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# Health Risk of Inhalation Exposure to Sub-10 µm Particulate Matter and Gaseous Pollutants in Urban Air

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## ABSTRACT

**Objective:** To access the health risks associated with exposure to particulate matter ($PM_{10}$), sulphur dioxide ($SO_2$), nitrogen dioxide ($NO_2$), carbon monoxide (CO) and ozone ($O_3$) in an urban air.

**Design:** This study utilised the year 2014 (January - December) hourly ambient pollution data.

**Setting:** The study was conducted in an industrial area located in Pretoria West, South Africa. The area accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter.

**Data and method:** Estimate of possible health risks from exposure to $PM_{10}$, $SO_2$, $NO_2$, CO and $O_3$ was done using the United States Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework. A scenario-assessment approach where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for both intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults). The normal acute (1-hour) exposure to these pollutants was also determined.

**Outcome measures:** Presence or absence of adverse health effects from exposure to airborne pollutants.

**Results:** Average annual ambient concentration of $PM_{10}$, $NO_2$ and $SO_2$ recorded were 48.3±43.4 µg/m$^3$, 11.50±11.6 µg/m$^3$ and 18.68±25.4 µg/m$^3$ respectively. Whereas, the South African National Ambient Air Quality recommended 40 µg/m$^3$, 40 µg/m$^3$ and 50 µg/m$^3$ for $PM_{10}$, $NO_2$ and $SO_2$ respectively. Exposure to an hour concentration of $NO_2$, $SO_2$, CO and $O_3$; 8-h concentration of CO, $O_3$; and 24-h concentration of $PM_{10}$, $NO_2$ and $SO_2$ will not likely produce adverse effects to sensitive exposed groups. Though, infants, and children, rather than adults, are

more likely to be affected. Moreover, for chronic annual exposure, $PM_{10}$, $NO_2$ and $SO_2$ posed a health risk to sensitive individuals, with the severity of risk varying across exposed groups.

**Conclusions:** Long-term chronic exposure to $PM_{10}$, $NO_2$, and $SO_2$ pollutants may results in health risks among the study population.

**Keywords**: particulate matter, gaseous pollutants, health risk assessment, exposure groups, South Africa

**Strengths and Limitations of this study**

- Large data set spanning hourly ambient concentration of pollutants for a whole year.
- This is the first study in Pretoria West, South Africa to estimate health risks of human exposure to airborne pollutants using US EPA assessment model.
- In our study, prediction of both long-term and short-term health effects in infants, children and adults resulting from inhalation of pollutants was possible.
- However, the health risk that could result from exposure to the combination of the pollutants could not be determined.

## INTRODUCTION

Air pollution is a multifaceted mix consisting of both suspended particulates and gaseous pollutants.[1] Globally, air pollution continues to be a major environmental problem that has been recognised as an important public health risk.[2] The upsurge in human population, industrialisation, urbanisation, modernisation and its attendant increase in vehicular emissions and activities are the major contributors to the rising urban air quality problems.[3]

The World Health Organization (WHO) in the year 2013, asserted that annually, the urban ambient air pollution was predicted to cause 2 million deaths in the world.[4] Epidemiological studies have linked exposure to ambient air pollution with adverse human health effects.[5-7] Exposure to air pollution can result in both acute (short-term) and chronic (long-term) health effects.[8, 9] The acute effects of air pollution on human health were sufficiently established in the 20th century, when severe air pollution scenarios in Europe and in the United States resulted in morbidities and mortalities in hundreds of thousands of people.[10]

Air pollution is a known trigger of Chronic Obstructive Pulmonary Disease (COPD)[11], and has informed the establishment of air quality standards in many countries [12, 13]. The broad legislative framework for air quality assessment in populated areas was put in place by the European Union Directive on Air Quality 2008/50/EC [14]. This framework recommended guideline limits for pollutants that have been identified to be injurious to the health of the public including the environment and the built infrastructure.[14] These injurious pollutants include particulate matter (PM) with a diameter of $\leq$ 10 μm ($PM_{10}$), nitrogen dioxide ($NO_2$), sulphur dioxide ($SO_2$) and carbon monoxide (CO).[15] The human health effects of exposure to $SO_2$, $NO_2$, $O_3$ and $PM_{10}$ have previously been reported.[7, 16-19] Ozone, $NO_2$ and $SO_2$ pollutants can all cause lethal effects on the airway[20] such as an increase in bronchial reactivity,[21, 22] airway oxidative stress,[23] pulmonary and systemic inflammation,[24] amplification of viral infections[25] and reduction in airway ciliary activity.[26]

South Africa has one of the largest industrialised economies in the Southern Hemisphere and is the only industrialised regional energy producer on the African continent with significant mining and metallurgical activities.[27] It is an arid country with high naturally-occurring dust levels, compounded by industrial and vehicular pollution emissions.[28] Excessive high particulate matter pollution levels have been observed in industrialised regions and urban areas which are said to contribute up to 30% of particulate pollution in the country.[29] Significant associations between exposure to particulate matter and respiratory, cardiovascular and cerebrovascular risks have been reported in South Africa.[30]

Therefore, increased emphasis on human health concerns from air pollution necessitates the need for estimating the association between exposure and adverse health effects. The United States

Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework is a handy tool that can be used to estimate human health risk that can result from exposure to a given pollutant.[31] In their studies,[32, 33] reported that health risk assessment is useful for estimating the occurrence of adverse health effects in children and adults resulting from the direct inhalation of atmospheric particulates in urban areas. This framework was first introduced by the National Research Council in 1994[34] and has been previously used in few studies in South Africa.[31, 35-37] However, an HHRA framework on $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ has never been previously used in Pretoria West, South Africa. Hence, in view of the known health effects of exposure to sub-10μm PM and other gaseous pollutants, this study aimed to quantify the health risk of people living in the urban area in Pretoria West using the HHRA framework.

## METHODS

### Study area and population

The study area was Pretoria West (25°44'46"S 28°11'17"E). Pretoria West is an industrial production area that accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter, fuel stations and a fuel tank farm. Pretoria is a city in the Northern part of Gauteng Province in Tshwane Metropolitan Municipality. It is situated approximately 55 km (34 mi) north-northeast of Johannesburg in the Northeast of South Africa, in a transitional belt between the plateau of the Highveld to the South and the lower-lying Bushveld to the North. Pretoria has a population of 741, 651 (49.75% males and 50.25% females) in 2011. This constitute 23.2% young (0-14 years) persons, 71.9% of working age (15-64 years) and 4.9% of elderly (65+ years) persons.[38]

### Data collection procedure

The study utilised secondary data obtained from the South African Weather Service (SAWS) through the South African Air Quality Information System (SAAQIS) website (www.saaqis.org.za) after the approval for its use was granted by the data originators, Environmental Management Services Department. The SAAQIS makes data available to stakeholders including the public and provides a mechanism to ensure uniformity in the way air quality data is managed i.e. captured, stored, validated, analysed and reported in South Africa.

The data originators obtained the data from a fixed ambient air quality monitoring station (Syntech Spectras GC955 series 600) located at Pretoria West at longitude 28.146108, latitude -25.7555 and 1329 m above sea level. Data requested by the researchers from the originators include hourly daily ambient level concentrations of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ for the year 2014.

Data analysis

SPSS version 20 was used for the statistical analyses of the data. Descriptive statistics such as mean and standard deviation was used to estimate the average concentration of pollutants that were monitored.

Human Health Risk Assessment

Health risk assessment is an inclusive procedure by which possible adverse effects of human exposure to toxic agents are characterised.[39] HHRA is predictive in nature and uses existing exposure data to measure health effects of exposure to a particular pollutant.[40] The HHRA framework used in this study has four components: hazard identification, dose-response assessment, exposure assessment and risk characterisation.

*Hazard identification*

The identification of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ as harmful and their attendant health risks was done through a review of existing literature.

*Dose-response assessment*

Here, the amount of the pollutant taken into the body is estimated as a function of concentration and the length of exposure [41] The dose-response assessment was not done in this study since it requires a full health screening and additional data from health records. Rather, we compared the measured ambient concentration of pollutants in the study area with the South African National ambient air quality standard which serves as the benchmark.

*Exposure assessment*

The exposure assessment identifies the population exposed to the hazard, the magnitude, and duration of exposure to the hazard. Our study assumed inhalation as the route of exposure to the monitored pollutants. As previously reported,[35] this study utilised a scenario assessment method where normal (average exposure) and worst-case (continuous exposure) scenarios were computed for both intermediate (24-hour) and chronic (annual) exposure periods for the different exposure groups. The normal acute (1-hour) exposure periods was also determined.

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the acute exposure rate equation is given as:

$$AHD = C \times IR/BW \qquad \text{(Equation 1)[41]}$$

Where *AHD* is the average hourly dose for inhalation (µg $kg^{-1}h^{-1}$), *C* the concentration of the chemical (µg $m^{-3}$), *IR* the inhalation rate ($m^3$ $h^{-1}$) and *BW* the body weight (kg).

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the chronic exposure equation used for the inhalation exposure route is:

ADD = (C x IR x ED) / (BW x AT) (Equation 2)[42]

Where ADD = average daily dose of the chemical of interest (µg$kg^{-1}day^{-1}$), C = concentration of the chemical in the atmosphere (µg$m^{-3}$), IR = inhalation rate ($m^3day^1$), ED = exposure duration (days), BW = average body weight of receptor over the exposure period (kg), AT = averaging time (days).

The exposure duration (ED) which is the length of time study population are exposed to a pollutant is expressed as:

ED = ET x EF x DE (Equation 3)[35]

Where ET = exposure time or event (hour $day^{-1}$), EF = exposure frequency (days $year^{-1}$), DE = duration of exposure (year)

The default values for EF, DE, and AT for each exposed groups are presented in table 1. The EF default value used was founded on the assumption that each population group will spend at least two weeks every year away from the study area.[43] The DE for an adult is estimated at 30 years while that of a child and an infant were 1 and 12 years respectively. The AT is estimated as the product of the duration of exposure by 365 days $year^{-1}$.

Table 1: Exposure frequency, exposure duration and averaging time for different exposure groups

| Exposed group | EF (days $yr^{-1}$) | DE (year) | AT (days) |
|---|---|---|---|
| Infant (Birth to 1 year) | 350 | 1 | 365 (1 x 365) |
| Child (6 to 12 years) | 350 | 12 | 4380 (12 x 365) |
| Adult (19 to 75 years) | 350 | 30 | 10950 (30 x 365) |

*EF = exposure frequency; DE = duration of exposure; AT = averaging time*
Source: Adapted from [35]

The ET for each population group is based on the normal and worst case scenarios for acute, intermediate, and chronic exposure periods (see table 2). Thus, the intermediate ET for adults

was estimated at 3hour day$^{-1}$, based on the notion that the remainder of their time is spent either at work, away from Pretoria West, or indoors. ET for children was assumed to be greater since they have more time to play outdoors at the end of the school days; infants were assumed to spend the majority of the day indoors. Default values were used for IR and BW[43] and are given in table 3 for each exposure groups.

