physical activity could lead to an increased breathing rate with a resultant increase in the absorption of these pollutants by the body.[26] For those driving or travelling by car, a high rate of in-vehicle exposure is likely due to emissions from vehicles in front and behind and a limited space for exchange and diffusion of in-vehicle air.

Empirical evidences have shown that these inhaled substances have strong pulmonary and systemic inflammatory potential and can cause irritation and allergy in the lungs and air passage of individuals who are exposed to them for a long time.[27 28] However, the type of disease developed may depend on the size of the particles or what is inhaled and where it ends up in the airway or lungs. In some cases, larger particles tend to end up trapped in the nose or larger airways.[29] Small particles on the order of 10 micrometers (PM_{10}) or less ($PM_{2.5}$ and UFP_s) can penetrate the deepest part of the lungs such as bronchioles or alveoli.[30] Sometimes, they get dissolved and absorbed into the blood stream,[31] eliciting greater biological effects. The composition, concentration and associated toxicity of specific ambient air pollutants as well as the duration and frequency of exposure will determine the adverse health effects and the clinical respiratory manifestations.[32] For example, ambient air pollutants with predominant particulate matter, ozone (O_3) and NO_2 have been shown to exacerbate airway oxidative stress,[33] bronchial reactivity,[34] respiratory viral infection[35] and reduced airway ciliary activity.[36] Also, particulate matters can facilitate the development of lung cancer and increase mortality.[37]

The spectrum and severity of adverse respiratory health effects of the inhaled pollutants may vary from subclinical effects to premature mortality,[38] depending on the degree of exposure by various occupations, environmental factors, sociodemographics and population sensitivities.[38] Lung diseases following occupational exposure among Nigerians have been extensively studied.[39] These include cement workers,[40] stone cutters,[41] tobacco industry workers,[42] street sweepers[3] and coal miners.[43] However, there exists paucity of information about the respiratory health effect of ambient air pollutants on city transit workers despite their high rate of exposure.[44] This research work was therefore, aimed to assess and compare the respiratory health effects of ambient air pollutants on transit (CMCs and TDs) and non-transit workers (civil servants (CSs)) in the Uyo metropolis, South-South Nigeria which hitherto has not been documented. The choice of CSs as occupationally unexposed group was based on the fact that, an ideal civil service routine is not associated with exposure to the traffic-related ambient air pollutants encountered by transit workers. We hope that the outcome of this study will help unfold the required interventions necessary to reduce incidence and hence complications associated with exposure to city air pollutants among transit workers in our city and similar cities all over the world.

METHODS

This was a cross-sectional study with 245 participants recruited in two primary healthcare promotion programmes, which took place between September 2009 and March 2010 at two designated centres within the Uyo metropolis, Nigeria.

Of this number, 168 (68.6%) met the inclusion criteria, which include a male subject, age 18–35 years, who has worked either as a CMC, TD or CS for at least a year prior to the study. Exclusion criteria include past or present history of respiratory diseases (asthma, tuberculosis and other chronic obstructive pulmonary diseases), cardiovascular diseases, anaemia, congenital anomalis, history of haemoptysis, drug history (such as antituberculosis and ant-asthmatics), declined participation and improper completion of the questionnaire. Women were also excluded because, in Nigeria, transit operation is regarded as a very stressful job and is dominated by men. Thus, in our study population, there was a complete absence of female transit workers, hence their exclusion from the study. Informed consent was obtained from all the participants and the University of Uyo Research and Ethics Committee approved the study protocol. Rules (Revised Helsinki Declaration, 2008) governing the conduct of human research in Nigeria and as accepted internationally were duly observed.

Of those eligible to participate, 50 (29.8%) were TDs, 60 (35.7%) were CMCs and 58 (34.5%) were CSs. Instruments of surveys were: pretested semistructured questionnaire, anthropometric indices, spirometric indices and air quality measures.

The questionnaire consisted of 26 items selected from the Compendium of Respiratory Standard Questionnaire for adults, which was chosen because of its high internal consistency and reliability.[45] The questionnaire was divided into three sections: A, B and C.

Section A gathered information regarding the participant's sociodemographic data such as age, educational level, ethnicity, area and nature of residence (number of rooms occupied and ventilation available for each room), exposure to second-hand smoke and exposure to biomass (eg, cooking fuel used and availability of a separate cooking apartment). Participants' medical history such as atopy, asthma, drug history, respiratory or any other diseases were also provided.

Section B contained questions regarding the participant's life style habits such as smoking status, alcohol intake, nutritional habits and baseline physical activity status.

Section C gathered information regarding the participants' work characteristics and environment such as duration and nature of job, mode of transport, transit time (in hours) per day, number of trips per day and exposure to work-related indoor air pollutants. The questionnaire was formulated to provide information covering 12 months prior to the study period.

To assess participants smoking habits, they were asked whether they had formerly smoked, currently smoke or never smoked. Former smokers were asked whether they had quit less than a year prior to the study period. Those who said they currently smoke at the day of the study were defined as current smokers. Thus, they were classified into three groups namely: never smokers, current smokers and ex-smokers. Current smokers were asked the number of cigarettes they smoke per day (<½ pack, ½ pack, 1 pack, 1½ packs, ≥2 packs). Former smokers were also asked, 'How many cigarettes they used to smoke per day (<½ pack, ½ pack, 1 pack, and ≥2 packs).

The classification of participants based on their smoking habits was because empirical evidences have shown that smoking is a strong maturely inclusive factor in the aetiology of most chronic respiratory disorders.[46] It has been shown to contribute as much as 73% of the causes of chronic respiratory disorders in developed countries and about 40% in developing countries.[46] A recent study has shown that the population attributable fraction for smoking as a cause of chronic obstructive pulmonary disorders (COPDs) ranged from 9.7 to 97.9%.[47]

In addition, there exist evidences of interactions between smoking and work place exposure in the aetiology of COPDs.[48] Literature documents a higher rate of smoking among blue-collar workers, and evidence-based studies have also shown that stressful occupations such as transit operation are associated with a higher rate of cigarette smoking than the white-collar or professional occupations.[49 50] Therefore, smoking is a strong confounding factor to air pollutants in the development of respiratory function impairment.

To assess the participants' baseline physical activity status, questions were asked based on the 2010 United State Healthy People Physical Activity Guideline Standards,[51] which recommends 150 min of moderate to severe intensity aerobic physical activity per week in bouts of 10 min or more for adults ages between 18 and 64 years. Based on this, the participants were classified into two groups: physically active or inactive.

Based on their alcohol intake, the participants were classified into two groups: current/regular drinkers and non-drinkers. Current/regular drinkers were defined as those who drink for ≥10 days in a month and even on the day of the survey. Dietary habits was assessed by assessing the intake of various macronutrients (fat, carbohydrate, vegetable, snacks, sweet drinks and portion size), for a period of 1 year. High to very high intake of macronutrients, full portion size consumption, high frequencies of fast food/restaurant patronage (>4–5 times per week) were regarded as poor dietary habit, whereas balance diet taken 2–3 times a day with enough vegetable and fruits in between was regarded as good dietary habits.

To assess the participants exposure to indoor air pollutants, questions were asked on marital status, number of rooms occupied, family members per room, specific number of windows per room, availability of a separate cooking apartment, cooking fuel used regularly (firewood, coal, kerosene, stove and gas cooker), regular use of mosquito coils, the presence of animals/pets in the house, the nature of carpet used if any. Individuals exposed to any of these indoor sources of air pollutants were classified as exposed, otherwise unexposed.

