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# 1 Individual exposure level following indoor and outdoor air 2 pollution exposure in Dakar (Senegal)

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4 A. NDONG BA<sup>1,2</sup>, A. VERDIN<sup>1\*</sup>, F. CAZIER<sup>3</sup>, G. GARCON<sup>4,5</sup>, J. THOMAS<sup>4</sup>, M.  
5 CABRAL<sup>2</sup>, D. DEWAELE<sup>3</sup>, P. GENEVRAY<sup>3</sup>, A. GARAT<sup>4,5</sup>, D. ALLORGE<sup>4,5</sup>, A. DIOUF<sup>2</sup>,  
6 J.M. LOGUIDICE<sup>4,5</sup>, D. COURCOT<sup>1</sup>, M. FALL<sup>2</sup>, M. GUALTIERI<sup>1#</sup>

7

8 <sup>1</sup> Unité de Chimie Environnementale et Interactions sur le Vivant (UCEIV) EA 4492, SFR  
9 Condorcet FR CNRS 3417, Maison de la Recherche en Environnement Industriel, Université  
10 du Littoral Côte d'Opale, Dunkerque

11 <sup>2</sup> Laboratoire de Toxicologie et d'Hydrologie, Faculté de Médecine, de Pharmacie et  
12 d'Odontologie, Université Cheikh Anta Diop, Dakar

13 <sup>3</sup> Centre Commun de Mesures, Maison de la Recherche en Environnement Industriel,  
14 Université du Littoral Côte d'Opale, Dunkerque

15 <sup>4</sup> Laboratoire de Toxicologie, Centre de Biologie-Pathologie-Génétique, CHRU de Lille

16 <sup>5</sup> IMPact de l'Environnement Chimique sur la Santé humaine (IMPECS), EA 4483, Univ.  
17 Lille

18 # Present address ENEA – SSPT – MET – INAT Via Martiri di Monte Sole, 4 Bologna Italy

19 \* Corresponding author: Anthony VERDIN (Anthony.Verdin@univ-littoral.fr)

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34 **Abstract**

35 The consequences of indoor and outdoor air pollution on human health are of great concern  
36 nowadays. In this study, we firstly evaluated indoor and outdoor air pollution levels (CO,  
37 CO<sub>2</sub>, NO, NO<sub>2</sub>, PM<sub>10</sub>) at an urban site in Dakar city center and at a rural site. Then, the  
38 individual exposure levels to selected pollutants and the variations in the levels of biomarkers  
39 of exposure were investigated in different groups of persons (bus drivers, traders working  
40 along the main roads and housemaids). Benzene exposure levels were higher for housemaids  
41 than for bus drivers and traders. High indoor exposure to benzene is probably due to cooking  
42 habits (cooking with charcoal), local practices (burning of incense), the use of cleaning  
43 products or solvent products which are important emitters of this compound. These results are  
44 confirmed by the values of S-PMA, which were higher in housemaids group compared to the  
45 others. Urinary 1-HOP levels were significantly higher for urban site housemaids compared to  
46 semirural district ones.

47 Moreover, urinary levels of DNA oxidative stress damage (8-OHdG) and inflammatory  
48 (interleukin-6 and -8) biomarkers were higher in urban subjects in comparison to rural ones.

49 The air quality measurement campaign showed that the bus interior was more polluted with  
50 PM<sub>10</sub>, CO, CO<sub>2</sub> and NO than the market and urban or rural households. However, the interior  
51 of households showed higher concentration of VOCs than outdoor sites confirming previous  
52 observations of higher indoor individual exposure level to specific classes of pollutants.

53  
54 **Keywords:** Air pollution; Volatile organic compounds exposure; Biomarkers of exposure;  
55 Indoor and outdoor air pollution exposure; Personal exposure evaluation.

56  
57 **Capsule:** Outdoor and indoor air pollution analysis and human biomarker exposure  
58 evaluation

## 59 **Introduction**

60 Indoor and outdoor air pollution is among the main causes of morbidity and mortality in the  
61 world (Cohen et al., 2017; IARC, 2014; Merbitz et al., 2012; Raaschou-Nielsen et al., 2013).

62 The assessment of environmental exposure to air pollution is, therefore, a major challenge for  
63 understanding and evaluating the potential impact of this pollutant in exposed population.

64 Road traffic is an important emission source of various organic compounds (such as volatile  
65 compounds - VOCs, like benzene, toluene, xylenes, and polycyclic aromatic hydrocarbons -  
66 PAHs) and others compounds deriving mainly from vehicle exhausts. Outdoor VOCs and  
67 PAHs extensively affect outdoor air quality and are mainly generated during the incomplete  
68 combustion of fuels (both gasoline and diesel), while indoor VOCs are related to specific  
69 sources which determines the type of molecules observed in confined environments (Sahlberg  
70 et al., 2013; Salthammer, 2016). Many of these organic compounds are known to be  
71 carcinogens (IARC, 2014; Filippini et al., 2015) and to cause pulmonary and cardiovascular  
72 diseases and immune system impairments (Alshaarawy et al., 2016; Hausmann, 2012). Since  
73 the majority of the studies on air pollution and related human exposure has been conducted in  
74 developed countries (Maitre et al., 2002; Schneider et al., 2001; Smith et al., 2007; Sorensen  
75 et al., 2003), there is a urgent need of similar information from studies performed in  
76 developing countries and especially from those areas (such as the sub-Saharan Africa) where,  
77 in the last years, an uncontrolled urbanization and old fleets of circulating vehicles generate a  
78 significant emission of air pollutants (Assamoi & Liousse, 2010; Baumbach et al., 1995).  
79 Dakar, the capital of Senegal, can be considered representative of these conditions. In Dakar  
80 the circulating vehicles are mainly characterized by old and inefficient combustion  
81 technologies. As a consequence, higher concentration of air pollutants are registered alongside  
82 roadways, and the individuals working along the major streets of the city may incur in  
83 substantial exposures to traffic-related air pollution (Knibbs & Morawska, 2012).

84 Beside outdoor air pollution, in the last years specific attention has been paid also to indoor  
85 air pollution and the consequent human exposure (Huang et al., 2019). Although the relation  
86 between outdoor and indoor air pollution, which vary depending on factors that can influence  
87 the indoor levels, e.g. climate, emission sources, human activity and building ventilation  
88 (Monn, 2001), it is well recognize that indoor emissions may greatly influence building air  
89 quality (Huang et al., 2019; Tang et al., 2016).

90 Epidemiological investigation may provide important information on the actual exposure of a  
91 population thanks to the selection of biomarkers of exposure/effect. Indeed biomarkers may  
92 reflect the total exposure of an individual to a selected (class of) pollutant. Several studies

93 used the urinary concentration of selected metabolites of organic compounds and/or other  
94 biomolecules to show a significant increase of these biomarkers in population close to the  
95 specific source of emission, such as traffic exhausts (Amodio-Cocchieri et al., 2001; Arayasiri  
96 et al., 2010; Fustinoni et al., 2005).

97 Among the different biomarkers of exposure S-phenylmercapturic acid (S-PMA), muconic  
98 acid (t,t-MA) and catechol are representative of the metabolic transformation of benzene,  
99 (Snyder et al., 1993; Waidyanatha et al., 2001). The urinary excretion of S-PMA and t,t-MA  
100 has been used in epidemiological investigation in occupational and environmental studies  
101 when benzene was considered the primary pollutant of interest (Ayi Fanou et al., 2006;  
102 Borgie et al., 2014; Fustinoni et al., 2005). Unfortunately, under environmental conditions,  
103 people are exposed to several airborne compounds among which also PAHs (Fan et al., 2012).  
104 One of the biomarkers normally used to evaluate the exposure to PAHs mixtures is the urinary  
105 concentration of 1-hydroxypyrene (1-OHP) (Ovrebo et al., 1995). Although biomarkers of  
106 exposure give significant information on the interaction of the selected population with the  
107 pollutants of interest, it is also important to evaluate also the levels of markers representative  
108 of the possible effects induced by air pollutants. Oxidative stress is considered a significant  
109 process of damage activated by air pollutants, such as PAHs and VOCs. Uncontrolled  
110 oxidative stress may in turn determine the oxidative damage of biological macromolecules,  
111 such as DNA, RNA and phospholipids. A largely used biomarker of oxidative damage is the  
112 level of 8-hydroxy-2'-deoxyguanosine (8-OHdG) in urine. This molecule results from  
113 oxidation of the DNA base guanine and has been already proposed as biomarker to evaluate  
114 the damages determined by ambient air pollution (Svecova et al., 2009) and indoor air  
115 pollution (Commodore et al., 2013). Finally, among the processes triggered by air pollution,  
116 lung epithelia inflammatory responses are considered of primary importance (Podechard et  
117 al., 2008; Umannova et al., 2011; Vogel et al., 2005) for understanding the health impacts of  
118 airborne pollutants. The inflammatory mediators, such as tumor necrosis factor alpha (TNF-  
119  $\alpha$ ), interleukin-6 (IL-6), interleukin-8 (IL-8), may be released also in the systemic circulation  
120 and finally excreted in urine (Sawyer, 2010; Van Eeden et al., 2001) and may therefore serve  
121 as additional biomarkers of the negative effects of air pollutants on exposed population.

