Cytotoxicity of Particulate Matter PM$_{10}$ Samples from Ouagadougou, Burkina Faso

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Particulate matter (PM) is one of the main air pollutants with 257,000 deaths per year in Africa. Studying their toxic mechanisms of action could provide a better understanding of their effects on the population health. The objective of this study was to describe the PM$_{10}$ toxic mechanism of action collected in 3 districts of Ouagadougou. Once per month and per site between November 2015 and February 2016, PM$_{10}$ was sampled for 24 hours using the MiniVol TAS (AirMetrics, Eugene, USA). The collected filters were then stored in Petri dishes at room temperature for in vitro toxicological studies using human pulmonary artery endothelial cells (HPAEC) at the Bordeaux INSERM-U1045 Cardio-thoracic Research Center. The three study districts were classified based on PM$_{10}$ level (high, intermediate, and low, respectively, for districts 2, 3, and 4). PM$_{10}$ induced a concentration-dependent decrease in cell viability. A significant decrease in cell viability was observed at 1 $\mu$g/cm$^2$, 10 $\mu$g/cm$^2$, and 25 $\mu$g/cm$^2$ for, respectively, districts 2, 3, and 4. A significant increase in the production of reactive oxygen species (ROS) was observed at 10 $\mu$g/cm$^2$ for district 2 versus 5 $\mu$g/cm$^2$ and 1 $\mu$g/cm$^2$ for districts 3 and 4, respectively. Finally, a significant production of IL-6 was recorded from 5 $\mu$g/cm$^2$ for district 4 versus 10 $\mu$g/cm$^2$ for districts 2 and 3. Consequently, Ouagadougou is subjected to PM$_{10}$ pollution, which can induce a significant production of ROS and IL-6 to cause adverse effects on the health of the population.

1. Introduction

Air pollution is one of the major environmental risk factors for the population health [1]. According to the WHO, a reduction in air pollution levels results in a significant decrease in morbidity from cardiovascular and respiratory diseases, including asthma [2]. The estimated number of deaths due to air pollution was 4.2 million worldwide in 2016 [2]. Most epidemiological studies in air pollution have been conducted in developed countries, and particulate matter (PM) is the main cause [3, 4].

The level of PM is an indicator of air pollution, and their size during inhalation exposure seems to determine the affected target organs as well as their impact on health [5]. Indeed, PM$_{2.5}$, fine particles with an aerodynamic diameter less than 2.5 $\mu$m, penetrate deeply into the lungs to the terminal bronchioles and alveoli, whereas PM$_{10}$, with a diameter of 10 $\mu$m, are deposited mainly in the upper respiratory tract [5, 6]. Autopsies performed on London smog air pollutant victims in 1952 revealed a high occurrence of chronic obstructive pulmonary disease (COPD) [7]. Epidemiological studies have shown the relationship between
exposure to PM air pollution and pulmonary pathology (asthma exacerbation, pulmonary cancer, and COPD) [8, 9].

Regarding the significant consequences of air pollution, the WHO organized the first international conference on air pollution in 2018 [10]. However, Africa has few air pollution studies [11]; although, the United Nations International Children’s Emergency Fund (UNICEF) recorded 258,000 deaths due to air pollution in 2017 in Africa [12]. Sub-Saharan African populations are the most affected by the air quality degradation effects [13]. Unfortunately, interest in air quality is of lesser importance due to the lack of political will and the absence of adequate tools for air quality monitoring [11]. Ouagadougou, the capital of Burkina Faso, is not excluded from this situation.

Located in the heart of the Sahel, Ouagadougou has, in addition to the factors mentioned above, a growing fleet of very old and poorly maintained vehicles in a context of increasing human activity [14]. Studies on air pollution are rare, incomplete, or old. They are mainly physicochemical studies on pollutants for environmental pollution [14–18]. Studies on PM$_{10}$ air pollution considered to be of concern for the health of populations and understanding their toxicological mechanisms of action at the cellular level are needed to guide decision-making about air pollution. In the current study, we report a possible toxicological mechanism of PM$_{10}$ action collected in Ouagadougou on pulmonary vascular target cells.