Measures

Three basic measurements were performed in this study. These include anthropometric, spirometric and air quality indices measures.

The anthropometric indices measured were weight in (kg) and height in (m) using standard protocols as approved by the WHO.[52] Body mass index was calculated as weight (kg)/height (m^2). Body mass index of ≤24.9 was regarded as normal, 25–29.9 overweight and ≥30 obese.

The pulmonary function assessment was performed by spirometric measures of lung function capacities specifically, force vital capacity (FVC) defined as the volume of air in litres that can forcefully and maximally exhaled. Force expiratory volume in 1 s (FEV_1), defined as the volume of air (in litres) that can be forcefully exhaled in 1 s. Ratios of FEV_1 to FVC (FEV_1/FVC) were calculated. The measurements were performed according to the methods suggested by the American Thoracic Society[53] using a dry vitalograph spirometer (Vitalograph Ltd. Buckingham, England).

Each subject performed at least three forced expiratory manoeuvres while sitting with free mobility and nose closed with a nose clip to prevent the passage of air through the nose to ensure reproducibility of results. The average of the three was taken as the actual value. The data were compared with predicted values based on age, sex, height and ethnic group, using Crapo *et al*[54] predicted equation for non-smoking male adults.

Reduced values of FVC, FEV_1 and FEV_1/FVC were the primary criteria used for diagnosing respiratory function impairment (restrictive or obstructive or both) based on the Global Initiative for Chronic Obstructive Lung Diseases (GOLD) and National Institute for Health and Clinical Excellence (NICE).[55 56]

The GOLD and NICE criteria were introduced by the collaborative effort between the National Institute of Health and the WHO. The criteria is used for diagnosing COPD using the values of FVC and FEV_1 obtained during pulmonary function tests to describe the severity of the obstruction or airway limitation in people with COPD. The worse the person's airflow limitation is, the lower their FEV_1 and FCV. These classifications have undergone updating over the years to take care of various aspects of diagnosing, severity classification and the management of stable diseases.[57]

FVC and FEV_1 less than 80% predicted were regarded as evidence of restrictive lung function impairment while FEV_1/FVC less than 70% predicted values was regarded evidence of obstructive lung function impairment. In combined lung disease (restrictive and obstructive) both the FVC and FEV_1/FVC ratio were decreased. FVC<80% and FEV_1/FVC<70% are considered to be combined lung function impairment.[58]

The concentrations of pollutants were recorded at six stations, each station located along each of the designated six routes (three high and three low traffic density routes). Measurements were also taken in cars and motorcycles while in motion along the designated routes about 7–10 km long within the metropolis. The measurement was taken between September 2009 and March 2010 using aerosol monitor 'DUST TRACK model 8520 (TS1 Inc., Minnesota, USA), with a coverage range of 0.001–100 mg/m^2 and particle size range of 0.1 to approximately 10 mm. The measurement took place along the six routes within the hours of 07:30 and 09:30 (peak traffic periods) and 15:30 to 17: 30 (low traffic periods). The designated points and routes were selected to cover the entire city and therefore the measurement was assumed to represent the entire city. In addition, the monitoring time and the concentration of pollutants were assumed to represent the concentration within the entire working hours.

The vehicles and motorbikes used were within 5–7 years old and used petrol or diesel engine. The in-vehicle measurements were taken with closed and open windows. The aerosol monitor had a multiple size and gas selective inlet. The sample intake was located in participant's breathing zone using conducive tubing attached to the device. Reading was taken every 5 min. The pollutants actually measured were carbon monoxide, sulphur dioxide, nitrogen dioxide, particulate matter ($PM_{2.5}$ and PM_{10}). This is because evidence-based studies have shown that, although air pollutants are categorised in a number of different ways, most air pollutants generally do not occur in isolation but in a complex mixture that creates the potential for synergistic effects among them.[59] Thus, assessing the levels of the priority pollutants gives a better understanding of the nature of the associated health hazards transit workers and commuters are exposed to.

STATISTICAL ANALYSIS

Descriptive analysis using simple percentages and reported as mean±standard deviation was computed for categorical and quantitative variables, respectively. Differences in continuous variables (age, height, BMI and duration on job) between job categories were computed using analysis of variance (ANOVA) and univariate relationship between two categorical variables tested with chi-square.

Multiple logistic regressions were also performed to test the association between exposure to outdoor ambient air pollution and its risk factor. Odd ratios and the corresponding 95% confidence intervals were computed. Statistical analysis was performed using SPSS 17.0 (Statistical package for social science).

RESULTS

Of the total number of participants in this survey, 48 (28.6%) met the criteria for the diagnosis of impaired lung functions using the GOLD and the NICE standards. Occupation-specific prevalence of impaired lung function was as follows: 14.3%, 10.7% and 3.6% for CMCs, TDs and CSs, respectively.

Results of the analysis showed a statistically significant difference in height, body mass index, educational statues, area of residence, exposure to biomass, physical activity, transit time and number of trips per day between the three occupations at $p<0.001$. Educational levels were also statistically different at $p<0.043$. However, age and duration on job were statistically non-significant at $p<0.295$ and 0.637, respectively, between the three occupations (table 1).

The odds for impaired respiratory functions following occupational exposure to ambient air pollution was highest in CMC for FVC<80% ($OR_{Adjusted}$ 3.10, CI 0.402 to 16.207) and $FEV_1/FVC<70\%$ ($OR_{Adjusted}$ 1.71, CI 0.61 to 4.76), whereas no difference was observed in the odds for $FEV_1<80\%$ between the two groups of transit workers. In addition, the CMC had the highest odds for incident respiratory symptoms than the TD with exceptions of frequent cough (table 2).

Additionally, results of multiple logistic regression analysis showed significant association between occupational exposure to ambient air pollution and duration on job (OR=2.54, CI 2.224 to 2.914) and (OR=2.42, CI 1.055 to 2.066), smoking habits (OR=1.68, CI 1.969 to 2.859) and (OR=1.59, CI 1.349 to 3.718), number of trip per day (OR=2.29, CI 2.238 to 19.931) and (OR=2.14, CI 2.08 to 13.473) and transit time (OR=2.53, CI 2.257 to 24.776) and (OR=2.37, CI 1.932 to 13.782) in CMCs and TDs, respectively.

Among CMCs and TDs, working for more than 5 years, with more than 10 trips per day, and transit time of more than 5 h per day were associated with higher odds for incident impaired respiratory functions, whereas in CSs, educational status was the only significant factor (OR=1.69, CI 1.079 to 5.354) for impaired respiratory function. Again, being obese, of low educational class, currently smoking and exposure to biomass were associated with higher odds for impaired respiratory functions in all work categories, with CMCs and TDs having higher odds than the CSs.

A non-significant association was obtained for other risk factors of impaired respiratory functions such as age, ethnicity, alcohol intake, area of residence and exposure to biomass in the multivariate analysis (table 3).

Results of the recorded mean values/concentrations of the priority pollutants along the six major routes (three heavy and three light traffic) within the Uyo metropolis using different modes of transportation showed a relatively higher value than the National[59] and the WHO[5] ambient air quality standards (table 4).