122 The aim of this study was therefore to assess, for the first time in a sub-Saharan region, the  
123 individual exposure level to air pollution, mainly volatile organic compounds (namely  
124 benzene, toluene, ethylbenzene and xylene – BTEX) and PM, and its potential health impact,  
125 by the quantification of selected biomarkers of exposure/effects. The biomarkers were  
126 quantified in groups of person representative of professionals (bus drivers and traders)

127 exposed mainly to outdoor air pollution and of housemaids mainly affected by indoor air  
128 pollution, selected in Dakar city center. Moreover the effects of indoor air pollution on the  
129 levels of biomarker of exposure/effects were also evaluated in a group of housemaids working  
130 in a semi-rural area located 45 km from the city center to evaluate possible differences  
131 between urban and non-urban areas on indoor air quality and related potential effects.

132

## 133 **1. Materials and methods**

### 134 **1.1. Study population**

135 This cross-sectional study took place in the city of Dakar, at the urban district of HLM where  
136 the road traffic is particularly intense and at a semirural district, Toubab Dialaw (TD), that is  
137 located 45 Km west from the urban site (**Fig. S1**).

138 A non-probability sampling technique was used to select subjects. 57 professionals and 59  
139 housemaids were selected. Professionals were representative of bus drivers (27 participants  
140 included) and traders (30 participants located along the street where traffic is more intense and  
141 in the main market of HLM). These two categories were considered exposed mainly during  
142 their working hours and mainly to outdoor air pollution. Housemaids (29 recruited at the  
143 urban site and 30 at rural site) were selected as working exclusively inside the houses during  
144 their working hours and therefore exposed mainly to indoor air pollution (for more details see  
145 supplementary material).

### 146 **1.2. Sample collection**

147 Individual exposure to BTEX (benzene, toluene, ethylbenzene and xylene) was monitored  
148 from March 13 to 31, 2017 using badges GABIE (Gas Adsorbent Badge for Individual  
149 Exposure). These passive samplers were worn by the subjects, as close as possible to the  
150 inhalation area (mouth/nose), daily during 8 h of the working time and then kept at -80 °C  
151 until chemical analysis. Urine spot samples were collected by each subject at the end of the 8  
152 h of monitoring period each day for the determination of S-phenylmercapturic acid (S-PMA),  
153 trans, trans-muconic acid (t,t-MA), 1-hydroxypyrene (1-OHP), 8-hydroxy-2- $\alpha$ -  
154 désoxyguanosine (8-OHdG) and inflammatory cytokines (TNF- $\alpha$ , IL-1 $\beta$ , IL-6 and IL-8). Each  
155 urine sample was divided into aliquots and quickly frozen at -80 °C until analyses.

### 156 **1.3. Analysis of BTEX**

157 BTEX were desorbed from the activated charcoal by using 5 mL of carbon disulfide under  
158 ultrasound for 15 min. The extract was transferred into a 2 mL vial then analyzed by gas  
159 chromatography (CP-3800, Varian USA) coupled to a flame ionization detection as reported

160 in Liu et al. (2009). Xylenes were resolved as ortho (o-) and meta (m-) + para (p-) xylene. As  
161 a result of the selected column used for the chromatography procedure, m- xylene and p-  
162 xylene were not discriminated.

#### 163 **1.4. Urinary metabolites**

##### 164 **1.4.1. S-phenylmercapturic acid (S-PMA)**

165 Urinary S-PMA was analyzed using a Waters Acquity ultra-performance liquid  
166 chromatography (UPLC) coupled with a tandem mass spectrometer detector (Waters XEVO  
167 TQS Waters, Manchester, UK). The analysis was performed at 40 °C using a gradient elution  
168 of ammonium formate and acetonitrile (**Table S1**). Results were collected by TargetLynx 4.1  
169 software (Waters) (for more details see supplementary material).

##### 170 **1.4.2. Trans,trans-muconic acid (t,t-MA)**

171 The t,t-MA was extracted from urine samples by a semi-liquid phase extraction using  
172 disposable cartridges (Varian bond elut SAX 500 mg 3 mL) and quantification of the benzene  
173 metabolite was carried out by a high pressure liquid chromatography (HPLC) coupled with a  
174 UV spectrometer (ALLIANCE 2695, Waters, Watford, UK) coupled with a PDA UV detector  
175 (2998 WATERS) at 259 nm wavelength (for more details see supplementary material).  
176

##### 177 **1.4.3. 1-Hydroxypyrene (1-HOP)**

178 Determination of 1-HOP was performed according to by Li et al. (2005) using a Waters  
179 Acquity ultra performance liquid chromatography (UPLC) coupled with a detector Waters  
180 XEVO TQS tandem mass spectrometer (Waters, Manchester, UK). Results were acquired by  
181 TargetLynx software 4.1 (Waters) (for more details see supplementary material).  
182

##### 183 **1.4.4. 8-Hydroxy-2- $\alpha$ -désoxyguanosine (8-OHdG)**

184 8-OHdG concentration in urine was studied using a commercially available enzyme  
185 immunoassay (Highly Sensitive 8-OHdG Check, Gentaur France SARL), as published  
186 elsewhere (Leclercq et al., 2017).

##### 187 **1.4.5. Urinary creatinine**

188 Values of urinary metabolites were related to those of creatinine, as evaluated by the Jaffé  
189 method using a Roche Diagnostics kit (Roche Diagnostics, France). Samples with urinary  
190 creatinine < 350 mg/L were excluded from the study.

#### 191 **1.5. Cytokines concentration measurement**

192 TNF- $\alpha$ , IL-1 $\beta$ , IL-6 and IL-8 concentrations in urine were determined using commercially  
193 available enzymatic immunoassays (Milliplex<sup>®</sup>Map, Human Cytokine/Chemokine Magnetic  
194 Bead Panel Luminex<sup>®</sup>, EDM Millipore, USA), scrupulously following the manufacturer's  
195 instructions. The results were expressed as pg.mL<sup>-1</sup>.

### 196 **1.6. Air quality analysis by EVM-7 and AQ Pro**

197 The campaign of the air quality measurement in Dakar was held from November 11, 2017 to  
198 March 30, 2018. Two different devices were adopted: the EVM-7 environmental monitor and  
199 the AQ Pro indoor air quality monitor. The EVM-7 (version 1.05 Quest Technologies) is a  
200 compact environmental monitor for the measurement of the airborne concentrations of PM<sub>10</sub>  
201 and gases (CO<sub>2</sub> and CO). It also measures meteorological parameters such as humidity and  
202 temperature. A simultaneous, multi-sensor gas detector, AQ Pro indoor air quality monitor,  
203 was configured to detect the concentration of NO and NO<sub>2</sub> in ppm.

204 As a first step, we measured the level of air quality at the four main roads of the HLM market,  
205 at the rate of 3 measurements per axis and in the 27 buses involved in the campaign. In a  
206 second step, we simultaneously assessed the indoor and outdoor air quality levels of the 29  
207 households at urban site (HLM) and the 30 households at rural site (TD). The devices were  
208 placed about 1 m above the ground, in the living room (inside) and on the sidewalk at the  
209 house's door (outside). The measurements were carried out for 8 hours.

### 210 **1.7. Statistical analysis**

211 Data processing was performed with the statistical analysis software SPSS for windows,  
212 v.22.0.0, Oct. 2016: p-values less than 0.05 were considered significant. One-factor ANOVA  
213 followed by Tukey tests were used to compare means values obtained between the different  
214 groups. Moreover, Spearman correlation analyses were used to evaluate the relationship  
215 between BTEX and urinary biomarkers. Data from multi-pollutants measurements between  
216 interior and exterior of dwellings are reported as mean and standard deviation and U non-  
217 parametric test Mann-Whitney was used to determine the statistical differences.

218

## 219 **2. Results and discussion**

### 220 **2.1. BTEX concentrations**

221 BTEX analysis by gas chromatography coupled to flame ionization detection revealed that  
222 toluene was the most encountered pollutant with concentrations ranging from 800 to 1950  
223  $\mu\text{g}/\text{m}^3$ . Benzene and xylenes were ranging from 150 to 650  $\mu\text{g}/\text{m}^3$  while ethylbenzene  
224 concentration was about 50  $\mu\text{g}/\text{m}^3$  (**Fig 1** and **Table S2** in supplementary information).



