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Health Risks Associated with Occupational Exposure to Ambient Air Pollution in Commercial Drivers: a Systematic Review

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Short title: Health risks in commercial drivers occupationally exposed to air pollution

Abstract:

Introduction: Ambient air pollution is major global health problem and commercial drivers are particularly exposed to it. No systematic assessment of the health risks associated with occupational exposure to ambient air pollution in this population has been carried out.

Methods: We conducted a systematic review using a protocol-driven strategy. Papers published from inception to 20th April 2018 in MEDLINE, EMBASE, CINAHL, African journals online, Cochrane library, ISRCTN and WHO ICTRP databases were screened for inclusion by two independent reviewers. Original articles with at least an available abstract in English or French were included.

Results: The initial search retrieved 1454 published articles of which 20 articles were included. 3 Studies reported a significant difference in white blood cells (10^6/L) among commercial motorcyclists compared to rural inhabitants (5.041±1.209 vs 5.900±1.213,p=0.001), an increased risk of lung cancer (RR=1.6, 95%CI 1.5-1.8) in bus drivers and an increased standardized mortality ratio (SMR) in bus drivers from Hodgkin's lymphoma (SMR 2.17, 95%CI 1.19-3.87) compared to white collar workers. Other studies also found that drivers had more oxidative DNA damage and chromosome breaks. 4 papers failed to demonstrate that the drivers were more exposed to air pollution than the controls. 3 other studies also reported no significant difference in lung function parameters and respiratory

symptoms. The genetic polymorphisms of detoxifying enzymes were not also homogeneously distributed compared to the controls.

Conclusion: There is some evidence that occupational exposure to ambient air pollution among commercial drivers is associated with adverse health outcomes but the existing literature is limited with few studies of small sample size, methodological weaknesses and contradictory findings. Further research is recommended.

Key words: Air pollution, Health risk, Driving, Automobile, Bus, Motorcycle

1. Introduction

Air pollution is a major global public health problem that caused 7 million deaths in 2012, including 3.7 million due to ambient air pollution (1). The majority (88%) of the deaths due to ambient air pollution occurred in low- and middle-income countries. Traffic air pollution is responsible for much of the ambient air pollution in cities with exhaust emissions alone accounting for up to 30% of all particulate matter (PM 2.5) emitted in urban areas (2). The International Agency for Research on Cancer (IARC) classified ambient air pollution (particularly particulate matter) as a group 1 carcinogen for the lungs (3). Commercial drivers of buses, cars, motorcycles in urban areas are commonly exposed to ambient air pollution in the course of their work. The vehicles they drive are both source of air pollution for the drivers and others who work in the outdoor environment. To our knowledge there has not yet been a systematic review of the literature on the health risks associated with occupational exposure to ambient air pollution amongst commercial drivers. This systematic review set out to fill this gap with a view to identifying knowledge gaps, opportunities for further research and guide policies to help protect the health of this vulnerable group.

2. Methods

Data Sources and search strategy

We searched MEDLINE, EMBASE, CINAHL, African journals online, Cochrane library, ISRCTN and WHO ICTRP databases, for English and French language papers published from inception to 20th April 2018 using a systematic search strategy with the removal of duplicate titles. Table 1 reported the key words that were used. Reference lists from published reviews and included publications, abstracts from major occupational and environmental medicine conference proceedings of major conferences on occupational and environmental health were also searched. The bibliography of studies included in the review were searched for any additional relevant titles.

Study selection

Original studies comparing the health effects of occupational air pollution in commercial drivers and a comparison group were included if they met the selection criteria detailed in table 2. Two (HL, MS) authors screened the titles and abstracts and made study selection decisions independently. A third author (LAY) reviewed in case of disagreement. There were no study design restrictions.

Data extraction, risk of bias assessment and analysis

Data was collected about study design and the location, type and number of drivers and their controls, the type of exposure and outcomes measured. A narrative synthesis was completed on all the included studies and reported the key points on each of the studied items.