Table 2: Exposure time for normal and worst case scenarios for acute, intermediate and chronic exposures

| | Exposure | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | 1 | 1 | 24 | 14.6 [(350/24) x 1] | 350 (1 x 350) |
| Child (6 to 12 years) | 1 | 6 | 24 | 1050.0 [(4200/24) x 6] | 4200 (12 x 350) |
| Adult (19 to 75 years) | 1 | 3 | 24 | 1312.5 [(10500/24) x 3] | 10500 (30 x 350) |

Source: Adapted from [35]

Table 3: Average Inhalation rates and Body weights of exposed population

| Exposed group | Mean inhalation rate ($m^3hr^{-1}$) | | Mean body weight |
|---|---|---|---|
| | Acute exposure | Chronic exposure | |
| Infant (Birth to 1 year) | 0.3 | 6.8 | 11.3 |
| Child (6 to 12 years) | 1.2 | 13.5 | 45.3 |
| Adult (19 to 75 years) | 1.2 | 13.3 | 71.8 |

Source: Adapted from [35]

*Risk characterisation*

Risk characterisation is the quantitative estimation of the health risk of exposure to a pollutant. Here, the non-carcinogenic health effects were expressed as a dimensionless ratio called a hazard quotient (HQ), which indicates the presence or absence of adverse health effects due to exposure.[36, 43] HQ also provides an indication of whether only sensitive individuals will be affected, or if both healthy and sensitive individuals will be affected. Non-cancer risks were calculated for both acute and chronic exposure scenarios as:

HQ = ADD/REL (Chronic exposure) or (Equation 4)

HQ = AHD/REL (Acute exposure) (Equation 5)

Where *REL* is the dose at which significant adverse health effects will occur in exposed subjects, compared to an unexposed group. In this study, we used the term "reference exposure level" (REL), as adopted by the Office of the Environmental Health Hazard Assessment (OEHHA).[44] The RELs that is used is presented in table 4.

An HQ of 1.0 is considered to be the benchmark of safety. An HQ that is < 1.0 indicates a negligible risk i.e. the pollutant under scrutiny is not likely to induce adverse health effects, even to a sensitive individual. An HQ > 1.0 indicates that there may be some risks to sensitive individuals as a result of exposure.[45]

Table 4: Reference Exposure Levels for different pollutants

| Pollutant | 1 hour ($\mu g/m^3$) | 8 Hours ($\mu g/m^3$) | 24 hours ($\mu g/m^3$) | Annual mean ($\mu g/m^3$) |
|---|---|---|---|---|
| $PM_{10}$ | - | | *75 | *40 |
| $NO_2$ | *200 | | **188 | *40 |
| $SO_2$ | *350 | | *125 | *50 |
| CO | ***29770 | ***10305 | - | - |
| $O_3$ | **226 | *120 | - | - |

**NAAQS (National ambient air quality standard for South Africa); ** South Africa standards – Air quality act (Act 39 of 2004); **Default value was converted from ppm to $\mu g/m^3$*
Source: Department of Environmental Affairs[46].

## RESULTS

*Particulate matter ($PM_{10}$) concentration*

The mean hourly, daily and annual concentration of $PM_{10}$ in the Pretoria West are 67.74 $\mu g/m^3$, 52.01 $\mu g/m^3$ and 48.26 $\mu g/m^3$ respectively (Table 5). Though, the daily (24 hours) guideline limit of 75 $\mu g/m^3$ set by the NAAQS was not exceeded, the annual recommended mean limit of 45 $\mu g/m^3$ that should not be exceeded was surpassed. The 1-h (acute) scenario was not considered as a 1-h REL value for $PM_{10}$ was not found in the literature. The hazard quotient (HQ) from the health risk characterisation from exposure to $PM_{10}$ is provided in Table 6. The results showed that under the normal and worst-case scenario for average and continuous exposures respectively, the risk of having health related problems by the exposed population is low (HQ < 1). This is because HQ of < 1.0 indicates that $PM_{10}$ is not likely to induce adverse health

outcomes. However, infants ($2.0 \times 10^{-2}$ vs $4.2 \times 10^{-1}$) followed by children ($1.1 \times 10^{-1}$ vs $4.2 \times 10^{-1}$) are likely to be affected from exposure to $PM_{10}$ than adults ($3.0 \times 10^{-2}$ vs $2.7 \times 10^{-1}$) under the normal and worst-case scenario respectively for intermediate exposure. For the chronic (annual) exposure scenario for normal and worst-case exposures, the HQ is > 1.0 for infants, children, and adults. These results show that sensitive exposed population may be at a risk of developing health related problems from chronic exposure to $PM_{10}$. Infants are more likely to be affected than children and adults under the normal chronic exposure while children will be more affected than infants and adults under the worst-case scenario.

Table 5: Summary statistics of ambient concentrations of pollutants

| Averaging period | $PM_{10}$ (µg/m$^3$) Mean ±SD | $NO_2$ (µg/m$^3$) Mean ±SD | $SO_2$ (µg/m$^3$) Mean ±SD | CO (µg/m$^3$) Mean ±SD | $O_3$ (µg/m$^3$) Mean ±SD |
|---|---|---|---|---|---|
| 1 Hour | 67.74 ± 61.63 | 17.44 ± 17.26 | 29.63 ± 33.64 | 1442.6 ± 1248.05 | 29.78 ± 8.69 |
| 8-Hours | - | - | - | 618.30 ± 618.30 | 22.15 ± 7.96 |
| 24-Hours | 52.01 ± 50.58 | 13.13 ± 13.21 | 21.48 ± 27.71 | - | - |
| Annual | 48.26 ± 43.41 | 11.50 ± 11.61 | 18.68 ± 25.36 | - | - |

*SD – Standard deviation*

Table 6: Hazard quotients for normal and worst-case exposure scenarios to $PM_{10}$

| Exposed group | Exposure | | | |
|---|---|---|---|---|
| | Intermediate | | Chronic | |
| | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-2}$ | $4.2 \times 10^{-1}$ | $1.0 \times 10^{1}$ | $2.44 \times 10^{2}$ |
| Child (6 to 12 years) | $1.1 \times 10^{-1}$ | $4.2 \times 10^{-1}$ | $3.62 \times 10^{2}$ | $1.45 \times 10^{3}$ |
| Adult (19 to 75 years) | $3.0 \times 10^{-2}$ | $2.7 \times 10^{-1}$ | $2.81 \times 10^{2}$ | $2.25 \times 10^{3}$ |

*The 1-h (acute) scenario was not considered since a 1-h REL value for $PM_{10}$ was not found in literature*

*Sulphur dioxide concentration*

The measured average concentration of $SO_2$ for 1-h, 24-hour and annual averages in the study area were 29.63 µg/m$^3$, 21.48 µg/m$^3$ and 18.68 µg/m$^3$ respectively (Table 5). These values are far less than the mean values of 350 µg/m$^3$, 125 µg/m$^3$ and 50 µg/m$^3$ as provided by NAAQS for 1-h, 24-hour and annual averages respectively that should not be exceeded (Table 4). Estimation of risk for acute and intermediate (normal and worst-case) exposures to $SO_2$ revealed that the HQ is

< 1.0 for infants, children, and adults (Table 7). This implies a negligible risk, even to a sensitive individual. For acute exposure, infants and children ($2.0 \times 10^{-3}$) are likely to be affected the same way from exposure to $SO_2$ compared to adults ($1.4 \times 10^{-3}$). Under the normal and worst-case scenarios for chronic exposure, the HQ was > 1.0 for all study population. This indicates that there may be some risks to sensitive individuals as a result of exposure to $SO_2$. The severity of exposures differs for different age groups.

Table 7: Hazard quotients for normal and worst-case exposure scenarios to $SO_2$ at different levels of exposures

| | Exposure | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-3}$ | $4.0 \times 10^{-3}$ | $1.1 \times 10^{-1}$ | $31.5 \times 10^{-1}$ | $7.55 \times 10^{1}$ |
| Child (6 to 12 years) | $2.0 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $1.0 \times 10^{-1}$ | $1.12 \times 10^{2}$ | $4.49 \times 10^{2}$ |
| Adult (19 to 75 years) | $1.4 \times 10^{-3}$ | $8.0 \times 10^{-3}$ | $7 \times 10^{-2}$ | $8.72 \times 10^{1}$ | $6.98 \times 10^{2}$ |

*Nitrogen dioxide concentration*

The monitored 1-h, 24-h and annual concentrations of $NO_2$ shown in Table 5 were 17.44 µg/m$^3$, 13.13 µg/m$^3$ and 11.50 µg/m$^3$. The NAAQS 1-h, 24-h and annual guideline of 200 µg/m$^3$, 188 µg/m$^3$ and 40 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The hazard quotients calculated for each of acute and intermediate (normal and worst-case scenarios) exposures (shown in Table 8) showed no likelihood of adverse health effects occurring at this level of exposure for an infant, child and adult (HQ < 1.0). However, there is likelihood that infants and children ($2.3 \times 10^{-3}$) might be affected by acute exposure to $NO_2$ than adults ($1.5 \times 10^{-3}$). Moreover, having an adverse health outcome from normal and worst-case chronic exposure to $NO_2$ was found to be higher (HQ > 1.0) for all age groups. Children ($3.05 \times 10^{2}$) appears more likely to be affected by normal chronic exposure than infants ($8.6 \times 10^{1}$) and adults ($2.37 \times 10^{2}$) whereas for worst-case chronic exposure, adults ($1.893 \times 10^{3}$) are more likely to be affected.

Table 8: Hazard quotients for normal and worst-case exposure scenarios to $NO_2$ at different levels of exposures

| Exposed group | Exposure | | | | |
|---|---|---|---|---|---|
| | Acute | Intermediate | | Chronic | |
| | | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.3 \times 10^{-3}$ | $6.0 \times 10^{-3}$ | $1.5 \times 10^{-1}$ | $8.6 \times 10^{1}$ | $2.05 \times 10^{2}$ |
| Child (6 to 12 years) | $2.3 \times 10^{-3}$ | $4 \times 10^{-2}$ | $1.5 \times 10^{-1}$ | $3.05 \times 10^{2}$ | $1.218 \times 10^{3}$ |
| Adult (19 to 75 years) | $1.5 \times 10^{-3}$ | $1.0 \times 10^{-2}$ | $9.0 \times 10^{-2}$ | $2.37 \times 10^{2}$ | $1.893 \times 10^{3}$ |

*Carbon monoxide concentration*

CO concentrations of 1442.6 µg/m$^3$ (1-h average) and 618.30 µg/m$^3$ (8-h average) (Table 5) were not exceeded in comparison with the NAAQS guideline of 29770 µg/m$^3$ for 1-h and 10305 µg/m$^3$ for 8-h exposure limit. Estimation of risk for acute exposure to CO revealed that the HQ is < 1.0 for infants, children, and adults (Table 9). This implies a negligible risk, even to sensitive infants, children, and adults. Though, infants, and children ($1.3 \times 10^{-3}$) may suffer the effects than adults ($8.0 \times 10^{-4}$). Additionally, infants, children and adults living in the study area are not likely to experience adverse health effects associated with normal and worst-case exposure scenarios to 8-h CO (HQ < 1.0).

Table 9: Hazard quotients for normal and worst-case exposure scenarios to CO at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $1.3 \times 10^{-3}$ | $2.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $1.3 \times 10^{-3}$ | $9.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $8.0 \times 10^{-4}$ | $3.0 \times 10^{-3}$ | $8.0 \times 10^{-4}$ |

**Intermediate – 8 Hour exposure period*

*Ozone concentration*

The monitored concentration of $O_3$ for 1-h and 8-h average in the study area are 29.78 µg/m$^3$ and 22.15 µg/m$^3$ respectively (Table 5). The NAAQS and annual guideline of 226 µg/m$^3$ and 120 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The HQ calculated for both the acute and intermediate (normal and worst-case) exposure scenarios shows no likelihood of

adverse health effects being experienced by any individuals (HQ < 1.0) (Table 10). During acute exposure, adults ($2.2 \times 10^{-2}$) are less likely to be affected than infants and children ($3.0 \times 10^{-3}$) while the reverse is the case for continuous exposure to $O_3$ for 8 hours.

Table 10: Hazard quotients for normal and worst-case exposure scenarios to $O_3$ at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $3.5 \times 10^{-3}$ | $5.0 \times 10^{-3}$ | $4.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $3.5 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $4.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $2.2 \times 10^{-2}$ | $9.0 \times 10^{-3}$ | $2.0 \times 10^{-2}$ |

**Intermediate – 8 Hour exposure period*

## DISCUSSION

Air pollution remains a global environmental threat and a public health risk. Researchers posited that health effects from exposure to ambient air pollution can occur at or below levels allowed by the national and international air quality standards. Findings from our study revealed that the 24-h $PM_{10}$ ambient quality standard of 75 $\mu g/m^3$ was not exceeded on any of the days during the monitoring period. This is in contrast with other studies conducted elsewhere in South Africa. A 24-h $PM_{10}$ of 157.37 $\mu g/m^3$ (highest peak) and 110 $\mu g/m^3$ was reported by [31] and [35] respectively. The average annual concentration of $PM_{10}$ recorded in our study was slightly above the guideline limit of 45 $\mu g/m^3$ set by the NAAQS. This may account for the chronic (annual) HQ > 1 recorded in our study, an indication of some level of risk to long-term exposure to $PM_{10}$.