2. Materials and Methods

2.1. Cell and Culture Conditions. PromoCell® human pulmonary artery endothelial cells (HPAECs) were used for all toxicology studies. The cells were grown in monolayers in 25 cm$^2$ culture flasks (Falcon®) at a density of 20,000 cells/cm$^2$ in Complete Red Medium (CRM).

CRM consisted of Endothelial Cell Growth Medium (ECGM), with phenol red, without antibiotics or fungicides, supplemented with Supplement Mix (PromoCell®). The Supplement Mix contains 2% fetal bovine serum (FBS), 0.4% endothelial cell growth supplement (ECGS), 0.1 ng/mL epidermal growth factor (EGF), 1 ng/mL basic fibroblast growth factor (BFGF), 90 µg/mL heparin, and 1 µg/mL hydrocortisone.

Cells were incubated at 37°C in a controlled humid atmosphere of 5% CO$_2$ at pH = 7.4. The culture medium was changed every other day. When the cells reached 80% confluence, they were subcultured. The cells were used between passages 2 and 8. Experiments with the cells were performed in a sterile environment under a laminar flow hood in a P2 laboratory.

2.2. Atmospheric Samples. PM$_{10}$ can directly penetrate the respiratory tract and alveoli to cause adverse health effects in the exposed population. The PM$_{10}$ level was evaluated in three districts of Ouagadougou, and the districts were then classified based on the PM$_{10}$ level (high PM$_{10}$ level, intermediate PM$_{10}$ level, and low PM$_{10}$ level). Particulate samples from each category (district 2 for high PM$_{10}$, district 3 for intermediate PM$_{10}$, and district 4 for low PM$_{10}$) were used for the toxicological studies. The MiniVol TAS (AirMetrics, Eugene, USA), a portable air sampling device, was used to sample PM$_{10}$ from ambient air with a Teflon filter. The filters were weighed before and after sample collection. The device was placed on a stand at the height of a 1.70 m human airway. An authorization (ref: 2016/Labio/XI-02/0135) was requested from the competent structures of the different districts for the PM$_{10}$ sampling. Four sampling campaigns (November 2016, December 2016, January 2017, and February 2017) were scheduled at each site within the different districts.

The mixture from the different campaigns per district was used as the sample from each district. The filters were stored in Petri dishes at room temperature for in vitro toxicology studies. The experimental (toxicological) study was performed at the Cardio-thoracic Research Center of Bordeaux (CRCTB) INSERM-U1045.

2.3. Extraction of Particles from Filters. The protocol used in the present study [19] has been adapted by the CRCTB (INSERM U 1045, University of Bordeaux) and the laboratory UMR-CNRS 8251 (University Paris Diderot) [20]. The filters without their periphery were cut into four pieces. Each piece was then placed in Eppendorf tubes supplemented with 500 µl of Complete White Medium (CWM), antibiotics (1% penicillin/streptomycin), and antifungals (1% amphotericin B).

The Eppendorf was then alternately vortexed and sonicated (Vibracell® 75186) using an induction probe. Then, the filter was removed and placed in a second Eppendorf tube containing 700 µl of CWM supplemented with antibiotics and antifungals. The same process was also applied to the second Eppendorf. Once all filter pieces were processed, the contents of the two Eppendorf tubes were combined to have a total volume of 1200 µl. The samples were then stored at -20°C for later use.

2.4. Cytotoxicity Assessment (WST-1 Test). The cytotoxicity of PM$_{10}$ was evaluated by the water-soluble tetrazolium test (WST-1). This colorimetric test is based on the cleavage of the colorless tetrazolium salts WST-1 (4-[3-(4-iodophenyl)-2-(4-nitrophenyl)-2H-5 tetrazolio]-1, 3-benzene disulfonate) into a formazan derivative, soluble in yellow color by mitochondrial succinate dehydrogenases of living cells. HPAEC cells were first seeded in ninety-six-well plates at a density of 20,000 cells/cm$^2$ and cultured for 24 h at 37°C with 5% CO$_2$. They were subsequently exposed to different concentrations of PM$_{10}$ (1 µg/cm$^2$, 5 µg/cm$^2$, 10 µg/cm$^2$, 25 µg/cm$^2$, and 50 µg/cm$^2$). The experiment was repeated three times with four wells per condition. After 24 hours of exposure, the cells are rinsed with CWM supplemented with antibiotics and antifungals and incubated for 3 hours at 37°C with 5% CO$_2$ with CWM supplemented with antifungals and antibiotics containing 10% of WST-1 [21].