Table 1: Keywords

MeSH Keywords		
- "Motorcycle" - "Motorbike" - "Automobile Driving" - "Taxi driver" - "Professional driver" - "Bus driver" - "worker" - "Commerce" - "Transit worker"	**And**	"Air pollution"

Table 2: Selection criteria

Inclusion criteria	Exclusion criteria
• Original articles • Focus on air pollution and drivers • Abstracts available • Abstract available in English, French • Study population must include at least one comparative group	• In vitro • Interventional studies • Studies on animal, cyclists or walkers

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3. Results

The initial search retrieved 1454 published articles of which 1416 were excluded based on their titles and abstracts. Full evaluation of 38 papers led to 20 articles relating to commercial motorcycle, car, bus and truck being included (Tables 3 to 6). The 18 non included papers were excluded on the basis of: 9 articles had no comparative group, 1 paper compared drivers with/without co morbidities, 6 articles did not report results for drivers only, 1 article compared indoor and outdoor vehicle exposure, 1 article compared air conditioned vs open air bus (Figure 1).

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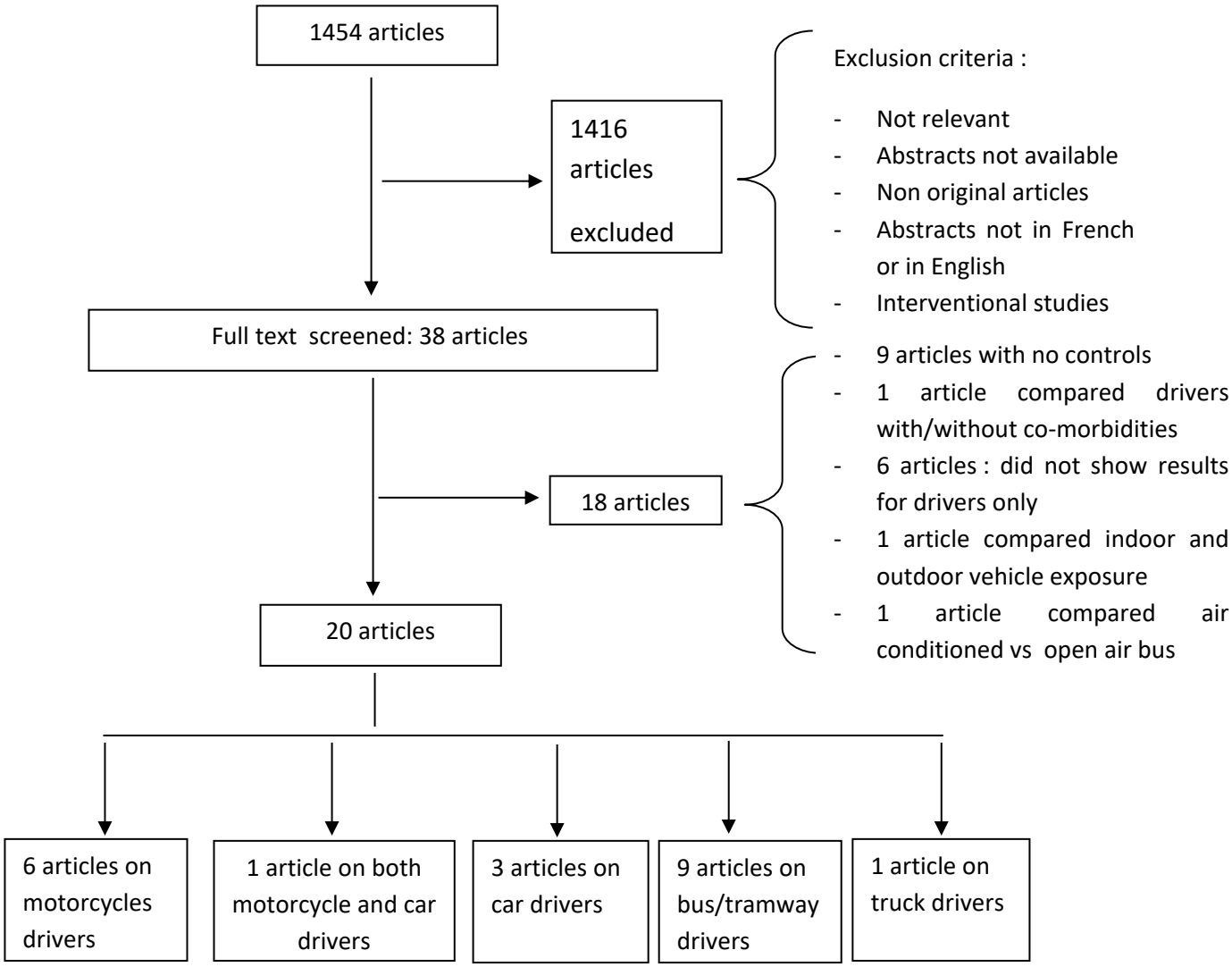
Figure 1: Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram



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3.1 Study design and site

All the included studies were observational – 17 cross-sectional and 3 cohort. All the cohort studies were implemented in high-income countries; almost one third of the cross-sectional studies were done in Africa.

3.2 Populations studied

Most of the articles (13 of 20) studied bus and commercial motorcycle drivers, three articles assessed car taxi drivers. The comparison groups varied considerably between studies e.g. drivers who were not occupationally exposed to ambient air pollution in the same location, rural/suburban inhabitants, administrative and office workers, policemen or civil servants. Only two studies used age and gender matching in the recruitment of the comparative group: one of these recruited the matched population from a rural area different to the working area of the exposed group; the other recruited the matched comparative group from the same locality.

3.3 Exposures variables measured in studies included in the review

Several types of pollutants were measured to characterize the exposure level in the drivers: particle matters (ultrafine, fine, coarse), volatiles organic compounds included benzene, toluene, ethyl benzene and xylene (BTEX), polycyclic aromatic hydrocarbons (PAHs) included benzo[a]pyrene (B[a]P) and gaseous pollutants

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(SO2, CO, NO). Most of the measurements were performed by using fixed monitoring stations. These fixed stations pre-existed or were set up for the purpose of the studies. Some personal measurements were performed by using urine excretion of PAHs or benzene and by the dosage of carboxyhemoglobin. The duration of measurement varied between studies but 24 to 48 hours or one week of exposure measurement duration were most frequently done. Some measurements were also performed before and after work. Two studies used the job title alone to define the exposure.

Of the 20 studies included in this review, four found no difference in air pollution exposures between the exposed and control groups. Brucker et al. (4) and Burgaz et al. (5) reported that exposure to carboxyhaemoglobin (almost 2% in the 2 groups) and 1 hydroxypyrene (0.32±0.25 vs 0.57±0.36 µmol/mol creatinine, $p>0.05$) were not different in a group of taxis drivers and controls. Brucker compared 39 automobile taxi drivers with 21 non-occupationally exposed controls and Burgaz 17 taxi drivers with 23 office workers. Ayi Fanou et al. (6) also reported a non-statistical significant difference in 1 hydroxypyrene level among 6 urban motorcycle taxi drivers and 5 rural inhabitants. Rossner et al. (7) reported that bus drivers were less exposed to B[a] P (1.3±0.7 vs 1.8±1.0 mg/m^3, $p<0.01$) and carcinogenic PAHs (7.1±3.7 vs 9.4±5.5 mg/m^3, $p<0.05$) than the

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controls. The controls were healthy male volunteers spending >90% of daily time indoors.