In South Africa it was estimated that outdoor air pollution was responsible for 3.7% of the national mortality attributable to cancers of the trachea, bronchus and lung in adults aged 30 years and older, and 1.1% of mortality in children under 5 years of age.[31] A review of 12 previous studies in year 2001 affirmed that a 10-$\mu g/m^3$ increase in $PM_{10}$ causes an increase in hospital admissions for congestive heart failure and ischemic heart disease.[47] Among the vulnerable population (elderly and people with previous medical history of respiratory and cardiovascular diseases), long-term exposure to $PM_{10}$ has been linked with an increase in morbidity and mortality from respiratory and cardiovascular diseases.[48] Also for adults, large population studies have shown an association between respiratory (admissions for asthma,

COPD, and pneumonia) hospitalization and ambient $PM_{10}$.[49] However, the effects seem to be stronger for elderly patients with even short-term exposures.[50]

This study further revealed that the 1-hour, 24-hours and annual mean concentration for $NO_2$ are below the national standard. Evidence from the risk characterisation assessment shows a negligible risk to acute and intermediate exposure to ambient levels of $NO_2$. However, 1-year exposure to ambient levels of $NO_2$ could pose some risks to the sensitive individual. Recent epidemiological studies have revealed that exposure to low levels of $NO_2$ could increase emergency room hospitalization for acute and obstructive lung diseases in the general population .[17, 51] Studies conducted in Canada, Denmark and Italy found a significant association between exposures to levels of $NO_2$ and acute ischemic stroke.[16, 52] However, some studies did not find significant associations between exposure to ambient and personal levels of $NO_2$ and health effects (Linaker *et al.,* 2000; Sarnat *et al.,* 2001).[53, 54]

Our study further shows low ambient value (compared to national standard) for $SO_2$ in Pretoria West. Similarly, there is no likelihood of health risk (HQ < 1) associated with 1-hour and 24-hours exposure to $SO_2$. Though, some levels of risk to sensitive individuals was found for chronic (annual) exposure to $SO_2$ in the study area. The possibility of $SO_2$ worsening childhood asthma at fairly modest concentration, that is well below the US EPA standards and WHO guidelines have been reported.[55] Multi-city studies conducted in Europe and Asia offer further proof supporting the short-term association of $SO_2$ with adverse health outcomes including both mortality[56] and morbidity.[57]

In this study, low ambient concentrations of CO and $O_3$ was recorded. Researchers are of the opinion that exposure to ambient levels of CO is often not recognized; its toxicity is mostly underreported and misdiagnosed due to its non-irritation and imperceptibility in the air we inhale.[18] CO remains the leading cause of poison correlated mortality in the United States.[18] On the other hand, $O_3$ is a strong oxidant that weakens biological tissues, thus resulting in increased use of medication, ailment and death.[58] It has even been previously established that no level of exposure to $O_3$ is safe since health risk has been found to be associated with $O_3$ even at concentrations below recommended standards.[58]

Furthermore, evidence from the risk characterisation assessment in this study shows that adults are less likely to be affected by acute and intermediate exposure to ambient concentrations of CO and O3 than infants and children. This was also true for acute and intermediate exposures to $NO_2$ and $SO_2$. It has been documented that children have a higher susceptibility to environmental pollutants than adults. They are considered a risk group for numerous reasons including their relative higher amount of air inhalation (the air intake per weight unit of a resting infant is twice that of an adult), their not fully developed immune system and lungs.[31]

## CONCLUSIONS

Ambient air pollution is composed of both suspended particulates and gaseous pollutants, with the gaseous components comprising $O_3$, CO, $NO_2$, and $SO_2$. The acute, intermediate and chronic ambient concentration of $PM_{10}$ and the gaseous pollutants recorded in Pretoria West were within the South African National Ambient Air Quality. No health risk was found to be associated with acute and intermediate exposure to the pollutants, though, infants and children than adults, are more likely to suffer the health effects. Long term chronic (annual) exposure to normal and worst-case exposure scenarios to each of the pollutants posed some levels of risks to sensitive individuals, with the severity of risk differing across groups. Identification of the possibility of these pollutants to pose health hazards, as measured through the human health risk assessment framework will make valuable contributions to government, environmental specialists and relevant stakeholders in taking more concrete steps to protect and prolong human lives. Additionally, these findings will assist policy makers in enforcing or strengthening existing legislation that limits the release of pollutants into the atmosphere, or institute risk management strategies.

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# Health Risk of Inhalation Exposure to Sub-10 µm Particulate Matter and Gaseous Pollutants in an Urban-industrial Area: a prospective study

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# Health Risk of Inhalation Exposure to Sub-10 µm Particulate Matter and Gaseous Pollutants in an Urban-industrial Area: a prospective study

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## ABSTRACT

**Objective:** To access the health risks associated with exposure to particulate matter ($PM_{10}$), sulphur dioxide ($SO_2$), nitrogen dioxide ($NO_2$), carbon monoxide (CO) and ozone ($O_3$) .

**Design:** The study is a prospective study that utilised the year 2014 hourly ambient pollution data.

**Setting:** The study was conducted in an industrial area located in Pretoria West, South Africa. The area accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter.

**Data and method:** Estimate of possible health risks from exposure to airborne $PM_{10}$, $SO_2$, $NO_2$, CO, and $O_3$ was done using the United States Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework. A scenario-assessment approach where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for both intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults). The normal acute (1-hour) exposure to these pollutants was also determined.

**Outcome measures:** Presence or absence of adverse health effects from exposure to airborne pollutants.

**Results:** Average annual ambient concentration of $PM_{10}$, $NO_2$ and $SO_2$ recorded were 48.3±43.4 µg/m$^3$, 11.50±11.6 µg/m$^3$ and 18.68±25.4 µg/m$^3$ respectively. Whereas, the South African National Ambient Air Quality recommended 40 µg/m$^3$, 40 µg/m$^3$ and 50 µg/m$^3$ for $PM_{10}$, $NO_2$ and $SO_2$ respectively. Exposure to an hour concentration of $NO_2$, $SO_2$, CO and $O_3$; 8-h concentration of CO, $O_3$; and 24-h concentration of $PM_{10}$, $NO_2$ and $SO_2$ will not likely produce adverse effects to sensitive exposed groups. Though, infants, and children, rather than adults, are

more likely to be affected. Moreover, for chronic annual exposure, $PM_{10}$, $NO_2$ and $SO_2$ posed a health risk to sensitive individuals, with the severity of risk varying across exposed groups.

**Conclusions:** Long-term chronic exposure to airborne $PM_{10}$, $NO_2$, and $SO_2$ pollutants may results in health risks among the study population.

**Keywords**: particulate matter, gaseous pollutants, health risk assessment, exposure groups, South Africa

## Strengths and Limitations of this study

- Large data set spanning hourly ambient concentration of pollutants for a whole year.
- This is the first study in Pretoria West, South Africa to estimate health risks of human exposure to airborne pollutants using US EPA assessment model.
- In our study, prediction of both long-term and short-term health effects in infants, children, and adults resulting from inhalation of pollutants was possible.
- However, the health risk that could result from exposure to the combination of the pollutants could not be determined.

## INTRODUCTION

Air pollution is a multifaceted mix consisting of both suspended particulates and gaseous pollutants.[1] Globally, air pollution continues to be a major environmental problem that has been recognised as an important public health risk.[2] The upsurge in human population, industrialisation, urbanisation, modernisation and its attendant increase in vehicular emissions and activities are the major contributors to the rising urban air quality problems.[3]

The World Health Organization (WHO) in the year 2013, asserted that annually, the urban ambient air pollution was predicted to cause 2 million deaths in the world.[4] Epidemiological studies have linked exposure to ambient air pollution with adverse human health effects.[5-7] Exposure to air pollution can result in both acute (short-term) and chronic (long-term) health effects.[8, 9] The acute effects of air pollution on human health were sufficiently established in the 20th century when severe air pollution scenarios in Europe and in the United States resulted in morbidities and mortalities in hundreds of thousands of people.[10]

Air pollution is a known trigger of Chronic Obstructive Pulmonary Disease (COPD)[11] and has informed the establishment of air quality standards in many countries [12, 13]. The broad legislative framework for air quality assessment in populated areas was put in place by the European Union Directive on Air Quality 2008/50/EC [14]. This framework recommended guideline limits for pollutants that have been identified to be injurious to the health of the public including the environment and the built infrastructure.[14] These injurious pollutants include particulate matter (PM) with a diameter of $\leq$ 10 μm ($PM_{10}$), nitrogen dioxide ($NO_2$), sulphur dioxide ($SO_2$) and carbon monoxide (CO).[15] The human health effects of exposure to $SO_2$, $NO_2$, $O_3$ and $PM_{10}$ have previously been reported.[7, 16-19] Ozone, $NO_2$ and $SO_2$ pollutants can all cause lethal effects on the airway[20] such as an increase in bronchial reactivity,[21, 22] airway oxidative stress,[23] pulmonary and systemic inflammation,[24] amplification of viral infections[25] and reduction in airway ciliary activity.[26]

South Africa has one of the largest industrialised economies in the Southern Hemisphere and is the only industrialised regional energy producer on the African continent with significant mining and metallurgical activities.[27] It is an arid country with high naturally-occurring dust levels, compounded by industrial and vehicular pollution emissions.[28] Excessive high particulate matter pollution levels have been observed in industrialised regions and urban areas which are said to contribute up to 30% of particulate pollution in the country.[29] Significant associations between exposure to particulate matter and respiratory, cardiovascular and cerebrovascular risks have been reported in South Africa.[30]

Therefore, increased emphasis on human health concerns from air pollution necessitates the need for estimating the association between exposure and adverse health effects. The United States

Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework is a handy tool that can be used to estimate human health risk that can result from exposure to a given pollutant.[31] In their studies,[32, 33] reported that health risk assessment is useful for estimating the occurrence of adverse health effects in children and adults resulting from the direct inhalation of atmospheric particulates in urban areas. This framework was first introduced by the National Research Council in 1994[34] and has been previously used in few studies in South Africa.[31, 35-37] However, an HHRA framework on $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ has never been previously used in Pretoria West, South Africa. Hence, in view of the known health effects of exposure to sub-10μm PM and other gaseous pollutants, this study aimed to quantify the health risk of people living in the urban area in Pretoria West using the HHRA framework.

## METHODS

### Study area and population

The study area was Pretoria West (25°44'46"S 28°11'17"E). Pretoria West is an industrial production area that accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter, fuel stations and a fuel tank farm. Pretoria is a city in the Northern part of Gauteng Province in Tshwane Metropolitan Municipality. It is situated approximately 55 km (34 mi) north-northeast of Johannesburg in the Northeast of South Africa, in a transitional belt between the plateau of the Highveld to the South and the lower-lying Bushveld to the North. Pretoria has a population of 741, 651 (49.75% males and 50.25% females) in 2011. This constitute 23.2% young (0-14 years) persons, 71.9% of working age (15-64 years) and 4.9% of elderly (65+ years) persons.[38]

### Data collection procedure

The study utilised secondary data obtained from the South African Weather Service (SAWS) through the South African Air Quality Information System (SAAQIS) website (www.saaqis.org.za) after the approval for its use was granted by the data originators, Environmental Management Services Department. The SAAQIS makes data available to stakeholders including the public and provides a mechanism to ensure uniformity in the way air quality data is managed i.e. captured, stored, validated, analysed and reported in South Africa.