Absorbance was measured directly by spectrophotometry at 450 nm corrected to 630 nm, using a SPEC-TROstarNano2.10 plate reader and MARS Data Analysis
Software (BMG LabTech®). The toxicity assessment determined the PM$_{10}$ concentrations to be used for the mechanistic studies. It was agreed not to exceed 10 µg/cm$^2$, a concentration resulting in less than 30% cell death. This choice was also made for this concentration to remain as realistic as possible regarding the concentrations to which humans could be exposed.

2.5. Assessment of the Production of Reactive Oxygen Species. Oxidative stress levels follow different steps over time, from antioxidant defense to cytotoxicity through inflammation [22]. Therefore, the ROS production was assessed after 4 h of exposure to PM$_{10}$ according to the method adapted from Deweirdt et al. [21].

The different reactive oxygen species (ROS) were quantified by spectrophotometric method using the CM-H$_2$DCF-DA probe (5-(and-6)-chloromethyl-2′,7′-dichlorodihydrofluorescein diacetate, acetyl ester), Fisher Scientific®. Non-fluorescent H$_2$DCF-FM-H$_2$DCF-DA CM-H$_2$DCF-DA in the acetylated and reduced state is cleaved at the 2′ and 7′ acetylated groups by an esterase to yield nonfluorescent H$_2$DCF. The molecule is then oxidized by ROS to a fluorescent derivative, DCF, which can easily be quantified by fluorimetry. Thus, the fluorescence intensity is directly proportional to the amount of ROS formed in the cell [23].

The probe and cells were protected from light. Cells were seeded into twenty-four-well plates at a density of 20,000 cells/cm$^2$ and cultured for 24 hours at 37°C with 5% CO$_2$. Prior to exposure, cells were rinsed and incubated for 20 minutes at 37°C in 5% CO$_2$ with the probe (20 µM) solubilized in serum-free blank medium. Once excess probe not incorporated by the cells had been removed, the cell layer was rinsed with CWM supplemented with antibiotics and antifungal at 1% and the cells were exposed to different concentrations of PM$_{10}$ such as 1 µg/cm$^2$, 5 µg/cm$^2$, and 10 µg/cm$^2$ from Ouagadougou districts 2, 3, and 4. A significant difference was observed at the concentration of 1 µg/cm$^2$ (Figure 1(a); 76.5% ± 2.5% cell viability), 10 µg/cm$^2$ for district 3 (Figure 1(b); 68.4% ± 7.2% cell viability), and 25 µg/cm$^2$ for district 4 (Figure 1(c); 73.3% ± 6% cell viability). At the highest concentration (50 µg/cm$^2$), a viability of 60.3% ± 4.1%, 66.9% ± 6.4%, and 69.5% ± 7% was noted, respectively, for districts 2, 3, and 4. A significant difference was observed at the concentration of 1 µg/cm$^2$ in HPAEC cells for districts 2 and 3 (76.5% ± 2.5% versus 93.4% ± 1.2% and for districts 2 and 4 (76.5% ± 2.5% versus 92.6% ± 0.39%). Overall, the results demonstrate that increasing the concentration decreases cell viability, and the threshold concentration to obtain a significant decrease in cell viability varies from one district to another.

2.6. Interleukin-6 (IL-6) Assay. Studies have shown that PM$_{10}$ induces the production of cytokines such as TNF-α, IL-1, IL-6, IL-8, or IL-10. However, IL-6 is a proinflammatory cytokine that intervenes during the acute phase of inflammation. Previous studies also demonstrated a correlation between IL-6 production and PM$_{10}$ exposure [24–26]. Based on these previous studies, we focused here on the assessment of IL-6 secretion.

Cells were seeded in twelve-well plates at a density of 20,000 cells/cm$^2$ and cultured for 24 hours at 37°C with 5% CO$_2$. Before exposure, the cell mat was rinsed. Then the cells were contacted with different PM$_{10}$ concentrations (1.5 and 10 µg/cm$^2$). The experiment was repeated three times ($n = 3$) with three wells per condition. After 24 hours of exposure, the supernatants were removed, centrifuged (10 minutes, at 15,000 g and at 4°C), and stored at −20°C until assay.