DISCUSSION

In this study, the adverse respiratory health effect of ambient air pollution was assessed among transit and non-transit workers. The overall prevalence of impaired respiratory functions among the participants was 28.6%. Occupation-specific prevalence was 14.3%, 10.7% and 3.6% for CMCs, TDs and CSs, respectively.

Globally, evidence-based studies have shown variations in the prevalence of respiratory impairment in the general population, with prevalence ranging from less than 5% among male and female population in Mexico to 16% and 22% in South Africa.[61] In Delhi, capital city of India, a prevalence rate of 47% was recorded.[58] In 2010, Binawara *et al*.[62] recorded 87% cases of restrictive lung function impairment in a study involving three-wheeler diesel TDs in Bikaner city. Several factors could account for this varying prevalence, ranging from inconsistencies in the definition diagnostic criteria as well as varying prevalence of risk factors in different countries. In addition, synergistic interactions among the risk factors with workplace exposure are implicated. Age, cultural practices, lifestyle and socioeconomic inequalities may influence exposure to air pollutants. The effect of these risk factors and the adverse health effect of air pollutants may be modified by individual's sensitivity to a given pollutant, and this varies from population to population. Thus, the respiratory function impairment in a given population or community is population specific and cannot be directly generalised from results of studies in other settings.[32]

Results of the present study showed that the prevalence of respiratory functions impairment was higher among transit than non-transit workers. Between the two groups of transit workers, the CMCs recorded higher odds for respiratory impairment than the TDs. Correlations of these findings with recorded values of ambient air pollutants by various commuting modes therefore suggest a higher rate of exposure in the transit than in non-transit workers. This assertion lends credence to previous studies that documented a higher exposure rate in CMCs than transit workers using other modes of transportation,[63–65] and gains validity from studies that recorded higher inhaled pollution doses by motorcyclists than TDs. Prior studies have also posited the higher minute ventilation observed among motorcyclists compared to TDs as the probable reason for their

Table 1 Sociodemographic characteristics of the participants

Variables	CS (n=58)	CMC (n=60)	TD (n=50)	p Value
Age (years)	40.50±0.13	39.88±1.08	37.92±1.33	0.295
Height (m)	1.60±0.010	1.65±0.009	1.60±0.009	**<0.001**
Weight (kg)	57.98±1.05	60.58±0.92	62.30±1.64	**0.043**
BMI (kg/m^2)	22.70±0.34	22.54±0.40	24.31±0.64	**0.014**
Duration on job (years)	6.09±0.32	6.08±0.31	5.64±0.50	0.637
Educational level				
Primary	15 (25.9)	31 (51.7)	19 (38.0)	**<0.001**
Secondary	16 (27.6)	23 (38.3)	22 (44.0)	
Tertiary	27 (46.5)	6 (10.0)	9 (18.0)	
Smoking habits				
Current smokers	5 (8.6)	15 (25.0)	10 (20.0)	0.084
Ex-smokers	19 (32.8)	17 (28.3)	9 (18.0)	
Non-smokers	34 (58.6)	28 (46.7)	31 (62.0)	
Alcohol intake				
Drinkers	35 (60.3)	36 (60.0)	32 (64.0)	0.896
Non-drinkers	23 (39.7)	24 (40.0)	18 (36.0)	
Ethnicity				
Ibibio	40 (69.0)	34 (56.7)	23 (46.0)	0.054
Non-Ibibio	18 (31.0)	26 (43.3)	27 (54.0)	
Area of residence				
Rural	8 (13.8)	39 (65.0)	17 (34.0)	**<0.001**
Urban	50 (86.2)	21 (35.0)	33 (66.0)	
Family history of respiratory disorder				
Yes	3 (5.2)	7 (11.7)	9 (18.0)	0.110
No	55 (94.8)	53 (88.3)	41 (82.0)	
Exposure to biomass				
Exposed	7 (12.1)	24 (40.0)	19 (38.0)	**<0.001**
Not-exposed	51 (87.9)	36 (60.0)	31 (62.0)	
Physical activity				
Active	7 (12.1)	29 (48.3)	18 (36.0)	**<0.001**
Inactive	51 (87.9)	31 (51.7)	32 (64.0)	
Dietary habits				
Good	31 (53.4)	21 (35.0)	24 (48.0)	0.118
Poor	27 (46.6)	39 (65.0)	26 (52.0)	
Transit time per day (h)				
<1	41 (70.7)	2 (3.3)	4 (8.0)	**<0.001**
1–5	12 (20.7)	17 (28.3)	12 (24.0)	
>5	5 (8.6)	41 (68.4)	34 (68.0)	
Number of trips per day				
1–2	25 (43.1)	3 (5.0)	2 (4.0)	**<0.001**
3–10	33 (56.9)	7 (11.7)	9 (18.0)	
>10	0 (0)	50 (83.3)	39 (78.0)	

Bold values are significant at ($p < 0.05$).
Values in parentheses are percentages.
CMC, commercial Motorcycle; CS, civil servants; TD, taxi drivers.

higher rate of exposure.[66] In many studies, motorcyclists have been noted to have a higher exposure to air pollution than taxi and bus drivers.[67] Thus, Zuurbier *et al*[44] recorded about 2 times higher pollution doses in cyclists for particle number count (PNC) than taxi and bus drivers. In addition, higher minute ventilation was recorded in cyclists than car drivers in that study, especially on a high traffic density road.[68] A similar recent study in Belgium showed increases in both ventilation rate and tidal volume with associated increase in minute ventilation, which was 4.3 times higher in cyclists than other transit workers.[69]

An additional view suggested that, the higher rate of exposure of the motorcyclists could be because, motorcyclists travel in close proximity to tail pipe exhaust emissions with little or no physical barrier between the exhaust and the motorcyclist's respiratory system.[70]

Morabia *et al*[71] attributed the less effect of air pollutants on car drivers to less physical activity while driving, but not necessarily less exposure to air pollutants. Others posited that, motorcyclists experienced significant higher average concentration because of high concentration and a very short duration peaks, which are not seen in traces of car and bus commuters.[72]

Table 2 Association between exposure to outdoor ambient air pollution by occupation and impaired lung function and chronic respiratory symptoms

Diagnostic criteria for impaired lung function (n=48)	CS (n=6)		CMC (n=24)		TD (n=18)	
	N (%)	(95% CI)	N (%)	$OR_{Adjusted}$ (95% CI)	N (%)	$OR_{Adjusted}$ (95% CI)
FVC<80% predicted	2 (33.3)	1.00	15 (62.5)	3.10 (0.402 to 16.207)	11 (61.1)	1.72 (0.408 to 4.732)
FEV_1<80% predicted	3 (50.0)	1.00	13 (54.2)	1.01 (0.942 to 1.081)	9 (50.0)	1.02 (0.953 to 1.091)
FEV_1/FVC<70% predicted	4 (66.7)	1.00	13 (37.5)	1.71 (0.611 to 4.76)	7 (38.9)	1.67 (0.28 to 4.368)
Respiratory symptoms						
Frequent cough	5 (8.6)	1.00	13 (21.7)	1.50 (1.682 to 4.920)	16 (32.0)	2.56 (1.624 to 5.478)
Frequent cough with phlegm	2 (3.4)	1.00	17 (28.3)	1.83 (1.329 to 6.413)	12 (24.0)	1.36 (0.869 to 2.126)
Wheezing	13 (22.4)	1.00	28 (46.7)	3.48 (1.932 to 8.416)	21 (42.0)	1.27 (0.706 to 2.286)
Breathlessness	9 (15.5)	1.00	19 (31.7)	2.64 (1.08 to 7.215)	17 (34.0)	2.06 (1.637 to 6.582)
Chest pain	14 (24.1)	1.00	35 (58.3)	1.78 (1.163 to 9.615)	28 (56.0)	1.36 (0.871 to 2.134)
Nasal discharge	2 (3.4)	1.00	11 (18.3)	1.45 (0.940 to 2.247)	8 (16.0)	1.09 (1.007 to 1.188)
Throat irritation	1 (1.7)	1.00	8 (13.3)	1.02 (0.953 to 1.091)	9 (18.0)	1.01 (0.969 to 1.041)