The data originators obtained the data from a fixed ambient air quality monitoring station (Syntech Spectras GC955 series 600) located at Pretoria West at longitude 28.146108, latitude -25.7555 and 1329 m above sea level. Data requested by the researchers from the originators include hourly daily ambient level concentrations of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ for the year 2014.

Data analysis

SPSS version 20 was used for the statistical analyses of the data. Descriptive statistics such as mean and standard deviation was used to estimate the average concentration of pollutants that were monitored.

Human Health Risk Assessment

Health risk assessment is an inclusive procedure by which possible adverse effects of human exposure to toxic agents are characterised.[39] HHRA is predictive in nature and uses existing exposure data to measure health effects of exposure to a particular pollutant.[40] The HHRA framework used in this study has four components: hazard identification, dose-response assessment, exposure assessment and risk characterisation.

*Hazard identification*

The identification of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ as harmful and their attendant health risks was done through a review of existing literature.

*Dose-response assessment*

Here, the amount of the pollutant taken into the body is estimated as a function of concentration and the length of exposure [41] The dose-response assessment was not done in this study since it requires a full health screening and additional data from health records. Rather, we compared the measured ambient concentration of pollutants in the study area with the South African National ambient air quality standard which serves as the benchmark.

*Exposure assessment*

The exposure assessment identifies the population exposed to the hazard, the magnitude, and duration of exposure to the hazard. Our study assumed inhalation as the route of exposure to the monitored pollutants. As previously reported,[35] this study utilised a scenario assessment method where normal (average exposure) and worst-case (continuous exposure) scenarios were computed for both intermediate (24-hour) and chronic (annual) exposure periods for the different exposure groups. The normal acute (1-hour) exposure periods was also determined.

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the acute exposure rate equation is given as:

$$AHD = C \times IR/BW \qquad \text{(Equation 1)[41]}$$

Where *AHD* is the average hourly dose for inhalation (µg $kg^{-1}h^{-1}$), *C* the concentration of the chemical (µg $m^{-3}$), *IR* the inhalation rate ($m^3$ $h^{-1}$) and *BW* the body weight (kg).

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the chronic exposure equation used for the inhalation exposure route is:

ADD = (C x IR x ED) / (BW x AT) (Equation 2)[42]

Where ADD = average daily dose of the chemical of interest (µg$kg^{-1}day^{-1}$), C = concentration of the chemical in the atmosphere (µg$m^{-3}$), IR = inhalation rate ($m^3day^1$), ED = exposure duration (days), BW = average body weight of receptor over the exposure period (kg), AT = averaging time (days).

The exposure duration (ED) which is the length of time study population are exposed to a pollutant is expressed as:

ED = ET x EF x DE (Equation 3)[35]

Where ET = exposure time or event (hour $day^{-1}$), EF = exposure frequency (days $year^{-1}$), DE = duration of exposure (year)

The default values for EF, DE, and AT for each exposed groups are presented in table 1. The EF default value used was founded on the assumption that each population group will spend at least two weeks every year away from the study area.[43] The DE for an adult is estimated at 30 years while that of a child and an infant were 1 and 12 years respectively. The AT is estimated as the product of the duration of exposure by 365 days $year^{-1}$.

Table 1: Exposure frequency, exposure duration and averaging time for different exposure groups

| Exposed group | EF (days $yr^{-1}$) | DE (year) | AT (days) |
|---|---|---|---|
| Infant (Birth to 1 year) | 350 | 1 | 365 (1 x 365) |
| Child (6 to 12 years) | 350 | 12 | 4380 (12 x 365) |
| Adult (19 to 75 years) | 350 | 30 | 10950 (30 x 365) |

*EF = exposure frequency; DE = duration of exposure; AT = averaging time*
Source: Adapted from [35]

The ET for each population group is based on the normal and worst case scenarios for acute, intermediate, and chronic exposure periods (see table 2). Thus, the intermediate ET for adults

was estimated at 3hour day$^{-1}$, based on the notion that the remainder of their time is spent either at work, away from Pretoria West, or indoors. ET for children was assumed to be greater since they have more time to play outdoors at the end of the school days; infants were assumed to spend the majority of the day indoors. Default values were used for IR and BW[43] and are given in table 3 for each exposure groups.

Table 2: Exposure time (hr.) for normal and worst case scenarios for acute, intermediate and chronic exposures

| | Exposure time (hr.) | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | 1 | 1 | 24 | 14.6 [(350/24) x 1] | 350 (1 x 350) |
| Child (6 to 12 years) | 1 | 6 | 24 | 1050.0 [(4200/24) x 6] | 4200 (12 x 350) |
| Adult (19 to 75 years) | 1 | 3 | 24 | 1312.5 [(10500/24) x 3] | 10500 (30 x 350) |

Source: Adapted from [35]

Table 3: Average Inhalation rates and Body weights of exposed population

| Exposed group | Mean inhalation rate ($m^3hr^{-1}$) | | Mean body weight (kg) |
|---|---|---|---|
| | Acute exposure | Chronic exposure | |
| Infant (Birth to 1 year) | 0.3 | 6.8 | 11.3 |
| Child (6 to 12 years) | 1.2 | 13.5 | 45.3 |
| Adult (19 to 75 years) | 1.2 | 13.3 | 71.8 |

Source: Adapted from [35]

*Risk characterisation*

Risk characterisation is the quantitative estimation of the health risk of exposure to a pollutant. Here, the non-carcinogenic health effects were expressed as a dimensionless ratio called a hazard quotient (HQ), which indicates the presence or absence of adverse health effects due to exposure.[36, 43] HQ also provides an indication of whether only sensitive individuals will be affected, or if both healthy and sensitive individuals will be affected. Non-cancer risks were calculated for both acute and chronic exposure scenarios as:

HQ = ADD/REL (Chronic exposure) or (Equation 4)

HQ = AHD/REL (Acute exposure) (Equation 5)

Where *REL* is the dose at which significant adverse health effects will occur in exposed subjects, compared to an unexposed group. In this study, we used the term "reference exposure level" (REL), as adopted by the Office of the Environmental Health Hazard Assessment (OEHHA).[44] The RELs that is used is presented in table 4.

An HQ of 1.0 is considered to be the benchmark of safety. An HQ that is < 1.0 indicates a negligible risk i.e. the pollutant under scrutiny is not likely to induce adverse health effects, even to a sensitive individual. An HQ > 1.0 indicates that there may be some risks to sensitive individuals as a result of exposure.[45]

Table 4: Reference Exposure Levels for different pollutants

| Pollutant | 1 hour ($\mu g/m^3$) | 8 Hours ($\mu g/m^3$) | 24 hours ($\mu g/m^3$) | Annual mean ($\mu g/m^3$) |
|---|---|---|---|---|
| $PM_{10}$ | - | | *75 | *40 |
| $NO_2$ | *200 | | **188 | *40 |
| $SO_2$ | *350 | | *125 | *50 |
| CO | ***29770 | ***10305 | - | - |
| $O_3$ | **226 | *120 | - | - |

**NAAQS (National ambient air quality standard for South Africa); ** South Africa standards – Air quality act (Act 39 of 2004); **Default value was converted from ppm to $\mu g/m^3$*
Source: Department of Environmental Affairs[46].

## RESULTS

*Particulate matter ($PM_{10}$) concentration*

The mean hourly, daily and annual concentration of $PM_{10}$ in the Pretoria West are 67.74 $\mu g/m^3$, 52.01 $\mu g/m^3$ and 48.26 $\mu g/m^3$ respectively (Table 5). Though, the daily (24 hours) guideline limit of 75 $\mu g/m^3$ set by the NAAQS was not exceeded, the annual recommended mean limit of 45 $\mu g/m^3$ that should not be exceeded was surpassed. The 1-h (acute) scenario was not considered as a 1-h REL value for $PM_{10}$ was not found in the literature. The hazard quotient (HQ) from the health risk characterisation from exposure to $PM_{10}$ is provided in Table 6. The results showed that under the normal and worst-case scenario for average and continuous exposures respectively, the risk of having health related problems by the exposed population is low (HQ < 1). This is because HQ of < 1.0 indicates that $PM_{10}$ is not likely to induce adverse health

outcomes. However, infants ($2.0 \times 10^{-2}$ vs $4.2 \times 10^{-1}$) followed by children ($1.1 \times 10^{-1}$ vs $4.2 \times 10^{-1}$) are likely to be affected from exposure to $PM_{10}$ than adults ($3.0 \times 10^{-2}$ vs $2.7 \times 10^{-1}$) under the normal and worst-case scenario respectively for intermediate exposure. For the chronic (annual) exposure scenario for normal and worst-case exposures, the HQ is > 1.0 for infants, children, and adults. These results show that sensitive exposed population may be at a risk of developing health related problems from chronic exposure to $PM_{10}$. Infants are more likely to be affected than children and adults under the normal chronic exposure while children will be more affected than infants and adults under the worst-case scenario.

Table 5: Summary statistics of ambient concentrations of pollutants

| Averaging period | $PM_{10}$ (µg/m$^3$) Mean ±SD | $NO_2$ (µg/m$^3$) Mean ±SD | $SO_2$ (µg/m$^3$) Mean ±SD | CO (µg/m$^3$) Mean ±SD | $O_3$ (µg/m$^3$) Mean ±SD |
|---|---|---|---|---|---|
| 1 Hour | 67.74 ± 61.63 | 17.44 ± 17.26 | 29.63 ± 33.64 | 1442.6 ± 1248.05 | 29.78 ± 8.69 |
| 8-Hours | - | - | - | 618.30 ± 618.30 | 22.15 ± 7.96 |
| 24-Hours | 52.01 ± 50.58 | 13.13 ± 13.21 | 21.48 ± 27.71 | - | - |
| Annual | 48.26 ± 43.41 | 11.50 ± 11.61 | 18.68 ± 25.36 | - | - |

*SD – Standard deviation*

Table 6: Hazard quotients for normal and worst-case exposure scenarios to $PM_{10}$

| Exposed group | Exposure | | | |
|---|---|---|---|---|
| | Intermediate | | Chronic | |
| | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-2}$ | $4.2 \times 10^{-1}$ | $1.0 \times 10^{1}$ | $2.44 \times 10^{2}$ |
| Child (6 to 12 years) | $1.1 \times 10^{-1}$ | $4.2 \times 10^{-1}$ | $3.62 \times 10^{2}$ | $1.45 \times 10^{3}$ |
| Adult (19 to 75 years) | $3.0 \times 10^{-2}$ | $2.7 \times 10^{-1}$ | $2.81 \times 10^{2}$ | $2.25 \times 10^{3}$ |

*The 1-h (acute) scenario was not considered since a 1-h REL value for $PM_{10}$ was not found in literature*

*Sulphur dioxide concentration*

The measured average concentration of $SO_2$ for 1-h, 24-hour and annual averages in the study area were 29.63 µg/m$^3$, 21.48 µg/m$^3$ and 18.68 µg/m$^3$ respectively (Table 5). These values are far less than the mean values of 350 µg/m$^3$, 125 µg/m$^3$ and 50 µg/m$^3$ as provided by NAAQS for 1-h, 24-hour and annual averages respectively that should not be exceeded (Table 4). Estimation of risk for acute and intermediate (normal and worst-case) exposures to $SO_2$ revealed that the HQ is

< 1.0 for infants, children, and adults (Table 7). This implies a negligible risk, even to a sensitive individual. For acute exposure, infants and children ($2.0 \times 10^{-3}$) are likely to be affected the same way from exposure to $SO_2$ compared to adults ($1.4 \times 10^{-3}$). Under the normal and worst-case scenarios for chronic exposure, the HQ was > 1.0 for all study population. This indicates that there may be some risks to sensitive individuals as a result of exposure to $SO_2$. The severity of exposures differs for different age groups.