The assay of IL-6 cytokines in culture supernatants was performed by the enzyme-linked immunosorbent assay (ELISA) technique using DuoSet kits (R&D Systems®), a SPECTROstarNano2.10 plate reader, and MARS 2.41 Data Analysis Software (BMG LabTech®). To relate the IL-6 cytokine level (ELISA) to the protein levels, a Lowry test was performed in the culture supernatants for each condition, using the Biorad® DC Protein assay reagent package kit. Absorbance was measured using a SPECTROstarNano2.10 plate reader 2.41 data analysis software (LabTech®) at 750 nm. For each test, the results are expressed as means ± the standard error of the mean (SEM). Each experiment was performed independently three times ($n = 3$). IL-6 was assayed in the culture supernatant of HPAEC cells after 24 hours of intoxication at different concentrations of PM$_{10}$ (1.5 and 10 µg/cm$^2$) in districts 2, 3, and 4.

2.7. Statistical Analyses. Differences between the means of HPAEC control and PM$_{10}$-exposed cells were evaluated using different tests: an ANOVA parametric test followed by a multiple comparison test with Dunnett’s correction. Statistical analyses and graphs were performed with GraphPad Prism 5.0 software. Differences were considered significant when $p < 0.05$(*), $p < 0.01$(**), and $p < 0.001$(***)

3. Results

3.1. The Effect of PM$_{10}$ on Cell Viability is District-Specific and Concentration-Dependent. The nature and chemical composition of PM$_{10}$ are important factors in their health effects. The current study results show that after 24 hours of exposing HPAEC cells to different PM$_{10}$ concentrations, there is a significant concentration-dependent decrease in cell viability from 1 µg/cm$^2$ for district 2 (Figure 1(a); 76.5% ± 2.5% cell viability), 10 µg/cm$^2$ for district 3 (Figure 1(b); 68.4% ± 7.2% cell viability), and 25 µg/cm$^2$ for district 4 (Figure 1(c); 73.3% ± 6% cell viability). At the highest concentration (50 µg/cm$^2$), a viability of 60.3% ± 4.1%, 66.9% ± 6.4%, and 69.5% ± 7% was noted, respectively, for districts 2, 3, and 4. A significant difference was observed at the concentration of 1 µg/cm$^2$ in HPAEC cells for districts 2 and 3 (76.5% ± 2.5% versus 93.4% ± 1.2% and for districts 2 and 4 (76.5% ± 2.5% versus 92.6% ± 0.39%). Overall, the results demonstrate that increasing the concentration decreases cell viability, and the threshold concentration to obtain a significant decrease in cell viability varies from one district to another.

3.2. PM$_{10}$-Induced ROS Production is District-Specific and Concentration-Dependent. PM$_{10}$-induced reactive oxygen species (ROS) production is concentration-dependent. The results show a concentration-dependent increase in ROS production in HPAEC cells compared to control cells (Figure 2).
This increase is significant at the highest concentration (10μg/cm²) for district 2 (Figure 2(a); 31.4% ± 6.1% increase in ERO production), whereas for districts 3 and 4 (Figure 2(b)) and 4 (Figure 2(c)), a significant increase is observed from 5μg/cm² (24.1% ± 6% increase in ERO production) and from 1μg/cm² (10.4% ± 0.4%), respectively. A comparison was made at the highest concentration (10μg/cm²) for the three districts. No significant difference was detected in ROS production in HPAEC cells after 4-hour exposure.