Adjusted for age, BMI, duration on job, educational status, smoking habits, alcohol intake, ethnicity, area of residence, exposure to biomass, number of trips per day, transit time per trip and family history of respiratory disorder.
GOLD criteria:[55] FEV_1/FVC ratio in % <70% predicted.
NICE criteria:[56] FEV_1/FVC<70% and FVC, FEV_1<80% predicted (equivalent to GOLD stage II).
Respiratory symptoms: any of cough, wheezing, breathlessness, chest pain, nasal discharge and throat irritation.
CMC, commercial motorcyclists; CS, civil servant; FVC: force vital capacity (ml/s); FEV_1: force expiratory volume in 1 s (ml/s); TD, taxi drivers.

Thus, the results of the present survey showing CMCs as the most affected group, with higher odds for respiratory function impairment and respiratory symptoms is therefore supported.

Contrary to the above findings, other studies have recorded a slightly higher in-vehicle air pollution exposure levels among TDs than motorcyclists.[73] This could probably be explained by the fact that, in congested traffic conditions, there is likely that much tailpipe emissions from the car in front are drawn into the air ventilation system, polluting the limited vehicle's passenger compartment.[72] Prolonged exposure time is likely in a very congested and air-polluted corridors such as checkpoints and hold-ups thereby maximising exposure doses and subsequent effects.

In addition to the activity status, it has also been shown that the transit time and number of trips per unit time are other important factors that could influence individual's pollutants dose inhaled.[65] Transit workers in this survey recorded a higher transit time and number of trips than the non-transit workers. This could imply exposure to higher pollutant doses among transit workers and subsequent effect on the respiratory system. This assertion is consistent with the result of other studies found in the literature.[72]

Also, the marked respiratory function impairment recorded among transit workers could probably be due to the synergistic interaction between risk factors of respiratory impairment and work place exposure to air pollution. Such risk factors as: low level of education, low level of income, exposure to biomass, area of residence, poor dietary habits, physical in-activity and other sources of indoor air pollutants are markers of low socioeconomic status, and empirical evidences have shown their predominance among the transit workers than the CSs. For example, about 90% of CMCs and 82% of TDs in this survey were of low educational level, compared with 52% of CSs. Also, according to prior studies, the average monthly income of a CMC in the Uyo metropolis stands at about 20 000-naira only.[74]

It is an established fact that there is a significant correlation between low socioeconomic status as mentioned above and poor respiratory health. Thus, Schikowski *et al*[75] in their study showed that participants of a lower educational level had a higher prevalence of respiratory disorders including impaired lung functions.

In other studies, educational levels have been shown to modify the effect of exposure to particulate matter on mortality, with higher risks among people of lower educational level.[76–79] Similarly, Samoli *et al*[80] found that in Europe and North America, a high percentage of unemployment was associated with a greater particulate matter health effect in both continents. Wheeler *et al*[81] in their studies in England showed that low social class and poor air quality were independently associated with decreased lung functions. These results suggest that apart from outdoor ambient air pollution, other aspects of urban environment could be important contributors to the development of pulmonary disorders[76] especially in those not exposed to ambient air pollutants. Therefore, the hypothesis that marked pulmonary functions impairment in transit workers was due to the synergistic effect of air pollutants and severally mutually inclusive factors is substantiated. These factors were modified by the higher socioeconomic status of the CSs, hence the low prevalence of respiratory functions impairment. This assertion gains support from previous studies with similar documentations.[12 77–79 82 83]

Table 3 Multiple logistic regression model showing association between exposure to outdoor ambient air pollution by different mode of transportation and risk factors of impaired respiratory functions

Risk factors of impaired respiratory functions	Occupationally unexposed to outdoor ambient air pollution			Occupationally exposed to outdoor ambient air pollution					
	Civil servants (CSs)			Commercial motorcyclists (CMCs)			Taxi drivers (TDs)		
	OR	CI	p	OR	CI	p	OR	CI	p
Age (years)									
18–25	1.00			1.00			1.00		
26–35	0.938	0.776 to 1.134	0.510	0.98	0.669 to 1.435	0.916	0.99	0.822 to 1.206	0.964
>35	1.22	0.783 to 1.916	0.375	0.84	0.496 to 1.214	0.528	1.04	0.849 to 1.275	0.703
BMI (kg/m^2)									
Not-obese	1.00			1.00			1.00		
Obese	1.25	0.357 to 4.405	0.714	1.08	0.703 to 1.650	0.733	1.44	0.526 to 3.911	0.480
Duration on job (years)									
<1	1.00			1.00			1.00		
1–5	0.839	0.521 to 1.352	0.472	1.71	1.919 to 3.184	**0.045**	1.69	1.165 to 2.453	**0.006**
>5	0.748	0.509 to 1.100	0.140	2.54	2.214 to 2.914	**<0.001**	2.42	1.055 to 2.066	**0.003**
Educational status									
Higher	1.00			1.00			1.00		
Low	1.69	1.079 to 5.354	0.002	1.90	1.071 to 3.354	**0.028**	1.83	1.526 to 3.911	**0.046**
Smoking habits									
Non-smokers	1.00			1.00			1.00		
Current smokers	1.21	0.371 to 3.964	0.750	1.68	1.969 to 2.859	**0.035**	1.59	1.349 to 3.718	**0.029**
Ex-smokers	1.19	0.064 to 6.139	0.905	1.42	0.823 to 2.437	0.209	1.31	0.671 to 3.512	0.423
Alcohol intake									
Non-drinkers	1.00			1.00			1.00		
Drinkers	0.91	6.242 to 3.434	0.892	1.35	0.805 to 2.272	0.253	1.04	0.849 to 1.275	0.703
Ethnicity									
Non-ibibio	1.00			1.00			1.00		
Ibibio	1.44	0.684 to 3.044	0.336	0.97	0.506 to 1.868	0.932	1.13	0.438 to 2.920	0.800
Area of Residence									
Rural	1.00			1.00			1.00		
Urban	1.32	0.536 to 3.271	0.543	0.78	0.621 to 0.969	0.075	0.982	0.378 to 2.552	0.971
Exposure to biomass									
Not-exposed	1.00			1.00			1.00		
Exposed	1.30	0.703 to 2.393	0.406	1.33	1.140 to 1.551	0.180	1.32	0.951 to 1.842	0.096
Number of trips per day									
1–2	1.00			1.00			1.00		
3–10	1.14	0.911 to 1.429	0.252	1.99	1.204 to 10.931	0.028	1.75	1.13 to 8.417	**0.034**
>10	0.94	0.790 to 1.109	0.444	2.29	2.238 to 19.931	**<0.001**	2.14	2.08 to 13.473	**<0.001**
Transit time per trip									
>1 h	1.00			1.00			1.00		
1–5	0.74	0.043 to 4.678	0.837	2.01	1.233 to 21.847	**0.014**	1.82	2.146 to 8.473	**0.038**
>5	0.56	0.174 to 1.826	0.339	2.53	2.257 to 24.776	**0.002**	2.37	1.932 to 13.782	**0.007**
Family history of respiratory disorder									
No	1.00			1.00			1.00		
Yes	0.97	0.443 to 1.362	0.378	1.21	0.743 to 1.980	0.441	1.16	0.543 to 2.476	0.703

Bold values are significant at ($p < 0.05$).