3.4 Outcomes variables measured in studies included in the review

Most of the outcomes measured were based on reactive oxygen species (ROS) that produce oxidative stress and DNA damage. The current studies reported measurement of oxidative DNA, oxidized protein and lipids, DNA adducts, chromosome aberrations and breakage. Cytochrome P4501A1 (CYP1A1), which is the main enzyme of the metabolic activation of PAHs, was measured. Genetic polymorphisms of glutathione S transferase (GSTs), which can detoxify the carcinogenic activity of the PAHs, were also measured. Inflammatory biomarkers (cytokines, high sensitivity C reactive protein) were also reported in a few studies. The results of these intermediate markers of health risks were contradictory. Four studies reported no difference in the distribution of these markers. Avogbe et al. (8) and Petchpoung et al. (9) reported that there was no statistical significant difference in the protective gene distribution (GST, CYP1A1, Glutathione peroxidase (GPX), NAD(P)H:quinone oxido-reductase 1) between the drivers and controls (rural and suburban residents). Although the level of chromosome break was higher among the urban taxi drivers (n=30), Taghizadeh et al. (10) did not show a statistical different (6.7% vs 3.3%, p=0.3) compared to the rural taxi drivers (n=30). Bagryantseva et al. (11) reported no difference in

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the level of oxidative DNA damage between the drivers and the administrative workers (2.35±2.17 vs 2.55±2.86 % of tail DNA damage, $p>0.05$).

Besides these intermediate outcomes, clinical endpoints were also measured. Lung function parameters, standardized mortality rate, ischemic heart disease mortality, blood cells count were reported. Three studies failed to demonstrate that the commercial drivers had more clinical health risks than their controls. Comparing motorcycle taxi drivers (n=85) and an individual matched control group in Cotonou (Benin), Lawin et al. (12) reported no difference in the prevalence of cough and/or phlegm (Adjusted Odds ratio (AOR) 1.57, 95% Confident Interval (CI) 0.51-4.84) and in lung function parameters (adjusted difference in forced expiratory volume in 1 second (FEV1) 0.12L, 95%CI -0.16-0.22; adjusted difference in forced vital capacity (FVC) 0.11, 95%CI −0.14-0.37). In the same area, Fourn et al. (13) also reported no difference in respiratory symptoms between drivers (n=250) and non-drivers in Cotonou (n=150) (Odds ratio 1.18, 95%CI 0.70-2.00). Ekpenyong et al. (14) in Uyo metropolis (Nigeria) reported a higher frequency of lung function disorders by comparing commercial motorcyclists (n=24) to 6 civil servants (FEV1<80% predicted AOR 1.01, 95%CI 0.942-1.081; FVC<80% predicted AOR 3.10, 95% CI 0.402-16.207) and car taxi drivers (n=18) to the same civil servants (FEV1<80% predicted AOR 1.02, 95%CI

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0.953-1.091; FVC<80% predicted AOR 1,72, 95% CI 0.408-4.732) although the difference was not statistically significant.

Four studies showed evidence of clinical health risks associated with occupational exposure to ambient air pollution in drivers. Avogbe et al. (15) reported a significant difference in white blood cells (10^6/L) among commercial motorcyclists in Cotonou (n=144) compared to 30 rural inhabitants (5.041±1.209 vs 5.900±1.213, p=0.001). Soll-Johanning et al. (16) found that Bus drivers in Copenhagen had an increased risk of lung cancer (Relative risk (RR) 1.6, 95%CI 1.5-1.8) and bladder cancer (RR 1.4, 95%CI 1.2-1.6]. Merlo et al. (17) also found an increased standardized mortality ratio (SMR) in bus drivers (n=6510) from Hodgkin's lymphoma (SMR 2.17, 95%CI 1.19-3.87) and lung cancer (SMR 1.16, 95 1.05-1.28) compared to white collar workers (n=601) in Italy. This risk mortality was increased after 30 years of employment. Hart et al. (18) also reported an increased risk of mortality from ischemic heart disease associated with at least one year of work in the US truck drivers (Hazard ratio 1.44, 95%CI 1.22-1.70).