3.3. PM₁₀-Induced IL-6 Secretion is District-specific and Concentration-Dependent. The results reveal a concentration-dependent increase in IL-6 after a 24-hour exposure to PM₁₀ in all three districts of Ouagadougou. This increase is significant compared to control cells at the highest concentration (10μg/cm²) for districts 2 (Figure 3(a)) and 3 (Figure 3(b)). At this concentration, the mean secretion of IL-6 was 317.2 pg/mg ± 38.2 pg/mg and 272.7 pg/mg ± 32.27 pg/mg, respectively for districts 2 and 3, that is, an increase of 71.8% ± 7.8% and 45.7% ± 7.2%, respectively, compared to control cells. The increase was significant for district 4 (Figure 3(c)) from 5μg/cm² with a mean IL-6 secretion of 222.5 pg/mg ± 43.14 pg/mg, an increase of 58.8% ± 9% relative to control cells. A comparison was performed at a concentration of 10μg/cm² for the three districts. A significant difference was observed between districts 3 and 4.

4. Discussion

The PM₁₀ collected in each of the three districts in the current study led to a concentration-dependent decrease in cell viability. Our results are consistent with those of Dieme et al., who had shown the ability of particles collected in Dakar (Senegal) to alter mitochondrial metabolism [27].
Many studies have also shown a concentration-dependent decrease in cell viability following exposure of epithelial cells to PM$_{10}$ [28]. The nature and chemical composition of PM$_{10}$ certainly determine the threshold amount toxic for cells.

Our results show that after 24 hours of exposure to PM$_{10}$, a significant (concentration-dependent) decrease in cell viability was observed from 1 $\mu$g/cm$^2$ for district 2. Cachon et al. in a study conducted on particles from Cotonou (Benin) had shown a significant decrease in cell viability from 3 $\mu$g/cm$^2$ [29]. The similarity of these results could be explained by the fact that Cotonou and Ouagadougou are two cities with comparable profiles (transportation, human activities, and city expansion). In our study, the significant decrease in cell viability was observed from 10 $\mu$g/cm$^2$ for district 3 and from 25 $\mu$g/cm$^2$ for district 4. Indeed, district 2 is a district with a high level of PM$_{10}$, while district 3 has a lower level of PM$_{10}$ than the first one. Only a difference in PM$_{10}$ chemical composition could explain the significant difference observed from 25 $\mu$g/cm$^2$. When comparing the cell viability of the three districts, for an exposure to a PM$_{10}$ concentration of 10 $\mu$g/cm$^2$, significant differences were observed between districts 2 and 3 and between districts 2 and 4. Toxicity terms are noted between the different districts depending on the PM$_{10}$ profiles.

Redox homeostasis is essential for cell function and viability [30]. Exposure to extrinsic stress factors such as PM can disrupt this balance. To assess whether the PM$_{10}$ samples collected in different districts of Ouagadougou could disrupt this balance, the production of ROS was studied. The results show that after 4 hours of exposure, there is a concentration-dependent increase in ROS production in HPAEC cells compared to control cells. The literature reports similar results found in neighboring countries of Burkina and throughout the world [29, 31, 32]. A previous study [20] conducted in Ouagadougou on a single sampling point within the framework of the French National Research Agency MEGATOX project in 2010 also showed a concentration-dependent increase in ROS production in HPAEC cells.

At the highest concentration (10 $\mu$g/cm$^2$), the increase in ROS production was approximately 16% in the MEGATOX project sample [20] compared to 31.4% ± 6% for district 2, 28.5% ± 1.1% for district 3, and 18.5% ± 0.7% for district 4. The values found in the present study are much higher than the previous studies of MEGATOX project. These results could be explained by an increase in PM pollution in recent years and changes in the chemical composition of PM$_{10}$. Indeed, between 2010 and 2017, the city of Ouagadougou experienced a significant development of anthropogenic activities that constitute a major pollution factor [33]. The comparison of ROS production induced by PM$_{10}$ between different districts revealed a significant increase in ROS production at the concentration of 10 $\mu$g/cm$^2$ for district 2, whereas for districts 3 and 4, a significant increase was observed from 5 $\mu$g/cm$^2$. ROS production can be due to several mechanisms [34]. The study focused on the assessment of ROS production using the CM-H$_2$DCFH-DA probe, which allows the evaluation of intracellular changes in H$_2$O$_2$. Other methods for evaluating oxidative stress such as the glutathione assay or the lipid peroxidation assay could explain this difference in results. Indeed, the difference in ROS production induced by PM$_{10}$ collected in districts 3 and 4 could be due to these two mechanisms. A difference in the chemical composition of PM$_{10}$ could also explain our results [1].