The CSs are generally known to use any available means of transportation depending on their socio-economic status. Similarly, compelling evidences have shown that most people receive a significant proportion of their daily air pollution dose while commuting to work, through walking, cycling, travelling by car or public transport.[27] This implies that the CSs were equally exposed to outdoor ambient air pollutants though transiently while commuting to, and from work and on few occasions while on pleasure or business trips. Prior studies have identified health effects including increased mortality,[84] increased respiratory

Table 4 Average concentrations of ambient air pollutants as recorded along six major routes compared with national (NAAQS)[59] and WHO[5] ambient air guideline values

Air pollutants	Average time of measurement	Average recorded values of ambient air pollutants using different modes of transportation			WHO[5] standard guideline values (PPM)	NAAQS
		Motorcycle recorded values	In-vehicular recorded values			
			Windows closed	Windows open		
Carbon monoxide (CO)	1 h	85.06	48.03	53.24	25	35 PPM
	30 min	–	–	–	50	–
	15 min	–	–	–	90	–
Nitrogen dioxide (NO_2)	1 h	0.621	0.349	0.456	0.11	0.04 PPM–0.06 PPM
Sulphur dioxide (SO_2)	1 h	0.297	0.195	0.103	0.175	0.10
Particulate matter						
$PM_{2.5}$	1 h	128.04	75.02	92.23	–	35 μg/m³
	24 h	–	–	–	25 μg/m³	–
PM_{10}	1 h	288.21	95.32	138.45	–	150 μg/m³
	24 h	–	–	–	50 μg/m³	–

NAAQS, National Ambient Air Quality Standards.[60]

symptoms[12 85] and decreased pulmonary functions[86] at concentration levels of air pollutants around or below current air quality guidelines recommended by the WHO.[5] However, the effects of these pollutants could have been attenuated by their higher socioeconomic status, hence the low prevalence. Additionally, the low prevalence among CSs could have been due to the short transit time and few numbers of trips embarked upon. These have been shown to correlate with the dose of the pollutants inhaled and subsequent adverse respiratory health effects.[44]

Apart from exposure by commuting mode and other risk factors, the exposure of the civil sarvents was further enhanced by their area of residence. A majority of them (86.2%) resided in the urban area, which has been shown to be associated with a higher air pollution level. This is consistent with the results of studies in Rome where wealthy residents of the city centre were more exposed to higher air pollution levels than the disadvantaged groups in the rural area.[87]

In Japan, higher levels of pollution were detected in the urban area than in rural area and were associated with adverse respiratory health effects.[88] According to the results of this survey, CSs who resided in the urban area had higher odds for exposure to air pollutants and subsequent development of respiratory function impairment. Reduced odds among transit workers residing in urban areas could be due to the actual location of residents within the urban setting. A great number of transit workers by virtue of their low socioeconomic status will choose to live in the city suburbs (less industrially polluted communities) than the centre city (more industrially polluted). The opposite is the case for CSs who are socioeconomically advantage. This assertion is consistent with studies that found a substantial decrease and statistically non-significant risk among adults who had an occupational exposure associated with impaired lung functions and living in less industrially polluted communities compared with those adults with similar occupational exposure but living in more industrially polluted communities.[61] Another interesting aspect of the results of the present study is that, not all transit and non-transit workers with similar exposure (occupationally, indoors, environmental) developed respiratory functions impairment. This clearly suggests a variation in population/individual sensitivity and genetic variability in the risk of developing lung function disorders. The validity of this assertion gains credence from prior studies that showed the association between deficiency of serine protease with COPDs and genes coding transforming growth factor B_1, tumor necrosis factor α and Microsomal epoxide hydrolase 1, being implicated in COPDs.[89]

The inconsistencies in the research literature are probably due to the effect of other confounders, which are noted to trigger the onset in a genetically predisposed individual.[89] This explains the higher odds for lung function impairment in transit workers with a family history of respiratory disorders compared with CSs with a similar history. Exposure to large doses of air pollutants could have triggered the onset of pulmonary function disorders in genetically predisposed transit workers.

Also, the smoking habits of transit workers in this survey could have contributed to the high prevalence of respiratory function impairment observed among them. About 45% of transit operators were current smokers. A significant association between current smoking and impaired respiratory functions was observed in transit workers but not so in non-transit workers, with CMCs having the highest odds for impaired lung functions. This finding stands in clear support to the previous literature that documents a higher rate of smoking among blue-collar jobs than white-collar and professional occupations,[90 91] which has been attributed to more occupational stress among them.[49 50]

Evidence-based studies have shown that, the occupation of transit operators relative to many other occupations is very stressful.[49] Thus, compared to other occupations, rates of smoking among transit operators are found to be elevated. Elevated rate of smoking may imply a higher risk for pulmonary function impairment in this group, since smoking is the most important single risk factor for pulmonary function impairment and accounts for more than 75% of cases of the disease.[46] Recent studies have shown that up to 50% of smokers actually develop COPDs.[89] Another interesting observation in this survey was that, there were no significant differences in the odds for developing respiratory function impairment between the transit and non-transit ex-smokers. This is probably because there were improvements in measured pulmonary function indices which were marked in CSs than in other groups. This explains the lowest odds for lung function impairment among CS ex-smokers in this survey. This is probably due to different exposure and attenuated effects from smoking following cessation. This effect is dependent on the number of years since quitting and number of cigarettes smoked. These findings are consistent with similar findings documented elsewhere.[92–95]

It supports the previous studies that showed that smoking cessation in smokers with minimal exposure to air pollution and without chronic symptoms slows the accelerated decline in lung functions towards that observed in non-smokers.[96 97] Additionally, it was observed that current smokers in all occupations recorded higher odds for impaired lung functions than the ex-smokers. This was more marked among transit than non-transit workers. These findings corroborate with prior studies that showed that, lung functions decline following smoking are strongly related to recent than previous exposure,[98] and that a significant decline in FVC and FEV_1 in current smokers over non-smokers, ex-smokers and quitters is expected.[97] It further demonstrated an established fact that, cigarette smoking and exposure to air pollutants are two independent risk factors with serious adverse respiratory health.[99] When they co-exist at the same time or at different times as in the current and ex-smoking transit workers, a more rapid and deleterious outcome ensues.[99 100]

Unlike previous studies,[101] there was a non-significant association between exposure to biomass and development of respiratory function impairment, even though a significant difference was noted in the proportion of participants exposed between the three occupations. This could be due to the effect of other confounders like gender. The absence of women (more vulnerable group) in this survey could have attenuated the effect of indoor air pollution particularly the use of biomass as a source of energy. This has been shown by prior studies to be more significant among women particularly the rural women.[89]

Also, another significant factor associated with higher odds for pulmonary function impairment among transit workers was the duration on job. Transit workers with more than 5 years on the job had higher odds for incident pulmonary function impairment. This could probably be due to the accumulated effect of air pollutants and other confounding factors.