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4. Discussion

This is the first systematic review to assess the health risks associated with occupational exposure to ambient air pollution in commercial drivers. We found few studies despite the increasing number of people in this occupation especially in LMIC. Most of the articles focused on bus and motorcycles taxi drivers which represent the main methods of public transport in LMICs especially in Africa (12). These methods of transport, especially in African settings, contribute substantially to the level of air pollution in urban cities due to the age of vehicles and fuel quality (19). Our findings showed that commercial drivers had decreased white blood cell counts (15), increased lung and bladder cancer risk (16) and also a higher mortality risk from Hodgkin's lymphoma, lung cancer (17) and ischemic heart disease (18). Oxidative DNA damage, DNA adducts and strand breaks, chromosome aberration that was found in these drivers may help to explain the increased cancer risk.

However, there was a wide variation in the methods and endpoints of the assessed studies. Seven studies found no significant differences between drivers and controls. Hence we were unable to conclude definitively that the health effects reported in drivers were fully attributable to occupational exposure to ambient air pollution

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The main methodological weaknesses of the included studies related to the choice of the comparative study population. In most of the studies drivers were compared to rural/suburban inhabitants, administrative and office workers, policemen, civil servants or other drivers in rural areas. As such the control groups were not always appropriate as they would also have experienced considerable air pollution exposures – for example to household air pollution - and the lack of adjustment for such exposures may have contributed to some of the contradictory findings seen between studies.

Four studies failed to demonstrate that drivers were more exposed than the controls. For three studies (4-6) this is possibly due to a lack of statistical power given their small sample sizes. The fourth study (7) found that controls were more exposed to B [a] P and carcinogenic PAHs than bus drivers. One possible explanation for this is that the use of closed bus driver cabin with closed windows may have reduced their exposure hence the small variation in the air pollution exposure due to a specific job activity. The use of a fixed station without a land use regression model (20) may not then adequately characterize individual exposures.

Three of the cross sectional studies found no difference in respiratory outcomes between drivers and controls (12-14). These contradictory results can be explained by the choice of controls and / or the lack of statistical power as noted

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above. The use of convenience sampling for both drivers and controls and a lack of controlling of confounders variables further explains the contradiction in these studies. The healthy worker effect commonly found in cross sectional study can also explain these results.

Taghizadeh et al. (10) and Bagryantseva et al. (11) also reported no difference in the frequency of the chromosome breaks and DNA damage compared to controls. The genetic polymorphisms of the detoxifying enzymes, their metabolic activation and their distribution in the population can also explain these contradictory results. These genes act as modulators or effect modifiers. GST, CYP1A1 contributed in the metabolic activation of the detoxification of the carcinogenic PAHs. Avogbe et al. (8) and Petchpoung et al. (9) reported that these genes were not homogeneously distributed in drivers and controls and may not be activated in low air pollution exposure especially in low PAH exposure.

There is a need to carry out studies with robust methods to define whether commercial driving is a risky job in relation to the occupational exposure to ambient air pollution. The intermediate health outcomes like the genetic polymorphism of GST, GPX and CYP1A1 may be considered in the assessment of the clinical health risks. They seem to modify the short term clinical health risks but their detoxification ability may be altered in long term. This reduction of their

ability may be associated with the health risks (cancer risk, increase mortality) that were reported in the cohort studies among drivers in Denmark, US and Italy (16-18).

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5. Conclusion

We found some evidence that occupational exposure to ambient air pollution among commercial drivers is associated with adverse health outcomes but the existing literature is limited with few studies of small sample size, methodological weaknesses and contradictory findings. We recommend that future research should have more robust method and take into account the distribution of the genetic polymorphisms. At the same time, there is evidence that exposure to air pollution is harmful to human health with established clean air interventions (including clean air legislation). Alongside further research in this area we recommend that effective interventions for cleaning up the air for all are implemented.