It has been shown in the literature that oxidative stress could trigger a proinflammatory response [35]. To assess these hypotheses, our cellular model was treated with PM$_{10}$ from the three districts of Ouagadougou, and the secretion of proinflammatory cytokines such as IL-6 was evaluated. The results show a concentration-dependent increase in IL-6 after 24 hours of PM$_{10}$ treatment. Ndong Ba et al. in a study carried out on BEAS-2B cells treated with PM from Dakar (Senegal)...
also found a significant dose-dependent increase in IL-6 production of about 750 pg/ml from 3 μg/cm² for urban sites and approximately 250 pg/ml from 3 μg/cm² for rural sites [31]. The results of the present study were significant for districts 2 and 3, at a concentration of 10 μg/cm² with a rate of 317.2 pg/mg ± 38.2 pg/mg and 272.7 pg/mg ± 32.27 pg/mg, respectively. For district 4, the results were significant from 5 μg/cm² with a value of 222.5 pg/mg ± 43.14 pg/mg. These differences could be due to the variable chemical composition between the PM₁₀ from Ouagadougou and those from Dakar. A significant difference was found between districts 3 and 4, after comparing the inflammatory response induced by PM₁₀ collected in these districts at the concentration of 10 μg/cm². This is in line with the classification of districts by PM₁₀ level with district 3 as PM₁₀ low level area and district 4 as PM₁₀ intermediate level area. Overall, the results of the present study suggest an association between PM₁₀ exposure and the occurrence of pulmonary disease. The toxicological study also supports this hypothesis through the correlation between PM₁₀ exposure and ROS production and the secretion of proinflammatory mediators (IL-6). Despite limitations such as the short collection period, the selection of patients based on voluntary participation, and the failure to evaluate some toxicological mechanisms of action, the present original study is of high relevance in a country such as Burkina Faso to document the toxicological profile of atmospheric pollution with PM₁₀.

5. Conclusions

The results of the present study revealed that PM₁₀ induced dose-dependent cytotoxicity to a greater or lesser extent by sampling district. Oxidative stress is a key source of toxic substances found in the endothelial cells of human pulmonary arteries that can trigger a proinflammatory response. PM₁₀ unlike larger particles that are filtered by the nasal and bronchial cilia, directly penetrate the upper respiratory tract and alveoli, causing inflammation and irritating the bronchi, thus affecting lung function, and exacerbating symptoms of respiratory diseases and other pathologies according to the intensity or duration of the inflammation.

The results of this study lead us to question the profile of atmospheric pollution by particulate pollutants in Ouagadougou (Burkina Faso), especially the other types of PM found there and their chemical composition. In addition, it would be interesting to conduct a study to better define the clinical effects of PM. Finally, to confirm our assumption, it would be necessary, through further studies, to better characterize, in the endothelial cells of human pulmonary arteries, the mechanisms responsible for the toxicity of PM and to assess whether oxidative stress could be a source of calcium signaling alterations or an apoptotic process.

Data Availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors’ Contributions

Joelle Nicole GUISSOU, Jacques SIMPORÉ, and Jean SAKANDE conceived and designed the study. Joelle Nicole GUISSOU, Abdoul Karim OUATTARA, and Isabelle BAUDRIMONT were involved in data generation, collection, and assembly. Joelle, Nicole GUISSOU, Isabelle BAUDRIMONT, Abdoul Karim OUATTARA, Jacques SIMPORÉ, and Jean SAKANDE were involved in data extraction, analysis, and interpretation. Joelle, Nicole GUISSOU, Isabelle BAUDRIMONT, Abdoul Karim OUATTARA, Jacques SIMPORÉ, and Jean SAKANDE were involved with drafting or revising the manuscript. Jacques SIMPORÉ and Jean SAKANDE provided administrative, technical, and material support. Supervision of the study was made by Jacques SIMPORÉ and Jean SAKANDE. All authors critically revised and approved the final version of this publication.