CONCLUSIONS

In summary, the findings of this study provide additional evidence and strength to few existing literature on the adverse respiratory health effect of ambient air pollution, faced by city transit operators in some Nigerian cities and similar cities elsewhere. The role of other confounders acting synergistically to cause a more serious and deleterious effect is obvious. The results have important policy implications for the introduction of stringent measures towards reducing ambient air pollution in our cities, such as better air quality management. This could include the establishment of a central pollution board, monitoring of personal exposure for the evaluation of health impact of air pollution and avoidance of building in cities air pollution hot spots.

Also, the workplace approach that addresses both individual and environmental factors could help ameliorate the adverse respiratory effect of ambient air pollution and its confounders on transit and non-transit workers. Individual intervention could include the avoidance of exposure to indoor air pollutants, life style modifications such as cessation of smoking, obesity preventive measures (eg, good nutrition, physical activity), avoidance of exposure to second-hand smoking, proper psychosocial stress management, adoption of good coping strategies and choice of residential area away from air pollution hot spots.

Also, effort should be directed towards tackling the problem of socioeconomic inequalities by encouraging early childhood education. Empirical evidences have shown that, the educational level can modify the effect of exposure to air pollutants on mortality, with higher risk among people with lower educational levels.[78 79]

STRENGTHS AND LIMITATIONS

The strength and precision of this study came from the fact that both the quantitative and qualitative variables were considered in assessing the adverse respiratory health effect of outdoor ambient air pollution on occupationally exposed and unexposed participants. Also, all the participants existed in the same environment, sharing similar environmental, cultural and ethnic factors effect. However, the study was limited by few confounding factors such as the effect of other psychosocial factors peculiar to their respective work environments, as well as differences in individual's genetic susceptibility to the effect of outdoor ambient air pollutants, which was not adjusted for in this study. Similarly, the energy level of the participants while on motion using different transportation modes was not reported because of poor results from faulty equipments. Again, the representativeness of the study subjects was difficult to determine, as efforts to get information regarding the total number of registered CMCs and TDs from the city authority was not successful. Being a cross-sectional study, it is possible to have oversampled the respiratory disorders that last for a long time and less likely to capture those that last for a short while. Also, this cross-sectional study cannot accurately attribute the chronic lung damage to the occupational exposure of participants to ambient air pollution.

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REFERENCES

1. Boko G, Joachim M. Air pollution and respiratory diseases in African Big Cities: the CASE of Cotonou in Benin. In: Bunch MJ, Suresh VM, Kumaran TV, eds. Proceedings of the Third International Conference on Environment and Health, Chennai, India; 15–17 December, 2003;32–43.
2. Chai ZF, Qian QF, Feng XQ, *et al*. Study of occupational health impact of atmospheric pollution on exposed workers at an iron and steel complex by using neutron activation analysis of scalp hair. *J Radioanal Nucl Chem* 2004;259:153–6.
3. Nku CO, Peters EJ, Eshiet AI, *et al*. Lung function, oxygen saturation and symptoms among street sweepers in Calabar-Nigeria. *Niger J Physiol Sci* 2005;20:79–84.
4. Wiwanitkit V, Suwansaksri J, Soorgarun S. Cancer risk for Thai traffic exposed to traffic benzene vapor. *Asian Pac J Cancer Prev* 2005;6:219–20.
5. WHO Air Quality Guidelines Global Update 2005. *Report on a working meeting*. Bonn, Germany, 18–20 October. World Health Organization, 2004.
6. Kennedy SM, Chambers R, Du W, *et al*. Environmental and occupational exposure: Do they affect chronic obstructive pulmonary disease differently in women and men? *Proc Am Thorac Soc* 2007;4:692–4.
7. Rojas-Rueda D, de Nazzelle A, Tainio M, *et al*. The health risk and benefits of cycling in urban environments compared with car use: health impact assessment study. *BMJ* 2011;343:d4521.
8. Olowoporoku D. How clean is the air Nigerians breathe? 2 February 2011. http://nigeriaworld.com/articles/2011/feb/012.htm/ (accessed 2 Feb 2011).
9. Jansen M (UNEP), Qian J (UNICEF), Wojciechowska-Shibuya M, *et al* (eds). Environmental threats to children. In: *Children in the New millennium Environmental impact on Health*. United Nations Environmental Programme, Children's Fund and World Health Organization, 2002:43–86.
10. Zanobetti A, Schwartz J. Are diabetics more susceptible to the health effects of air borne particles? *Am J Respir Crit Care Med* 2001;164:831–3.
11. Dockery DW. Epidemiologic evidence of cardiovascular effects of particulate air pollution. *Environ Health Perspect* 2001;109(Suppl. 4):483–6.
12. Pope CA III, Burnett RT, Thun MJ, *et al*. Lung cancer, cardiopulmonary mortality, and long term exposure to fine particulate air pollution. *JAMA* 2002;287:1132–41.
13. Epton MJ, Drawson RD, Brooks WM, *et al*. The effect of ambient air pollution on respiratory health of school children: a panel study. *Environ Health* 2008;7:16.
14. Müeller D, Klingelhöfer D, Uibel S, *et al*. Car indoor air pollution–analysis of potential sources. *J Occup Med Toxicol* 2011;6:33.
15. Efe SI. Particulate pollution and its health implications in Warri Metropolis, Delta State, Nigeria. *Env Anal* 2006;11:1339–51.
16. Olobaniyi SB, Efe SI. Comparative assessment of rain water and ground water quality in an oil producing area of Nigeria: environmental and health implications. *J Environ Health Res* 2007;6:111–18.
17. Ana GR, Sridhar MK, Bamgboye EA. Environmental risk factors and health outcomes in selected communities of the Niger Delta area, Nigeria. *Perspect Public Health* 2009;129:183–91.
18. Han X, Naeher LP. A review of traffic—related air pollution exposure assessment studies in the developing world. *Environ Int* 2006;32:106–20.
19. Khan SA, Saadia A. Pulmonary function studies in Pakistani cotton ginners. *Pak J Physiol* 2006;2:50–4.
20. Toren K, Bergdahl IA, Nilsson T, *et al*. Occupational exposure to particulate air pollution and mortality due to ischemic heart disease and cerebrovascular disease. *Occup Environ Med* 2007;64:515–9.
21. Gupta S, Mittal S, Kumar A, *et al*. Respiratory effects of air pollutants among non smoking traffic policemen of Patiala, India. *Lung India* 2011;28:253–7.
22. Seneca JJ, Tausig MK. *Environmental economics*. 3rd edn. Eagle Wood Cliffs, NJ: Prentice-Hall, 1984.
23. Obot JU, Etim EE, Atser J. Intra-urban traffic and parking demand in Uyo Urban Area. *Global J Soc Sci* 2009;8:61–8.
24. Brunekreef B. Out of American commentary occupation. *Environ Med* 2005;62:351–2.
25. Savile SB. Automotive options and quality management in developing countries. *Ind Environ* 1993;16:20–32.
26. Dirks KN, Longley I, Fernandes K, *et al*. Blood carboxyhemoglobin levels as a biomarker for urban air pollution exposure. Paper presented at the International Conference on Southern Hemisphere Meteorology and Oceanography; Melbourne, 9–13 February, 2009.
27. Pravati P, John RA, Dutta TK, *et al*. Pulmonary function test in traffic police personnel in Pondicherry. *Indian J Physiol Pharmacol* 2010;54:329–36.
28. Happo MS, Salonen RO, Halinen AI, *et al*. Inflammation and tissue damage in mouse lung by single and repeated dosing of urban air coarse and fine particles collected from six European Cities. *Inhal Toxicol* 2010;22:402–12.
29. Budinger GR, Mc Kell J, Urich D, *et al*. Particulate matter-induced lung inflammation increases systemic levels of PAI-1 and activates coagulation through distinct mechanisms. *PLoS One* 2011;6:e18525.
30. Bakke P, Eide GE, Honoa R, *et al*. Occupational dust or gas exposure and prevalence of respiratory symptoms and asthma in a general population. *Eur Respir J* 1991;3:273–8.
31. Tomas ML, Amund G, Eide GE, *et al*. Occupational airborne exposure and the incidence of respiratory symptoms and asthma. *Am J Respir Crit Care Med* 2002;166:933–8.
32. Nandasena YL, Wickremasinghe AR, Sathiakumar N. Air pollution and health in Sri Lanka: a review of epidemiologic studies. *BMC Public Health* 2010;10:300.
33. Oh SM, Kim HR, Park YJ, *et al*. Organic extracts of urban air pollution particulate matter ($PM_{2.5}$)-induced genotoxicity and oxidative stress in human lung bronchial epithelial cells (BEAS-2B cells). *Mutat Res* 2011;723:142–51.
34. Foster WM, Brown RH, Macri K, *et al*. Bronchial reactivity of health subjects: 18–20 h post-exposure to ozone. *J Appl Physiol* 2000;89:1804–10.
35. Wong CM, Thach TQ, Chau PY, *et al*. Part 4: Interaction between air pollution and respiratory viruses: time-series study of daily mortality and hospital admissions in Hong Kong. *Res Rep Health Effect Inst* 2010:283–362.
36. Kakinoki Y, Ohashi Y, Tanaka A, *et al*. Nitrogen dioxide compromises defense functions of the airway epithelium. *Acta Otolaryngol Suppl* 1998;538:221–6.