225 These results are in agreement with previous works where toluene was reported to have the  
226 highest concentration of BTEX in urban areas: Miri et al. (2016) ( $16 \mu\text{g}/\text{m}^3$ ) in Tehran (Iran),  
227 Pekey and Yılmaz (2011) ( $35 \mu\text{g}/\text{m}^3$ ) in Turkey, Hazrati et al. (2016) ( $45 \mu\text{g}/\text{m}^3$ ) in Ardabil  
228 (Iran) and Borgie et al. (2014) ( $11520 \mu\text{g}/\text{m}^3$ ) in Lebanon. **Fig. 1B** highlights the differences  
229 of toluene concentrations between outdoor professional groups ( $1922$  and  $1023 \mu\text{g}/\text{m}^3$  for  
230 traders and bus drivers respectively) and TD housemaids ( $867 \mu\text{g}/\text{m}^3$ ). The outdoor  
231 professional activities might determine a prolonged proximity to main streets and/or  
232 congested vehicular traffic and therefore a substantial increased occupational exposures to  
233 traffic-related air pollution (Knibbs & Morawska, 2012) during the working hours of the  
234 selected participants, as also suggested by the toluene concentration exposure here reported.  
235 However, the average concentration of BTEX found in HLM housemaids group ( $1454 \mu\text{g}/\text{m}^3$ )  
236 was significantly higher than those of drivers. This result may suggest a major exposure for  
237 those groups of participants spending their working hours at a specific urban site (regardless  
238 of whether the activity is indoor or outdoor) rather than the one moving around the city.

239 Benzene concentrations (**Fig. 1A**) were significantly higher for housemaids ( $649$  and  $568$   
240  $\mu\text{g}/\text{m}^3$  respectively for urban and rural sites) than for professionals ( $192$  and  $425 \mu\text{g}/\text{m}^3$   
241 respectively for bus drivers and traders). This difference is probably due to local culinary  
242 habits (coal cooking), specific indoor practices (important incense burning) and use of  
243 products that are important emitters of benzene (Olsson & Petersson, 2003), but is also likely  
244 the indoor accumulation and concentration of outdoor emissions (Weisel, 2010). Benzene  
245 concentration in our study was higher than the level reported in Cotonou, Benin ( $60 \mu\text{g}/\text{m}^3$ )  
246 (Ayi Fanou et al., 2006), in Lagos, Nigeria ( $250 \mu\text{g}/\text{m}^3$ ) (Baumbach et al., 1995), in Torino,  
247 Italy ( $10.3 \mu\text{g}/\text{m}^3$ ) (Bono et al., 2003), in Grenoble, France ( $23.5 \mu\text{g}/\text{m}^3$ ) (Maitre et al., 2002)  
248 and in Copenhagen, Denmark ( $2.5 \mu\text{g}/\text{m}^3$ ) (Sorensen et al., 2003).

249 Ethylbenzene was the least representative VOC with concentration of about  $50 \mu\text{g}/\text{m}^3$   
250 whatever the population considered ( $53$ ,  $59$ ,  $63$  and  $45 \mu\text{g}/\text{m}^3$  for rural and urban housemaids,  
251 traders and bus drivers, respectively) (**Fig. 1C**).

252 Finally, among the xylene isomers (**Table S2** and **Fig. 1 D-E**), o-xylene was the most  
253 abundant in all the analyzed samples and higher for the urban groups (traders and  
254 housemaids) than for the other groups. In Borgie et al. (2014) traffic policemen were exposed  
255 mainly to m- and o-xylene, similarly Kim et al. (2012) reported higher urban air concentration  
256 of m+p xylene than o-xylene. This difference may be related to the different sources of  
257 emissions or to different secondary reaction in the atmosphere of the xylene isomers (Zhang  
258 et al., 2019).

259 Although below the recommended values by the Occupational exposure limit in France (3250,  
260 76800, 88000 and 221000  $\mu\text{g}/\text{m}^3$  for benzene, toluene, ethylbenzene and xylene,  
261 respectively), BTEX concentrations here reported go beyond ATSDR's chronic MRL  
262 (Minimal Risk Level according to the Agency for Toxic Substances and Disease Registry):  
263 9.8  $\mu\text{g}/\text{m}^3$  for benzene, 300  $\mu\text{g}/\text{m}^3$  for toluene and 200  $\mu\text{g}/\text{m}^3$  for xylene (INRS, 2016).  
264 Just a minor correlation (between o-toluene and xylene) was found among the parameters  
265 analysed for the drivers and housemaids in rural site, whereas excellent spearman correlation  
266 ( $p < 0.001$ ) between the different BTEX for the traders and housemaids of the urban site is  
267 reported (**Table S3**). These results are in agreement with previous studies that reported strong  
268 correlations between BTEX compounds (Dehdari et al., 2014; Hoque et al., 2008; Miller et  
269 al., 2012) at urban sites of sampling.

270 This means that the BTEX of the first two groups (traders and urban housemaids) are likely to  
271 come from common urban emission sources, and therefore it is likely that indoor levels are  
272 influences also by outdoor emission sources, while those of the rural housemaids probably  
273 have different sources of emission. Drivers' case differs from the others since this group is  
274 representative of persons that are always moving around the city and their exposure to BTEX  
275 can hugely be influenced by other local/transient sources.

276 In a general, the total BTEX concentration found in this study was considerably higher than  
277 that obtained by other studies conducted in developed countries, such as Germany (46.5  
278  $\mu\text{g}/\text{m}^3$ ) (Schneider et al., 2001) and USA (6.7  $\mu\text{g}/\text{m}^3$ ) (Smith et al., 2007). The differences  
279 observed can be explained by specific characteristics of traffic such as the quality of fuels  
280 and/or the number and the mean technology of circulating vehicles while domestic activities  
281 significantly impact the levels of indoor pollution.

282

## 283 **2.2. Biomarkers of exposure**

284 Benzene can be transformed into primary and secondary metabolites that are usually excreted  
285 in urine and can serve as biomarkers of professional or environmental exposure. In this study,  
286 urinary S-PMA and t,t-MA levels were considered as biomarkers of environmental exposure  
287 to benzene for the enrolled subjects at the urban site (drivers, traders and HLM housemaids)  
288 and the ones enrolled at the semirural district (TD housemaids).

289 Due to low sensitivity of the technique, the urinary concentration of t,t-MA was not detected  
290 (values below the LOD) in all the collected samples. S-PMA concentration was found, for the  
291 sites we selected, to be a better biomarker of benzene exposure than t,t-MA. Results showed  
292 that the median S-PMA values were higher for housemaids (1.3 and 1.2  $\mu\text{g}/\text{g}$  creatinine for

293 urban and rural group, respectively) than for professionals (1.1 and 0.8  $\mu\text{g/g}$  creatinine for  
294 traders and drivers, respectively). However, post hoc analysis indicated that only city  
295 housemaids had a significant higher urinary excretion of S-PMA compared to the traders (**Fig.**  
296 **2A**). S-PMA contents were lower than those found in a previous study conducted in Benin,  
297 where a mean S-PMA concentration of 9.3  $\mu\text{g/g}$  creatinine was reported for motorcycle taxi  
298 drivers compared to 4.2  $\mu\text{g/g}$  for residents living in a village without traffic emissions (Ayi  
299 Fanou et al., 2006). Similarly, in a previous study in Lebanon (Borgie et al., 2014), the pre  
300 and post-shift S-PMA levels for traffic policemen (6.2 and 5.8  $\mu\text{g/g}$  creatinine, respectively)  
301 and for office policemen (2.6 and 2.7  $\mu\text{g/g}$  creatinine) were higher than those reported in this  
302 study despite here we reported higher individual levels of benzene exposure. This difference  
303 could be related to a co-exposure of benzene with other chemicals and additives emitted from  
304 gasoline vehicles (Barbieri et al., 2008) such as toluene (a competitive inhibitor of benzene  
305 for CYP metabolism) which could affect the pharmacokinetics and metabolism of benzene,  
306 including conjugation with glutathione and subsequent mercapturic acid excretion (Johnson et  
307 al., 2007).

308 The median level of 1-HOP, used to assess pyrene exposure was slightly higher in drivers (0.4  
309  $\mu\text{g/g}$  creatinine) compared to others groups (0.2, 0.3 and 0.2  $\mu\text{g/g}$  creatinine respectively for  
310 traders, urban housemaids and semirural housemaids), but without significant differences.  
311 Statistical analysis showed that urinary 1-HOP levels were significantly higher for urban site  
312 housemaids compared to semirural district ones (**Fig. 2B**). These values were higher than  
313 those observed in diesel exhaust-exposed workers in Denmark (0.11  $\mu\text{g/g}$ ; Nielsen et al.,  
314 1996), in Brazil (0.11  $\mu\text{g/g}$ ; Brucker et al., 2013) and in Copenhagen (0.25  $\mu\text{g/g}$ ; Autrup et al.,  
315 1999) but lower compared to the level reported for workers of coke oven (5.5  $\mu\text{g/g}$ ; Ovrebo et  
316 al., 1995).