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Table 3: Studies on motorcycle taxi drivers included in the review

Authors	Study Design/ Site	Type of Drivers (number)	Comparative Study Population (number)	Exposure	Outcomes	Key findings
Avogbe, Ayi-Fanou (8)	Cross sectional /Cotonou (Benin)	Motor cycle (n=29)	1.Rural subjects (n=27) 2.Roadside residents (n=37) 3.Suburban subjects (n=42)	1.PM0.1 (fixed site) measured during the working day 2.S-phenylmercapturic acid (S-PMA)	1. oxidative DNA damage in mononuclear blood cells: strand breaks (SB) and formamidopyrimidine glycosylase (FPG) 2. Glutathione S-transferase (GST) 3.Glutathione peroxidase (GPX) 4.NAD(P)H:quinone oxidoreductase 1 (NQO1)	1.Stepwise exposure gradient (rural subjects<suburban subjects<Roadside residents <taxi-moto drivers) 2. NSD in the distribution of most of the genes + inhomogeneous distribution 3. SD in the distribution of SB and FPG sensitive sites
Ayi Fanou, Mobio (6)	Panel study /Cotonou (Benin)	Motorcycle stage 1 (n=35) stage 2 (n=6)	Stage 1 1. Rural subjects (n=6) Stage 2 1. Rural subjects (n=5) 2.Roadside residents (n=12)	1. urine benzene 2. S-PMA 3. 1-hydroxypyrene (1-OHP) 4. Personal exposure to Benzene Toluene Ethyl benzene Xylene (BTEX) per week	1. DNA adducts 2. DNA fragmentation 3. oxidized DNA : 8-hydroxy-2V-deoxyguanosine (8-oxodG) and 5-methylcytosine (m5dC)	1. BTEX and S-PMA in urban drivers >BTEX in rural residents 2. NSD of BTEX and S-PMA difference in taxi-drivers and roadside residents 3. NSD of 1-hydroxypyrene(urban drivers vs. Rural area) 4. SD in DNA damage (urban drivers vs. Rural area inhabitants) but NSD in oxidized DNA (urban drivers vs. Rural area inhabitants)

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Ayi-Fanou, Avogbe (21)	Cross sectional/ Cotonou (Benin)	Motorcycle (n=13)	1. Street vendors (n=16) 2.Gasoline sellers (n=17) 3.Roadside residents(n=11) 4. Suburban residents(n=20) 5. Rural inhabitants	1.Benzene (fixed site)/working day 2.Polycyclic aromatic hydrocarbons (PAHs) mainly benzo(a)pyrene (B[a]P) (6h/day/3 consecutive days) 4. 1-OHP 5. Phenol (urine)	DNA adducts	1. urban drivers >exposed rural inhabitants 2. NSD in Phenol and 1-OH : urban drivers vs Street vendors vs gasoline sellersvs roadside residents 3.DNA adducts of Urban drivers>rural inhabitants
(Ekpenyong CE et al., 14)*	Cross sectional/ Uyo metropolis, (South-South Nigeria)	Motorcycle (n=24) Automobile taxi (n=18)	Civil servants (n=6)	1. CO 2.SO2 3. NO2 4.PM2.5 and PM10 Fixed station /07:30 and 09:30 (peak traffic periods) and 15:30 to 17: 30 (low traffic periods) and some personal exposure	1.Respiratory symptoms 2. Lung function	1. NSD in lung function impairment in drivers vs civil servants 2. More respiratory symptoms among drivers
Fourn and Fayomi (13)	Cross sectional/ Cotonou and Lokossa (Benin)	Motorcycle (n=250 in Cotonou n=150 in Lokossa)	Non drivers in each location	1. personal Carboxyhaemoglobin 2. CO/morning and afternoon/ Fixed station 3.Benzène/morning/ Fixed station	Health disorders (headache, arterial hypertension, respiratory symptoms, digestive disorders, conjunctival hyperemia, photophobia)	1.More health disorders in Cotonou drivers 2. NSD for most of the health disorders especially respiratory symptoms(Drivers vs non drivers in Cotonou)