37. Abam FI, Unachukwu GO. Vehicular Emissions and Air Quality Standards in Nigeria. *Eur J Sci Res* 2009;34:550–60.
38. American Thoracic Society. What constitutes an adverse health Effect of Air Pollution? *Am J Respir Crit Care Med* 2000;161:665–73.
39. Akanbi MO, Ukoli CO, Akanbi FO, *et al*. The burden of respiratory disease in Nigeria. *Afr J Respir Med* 2009:10–17.
40. Kalaic I. Chronic non-specific lung disease in cement workers. *Arch Environ Health* 1973;26:78–88.
41. Urom SE, Antai AB, Osim EE. Symptoms and lung function values in Nigeria men and women exposed to dust generated from crushing of granite rocks in Calabar, Nigeria. *Niger J Physiol Sci* 2004;19:41–7.
42. Maduka SO, Osim EE, Nneli RO, *et al*. Effect of occupational exposure to local powered tobacco (Snuff) on pulmonary function in south Eastern Nigeria. *Niger J Physiol Sci* 2009;24:195–202.
43. Ogakwu MA. Pilot health survey among Enugu coal miners. *Niger J Med* 1973;3:97–9.
44. Zuurbier M, Hoek G, Oldenwening M, *et al*. Commuters exposure to particulate matter air pollutions is affected by mode of transport, fuel type and route. *Environ Health Perspect* 2010;118:783–9.
45. Pistelli F, Viegi G, Carrozzi L, *et al*. Appendix 3. Compendium of respiratory standard questionnaires for adults (CORSQ). *Eur Respir Rev* 2001;11:118–43.
46. Lopez AD, Mathers CD, Ezzati M, *et al*. *Global burden of disease and risk factors*. Washington: The World Bank, 2006.
47. Eisner DM, Anthonisen N, Coulta D, *et al*. An official American Thoracic Society public policy statement: novel risk factor and the global burden of chronic obstructive pulmonary diseases. *Am J Respir Crit Care Med* 2010;182:693–718.
48. Morgan WK. Bronchitis, airways obstruction and occupation. In: Parkes WR, ed. Occupational lung disorders. 3rd edn. Oxford: Butterworth Heinemann, 1994: 238–52.
49. Aronson G, Rissler A. Psycho physiological stress reactions in female and male urban bus drivers. *J Occup Health Psychol.* 1998;3:122–9.
50. Cunradi CB, Greiner BA, Ragland DR, *et al*. Burnout and alcohol problems among urban transit operators in San Francisco. *Addict Behav* 2003;28:91–109.
51. Center for Disease Control and Prevention. State indicator report on physical activity, 2010. Atlanta, GA: US Department of Health and Human Services, 2010. http:// www.cdc.gov/physical activity.
52. World Health Organization. *Screening for type 2 diabetes. Report of a WHO and IDF meeting*. Geneva: World Health Organization, 2003.
53. American Thoracic Society. Hospital—acquired pneumonia in adults, diagnosis, assessment of severity, initial antimicrobial therapy, preventive strategies: a consensus statement. *Am J Repir Crit Care Med* 1995;153:1711–35.
54. Crapo RO, Morris AH, Clayton PD, *et al*. Lung volumes in health non-smoking adults. *Bull Eur Physiopathol Respir* 1982;18:419–25.
55. Global strategy for the diagnosis, management and prevention of COPD, *Global Initiative for Chronic Obstructive Disease (GOLD)*, 2008. Available from http://www.goldcopd.org
56. The National Collaborating Centre for Chronic Conditions. Chronic obstructive pulmonary disease. National Clinical guideline on management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax* 2004;59(Suppl 1):1–232.
57. O' Reilly J, Rudolf M. What is nice about the new NICE guideline? *Thorax* 2011;66:93–6.
58. Central Pollution Control Board, Ministry of Environment and Forests, Government of India, Parivesh Bhavan, East Arjun Nagar Delhi-110032. Epidemiological study on effect of IR Pollution on Human health (Adults) in Delhi August 2008. http://www.cpcb.nk.in/ e-mail:cpcb@nic.in
59. HEI. Special Report 15, Health effect of outdoor air pollution in developing countries of Asia: a literature review. Health Effects Institute, 2004.
60. U. S. Environmental Protection Agency. Air and Radiation. National Ambient Air Quality Standards (NAAQS). Available from http://epa. gov/air/criteria.html. Updated 28 April 2011.
61. Naidoo RN, Robins TG, Batterman S, *et al*. Respiratory out comes, work and ambient air pollution. *Presentation to the International Commission for Occupational Health Conference*; Cape Town, 2009.
62. Binawara BK, Gablot S, Mathur KC, *et al*. Pulmonary function tests in three wheeler diesel taxi drivers in Bikaner city. *Pak J Physiol* 2010;6:28–31.
63. Vellopoulou AV, Ashmore MR. Personal exposures to carbon monoxide in the city of Athens. I: Commuters' exposures. *Environ Int* 1998;24:713–20.
64. De Bruin YB, Carrer P, Jantunen M, *et al*. Personal carbon monoxide exposure levels contribution of local sources to exposures and microenvironment concentrations in Milan. *J Exp Anal Environ Epidemiol* 2004;14:312–22.
65. Saksena S, Quang TN, Nguyen T, *et al*. Commuter's exposure to particulate matter and carbon monoxide in Hanoi, Vietnam transportation research. Part D. *Transp Environ* 2008;13:206–11.
66. Zuurbier M, Hoek G, Van den Hazel P, *et al*. Minute ventilation of cyclist, car and bus passengers: an experimental study. *Environ Health* 2009;8:48.
67. de Hartog JJ, Boogaard H, Hoek G. Cycling: de Hartog *et al* Respond. *Environ Health Perspect* 2011;119:144–55.
68. Van Wijnen JH, Verhoeff AP, Jans HW, *et al*. The exposures of cyclists can drivers and pedestrians to traffic-related air pollutants. *Int Arch Occup Environ Health* 1995;67:187–93.
69. Int. Panis L, de Geus B, Vandenbuleke G, *et al*. Exposure to particulate matter in traffic; a comparison of cyclists and car passengers. *Atmos Environ* 2010;44:2263–70.
70. Liu JJ, Chan CC, Jeng FT. Predicting personal exposure levels to carbon monoxide (CO) in Taipei, based on actual CO measurements in microenvironments and a Monte Carlo simulation method. *Atmos Environ* 1994;28:2361–8.
71. Morabia A, Philippe NA, Franklin EM, *et al*. Air pollution and activity during transportation by car, subway and walking. *Am J Prev Med.* 2009;37:72–7.
72. Dirks KN, Sharma P, Salmond JA, *et al*. Personal exposure to air pollution for various modes of transportation in Auckland, New Zealand. *Open Atmos Sci J* 2012;6(Suppl 1:M5):84–92.
73. Kaur S, Nieuwenhuijsen MJ, Colvile RN. Fine particulate matter and carbon monoxide exposure concentrations in urban street transport microenvironments. *Atmos Environ* 2007;41:4781–810.
74. Umoren V, Jacob A, Ekong F. Motorcycle operation and inter-urban accident rate in Uyo, Akwa Ibom State, Nigeria. *JED* 2008;3:47–54.
75. Schikowski T, Suguri D, Reimann V, *et al*. Contribution of smoking and air pollution exposure in urban areas to social differences in respiratory health. *BMC Public Health* 2008;8:179.
76. Finkelstein MM, Jerrett M, De Luca P, *et al*. Relation between income, air pollution and mortality: a cohort study. *Can Med Assoc J* 2003;169:397–402.
77. Miller KA, Siscovick DS, Sheppard L, *et al*. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 2007;356:447–58.
78. Beelen R, Hoek G, van den Brandt PA, *et al*. Long term effects of traffic related air pollution on mortality in a Dutch cohort (NLCS-Air Study). *Environ Health Perspect* 2008;116:196–202.
79. Krewski D, Jerrett M, Burnett RT, *et al*. Extended follow up and spatial analysis of the American Cancer Society Study linking particulate air pollution and mortality. *Health Eff Inst* 2009;140:5–144.
80. Samoli E, Peng R, Ramsay T, *et al*. Acute effects of Ambient Particulate Matter on mortality in Europe and North America: results from the APHENA study. *Environ Health Perspect* 2008;116:1480–6.
81. Wheeler BW, Ben-Shlomo Y. Environmental equity, air quality, socio-economic status, and respiratory health: a linkage analysis of routine data from Health Survey for England. *J Epidemiol Commun Health* 2005;59:948–54.
82. Cesaroni G, Boogaard H, Jonkers S, *et al*. Health benefits of traffic-related air pollution reduction in different socioeconomic groups: the effect of low emission zoning in Rome. *Occup Environ Med* 2012;69:133–9.
83. Katsouyanni K, Karakatsanni A, Messari I, *et al*. Air pollution and caused specific mortality in Athens. *J Epidemiol Commun Health* 1990;44:321–4.
84. Roemer W, Hoek G, Brunekreef B. Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. *Am Rev Respir Dis* 1993;147:118–24.
85. Hoek G, Brunekreef B. Effects of low-level winter air pollution concentrations in respiratory health of Dutch children. *Environ Res* 1994;64:136–50.
86. Ren C, Tong S. Health effect of ambient air pollution- recent research development and contemporary methodological challenges. *Environ Health* 2008;7:56.
87. Jerret M. Global geographies of injustice in traffic related air pollution exposure. *Epidemiology* 2009;20:231–3.
88. Sekine K, Shima M, Nitta Y, *et al*. Long term effect of exposure to automobile exhaust on pulmonary functions of female adult in Tokyo, Japan . *Occup Environ Med* 2004;61:350–7.
89. Mannino DM, Buist AS. Global burden of COPD risk factors, prevalence and future trend. *Lancet* 2007;370:765–73.
90. Park H, Sprince NL, Jensen C, *et al*. Health risk factors and occupation among lower workers. *Am J Prev Med* 2001;21:203–8.