317 The excretion of 1-OHP in urine can be significantly influenced also by lifestyle (Castano-  
318 Vinyals et al., 2004). Food has previously been shown to be an important source of pyrene  
319 (Knize et al., 1999) and specific cooking activities may determine a high exposure to organic  
320 compounds known to pose significant risks to health (Weinstein et al., 2017).

321 The high concentrations of BTEX here reported may be related to common local practices,  
322 such as the use of wood and charcoal as cooking fuels, incense burning and lack of ventilation  
323 of indoor environments. Also the use of cleaning products or solvents may increase the  
324 personal exposure to volatile compounds. Lifestyle is therefore an important contributor to  
325 PAHs and VOCs exposure and might explain the differences between the pyrene biomarker of  
326 exposure between the indoor urban and rural population (**Table S3**). The correlation of 1-

327 HOP with other VOCs (toluene and xylene concentrations  $p < 0.05$ ) for housemaids at the  
328 urban site may indicate that this population is exposed at the same time to indoor and outdoor  
329 sources. Interestingly, S-PMA had significant correlation with benzene among housemaids in  
330 rural site ( $p < 0.05$ ) and traders ( $p < 0.01$ ) therefore confirming the importance of this  
331 metabolite for assessing personal exposure to benzene (**Table S3**).  
332 These results clearly showed that indoor air pollution was a major driver for the personal  
333 exposure to BTEX in the selected populations and as consequence this poses a urgent need to  
334 regulate or, at least, to activate campaigns of information to share the potential impact of  
335 indoor activities in generating volatile organic pollutants.

### 336 **2.3. Oxidative stress and inflammation biomarkers**

337 Urinary levels of 8-OHdG were higher for urban site housemaids ( $2.7 \mu\text{g/g creatinine}$ ) when  
338 compared to rural site ones ( $1 \mu\text{g/g creatinine}$ ) (**Fig. 3**). These results support the importance  
339 of urban air pollution emissions in determining biological effects and the higher impact of  
340 urban pollution compared to rural one. Road traffic is an important source of air pollution in  
341 African cities. United Nations estimates that roads account for up to 90% of urban air  
342 pollution in developing countries (UNEP, 2011). We have recently reported (Ndong et al,  
343 2019) that the traffic emissions are major contributors to air pollution at the urban site  
344 compared to the rural one. These emissions contribute to the risk of chronic respiratory  
345 diseases such as asthma and Chronic Obstructive Pulmonary Disease (COPD) among  
346 professionals exposed to air pollution related to road traffic. Indeed, recent studies found a  
347 high prevalence of allergic rhinitis among vendors in Dakar city (Sylla et al., 2017) and  
348 showed a higher prevalence of chronic respiratory diseases and a decreased lung function in  
349 bus drivers in the HLM neighborhood in Dakar (Sylla et al., 2018). The results here reported  
350 are in agreement with these studies showing that urinary level of 8-OHdG is significantly  
351 higher in HLM housemaids in comparison to the ones of the rural site Toubab Dialaw. The  
352 lack of high positive correlation (**Table S3**) between 8-OHdG and the VOCs we analysed  
353 suggests also that the increase in 8-OHdG may be related to other organic and/or inorganic  
354 compounds not analysed in this study and suggest the possibility that other air pollution  
355 parameters (such as fine particulate matter) should be taken into account for understanding the  
356 observed levels of this biomarker.  
357 The 8-OHdG values found in this study were lower compared to the level in women exposed  
358 to wood smoke in a cook stove emissions reported in Commodore et al. (2013) which  
359 analysed this biomarker in San Marcos, Peru ( $132.6 \mu\text{g/g creatinine}$ ; 2013), and lower than the

360 values reported for pregnant rural Guatemalan women cooking and heating with solid fuels  
361 (91.3  $\mu\text{g/g}$  creatinine; Weinstein et al., 2017) and in a cross-sectional sample of 48 green  
362 space workers in Brussels, Belgium (between 7.05 and 20.92  $\mu\text{g/g}$  creatinine; Guilbert et al.,  
363 2019). The variability in results may be due to the variety of confounding factors associated  
364 with urinary oxidant concentrations, such as age, physical activity and vitamin status (Pilger  
365 and Rudiger, 2006; Romanazzi et al., 2013) and above all to the exposure to oxidative  
366 damaging compounds inhaled or intake by diet or other exposure routes.

367 As shown in **Fig. 4**, a release of pro-inflammatory biomarkers, such as IL-6 and IL-8 was  
368 higher for housemaids at urban and rural sites than for professionals but no significant  
369 differences were found between the different groups. IL-1 $\beta$  and TNF- $\alpha$  were low and did not  
370 vary between subjects. Mean IL-6 values here reported (6.7, 6.1, 8.3 and 6.4 pg/mL for  
371 drivers, traders, city and semirural housemaids, respectively) were similar to those observed  
372 in healthy control subjects population (8 pg/mL) and clearly lower than subjects with  
373 congestive heart failure (24 pg/mL) assessed in Spain while TNF- $\alpha$  concentrations were  
374 slightly higher (2 and 4 pg/mL respectively for control and sick subjects) (Sirera et al., 2003).  
375 This might suggest that specific indoor contaminants could determine the increase of the  
376 inflammatory response.

#### 377 **2.4. Air quality measurements**

378 The results of air quality measurements showed that CO and CO<sub>2</sub> concentrations were  
379 significantly higher for the drivers population (1.7 ppm for CO and 633.2 ppm for CO<sub>2</sub>) than  
380 in others environments (**Fig. 5A** and **5B**). Similar CO concentration rates were found in Hong  
381 Kong buses (1.9 ppm) (Chan et al., 2002), therefore suggesting a specific occupational  
382 exposure. However, the values reported by Limasset et al. (1993) were 11.3 ppm and 12.5  
383 ppm in Paris and Bordeaux buses, respectively. In a comparative study between various  
384 travelling habits (walking, motorcycles, light vehicles and buses), Saksena et al. (2008)  
385 reported a mean concentration of 11.5 ppm for CO in buses. the American Conference of  
386 Governmental Industrial Hygienists (ACGIH) has set a 25-ppm TWA (Time-Weighted  
387 Average) threshold in the workplace, and the National Institute for Occupational Safety and  
388 Health (NIOSH) has proposed a maximum average concentration (TWA-8 hours) of 35 ppm  
389 with a maximum acceptable limit of 200 ppm for 8 hours of work. Considering these limits,  
390 the CO concentrations here reported comply with international standards on indoor air quality  
391 and occupational exposures and should not pose any risk for the exposed population.

392 NO, released into the atmosphere in presence of ozone from the air and under the influence of  
393 solar radiation, becomes NO<sub>2</sub>. **Figure 5C** revealed that NO levels were significantly higher in

394 the buses (0.1 ppm) than those found at the urban site houses (0.0 ppm). No significant  
395 difference was found between NO<sub>2</sub> concentrations at the different environments (**Fig. 5D**).  
396 NO concentrations in our study are lower than the accepted average exposure value of 25 ppm  
397 (INRS, 2006). Our results agree with those reported in Limasset et al. (1993) which found NO  
398 concentrations ranging from 0.5 to 0.6 ppm over a 90 to 130 minute paths in Paris and  
399 concentrations from 0.07 to 0.1 ppm for NO<sub>2</sub> for the same travel times.

400 PM<sub>10</sub> (Particulate Matter) are particulates with aerodynamic diameter less than 10 µm. Air  
401 pollution guidelines, proposed by the WHO and the European Union, define a threshold of 50  
402 µg/m<sup>3</sup> of PM<sub>10</sub> (calculated as daily average) to protect the health of exposed population  
403 (WHO, 2005). On the other hand the Senegalese air quality law has set a higher limit value  
404 (260 µg/m<sup>3</sup> for 24 hours). The results of this study showed levels of PM<sub>10</sub> concentration lower  
405 at the rural site houses (30 µg/m<sup>3</sup>) than those found at urban sites, houses (200 µg/m<sup>3</sup>), market  
406 (100 µg/m<sup>3</sup>) and buses (300 µg/m<sup>3</sup>) (**Fig. 6A**). Concentration levels of PM<sub>10</sub> in the buses  
407 exceeded the national recommended limit values and are significantly higher than the  
408 international standards. Similar results were obtained by Saksena et al. (2008) who showed  
409 PM<sub>10</sub> concentrations as high as 262 µg/m<sup>3</sup> in buses in Vietnam. Similarly Praml et Schierl  
410 (2000) found an average concentration of 155 µg/m<sup>3</sup> considering a multi buses trip analysis in  
411 Munich.

412 Median temperature values were slightly lower for the urban housemaid population (25 °C)  
413 than all the other populations studies (29.2, 28 and 28 °C for drivers, commerciants and rural  
414 housemaids, respectively) (**Fig. 6B**) possibly in relation to air conditioning or other type of  
415 temperature controlling systems.