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References

[1] K. H. Kim, E. Kabir, and S. Kabir, “A review on the human health impact of airborne particulate matter,” Environment International, vol. 74, pp. 136–143, 2015.
[2] WHO, “Ambient (Outdoor) Air Pollution,” 2021, https://www.who.int/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health.
[3] J. M. Samet, F. Dominici, F. C. Curriero, I. Courser, and S. L. Zeger, “Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994,” New England Journal of Medicine, vol. 343, no. 24, pp. 1742–1749, 2000.
[4] J. Schwartz and A. Zanobetti, “Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death,” Epidemiology, vol. 11, no. 6, pp. 666–672, 2000.
[5] K. L. Huang, S. Y. Liu, C. C. K. Chou, Y. H. Lee, and T. J. Cheng, “The effect of size-segregated ambient particulate matter on Th1/Th2-like immune responses in mice,” PLoS One, vol. 12, no. 2, Article ID e0173158, 2017.
[6] S. Feng, D. Gao, F. Liao, F. Zhou, and X. Wang, “The health effects of ambient PM2.5 and potential mechanisms,” Ecotoxicology and Environmental Safety, vol. 128, pp. 67–74, 2016.
[7] M. L. Bell and D. L. Davis, "Reassessment of the lethal London fog of 1952: Novel indicators of acute and chronic consequences of acute exposure to air pollution," *Environmental Health Perspectives*, vol. 109, pp. 389–394, 2001.

[8] O. K. Kurt, J. Zhang, and K. E. Pinkerton, "Pulmonary health effects of air pollution," *Current Opinion in Pulmonary Medicine*, vol. 22, no. 2, pp. 138–143, 2016.

[9] A. S. V. Shah, K. K. Lee, D. A. McAllister et al., "Short term exposure to air pollution and stroke: Systematic review and meta-analysis," *British Medical Journal*, vol. 350, 2015.

[10] M. J. Gaturi, "First WHO global conference on air pollution and health: A brief report," *Clean Air Journal*, vol. 29, no. 1, 2019.

[11] A. K. Amegah and S. Agyei-Mensah, "Urban air pollution in sub-saharan Africa: Time for action," *Environmental Pollution*, vol. 220, pp. 738–743, 2017.

[12] UNICEF. Silent suffocation in Africa, "Air pollution is a growing menace," 2019, https://www.unicef.org/reports/silent-suffocation-in-africa-pollution-2019.

[13] P. J. Landrigan, R. Fuller, N. J. R. Acosta et al., "The Lancet Commission on pollution and health," *Lancet*, vol. 391, no. 10119, pp. 462–512, 2018.

[14] B. Nanaa, O. Sanogob, P. Savadogoa, T. Dahoa, M. Boudad, and J. Koulidiatia, "Air quality study in urban centers: Case study of Ouagadougou Burkina Faso," *FUTY Journal of the Environment*, vol. 7, no. 1, pp. 1–18, 2012.

[15] J. Lindén, S. Thorsson, and I. Eliasson, "Carbon monoxide in Ouagadougou, Burkina Faso—a comparison between urban background, roadside and in-traffic measurements," *Water, Air, and Soil Pollution*, vol. 188, no. 1-4, pp. 345–353, 2008.

[16] I. Eliasson, P. Jonsson, and B. Holmer, "Diurnal and intra-urban particle concentrations in relation to windspeed and stability during the dry season in three African cities," *Environmental Monitoring and Assessment*, vol. 154, no. 1-4, pp. 309–324, 2009.

[17] J. Boman, J. Lindén, S. Thorsson, B. Holmer, and I. Eliasson, "A tentative study of urban and suburban fine particles (PM2.5) collected in Ouagadougou, Burkina Faso," *X-Ray Spectrometry*, vol. 38, no. 4, pp. 354–362, 2009.

[18] J. Lindén, J. Boman, B. Holmer, S. Thorsson, and I. Eliasson, "Intra-urban air pollution in a rapidly growing Sahelian city," *Environment International*, vol. 40, pp. 51–62, 2012.

[19] N. A. Janssen, A. Yang, M. Strak et al., "Oxidative potential of particulate matter collected at sites with different source characteristics," *The Science of the Total Environment*, vol. 472, pp. 572–581, 2014.

[20] M. Leroux, "Caractérisation de la toxicité des particules atmosphériques de deux mégacités sur les cellules épithéliales bronchiques humaines 16HBE," *Université Paris Diderot—Paris*, vol. 7, Paris, France, 2009.