91. Bank KM, Kim JH. Prevalence of cigarette smoking by occupation and industry in the United State. *Am J Ind Med* 2001;40:233–9.
92. Lange P, Groth S, Nyboe GJ, *et al*. Effect of smoking and changes in smoking habits on the decline of FEV_1. *Eur Respir J* 1989;2:811–6.
93. Sherrill DL, Holberg CJ, Enright PL, *et al*. Longitudinal analysis of the effects of smoking onset and cessation on pulmonary faction. *Am J Respir Crit Care Med* 1994;149:591–1.
94. Connett JE, Murray RP, Buist AS, *et al*. Changes in smoking status affect women more than men: results of the lung health study. *Am J Epidemiol* 2003;157:973–9.
95. Goodman P, Agnew M, McCaffrey M, *et al*. Effects of the Irish smoking ban on respiratory health of bar workers and Air Quality in Dublin Pubs. *Am J Respir Crit Care Med* 2007;175:840–5.
96. Burchfiel CM, Marcus EB, Curb JD, *et al*. Effects of smoking and smoking cessation on longitudinal decline in pulmonary function. *Am J Respir Crit Care Med* 1995;151:1778–85.
97. Min JY, Min KB, Cho S, *et al*. Combined effect of cigarette smoking and sulfur dioxide on lung function in Koreans. *J Toxicol Environ Health* 2008;4:301–8.
98. Samet JM, Lange P. Longitudinal studies of active and passive smoking. *Am J Respir Crit Care Med* 1996;154(6 pt 2):S257–65.
99. Xu X, Wang L. Synergistic effects of air pollution and personal smoking on adult pulmonary function. *Arch Environ Health* 1998;53:44–53.
100. Roger SM, Franklyn NJ, Joseph P, *et al*. Health effects of urban air pollution special consideration at 1500 m and above. *JAMA* 1979;242:1163–8.
101. Lankathilaka KN, Seneviratne SR, Fernando DN. Indoor air quality and respiratory symptoms among children and women. Sri Lanka Association for the Advancement of Sciences-56th Annual Session. Volume Part-1 Abstract. Colombo 2000.