Table 7: Hazard quotients for normal and worst-case exposure scenarios to $SO_2$ at different levels of exposures

| | Exposure | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-3}$ | $4.0 \times 10^{-3}$ | $1.1 \times 10^{-1}$ | $31.5 \times 10^{-1}$ | $7.55 \times 10^{1}$ |
| Child (6 to 12 years) | $2.0 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $1.0 \times 10^{-1}$ | $1.12 \times 10^{2}$ | $4.49 \times 10^{2}$ |
| Adult (19 to 75 years) | $1.4 \times 10^{-3}$ | $8.0 \times 10^{-3}$ | $7 \times 10^{-2}$ | $8.72 \times 10^{1}$ | $6.98 \times 10^{2}$ |

*Nitrogen dioxide concentration*

The monitored 1-h, 24-h and annual concentrations of $NO_2$ shown in Table 5 were 17.44 µg/m$^3$, 13.13 µg/m$^3$ and 11.50 µg/m$^3$. The NAAQS 1-h, 24-h and annual guideline of 200 µg/m$^3$, 188 µg/m$^3$ and 40 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The hazard quotients calculated for each of acute and intermediate (normal and worst-case scenarios) exposures (shown in Table 8) showed no likelihood of adverse health effects occurring at this level of exposure for an infant, child and adult (HQ < 1.0). However, there is the likelihood that infants and children ($2.3 \times 10^{-3}$) might be affected by acute exposure to $NO_2$ than adults ($1.5 \times 10^{-3}$). Moreover, having an adverse health outcome from normal and worst-case chronic exposure to $NO_2$ was found to be higher (HQ > 1.0) for all age groups. Children ($3.05 \times 10^{2}$) appears more likely to be affected by normal chronic exposure than infants ($8.6 \times 10^{1}$) and adults ($2.37 \times 10^{2}$) whereas for worst-case chronic exposure, adults ($1.893 \times 10^{3}$) are more likely to be affected.

Table 8: Hazard quotients for normal and worst-case exposure scenarios to $NO_2$ at different levels of exposures

| Exposed group | Exposure | | | | |
|---|---|---|---|---|---|
| | Acute | Intermediate | | Chronic | |
| | | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.3 \times 10^{-3}$ | $6.0 \times 10^{-3}$ | $1.5 \times 10^{-1}$ | $8.6 \times 10^{1}$ | $2.05 \times 10^{2}$ |
| Child (6 to 12 years) | $2.3 \times 10^{-3}$ | $4 \times 10^{-2}$ | $1.5 \times 10^{-1}$ | $3.05 \times 10^{2}$ | $1.218 \times 10^{3}$ |
| Adult (19 to 75 years) | $1.5 \times 10^{-3}$ | $1.0 \times 10^{-2}$ | $9.0 \times 10^{-2}$ | $2.37 \times 10^{2}$ | $1.893 \times 10^{3}$ |

*Carbon monoxide concentration*

CO concentrations of 1442.6 µg/m$^3$ (1-h average) and 618.30 µg/m$^3$ (8-h average) (Table 5) were not exceeded in comparison with the NAAQS guideline of 29770 µg/m$^3$ for 1-h and 10305 µg/m$^3$ for 8-h exposure limit. Estimation of risk for acute exposure to CO revealed that the HQ is < 1.0 for infants, children, and adults (Table 9). This implies a negligible risk, even to sensitive infants, children, and adults. Though, infants, and children ($1.3 \times 10^{-3}$) may suffer the effects than adults ($8.0 \times 10^{-4}$). Additionally, infants, children and adults living in the study area are not likely to experience adverse health effects associated with normal and worst-case exposure scenarios to 8-h CO (HQ < 1.0).

Table 9: Hazard quotients for normal and worst-case exposure scenarios to CO at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $1.3 \times 10^{-3}$ | $2.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $1.3 \times 10^{-3}$ | $9.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $8.0 \times 10^{-4}$ | $3.0 \times 10^{-3}$ | $8.0 \times 10^{-4}$ |

**Intermediate – 8 Hour exposure period*

*Ozone concentration*

The monitored concentration of $O_3$ for 1-h and 8-h average in the study area are 29.78 µg/m$^3$ and 22.15 µg/m$^3$ respectively (Table 5). The NAAQS and annual guideline of 226 µg/m$^3$ and 120 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The HQ calculated for both the acute and intermediate (normal and worst-case) exposure scenarios shows no likelihood of

adverse health effects being experienced by any individuals (HQ < 1.0) (Table 10). During acute exposure, adults ($2.2 \times 10^{-2}$) are less likely to be affected than infants and children ($3.0 \times 10^{-3}$) while the reverse is the case for continuous exposure to $O_3$ for 8 hours.

Table 10: Hazard quotients for normal and worst-case exposure scenarios to $O_3$ at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $3.5 \times 10^{-3}$ | $5.0 \times 10^{-3}$ | $4.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $3.5 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $4.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $2.2 \times 10^{-2}$ | $9.0 \times 10^{-3}$ | $2.0 \times 10^{-2}$ |

**Intermediate – 8 Hour exposure period*

## DISCUSSION

Air pollution remains a global environmental threat and a public health risk. Researchers posited that health effects from exposure to ambient air pollution can occur at or below levels allowed by the national and international air quality standards. Findings from our study revealed that the 24-h $PM_{10}$ ambient quality standard of 75 $\mu g/m^3$ was not exceeded on any of the days during the monitoring period. This is in contrast with other studies conducted elsewhere in South Africa. A 24-h $PM_{10}$ of 157.37 $\mu g/m^3$ (highest peak) and 110 $\mu g/m^3$ was reported by [31] and [35] respectively. The average annual concentration of $PM_{10}$ recorded in our study was slightly above the guideline limit of 45 $\mu g/m^3$ set by the NAAQS. This may account for the chronic (annual) HQ > 1 recorded in our study, an indication of some level of risk to long-term exposure to $PM_{10}$. The low concentration of pollutants recorded in our study may be due to the fact that industries in South Africa are required to submit their emission inventory to regulatory agencies monthly. This may compel these industries to ensure that their emission into the atmosphere is within stipulated guideline limits.

In South Africa, it was estimated that outdoor air pollution was responsible for 3.7% of the national mortality attributable to cancers of the trachea, bronchus and lung in adults aged 30 years and older, and 1.1% of mortality in children under 5 years of age.[31] A review of 12 previous studies in the year 2001 affirmed that a 10-$\mu g/m^3$ increase in $PM_{10}$ causes an increase in hospital admissions for congestive heart failure and ischemic heart disease.[47] Among the vulnerable population (elderly and people with a previous medical history of respiratory and

cardiovascular diseases), long-term exposure to $PM_{10}$ has been linked with an increase in morbidity and mortality from respiratory and cardiovascular diseases.[48] Also for adults, large population studies have shown an association between respiratory (admissions for asthma, COPD, and pneumonia) hospitalization and ambient $PM_{10}$.[49] However, the effects seem to be stronger for elderly patients with even short-term exposures.[50]

This study further revealed that the 1-hour, 24-hours and annual mean concentration for $NO_2$ are below the national standard. Evidence from the risk characterisation assessment shows a negligible risk to acute and intermediate exposure to ambient levels of $NO_2$. However, 1-year exposure to ambient levels of $NO_2$ could pose some risks to the sensitive individual. Recent epidemiological studies have revealed that exposure to low levels of $NO_2$ could increase emergency room hospitalization for acute and obstructive lung diseases in the general population .[17, 51] Studies conducted in Canada, Denmark and Italy found a significant association between exposures to levels of $NO_2$ and acute ischemic stroke.[16, 52] However, some studies did not find significant associations between exposure to ambient and personal levels of $NO_2$ and health effects (Linaker *et al.,* 2000; Sarnat *et al.,* 2001).[53, 54]

Our study further shows low ambient value (compared to national standard) for $SO_2$ in Pretoria West. Similarly, there is no likelihood of health risk (HQ < 1) associated with 1-hour and 24-hours exposure to $SO_2$. Though, some levels of risk to sensitive individuals was found for chronic (annual) exposure to $SO_2$ in the study area. The possibility of $SO_2$ worsening childhood asthma at fairly modest concentration, that is well below the US EPA standards and WHO guidelines have been reported.[55] Multi-city studies conducted in Europe and Asia offer further proof supporting the short-term association of $SO_2$ with adverse health outcomes including both mortality[56] and morbidity.[57]

In this study, low ambient concentrations of CO and $O_3$ was recorded. Researchers are of the opinion that exposure to ambient levels of CO is often not recognized; its toxicity is mostly underreported and misdiagnosed due to its non-irritation and imperceptibility in the air we inhale.[18] Exposure to CO has been linked to poison correlated mortality in the United States.[18] On the other hand, $O_3$ is a strong oxidant that weakens biological tissues, thus resulting in increased use of medication, ailment, and death.[58] It has even been previously established that no level of exposure to $O_3$ is safe since health risk has been found to be associated with $O_3$ even at concentrations below recommended standards.[58]

Furthermore, evidence from the risk characterisation assessment in this study shows that adults are less likely to be affected by acute and intermediate exposure to ambient concentrations of CO and O3 than infants and children. This was also true for acute and intermediate exposures to $NO_2$ and $SO_2$. It has been documented that children have a higher susceptibility to environmental pollutants than adults. They are considered a risk group for numerous reasons including their

relative higher amount of air inhalation (the air intake per weight unit of a resting infant is twice that of an adult), their not fully developed immune system and lungs.[31]

Uncertainties and Limitations

Uncertainty is the inadequate knowledge that a person has about the value of a variable or the variability within an individual or a population.[59] It occurs basically because a risk assessment incorporates information of the pollutants released into the environment; the fate and transport of pollutants in changeable environments through poorly understood and often unquantifiable methods; the potential for adverse human health effects obtained through extrapolation from human and animal studies and the probability of adverse human health effects given the genetic and other causes of diversity within the human population.[59]

Although uncertainties occur in risk assessment, the risk assessment application has found usefulness in providing a quantitative and consistent framework for systematically evaluating environmental health risks and decisions for their control. Human health risk assessment as used in this study is usually conservative as it include many safety factors that are built into the process. The final risk estimate is therefore likely to overstate the actual risk. To address these uncertainties in our study, we adopted equations from the US-EPA, and applied benchmark values that were based on national and international standards and guidelines which were set based on the resulting human health effects from exposure to known pollutants.

## CONCLUSIONS

Ambient air pollution is composed of both suspended particulates and gaseous pollutants, with the gaseous components comprising $O_3$, CO, $NO_2$, and $SO_2$. The acute, intermediate and chronic ambient concentration of $PM_{10}$ and the gaseous pollutants recorded in Pretoria West were within the South African National Ambient Air Quality. No health risk was found to be associated with acute and intermediate exposure to the pollutants, though, infants and children than adults, are more likely to suffer the health effects. Long term chronic (annual) exposure to normal and worst-case exposure scenarios to each of the pollutants posed some levels of risks to sensitive individuals, with the severity of risk differing across groups. Identification of the possibility of these pollutants to pose health hazards, as measured through the human health risk assessment framework will make valuable contributions to government, environmental specialists and relevant stakeholders in taking more concrete steps to protect and prolong human lives. Additionally, these findings will assist policy makers in enforcing or strengthening existing legislation that limits the release of pollutants into the atmosphere or institutes risk management strategies.

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# Health Risk of Inhalation Exposure to Sub-10 µm Particulate Matter and Gaseous Pollutants in an Urban-industrial Area in South Africa: an ecological study

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# Health Risk of Inhalation Exposure to Sub-10 µm Particulate Matter and Gaseous Pollutants in an Urban-industrial Area in South Africa: an ecological study

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## ABSTRACT

**Objective:** To access the health risks associated with exposure to particulate matter ($PM_{10}$), sulphur dioxide ($SO_2$), nitrogen dioxide ($NO_2$), carbon monoxide (CO) and ozone ($O_3$).

**Design:** The study is an ecological study that utilised the year 2014 hourly ambient pollution data.

**Setting:** The study was conducted in an industrial area located in Pretoria West, South Africa. The area accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter.