416 This study clearly shows that the interior of the buses circulating in the highly busy urban area  
417 of Dakar determines a higher exposure to gases and particles compared to the other  
418 environments here investigated. These differences could be explained by the congestion of the  
419 roads due to the density of traffic, traffic jams and high emission from old combustion  
420 technologies of the circulating vehicles fleet, as well as local topographical properties (such as  
421 street canyons) that prevent aeration and dispersion of pollutants and promoting their  
422 accumulation (Saksena et al., 2008; Morin et al., 2009; Zagury et al., 2000).

423 The comparison of indoor and outdoor air quality showed that average PM<sub>10</sub> concentrations  
424 were significantly higher indoors (0.5 mg/m<sup>3</sup>) than outdoors (0.2 mg/m<sup>3</sup>) at the urban site.  
425 Similarly, CO levels were significantly higher indoors (3.9 ppm) than outdoors (0.5 ppm). For  
426 CO<sub>2</sub>, the concentrations found were significantly higher within households in the rural site  
427 (**Fig. S2**). No significant differences were observed for the meteorological parameters

428 (temperature and humidity) between indoor and outdoor sampling areas, both for the urban  
429 and the rural site.

430 These results were similar with other studies where many pollutants were found at higher  
431 concentrations in indoor sampling sites in comparison to outdoor ones (Bardana et al., 2001;  
432 Kirchner et al., 2011). The accumulation of pollutants in indoor environments may be related  
433 to several conditions such as the ventilation of the houses; the season of sampling; the level of  
434 outdoor air pollution; the proximity to high traffic roads and the presence of specific indoor  
435 emission sources such as gas or wood equipment for cooking (Gerber et al., 1997).

436

### 437 **Conclusion**

438 For the first time here we report data on the personal exposure levels of different selected  
439 populations to indoor and outdoor air pollution in the sub-Saharan region of Dakar. The data  
440 provide an important milestone for the knowledge of the exposure levels to VOCs, PAHs and  
441 other air pollutants in developing countries.

442 Housemaids at urban and rural sites were more exposed than professionals to volatile organic  
443 air pollution. Benzene indoor concentrations are probably due to local indoor practices  
444 (charcoal cooking, incense burning etc...) and this exposure correlates with the median values  
445 of S-PMA (**Table S3**), which were higher among housemaids compared to the other groups.

446 In addition, urinary levels of oxidative stress (8-OHdG) and inflammatory (interleukin-6 and -  
447 8) biomarkers were higher in urban subjects comparing to rural ones therefore suggesting a  
448 general major impact of urban air pollution compared to the rural one.

449 The air quality measurement campaign showed that the bus interior was more polluted with  
450 PM<sub>10</sub>, CO, CO<sub>2</sub> and NO than the market and urban and rural indoor sites. The comparison of  
451 air quality between indoor and outdoor sites revealed that, especially at the urban site, indoor  
452 air quality may be worse than the corresponding outdoor one, confirming previous  
453 observations of potential higher individual exposure level to pollutants in indoor environment.

454

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465

466 **Declaration of interest**

467 The authors declare no conflict of interest.



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**Table S1**

Gradient elution of ammonium formate (A1) and acetonitrile (B1).

**Table S2**

Mean concentration (and number of measurements), minimum, maximum, median first (Q1) and third (Q3) quartile of exposed population (Housemaids-HLM, Housemaids-TD, Traders and Bus drivers).

**Table S3**

Spearman correlation coefficients between different variables in bus drivers, traders, in housemaids in urban and rural site (\*=  $p < 0.05$ ; \*\*=  $p < 0.01$ ; \*\*\*=  $p < 0.001$ ).

**Fig. 1.** Distribution of individual Benzene (A), Toluene (B), Ethylbenzene (C), m+p Xylenes (D) and o-Xylene (E) exposure in between bus drivers, traders and housemaids in urban and rural sites. Values are expressed as median, upper and lower quartiles, range and the outliers (<sup>o</sup>) (ANOVA–Tukey test: **a** =  $p < 0.05$ ; **b** =  $p < 0.01$ , **c** =  $< 0.001$ ).

**Fig. 2.** Exposure biomarkers (S-PMA(A) and 1-HOP (B)) in urine between bus drivers, traders and housemaids in urban and rural sites illustrated by the median, the upper and lower quartiles, the range and the outliers (\*, <sup>o</sup>) (ANOVA–Tukey test: **a** =  $p < 0.05$ ; **b** =  $p < 0.01$ ).

**Fig. 3.** 8-Hydroxy-2- $\alpha$ -deoxyguanosine (8-OHdG) in urine between bus drivers, traders and housemaids in urban and rural sites. These values are depicted as median, upper and lower quartiles, range and the outliers (\*, <sup>o</sup>) (ANOVA–Tukey test: **a** =  $p < 0.05$ ).

**Fig. 4.** Protein secretion of Tumor Necrosis Factor-alpha (TNF- $\alpha$ ) (A), Interleukin 1-bêta (IL-1 $\beta$ ) (B), Interleukin 6 (IL-6) (C) and Interleukin 8 (D) in urine between bus drivers, traders and housemaids in urban and rural sites. These values are depicted as median, upper and lower quartiles, range and the outliers (\*, <sup>o</sup>) (ANOVA–Tukey test).

**Fig. 5.** Distribution of CO<sub>2</sub> (A), CO (B), NO (C) and NO<sub>2</sub> (D) in between bus market, houses in urban (HLM) and rural (TD) sites. Values are expressed as median, upper and lower quartiles, range and outliers (\*, <sup>o</sup>) (ANOVA–Tukey test: **a** =  $p < 0.05$ ; **b** =  $p < 0.01$ , **c** =  $< 0.001$ ).

**Fig. 6.** Distribution of PM<sub>10</sub> (A), Temperature (B), and Humidity (C) in between bus market, houses in urban (HLM) and rural (TD) sites. Values are expressed as median, upper and lower quartiles, range and outliers (\*, <sup>o</sup>) (ANOVA–Tukey test: **a** =  $p < 0.05$ ; **b** =  $p < 0.01$ , **c** =  $< 0.001$ ).

**Fig. S1.** Location map of the study

**Fig. S2.** PM<sub>10</sub> (A), CO<sub>2</sub> (B), CO (C), Temperature (D), and Humidity (E) levels between outdoor and indoor air in urban (HLM) and rural (TD) sites. Values are reported as mean and standard deviations. \*=  $p < 0.05$ ; \*\*=  $p < 0.01$ ; \*\*\*=  $p < 0.001$  (Mann–Whitney U; outdoor versus indoor). p indicates differences between HLM outdoor and TD outdoor and a =  $p < 0.05$ ; c =  $p < 0.001$ . t stays for differences between HLM indoor and TD indoor and b =  $p < 0.01$ ; c =  $p < 0.001$ .