Patrice H. Avogbe, Lucie Ayi-Fanou (15)	Cross sectional/ Cotonou (Benin)	Motorcycle (n=144)	"Age and sex matched" Rural inhabitants (n=30)	1. benzene (personal) 3. BTEX	12 parameters from complete blood counts: total white blood cells (WBC) with four WBC subtypes (neutrophils, eosinophils, monocytes, and lymphocytes), total red blood cells (RBC) with five red cell-related measures (hemoglobin, hematocrit, mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC) and mean corpuscular hemoglobin (MCH)) and platelets	1.Drivers were more exposed than rural inhabitants 2. Decrease only in white blood cells, lymphocyte and eosinophil counts
Lawin, Agodokpessi (12)	Cross sectionnal / Cotonou (Benin)	Motorcycle (n=85)	Individual matched group (n=85)	CO	Lung function	1. drivers more exposed 2. NSD in lung function and respiratory symptoms

***study on both motorcycle and car taxis drivers SD= statistical difference NSD = No significant statistical difference**

Table 4: Studies on car taxi drivers included in the review

Authors	Study Design/ Site	Type of Drivers (number)	Comparative Study Population (number)	Exposure	Outcomes	Key Findings
Brucker, Moro (4)	Cross sectional /Porto Alegre, Brazil	Automobile Taxi (n=39)	Non occupationally exposed (n=21)	1. Carboxyhaemoglobin (COHb) 2. 1-OHP	1. Platelets 2.Glucose (mg dL−1) 3.Total cholesterol ,HDL cholesterol , LDL cholesterol, Total cholesterol/HDL-c ratio, Triglycerides 3. oxidized-LDL (Ox-LDL) and autoantibodies against ox-LDL (Ox-LDL-Ab) 4. Malondialdehyde (MDA) 5. Protein carbonyl (PCO) 6. Catalase (CAT) 7.Glutathione peroxidase (GPX) 8.GST 9. high sensitivity C reactive protein (hs-CRP) 10. homocysteine (Hcy) 11. Cytokines : Interleukin-1β (IL-1β), IL-6,IL-10, tumour necrosis factor-α (TNF-α), interferon-γ (IFN-γ) 12. Vitamin C	1. drivers >controls for 1-OHP not for COHb 2. NSD for platelets, glucose, total cholesterol 3. drivers > controls for ox-LDL and Ox-LDL-Ab , cytokines,hs-CRP,MDA, PCO 4. Decrease in CAT, GPX, GST, vitamin C among drivers
Burgaz, Demircigil (5)	Cross sectional / Ankara (Turkey)	Automobile drivers (n=7)	Traffic policemen (n=5) Office workers (n=9)	1-OHP	Chromosomal aberration (CA)	1. Controls excreted more 1-OHP than drivers and traffic policemen 2. Drivers had more CA

Taghizadeh, Najmabadi (10)	Cross sectional /Teheran (Iran)	Urban Taxi (n=30)	Rural taxi drivers (n=30)	N/A	1.Chromosome breakage (CB) 2.Cchromosome aberration (CA) rate (including both chromosome and chromatid gaps)	1.Urban drivers had more CA 2.NSD in urban vs rural drivers regarding CB

SD= statistical difference NSD = No significant statistical difference

Table 5: Study on truck drivers included in the review

Authors	Study Design/ Site	Type of Drivers (number)	Comparative Study Population (number)	Exposure	Outcomes	Key Findings
Hart, Garshick (18)	Cohort study (1985-2000)/US	Long haul up (n= 13,752) and Pick-and delivery (P&D) drivers (n=8,930)	Non drivers in trucking industry	Job title and residential exposure to PM10, NO2 and SO2	Ischemic heart Disease(IHD) deaths (number and Hazard ratios for IHD mortality associated with at least one year of work in each specific job category)	Long haul drivers had more IHD deaths Hazard ratio=1.44 [1.22, 1.70]

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Table 6: Studies on Bus drivers included in the review