**Data and method:** Estimate of possible health risks from exposure to airborne $PM_{10}$, $SO_2$, $NO_2$, CO, and $O_3$ was done using the United States Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework. A scenario-assessment approach where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for both intermediate (24-hour) and chronic (annual) exposure periods for different exposure groups (infants, children, adults). The normal acute (1-hour) exposure to these pollutants was also determined.

**Outcome measures:** Presence or absence of adverse health effects from exposure to airborne pollutants.

**Results:** Average annual ambient concentration of $PM_{10}$, $NO_2$ and $SO_2$ recorded were 48.3±43.4 µg/m$^3$, 11.50±11.6 µg/m$^3$ and 18.68±25.4 µg/m$^3$ respectively. Whereas, the South African National Ambient Air Quality recommended 40 µg/m$^3$, 40 µg/m$^3$ and 50 µg/m$^3$ for $PM_{10}$, $NO_2$ and $SO_2$ respectively. Exposure to an hour concentration of $NO_2$, $SO_2$, CO and $O_3$; 8-h concentration of CO, $O_3$; and 24-h concentration of $PM_{10}$, $NO_2$ and $SO_2$ will not likely produce adverse effects to sensitive exposed groups. Though, infants, and children, rather than adults, are

more likely to be affected. Moreover, for chronic annual exposure, $PM_{10}$, $NO_2$ and $SO_2$ posed a health risk to sensitive individuals, with the severity of risk varying across exposed groups.

**Conclusions:** Long-term chronic exposure to airborne $PM_{10}$, $NO_2$, and $SO_2$ pollutants may results in health risks among the study population.

**Keywords**: particulate matter, gaseous pollutants, health risk assessment, exposure groups, South Africa

## Strengths and Limitations of this study

- Large data set spanning hourly ambient concentration of pollutants for a whole year.
- This is the first study in Pretoria West, South Africa to estimate health risks of human exposure to airborne pollutants using US EPA assessment model.
- In our study, prediction of both long-term and short-term health effects in infants, children, and adults resulting from inhalation of pollutants was possible.
- However, the health risk that could result from exposure to the combination of the pollutants could not be determined.

## INTRODUCTION

Air pollution is a multifaceted mix consisting of both suspended particulates and gaseous pollutants.[1] Globally, air pollution continues to be a major environmental problem that has been recognised as an important public health risk.[2] The increase in human population, industrialisation, urbanisation, modernisation and its attendant increase in vehicular emissions and activities are the major contributors to the rising urban air quality problems.[3]

The World Health Organization (WHO) in the year 2013, asserted that annually, the urban ambient air pollution was predicted to cause 2 million deaths in the world.[4] Epidemiological studies have linked exposure to ambient air pollution with adverse human health effects.[5-7] Exposure to air pollution can result in both acute (short-term) and chronic (long-term) health effects.[8, 9] The acute effects of air pollution on human health were sufficiently established in the 20th century when severe air pollution scenarios in Europe and in the United States resulted in morbidities and mortalities in hundreds of thousands of people.[10]

Air pollution is a known trigger of Chronic Obstructive Pulmonary Disease (COPD)[11] and has informed the establishment of air quality standards in many countries [12, 13]. The broad legislative framework for air quality assessment in populated areas was put in place by the European Union Directive on Air Quality 2008/50/EC [14]. This framework recommended guideline limits for pollutants that have been identified to be injurious to the health of the public including the environment and the built infrastructure.[14] These injurious pollutants include particulate matter (PM) with a diameter of $\leq$ 10 μm ($PM_{10}$), nitrogen dioxide ($NO_2$), sulphur dioxide ($SO_2$) and carbon monoxide (CO).[15] The human health effects of exposure to $SO_2$, $NO_2$, $O_3$ and $PM_{10}$ have previously been reported.[7, 16-19] Ozone, $NO_2$ and $SO_2$ pollutants can all cause lethal effects on the airway[20] such as an increase in bronchial reactivity,[21, 22] airway oxidative stress,[23] pulmonary and systemic inflammation,[24] amplification of viral infections[25] and reduction in airway ciliary activity.[26]

South Africa has one of the largest industrialised economies in the Southern Hemisphere and is the only industrialised regional energy producer on the African continent with significant mining and metallurgical activities.[27] It is an arid country with high naturally-occurring dust levels, compounded by industrial and vehicular pollution emissions.[28] Excessive high particulate matter pollution levels have been observed in industrialised regions and urban areas which are said to contribute up to 30% of particulate pollution in the country.[29] Significant associations between exposure to particulate matter and respiratory, cardiovascular and cerebrovascular risks have been reported in South Africa.[30]

Therefore, increased emphasis on human health concerns from air pollution necessitates the need for estimating the association between exposure and adverse health effects. The United States

Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework is a handy tool that can be used to estimate human health risk that can result from exposure to a given pollutant.[31] In their studies,[32, 33] reported that health risk assessment is useful for estimating the occurrence of adverse health effects in children and adults resulting from the direct inhalation of atmospheric particulates in urban areas. This framework was first introduced by the National Research Council in 1994[34] and has been previously used in few studies in South Africa.[31, 35-37] However, an HHRA framework on $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ has never been previously used in Pretoria West, South Africa. Hence, in view of the known health effects of exposure to sub-10µm PM and other gaseous pollutants, this study aimed to quantify the health risk of people living in the urban area in Pretoria West using the HHRA framework.

## METHODS

### Study area and population

The study area was Pretoria West is situated at 25°44'46"S 28°11'17"E (Figure 1). Pretoria West is an industrial production area that accommodates a coal-fired power station, metallurgical industries such as a coke plant and a manganese smelter, fuel stations and a fuel tank farm. Pretoria is a city in the Northern part of Gauteng Province in Tshwane Metropolitan Municipality. It is situated approximately 55 km (34 mi) north-northeast of Johannesburg in the Northeast of South Africa, in a transitional belt between the plateau of the Highveld to the South and the lower-lying Bushveld to the North. Pretoria has a population of 741, 651 (49.75% males and 50.25% females) in 2011. This constitute 23.2% young (0-14 years) persons, 71.9% of working age (15-64 years) and 4.9% of elderly (65+ years) persons.[38]

### Data collection procedure

The study was an ecological study that focused on the comparison of groups, rather than individuals. It makes biologic inferences about effects on individual risks or groups to make ecologic inferences about effects on group rate. The study utilised secondary data obtained from the South African Weather Service (SAWS) through the South African Air Quality Information System (SAAQIS) website (www.saaqis.org.za) after the approval for its use was granted by the data originators, Environmental Management Services Department. The SAAQIS makes data available to stakeholders including the public and provides a mechanism to ensure uniformity in the way air quality data is managed i.e. captured, stored, validated, analysed and reported in South Africa.

The data originators obtained the data from a fixed ambient air quality monitoring station (Syntech Spectras GC955 series 600) located at Pretoria West at longitude 28.146108, latitude -25.7555 and 1329 m above sea level. To ensure quality control of the data, the South African Weather Service conducts a quarterly quality control (calibration verification) of the monitoring

station at three intermediate point checks using known concentration of certified reference gases. Moreover, the South African National Accreditation System conducts an annual calibration of the monitoring station. This is addition to the weekly routine visits to the monitoring station to ensure its proper functioning and maintenance. Data requested by the researchers from the originators include hourly daily ambient level concentrations of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ for the year 2014.

Data analysis

SPSS version 20 was used for the statistical analyses of the data. Descriptive statistics such as mean and standard deviation was used to estimate the average concentration of pollutants that were monitored.

Human Health Risk Assessment

Health risk assessment is an inclusive procedure by which possible adverse effects of human exposure to toxic agents are characterised.[39] HHRA is predictive in nature and uses existing exposure data to measure health effects of exposure to a particular pollutant.[40] The HHRA framework used in this study has four components: hazard identification, dose-response assessment, exposure assessment and risk characterisation.

*Hazard identification*

The identification of $PM_{10}$, CO, $NO_2$, $SO_2$ and $O_3$ as harmful and their attendant health risks was done through a review of existing literature.

*Dose-response assessment*

Here, the amount of the pollutant taken into the body was estimated as a function of concentration and the length of exposure [41] The dose-response assessment was not done in this study since it requires a full health screening and additional data from health records. Rather, we compared the measured ambient concentration of pollutants in the study area with the South African National ambient air quality standard which serves as the benchmark.

*Exposure assessment*

The exposure assessment identifies the population exposed to the hazard, the magnitude, and duration of exposure to the hazard. Our study assumed inhalation as the route of exposure to the monitored pollutants. As previously reported,[35] this study utilised a scenario assessment method where normal (average exposure) and worst-case (continuous exposure) scenarios were computed for both intermediate (24-h) and chronic (annual) exposure periods for the different exposure groups. The normal acute (1-h) exposure periods was also determined.

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the acute exposure rate equation is given as:

$$AHD = C \times IR/BW \qquad \text{(Equation 1)[41]}$$

Where *AHD* is the average hourly dose for inhalation ($\mu g\ kg^{-1}h^{-1}$), *C* the concentration of the chemical ($\mu g\ m^{-3}$), *IR* the inhalation rate ($m^3\ h^{-1}$) and *BW* the body weight (kg).

For exposure to non-carcinogenic pollutants ($PM_{10}$, CO, $NO_2$, $SO_2$, $O_3$), the chronic exposure equation used for the inhalation exposure route is:

$$ADD = (C \times IR \times ED) / (BW \times AT) \qquad \text{(Equation 2)[42]}$$

Where ADD = average daily dose of the chemical of interest ($\mu gkg^{-1}day^{-1}$), C = concentration of the chemical in the atmosphere ($\mu gm^{-3}$), IR = inhalation rate ($m^3day^1$), ED = exposure duration (days), BW = average body weight of receptor over the exposure period (kg), AT = averaging time (days).

The exposure duration (ED) which is the length of time study population are exposed to a pollutant is expressed as:

$$ED = ET \times EF \times DE \qquad \text{(Equation 3)[35]}$$

Where ET = exposure time or event (hour $day^{-1}$), EF = exposure frequency (days $year^{-1}$), DE = duration of exposure (year)

The default values for EF, DE, and AT for each exposed groups are presented in table 1. The EF default value used was founded on the assumption that each population group will spend at least two weeks every year away from the study area.[43] The DE for an adult is estimated at 30 years while that of a child and an infant were 1 and 12 years respectively. The AT is estimated as the product of the duration of exposure by 365 days $year^{-1}$.

Table 1: Exposure frequency, exposure duration and averaging time for different exposure groups

| Exposed group | EF (days $yr^{-1}$) | DE (year) | AT (days) |
|---|---|---|---|

| | | | |
|---|---|---|---|
| Infant (Birth to 1 year) | 350 | 1 | 365 (1 x 365) |
| Child (6 to 12 years) | 350 | 12 | 4380 (12 x 365) |
| Adult (19 to 75 years) | 350 | 30 | 10950 (30 x 365) |

*EF = exposure frequency; DE = duration of exposure; AT = averaging time*
Source: Adapted from [35]

The ET for each population group is based on the normal and worst case scenarios for acute, intermediate, and chronic exposure periods (see table 2). Thus, the intermediate ET for adults was estimated at 3hour $day^{-1}$, based on the notion that the remainder of their time is spent either at work, away from Pretoria West, or indoors. ET for children was assumed to be greater since they have more time to play outdoors at the end of the school days; infants were assumed to spend the majority of the day indoors. Default values were used for IR and BW[43] and are given in table 3 for each exposure groups.