Figure 1

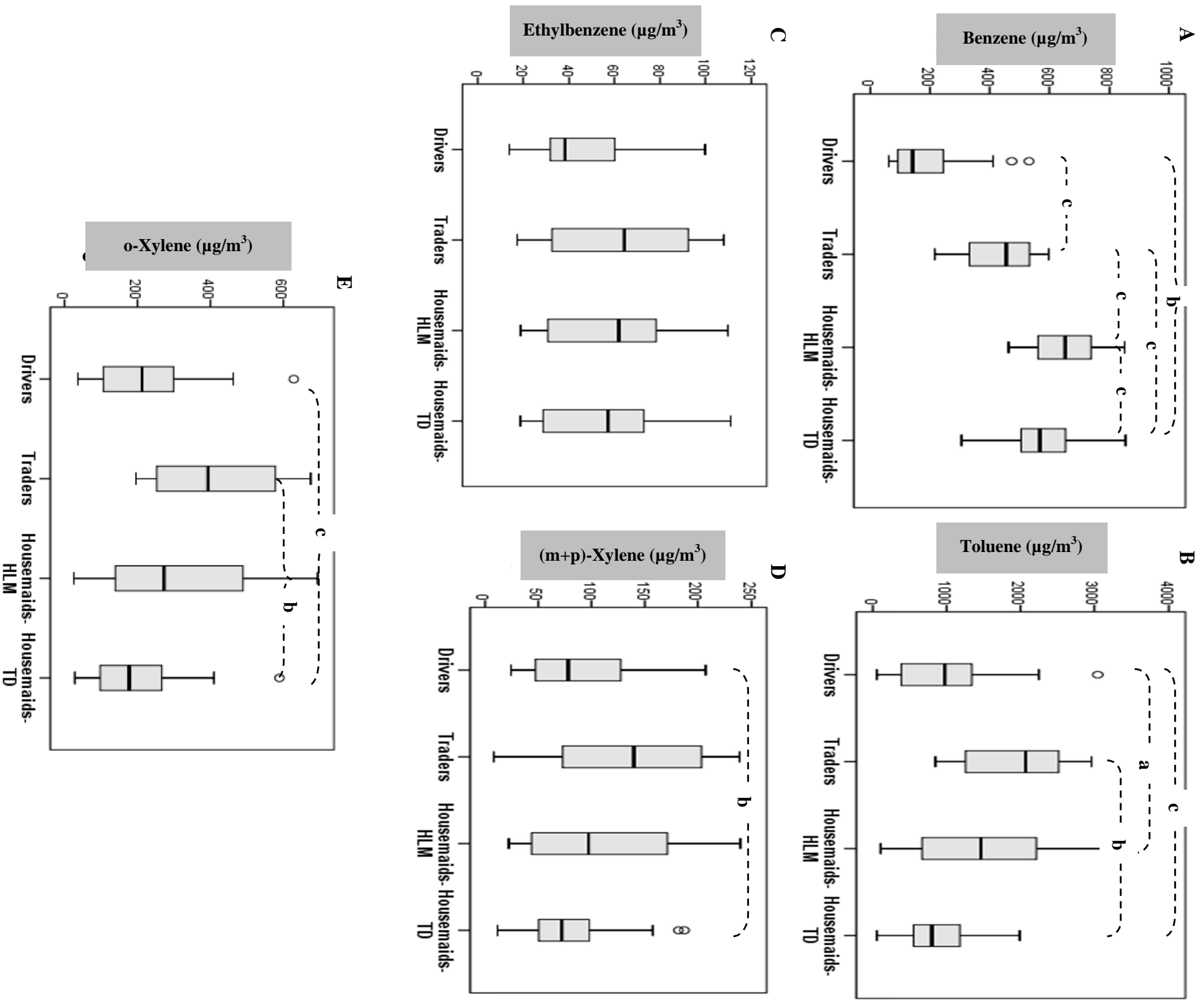


Figure 2

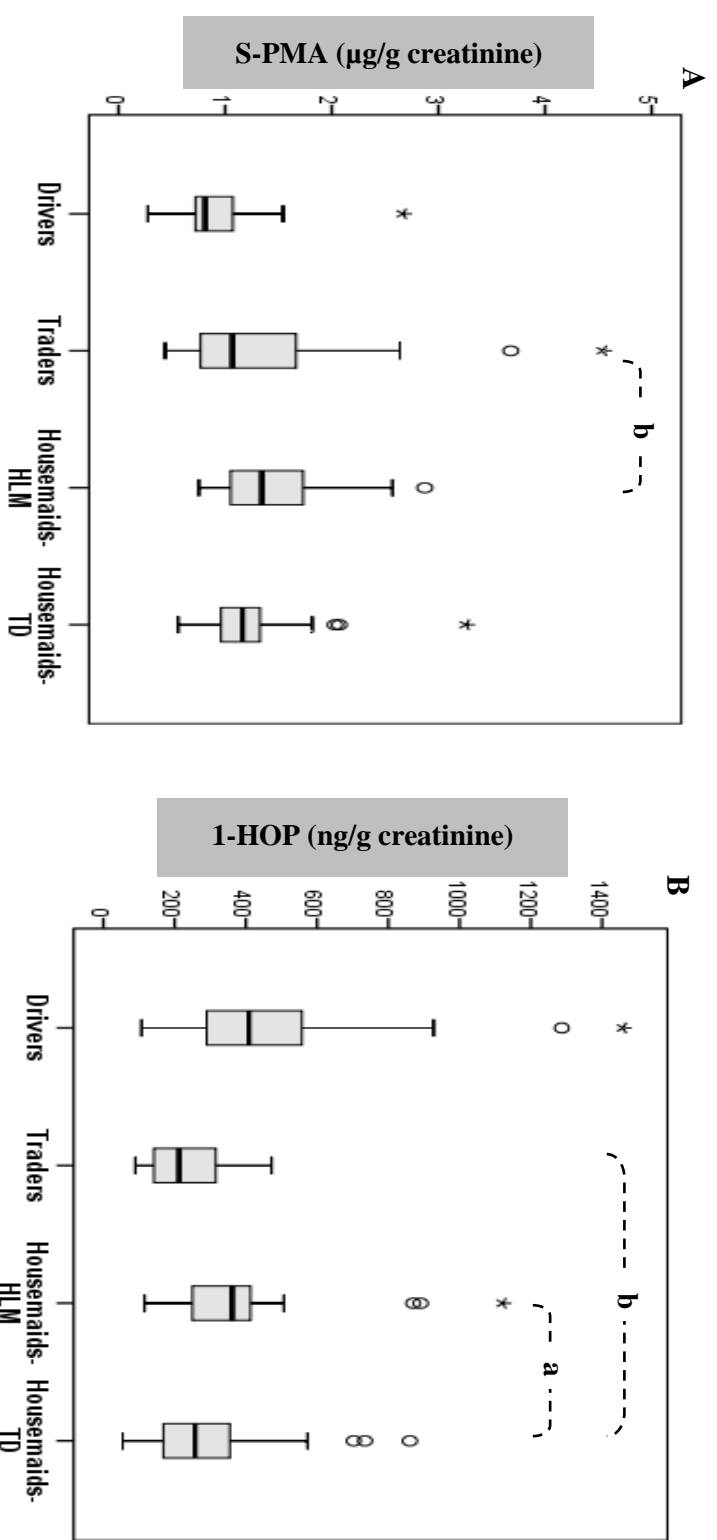


Figure 3

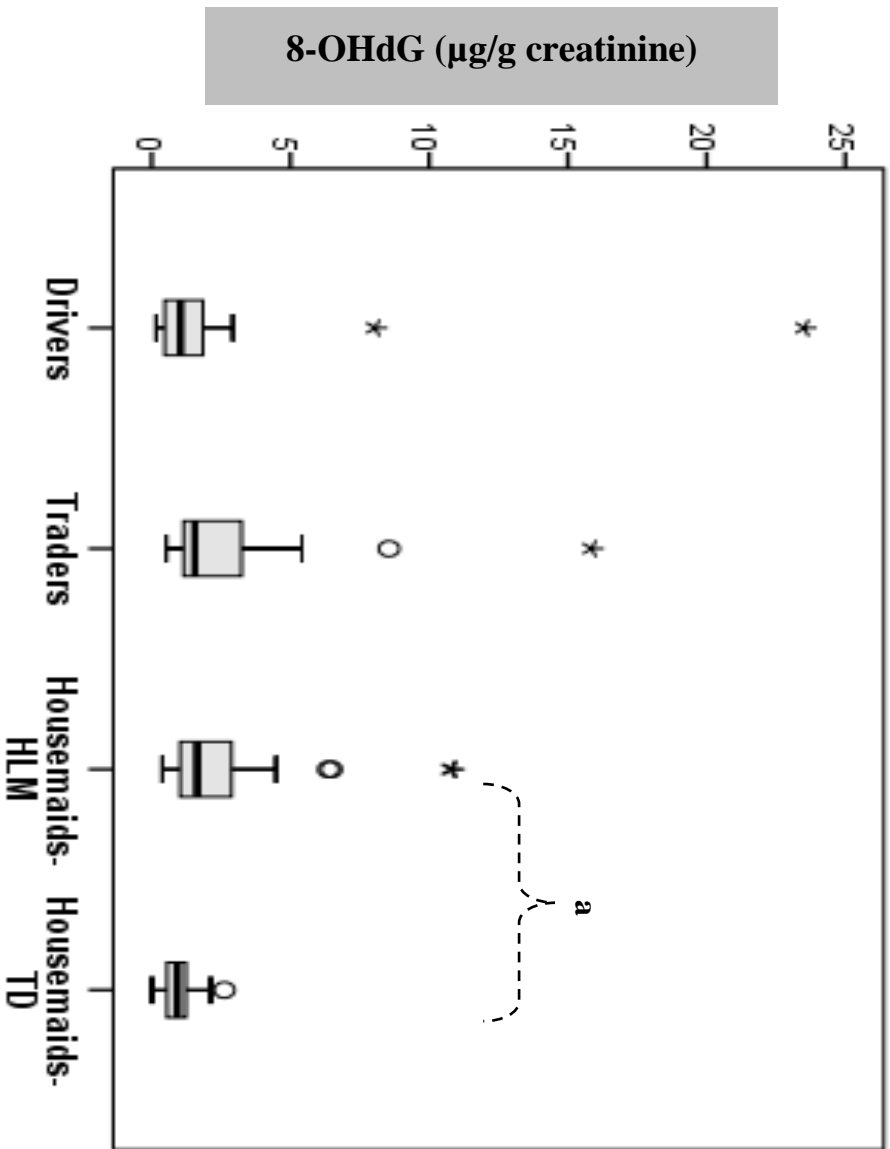


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