[21] J. Deweirdt, J. Quignard, B. Crobeddu et al., "Involvement of oxidative stress and calcium signaling in airborne particulate matter—induced damages in human pulmonary artery endothelial cells," *Toxicology in Vitro*, vol. 45, no. Pt 3, pp. 340–350, 2017.

[22] S. Hussain, L. C. Thomassen, I. Ferecatu et al., "Carbon black and titanium dioxide nanoparticles elicit distinct apoptotic pathways in bronchial epithelial cells," *Particle and Fibre Toxicology*, vol. 7, no. 1, p. 10, 2010.

[23] X. Chen, Z. Zhong, Z. Xu, L. Chen, and Y. Wang, "Dichlorodihydrofluorescein as a fluorescent probe for reactive oxygen species measurement: forty years of application and controversy," *Free Radical Research*, vol. 44, no. 6, pp. 587–604, 2010.

[24] H. Tilg, E. Trehu, M. Atkins, C. Dinarello, and J. Mier, "Interleukin-6 (IL-6) as an anti-inflammatory cytokine: induction of circulating IL-1 receptor antagonist and soluble tumor necrosis factor receptor p55," *Blood*, vol. 83, no. 1, pp. 113–118, 1994.

[25] J. Y. Yang, J. Y. Kim, J. Y. Jang et al., "Exposure and toxicity assessment of ultrafine particles from nearby traffic in urban air in seoul, Korea," *Environ Health Toxicol*, vol. 28, Article ID e2013007, 2013.

[26] T. Fujii, S. Hayashi, J. C. Hogg, R. Vincent, and S. F. Van Eeden, "Particulate matter induces cytokine expression in human bronchial epithelial cells," *American Journal of Respiratory Cell and Molecular Biology*, vol. 25, no. 3, pp. 265–271, 2001.

[27] D. Dienie, M. Cabral-Ndior, G. Garcon et al., "Relationship between physicochemical characterization and toxicity of fine particulate matter (PM2.5) collected in Dakar city (Senegal)," *Environmental Research*, vol. 113, pp. 1–13, 2012.

[28] R. Van Den Heuvel, J. Staelens, G. Koppen, and G. Schoeters, "Toxicity of urban PM(10) and relation with tracers of biomass burning," *International Journal of Environmental Research and Public Health*, vol. 15, no. 2, p. 320, 2018.

[29] B. F. Cachon, S. Firmin, A. Verdin et al., "Inflammatory effects and oxidative stress within human bronchial epithelial cells exposed to atmospheric particulate matter (PM(2.5) and PM(>2.5)) collected from Cotonou, Benin," *Environmental Pollution*, vol. 185, pp. 340–351, 2014.

[30] H. Sies, "Oxidative eustress: On constant alert for redox homeostasis," *Redox Biology*, vol. 41, Article ID 101867, 2021.

[31] A. Ndong Ba, F. Cazier, A. Verdin et al., "Physico-chemical characterization and in vitro inflammatory and oxidative potency of atmospheric particles collected in Dakar city’s (Senegal)," *Environmental Pollution*, vol. 245, pp. 568–581, 2019.

[32] A. M. Spagnolo, G. Ottria, F. Perdelli, and M. Cristina, "Chemical characterisation of the coarse and fine particulate matter in the environment of an underground railway system: cytotoxic effects and oxidative stress—a preliminary study," *International Journal of Environmental Research and Public Health*, vol. 12, no. 4, pp. 4031–4046, 2015.

[33] B. Y. Yameogo, G. Honoré, and Georges, "Production et gestion des huiles usées de la ville de Ouagadougou, Burkina Faso," *Journal of Biological Chemistry*, vol. 25, no. 3, pp. 1176–1190, 2021.

[34] A. J. Lambert and M. D. Brand, "Reactive oxygen species production by mitochondria," *Methods in Molecular Biology*, vol. 554, pp. 165–181, 2009.

[35] S. Robertson, P. Douglas, D. Jarvis, and E. Marczylo, "Bioaerosol exposure from composting facilities and health outcomes in workers and in the community: A systematic review update," *International Journal of Hygiene and Environmental Health*, vol. 222, no. 3, pp. 364–386, 2019.