Table 2: Exposure time (h) for normal and worst case scenarios for acute, intermediate and chronic exposures

| | Exposure time (h) | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | 1 | 1 | 24 | 14.6 [(350/24) x 1] | 350 (1 x 350) |
| Child (6 to 12 years) | 1 | 6 | 24 | 1050.0 [(4200/24) x 6] | 4200 (12 x 350) |
| Adult (19 to 75 years) | 1 | 3 | 24 | 1312.5 [(10500/24) x 3] | 10500 (30 x 350) |

Source: Adapted from [35]

Table 3: Average Inhalation rates and Body weights of exposed population

| Exposed group | Mean inhalation rate ($m^3hr^{-1}$) | | Mean body weight (kg) |
|---|---|---|---|
| | Acute exposure | Chronic exposure | |
| Infant (Birth to 1 year) | 0.3 | 6.8 | 11.3 |
| Child (6 to 12 years) | 1.2 | 13.5 | 45.3 |
| Adult (19 to 75 years) | 1.2 | 13.3 | 71.8 |

Source: Adapted from [35]

*Risk characterisation*

Risk characterisation is the quantitative estimation of the health risk of exposure to a pollutant. Here, the non-carcinogenic health effects were expressed as a dimensionless ratio called a hazard quotient (HQ), which indicates the presence or absence of adverse health effects due to exposure.[36, 43] HQ also provides an indication of whether only sensitive individuals will be affected, or if both healthy and sensitive individuals will be affected. Non-cancer risks were calculated for both acute and chronic exposure scenarios as:

HQ = ADD/REL (Chronic exposure) or (Equation 4)

HQ = AHD/REL (Acute exposure) (Equation 5)

Where *REL* is the dose at which significant adverse health effects will occur in exposed subjects, compared to an unexposed group. In this study, we used the term "reference exposure level" (REL), as adopted by the Office of the Environmental Health Hazard Assessment (OEHHA).[44] The RELs that is used is presented in table 4.

An HQ of 1.0 is considered to be the benchmark of safety. An HQ that is < 1.0 indicates a negligible risk i.e. the pollutant under scrutiny is not likely to induce adverse health effects, even to a sensitive individual. An HQ > 1.0 indicates that there may be some risks to sensitive individuals as a result of exposure.[45]

Table 4: Reference Exposure Levels for different pollutants

| Pollutant | 1 hour ($\mu g/m^3$) | 8 Hours ($\mu g/m^3$) | 24 hours ($\mu g/m^3$) | Annual mean ($\mu g/m^3$) |
|---|---|---|---|---|
| $PM_{10}$ | - | | *75 | *40 |
| $NO_2$ | *200 | | **188 | *40 |
| $SO_2$ | *350 | | *125 | *50 |
| CO | ***29770 | ***10305 | - | - |
| $O_3$ | **226 | *120 | - | - |

**NAAQS (National ambient air quality standard for South Africa); ** South Africa standards – Air quality act (Act 39 of 2004); **Default value was converted from ppm to $\mu g/m^3$*
Source: Department of Environmental Affairs[46].

## RESULTS

*Particulate matter ($PM_{10}$) concentration*

The mean hourly, daily and annual concentration of $PM_{10}$ in the Pretoria West are 67.74 µg/m$^3$, 52.01 µg/m$^3$ and 48.26 µg/m$^3$ respectively (Table 5). Though, the daily (24 hours) guideline limit of 75 µg/m$^3$ set by the NAAQS was not exceeded, the annual recommended mean limit of 45 µg/m$^3$ that should not be exceeded was surpassed. The 1-h (acute) scenario was not considered as a 1-h REL value for $PM_{10}$ was not found in the literature. The hazard quotient (HQ) from the health risk characterisation from exposure to $PM_{10}$ is provided in Table 6. The results showed that under the normal and worst-case scenario for average and continuous exposures respectively, the risk of having health related problems by the exposed population is low (HQ < 1). This is because HQ of < 1.0 indicates that $PM_{10}$ is not likely to induce adverse health outcomes. However, infants ($2.0 \times 10^{-2}$ vs $4.2 \times 10^{-1}$) followed by children ($1.1 \times 10^{-1}$ vs $4.2 \times 10^{-1}$) are likely to be affected from exposure to $PM_{10}$ than adults ($3.0 \times 10^{-2}$ vs $2.7 \times 10^{-1}$) under the normal and worst-case scenario respectively for intermediate exposure. For the chronic (annual) exposure scenario for normal and worst-case exposures, the HQ is > 1.0 for infants, children, and adults. These results show that sensitive exposed population may be at a risk of developing health related problems from chronic exposure to $PM_{10}$. Infants are more likely to be affected than children and adults under the normal chronic exposure while children will be more affected than infants and adults under the worst-case scenario.

Table 5: Summary statistics of ambient concentrations of pollutants

| Averaging period | $PM_{10}$ (µg/m$^3$) Mean ±SD | $NO_2$ (µg/m$^3$) Mean ±SD | $SO_2$ (µg/m$^3$) Mean ±SD | CO (µg/m$^3$) Mean ±SD | $O_3$ (µg/m$^3$) Mean ±SD |
|---|---|---|---|---|---|
| 1 h | 67.74 ± 61.63 | 17.44 ± 17.26 | 29.63 ± 33.64 | 1442.6 ± 1248.05 | 29.78 ± 8.69 |
| 8h | - | - | - | 618.30 ± 618.30 | 22.15 ± 7.96 |
| 24h | 52.01 ± 50.58 | 13.13 ± 13.21 | 21.48 ± 27.71 | - | - |
| Annual | 48.26 ± 43.41 | 11.50 ± 11.61 | 18.68 ± 25.36 | - | - |

*SD – Standard deviation*

Table 6: Hazard quotients for normal and worst-case exposure scenarios to $PM_{10}$

| Exposed group | Exposure | | | |
|---|---|---|---|---|
| | Intermediate | | Chronic | |
| | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-2}$ | $4.2 \times 10^{-1}$ | $1.0 \times 10^{1}$ | $2.44 \times 10^{2}$ |
| Child (6 to 12 years) | $1.1 \times 10^{-1}$ | $4.2 \times 10^{-1}$ | $3.62 \times 10^{2}$ | $1.45 \times 10^{3}$ |
| Adult (19 to 75 years) | $3.0 \times 10^{-2}$ | $2.7 \times 10^{-1}$ | $2.81 \times 10^{2}$ | $2.25 \times 10^{3}$ |

*The 1-h (acute) scenario was not considered since a 1-h REL value for $PM_{10}$ was not found in literature*

*Sulphur dioxide concentration*

The measured average concentration of $SO_2$ for 1-h, 24-h and annual averages in the study area were 29.63 µg/m$^3$, 21.48 µg/m$^3$ and 18.68 µg/m$^3$ respectively (Table 5). These values are far less than the mean values of 350 µg/m$^3$, 125 µg/m$^3$ and 50 µg/m$^3$ as provided by NAAQS for 1-h, 24-h and annual averages respectively that should not be exceeded (Table 4). Estimation of risk for acute and intermediate (normal and worst-case) exposures to $SO_2$ revealed that the HQ is < 1.0 for infants, children, and adults (Table 7). This implies a negligible risk, even to a sensitive individual. For acute exposure, infants and children ($2.0 \times 10^{-3}$) are likely to be affected the same way from exposure to $SO_2$ compared to adults ($1.4 \times 10^{-3}$). Under the normal and worst-case scenarios for chronic exposure, the HQ was > 1.0 for all study population. This indicates that there may be some risks to sensitive individuals as a result of exposure to $SO_2$. The severity of exposures differs for different age groups.

Table 7: Hazard quotients for normal and worst-case exposure scenarios to $SO_2$ at different levels of exposures

| Exposed group | Exposure | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.0 \times 10^{-3}$ | $4.0 \times 10^{-3}$ | $1.1 \times 10^{-1}$ | $31.5 \times 10^{-1}$ | $7.55 \times 10^{1}$ |
| Child (6 to 12 years) | $2.0 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $1.0 \times 10^{-1}$ | $1.12 \times 10^{2}$ | $4.49 \times 10^{2}$ |
| Adult (19 to 75 years) | $1.4 \times 10^{-3}$ | $8.0 \times 10^{-3}$ | $7 \times 10^{-2}$ | $8.72 \times 10^{1}$ | $6.98 \times 10^{2}$ |

*Nitrogen dioxide concentration*

The monitored 1-h, 24-h and annual concentrations of $NO_2$ shown in Table 5 were 17.44 µg/m$^3$, 13.13 µg/m$^3$ and 11.50 µg/m$^3$. The NAAQS 1-h, 24-h and annual guideline of 200 µg/m$^3$, 188 µg/m$^3$ and 40 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The HQ calculated for each of the acute and intermediate (normal and worst-case scenarios) exposures (shown in Table 8) showed no likelihood of adverse health effects occurring at this level of exposure for an infant, child and adult (HQ < 1.0). However, there is the likelihood that infants and children ($2.3 \times 10^{-3}$) might be affected by acute exposure to $NO_2$ than adults ($1.5 \times 10^{-3}$). Moreover, having an adverse health outcome from normal and worst-case chronic exposure to $NO_2$ was found to be higher (HQ > 1.0) for all age groups. Children ($3.05 \times 10^{2}$) appears more likely to be affected by normal chronic exposure than infants ($8.6 \times 10^{1}$) and adults ($2.37 \times 10^{2}$) whereas for worst-case chronic exposure, adults ($1.893 \times 10^{3}$) are more likely to be affected.

Table 8: Hazard quotients for normal and worst-case exposure scenarios to $NO_2$ at different levels of exposures

| | Exposure | | | | |
|---|---|---|---|---|---|
| | | Intermediate | | Chronic | |
| Exposed group | Acute | Normal | Worst case | Normal | Worst case |
| Infant (Birth to 1 year) | $2.3 \times 10^{-3}$ | $6.0 \times 10^{-3}$ | $1.5 \times 10^{-1}$ | $8.6 \times 10^{1}$ | $2.05 \times 10^{2}$ |
| Child (6 to 12 years) | $2.3 \times 10^{-3}$ | $4 \times 10^{-2}$ | $1.5 \times 10^{-1}$ | $3.05 \times 10^{2}$ | $1.218 \times 10^{3}$ |
| Adult (19 to 75 years) | $1.5 \times 10^{-3}$ | $1.0 \times 10^{-2}$ | $9.0 \times 10^{-2}$ | $2.37 \times 10^{2}$ | $1.893 \times 10^{3}$ |

*Carbon monoxide concentration*

CO concentrations of 1442.6 µg/m$^3$ (1-h average) and 618.30 µg/m$^3$ (8-h average) (Table 5) were not exceeded in comparison with the NAAQS guideline of 29770 µg/m$^3$ for 1-h and 10305 µg/m$^3$ for 8-h exposure limit. Estimation of risk for acute exposure to CO revealed that the HQ is < 1.0 for infants, children, and adults (Table 9). This implies a negligible risk, even to sensitive infants, children, and adults. Though, infants, and children ($1.3 \times 10^{-3}$) may suffer the effects than adults ($8.0 \times 10^{-4}$). Additionally, infants, children and adults living in the study area are not likely to experience adverse health effects associated with normal and worst-case exposure scenarios to 8-h CO (HQ < 1.0).

Table 9: Hazard quotients for normal and worst-case exposure scenarios to CO at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $1.3 \times 10^{-3}$ | $2.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $1.3 \times 10^{-3}$ | $9.0 \times 10^{-3}$ | $1.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $8.0 \times 10^{-4}$ | $3.0 \times 10^{-3}$ | $8.0 \times 10^{-4}$ |

**Intermediate – 8 h r exposure period*

*Ozone concentration*

The monitored concentration of $O_3$ for 1-h and 8-h average in the study area are 29.78 µg/m$^3$ and 22.15 µg/m$^3$ respectively (Table 5). The NAAQS and annual guideline of 226 µg/m$^3$ and 120 µg/m$^3$ respectively were not exceeded at Pretoria West (Table 4). The HQ calculated for both the acute and intermediate (normal and worst-case) exposure scenarios shows no likelihood of adverse health effects being experienced by any individuals (HQ < 1.0) (Table 10). During acute exposure, adults ($2.2 \times 10^{-2}$) are less likely to be affected than infants and children ($3.0 \times 10^{-3}$) while the reverse is the case for continuous exposure to $O_3$ for 8 hours.