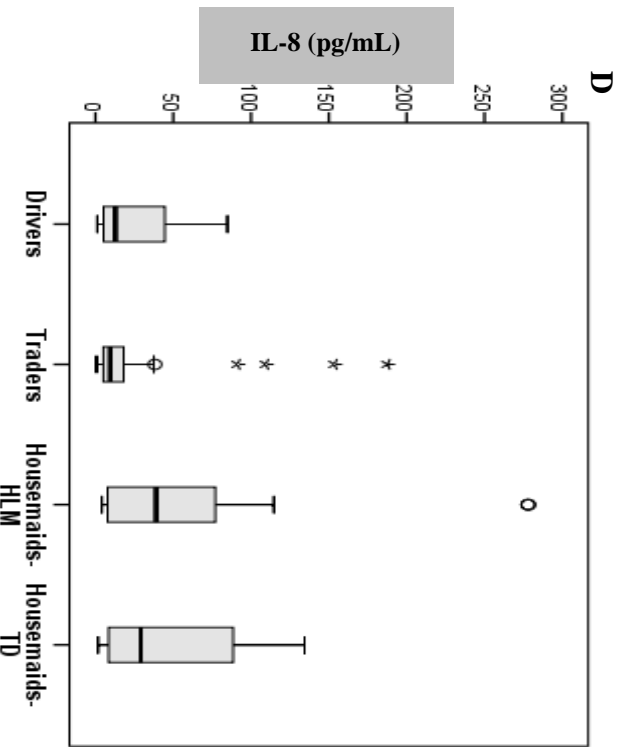
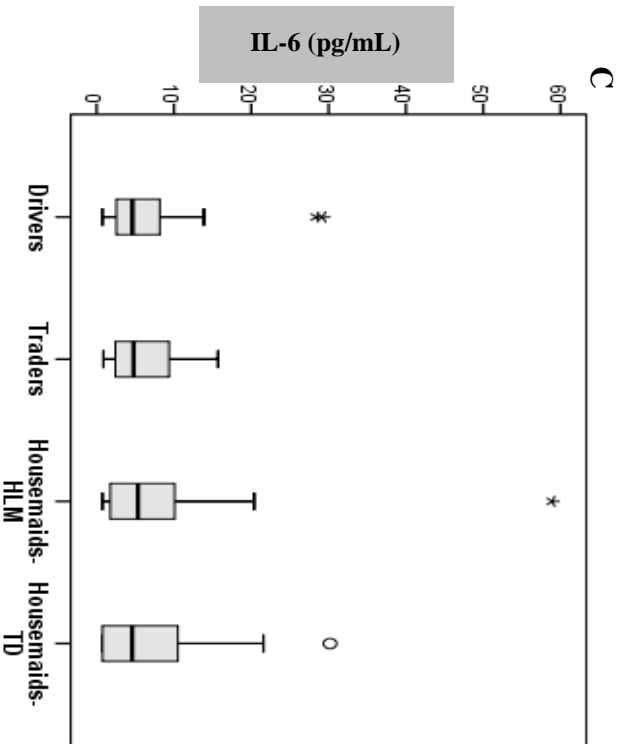
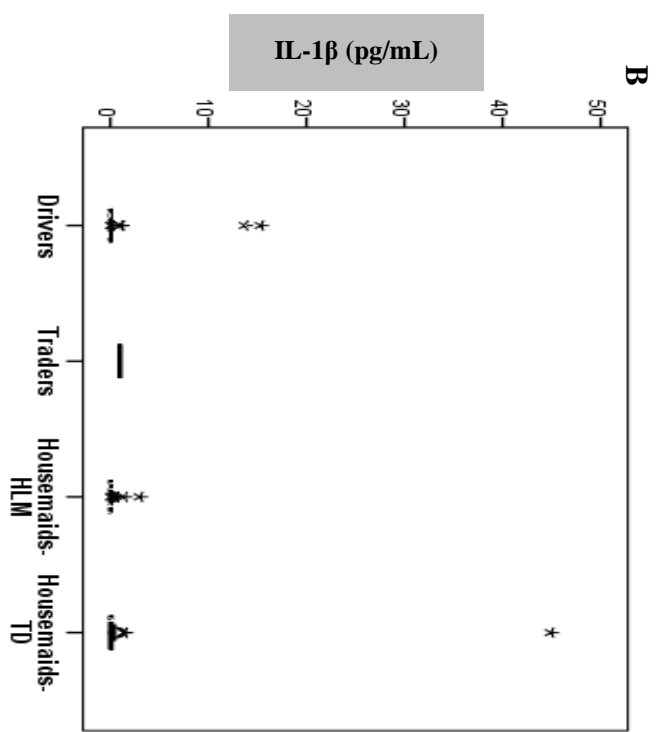
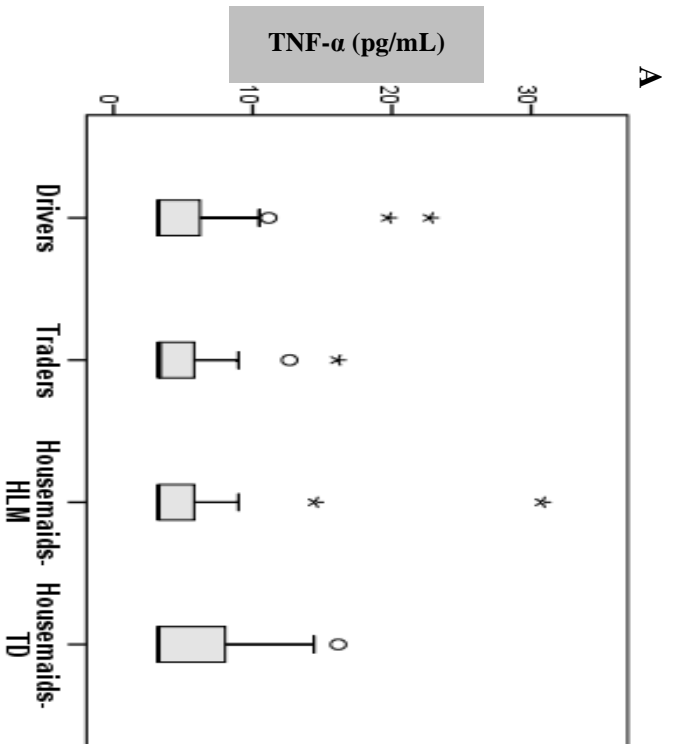