Authors	Study Design/ Site	Type of Drivers (number)	Comparative Study Population (number)	Exposure	Outcomes	Key Findings
Bagryantseva, Novotna (11)	Cross sectional/ Prague (Czech Republic)	Bus (n=50)	1. Garagmen (n=20) 2. Administrative workers (n=50)	1. total carcinogenic PAHs including B[a]P)/ 48h 2. BTEX / 24h	1. Percentage of DNA in the tail (Tail DNA %). 2.Total DNA damage (with enzymes) 3. DNA-SB or unspecified DNA damage; without enzymes) 3. urinary excretion of 8-oxodG 4. Urinary 15-F2t-IsoP (oxidative damage to lipids) 5. protein carbonyl 6. Polymorphisms of metabolic genes (CYP1A1, GSTM1, GSTP1, GSTT1, EPHX3,4), folic acid metabolism genes(MS,MTHFR)and DNA repair genes(XRCC1,XPD6,XPD23, hOGG1)	1.Drivers were more exposed than administrative workers 2. Almost the same exposure for drivers and garagmen (p value not shown) 3. NSD in Tail DNA% (drivers vs administrative workers) 4.Drivers had more DNA-SB, 8-oxodG, 15-F2t-IsoP than administrative workers 5.Almost the same oxidative damage (drivers vs garagmen, p value not shown)
Han, Donovan (22)	Cross sectional/ Taiwan	Bus (n=120)	Office workers (n=58)	N/A	8-oxodG (24 hours sampling)	drivers>office workers

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Hansen, Wallin (23)	Cross sectional(Denmark)	Bus (n=60)	Mail carriers (n=88)	1-OHP (working day and day off)	N-acetyltransferase (NAT2) phenotype	Drivers were more exposed than mail carriers
Merlo, Stagi (17)	Cohort study/ Genoa (Italy) 1970-2005	Bus (n=6510)	1.Maintenance workers (n=2073) 2. White collar (n=601)	Job title	Standardized mortality ratios (SMRs)	SMRs for all causes of deaths and lung diseases: maintenance workers>drivers>white collar
Nielsen, de Pater (24)	Cross-sectional/ Copenhagen (Denmark)	Bus(n=90) Divided regarding gradient of exposition (central, dormitory and suburban	Rural inhabitants (n=60)	N/A	DNA adducts	Drivers had more DNA adducts
Petchpoung, Kaojaren (9)	Cross sectional/ Bangkok (Thailand)	Bus (n=100)	Rural inhabitants (n=100)	1-OHP	1. cytochrome P4501A1 (CYP1A1) 2. GSTM1 3.GSTP1 4.GSTT1	1. Driver excreted more 1-OHP 2. The genotype distribution was almost the same
Rossner, Svecova (7)	Cross sectional / Prague	Bus (n=50)	controls (n=50) healthy male volunteers	PM 2.5 PM 10 cPAHs (B[a]P)	1.PCO 2.8-oxodG 3.15-F2t-IsoP 4.nitrotyrosine (NT)	1. cPAHs: controls>drivers 2. More oxidative stress in drivers

	(Czech Republic)		spending >90% of daily time indoors			
Rossner, Svecova (25)	Cohort/ Prague (Czech Republic) 03 seasons	Bus (n=50)	controls (n=50) healthy male volunteers spending >90% of daily time indoors	PM 2.5 PM 10 cPAHs (B[a]P) BTEX Personal/fixed monitoring	1. PCO 2. 15-F2t-IsoP	PCO and 15-F2t-IsoP: drivers>controls in both winter(2005-2006) but not in summer
Soll-Johanning, Bach (16)	Cohort/Copenhagen (Denmark)	Bus (n=18 120)	Other people in Denmark	Job title	Cancer risk	drivers>general population lung cancer rates [relative risk (RR) = 1.6,95%confidence interval (95%CI) = 1.5–1.8] and bladder cancer rates (RR = 1.4, 95% CI = 1.2–1.6)

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