Table 10: Hazard quotients for normal and worst-case exposure scenarios to $O_3$ at different levels of exposures

| Exposed group | Exposure | | |
|---|---|---|---|
| | Acute | *Intermediate | |
| | | Normal | Worst |
| Infant (Birth to 1 year) | $3.5 \times 10^{-3}$ | $5.0 \times 10^{-3}$ | $4.0 \times 10^{-2}$ |
| Child (6 to 12 years) | $3.5 \times 10^{-3}$ | $3.0 \times 10^{-2}$ | $4.0 \times 10^{-2}$ |
| Adult (19 to 75 years) | $2.2 \times 10^{-2}$ | $9.0 \times 10^{-3}$ | $2.0 \times 10^{-2}$ |

**Intermediate – 8 h exposure period*

## DISCUSSION

Air pollution remains a global environmental threat and a public health risk. Researchers posited that health effects from exposure to ambient air pollution can occur at or below levels allowed by the national and international air quality standards. Findings from our study revealed that the 24-h $PM_{10}$ ambient quality standard of 75 µg/m$^3$ was not exceeded on any of the days during the monitoring period. This is in contrast with other studies conducted elsewhere in South Africa. A 24-h $PM_{10}$ of 157.37 µg/m$^3$ (highest peak) and 110 µg/m$^3$ was reported by [31] and [35] respectively. The average annual concentration of $PM_{10}$ recorded in our study was slightly above

the guideline limit of 45 µg/m$^3$ set by the NAAQS. This may account for the chronic (annual) HQ > 1 recorded in our study, an indication of some level of risk to long-term exposure to $PM_{10}$. The low concentration of pollutants recorded in our study may be due to the fact that industries in South Africa are required to submit their emission inventory to regulatory agencies monthly. This may compel these industries to ensure that their emission into the atmosphere is within stipulated guideline limits.

In South Africa, it was estimated that outdoor air pollution was responsible for 3.7% of the national mortality attributable to cancers of the trachea, bronchus and lung in adults aged 30 years and older, and 1.1% of mortality in children under 5 years of age.[31] A review of 12 previous studies in the year 2001 affirmed that a 10-µg/m$^3$ increase in $PM_{10}$ causes an increase in hospital admissions for congestive heart failure and ischemic heart disease.[47] Among the vulnerable population (elderly and people with a previous medical history of respiratory and cardiovascular diseases), long-term exposure to $PM_{10}$ has been linked with an increase in morbidity and mortality from respiratory and cardiovascular diseases.[48] Also for adults, large population studies have shown an association between respiratory (admissions for asthma, COPD, and pneumonia) hospitalization and ambient $PM_{10}$.[49] However, the effects seem to be stronger for elderly patients with even short-term exposures.[50]

This study further revealed that the 1-h, 24-h and annual mean concentration for $NO_2$ are below the national standard. Evidence from the risk characterisation assessment shows a negligible risk to acute and intermediate exposure to ambient levels of $NO_2$. However, 1-year exposure to ambient levels of $NO_2$ could pose some risks to the sensitive individual. Recent epidemiological studies have revealed that exposure to low levels of $NO_2$ could increase emergency room hospitalization for acute and obstructive lung diseases in the general population .[17, 51] Studies conducted in Canada, Denmark and Italy found a significant association between exposures to levels of $NO_2$ and acute ischemic stroke.[16, 52] However, some studies did not find significant associations between exposure to ambient and personal levels of $NO_2$ and health effects (Linaker *et al.,* 2000; Sarnat *et al.,* 2001).[53, 54]

Our study further shows low ambient value (compared to national standard) for $SO_2$ in Pretoria West. Similarly, there is no likelihood of health risk (HQ < 1) associated with 1-h and 24-h exposure to $SO_2$. Though, some levels of risk to sensitive individuals was found for chronic (annual) exposure to $SO_2$ in the study area. The possibility of $SO_2$ worsening childhood asthma at fairly modest concentration, that is well below the US EPA standards and WHO guidelines have been reported.[55] Multi-city studies conducted in Europe and Asia offer further proof supporting the short-term association of $SO_2$ with adverse health outcomes including both mortality[56] and morbidity.[57]

In this study, low ambient concentrations of CO and $O_3$ was recorded. Researchers are of the opinion that exposure to ambient levels of CO is often not recognized; its toxicity is mostly underreported and misdiagnosed due to its non-irritation and imperceptibility in the air we inhale.[18] Exposure to CO has been linked to poison correlated mortality in the United States.[18] On the other hand, $O_3$ is a strong oxidant that weakens biological tissues, thus resulting in increased use of medication, ailment, and death.[58] It has even been previously established that no level of exposure to $O_3$ is safe since health risk has been found to be associated with $O_3$ even at concentrations below recommended standards.[58]

Furthermore, evidence from the risk characterisation assessment in this study shows that adults are less likely to be affected by acute and intermediate exposure to ambient concentrations of CO and $O_3$ than infants and children. This was also true for acute and intermediate exposures to $NO_2$ and $SO_2$. It has been documented that children have a higher susceptibility to environmental pollutants than adults. They are considered a risk group for numerous reasons including their relative higher amount of air inhalation (the air intake per weight unit of a resting infant is twice that of an adult), their not fully developed immune system and lungs.[31]Uncertainties and Limitations

Although uncertainties occur in risk assessment, the risk assessment application has found usefulness in providing a quantitative and consistent framework for systematically evaluating environmental health risks and decisions for their control. Human health risk assessment as used in our study is conservative as it include many safety factors that are built into the process. The final risk estimate is therefore likely to overstate the actual risk. To address these uncertainties in our study, we adopted equations from the US-EPA, and applied benchmark values that were based on national and international standards and guidelines which were set based on the resulting human health effects from exposure to known pollutants.

Findings in our study should be interpreted in the light of the following limitations. The ecological nature of this study used population or groups of people as the unit of analysis rather than individuals. Ecologic technique assumes that individuals in the study area are all exposed to the same concentration of air pollutants without recourse to individual risk factors that may trigger the occurrence of disease outcomes. Such risk factors include socio-demographic factors, genetics, smoking habits and occupational exposure to respiratory hazards and pollutants in the workplace. Also, the health risk that could possibly result from exposure to the combination of the pollutants rather than individual pollutants as measured in our study could not be determined.

The strengths of this study are worthy of mention. First, the uniqueness of this study being the first that was conducted in Pretoria industrial area in South Africa that described the health risk associated with human exposure to particulate matter and other gaseous pollutants. The study leverages on the use of an hourly ambient pollution data, a data whose method of collection has

been through a validated process, and its outcome generalizable. Also, the use of the US EPA human health risk assessment framework which was first adopted by the National Research Council in 1994 allows our findings to be comparable to other studies.

## CONCLUSIONS

Ambient air pollution is composed of both suspended particulates and gaseous pollutants, with the gaseous components comprising $O_3$, CO, $NO_2$, and $SO_2$. The acute, intermediate and chronic ambient concentration of $PM_{10}$ and the gaseous pollutants recorded in Pretoria West were within the South African National Ambient Air Quality. No health risk was found to be associated with acute and intermediate exposure to the pollutants, though, infants and children than adults, are more likely to suffer the health effects. Long term chronic (annual) exposure to normal and worst-case exposure scenarios to each of the pollutants posed some levels of risks to sensitive individuals, with the severity of risk differing across groups. Identification of the possibility of these pollutants to pose health hazards, as measured through the human health risk assessment framework will make valuable contributions to government, environmental specialists and relevant stakeholders in taking more concrete steps to protect and prolong human lives. Additionally, these findings will assist policy makers in enforcing or strengthening existing legislation that limits the release of pollutants into the atmosphere or institutes risk management strategies.

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**Contributors** OMM, MIM and MSM conceptualised the study. OMM designed the study, acquired the data and wrote the initial manuscript. ASA analysed the data and revised the manuscript. MIM and MSM critically reviewed the manuscript for important intellectual content. All authors proofread and approved the final version of the manuscript.

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**Competing interests** None declared.

**Ethics approval** Human subject was not used in this study. However, the study was approved by the Tshwane University of Technology Senate Committee for Research Ethics, with reference number FCRE 2015/11/006 (SCI).

**Provenance and peer review** Not commissioned; externally peer reviewed.

**Data sharing statement** No additional data are available.



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Figure Legend

Figure 1: Map of Pretoria West industrial area. The area is located in the Tshwane Metro and boasts of a coal-fired power station, metallurgical industries and a fuel tank farm.

STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

| | Item No | Recommendation | |
|---|---|---|---|
| **Title and abstract** | 1 | (*a*) Indicate the study's design with a commonly used term in the title or the abstract | The study design is indicated on page 1 |
| | | (*b*) Provide in the abstract an informative and balanced summary of what was done and what was found | This was appropriately done. See abstract section on page 1 and 2. |
| **Introduction** | | | |
| Background/rationale | 2 | Explain the scientific background and rationale for the investigation being reported | The scientific background for the study was discussed on page 3 and 4. The rationale for the investigation was included on page 4. |
| Objectives | 3 | State specific objectives, including any prespecified hypotheses | The objective of our study is included on page 4. |
| **Methods** | | | |
| Study design | 4 | Present key elements of study design early in the paper | This has been captured under data collection procedure section on page 4 |
| Setting | 5 | Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection | The study area and location of study was discussed under "study area and population" in page 4.<br><br>Relevant days and data collection procedure were discussed on page 4. |
| Participants | 6 | (*a*) Give the eligibility criteria, and the sources and methods of selection of participants | Human subjects was not used in our study. |
| Variables | 7 | Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable | The study outcomes were discussed under the heading "Risk characterisation" on page 8.<br><br>Exposure outcome was discussed under the heading "exposure assessment" on page 5 and on the whole of pages 6 and 7. |
| Data sources/ measurement | 8* | For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group | This was sufficiently discussed on page 5 to 8 under the headings "Risk characterisation" and "exposure assessment." |
| Bias | 9 | Describe any efforts to address potential sources of bias | This was discussed under data collection procedure on page 4 and page 5. |
| Study size | 10 | Explain how the study size was arrived at | Not applicable |
| Quantitative variables | 11 | Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why | This was captured under exposure assessment on page 5 to 7. |
| Statistical methods | 12 | (*a*) Describe all statistical methods, including those used to control for confounding | This was sufficiently discussed as equations 1, 2 and 3 on page 6, and equations 4 and 5 on page 8. |
| | | (*b*) Describe any methods used to examine subgroups and interactions | This was discussed on page 5 to 8 under the headings "Risk characterisation" and "exposure assessment." |
| | | (*c*) Explain how missing data were addressed | Not applicable |
| | | (*d*) If applicable, describe analytical methods taking account of sampling strategy | Not applicable |

| | | | |
|---|---|---|---|
| | | (*e*) Describe any sensitivity analyses | Not applicable |
| **Results** | | | |
| Participants | 13* | (a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed | Not applicable |
| | | (b) Give reasons for non-participation at each stage | Not applicable |
| | | (c) Consider use of a flow diagram | Not applicable |
| Descriptive data | 14* | (a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders | Not applicable |
| | | (b) Indicate number of participants with missing data for each variable of interest | Not applicable |
| Outcome data | 15* | Report numbers of outcome events or summary measures | Not applicable |
| Main results | 16 | (*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included | Not applicable |
| | | (*b*) Report category boundaries when continuous variables were categorized | Not applicable |
| | | (*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period | Not applicable |
| Other analyses | 17 | Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses | This was sufficiently discussed on page 5 to 8. |
| **Discussion** | | | |
| Key results | 18 | Summarise key results with reference to study objectives | This was discussed under the discussion section on page 12 to 14. |
| Limitations | 19 | Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias | This was discussed under the uncertainties and limitations' section on page 14. |
| Interpretation | 20 | Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence | This was discussed under the discussion section on page 12 to 14. |
| Generalisability | 21 | Discuss the generalisability (external validity) of the study results | This was discussed under the uncertain and limitation section on page 14 and page 15. |
| **Other information** | | | |
| Funding | 22 | Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based | This was discussed on page 15. |

*Give information separately for exposed and unexposed groups.

**Note:** An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.