Figure 5

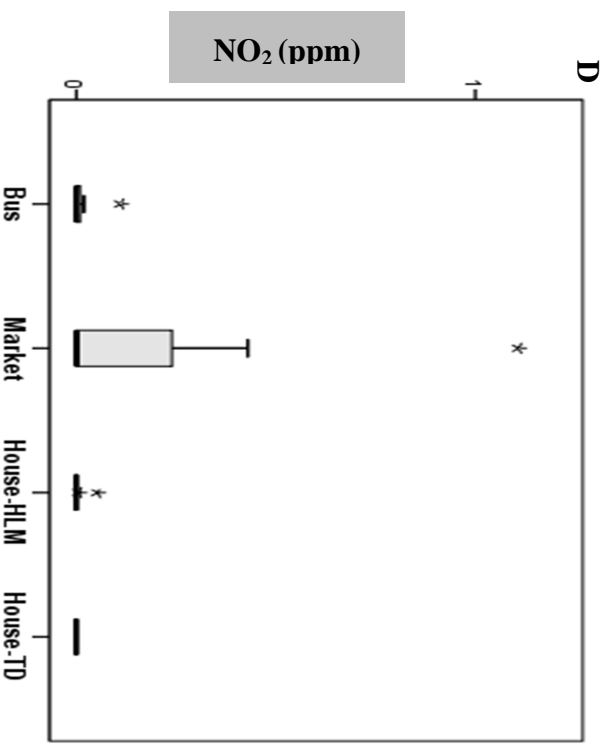
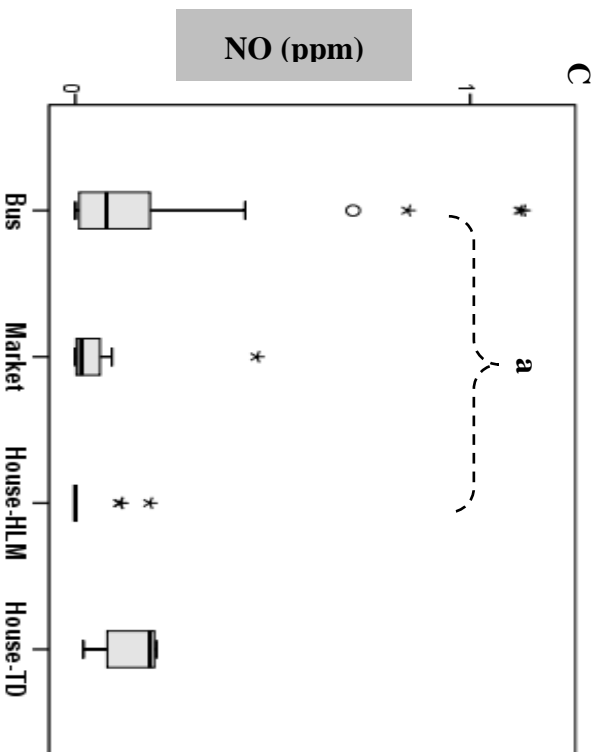
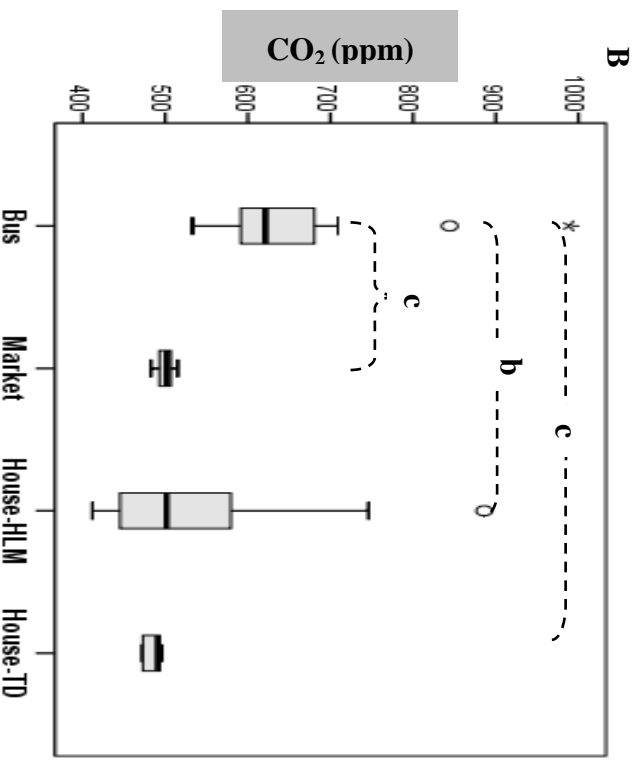
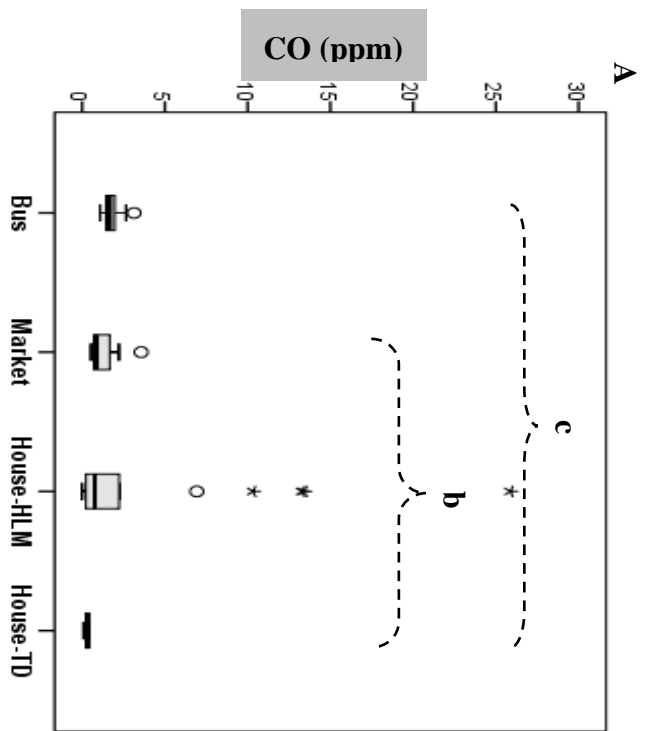
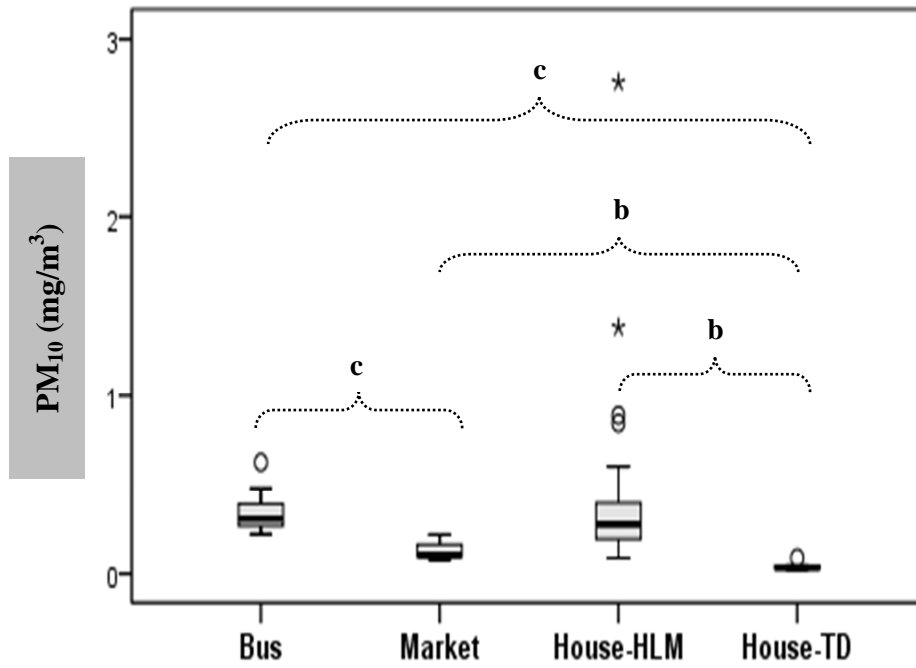
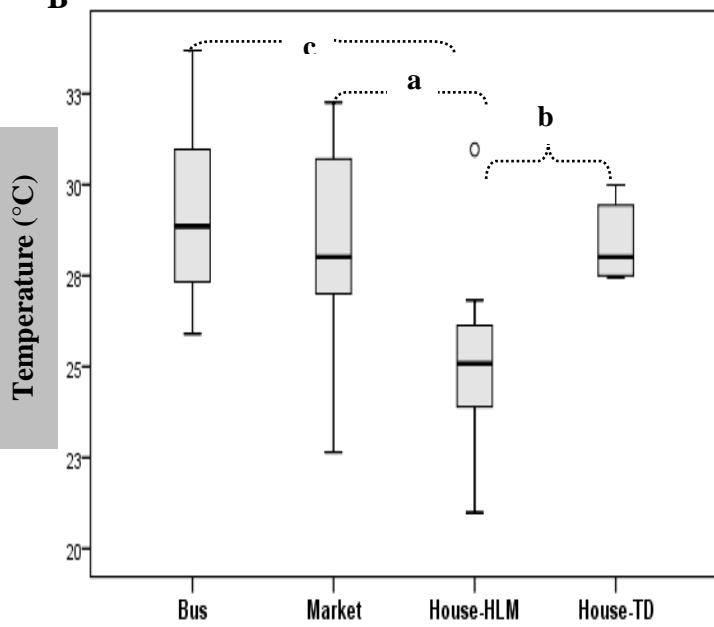


Figure 6

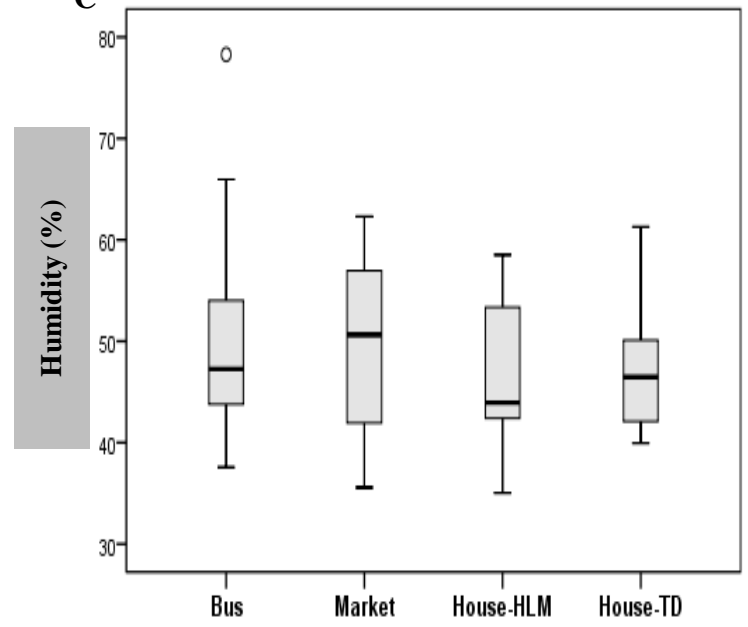
A



B



C



- Indoor organic volatile pollution higher than outdoor one
- Highest urinary levels of oxidative stress and inflammatory biomarkers in urban subjects
- Importance of indoor air pollution on the benzene metabolite S-PMA
- Relevance of indoor habit to determine the exposure levels to selected VOCs
- Correlation of biomarker of exposure with selected VOCs



**Urban site**



**Rural site**

**Air quality analysis**



**EVM-7**



**AQ Pro**

**Human exposure**



**GABIE badges: BTEX analysis**



**Urine sampling: biomarkers determination**