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 assess the health risks associated with exposure to particulate matter (PM10), sulphur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO) and ozone (O3).

Design: The study is an ecological study that used 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 PM10, SO2, NO2, CO and O3 was performed using the US Environmental Protection Agency human health risk assessment framework. A scenario-assessment approach where normal (average exposure) and worst-case (continuous exposure) scenarios were developed for 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 PM10, NO2 and SO2 recorded was 48.3±43.4, 11.50±11.6 and 18.68±25.4 µg/m3, respectively, whereas the South African National Ambient Air Quality recommended 40, 40 and 50 µg/m3 for PM10, NO2 and SO2, respectively. Exposure to an hour’s concentration of NO2, SO2, CO and O3, an 8-hour concentration of NO2 and SO2, and a 24-hour concentration of PM10, NO2 and SO2 will not likely produce adverse effects to sensitive exposed groups. However, infants and children, rather than adults, are more likely to be affected. Moreover, for chronic annual exposure, PM10, NO2 and SO2 posed a health risk to sensitive individuals, with the severity of risk varying across exposed groups.

Conclusions: Long-term chronic exposure to airborne PM10, NO2 and SO2 pollutants may result in health risks among the study population.

INTRODUCTION

Air pollution is a multifaceted mix consisting of 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 and modernisation, and its attendant increase in vehicular emissions and activities are the major contributors to the rising urban air quality problems.3 WHO in the year 2013 asserted that urban ambient air pollution resulted in 2 million deaths in the world.4 Epidemiological studies have linked exposure to ambient air...
pollution with adverse human health effects. Exposure to air pollution can result in acute (short-term) and chronic (long-term) health effects. The acute effects of air pollution on human health were sufficiently established in the twentieth century when severe air pollution scenarios in Europe and in the USA resulted in disease morbidities and mortalities in hundreds of thousands of people.

Air pollution is a known trigger of chronic obstructive pulmonary disease (COPD) and has informed the establishment of air quality standards in many countries. 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. 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. These injurious pollutants include particulate matter (PM) with a diameter of ≤10 µm (PM10), nitrogen dioxide (NO₂), sulphur dioxide (SO₂) and carbon monoxide (CO). The human health effects of exposure to SO₂, NO₂, O₃ and PM10 have previously been reported. Ozone, NO₂ and SO₂ pollutants can all cause lethal effects on the airway such as an increase in bronchial reactivity, airway oxidative stress, pulmonary and systemic inflammation, amplification of viral infections and reduction in airway ciliary activity.

South Africa is 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. It is an arid country with high naturally occurring dust levels, compounded by industrial and vehicular pollution emissions. Excessive high PM 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. Significant associations between exposure to PM and respiratory, cardiovascular and cerebrovascular risks have been reported in South Africa.

Increased emphasis on human health concerns resulting from air pollution necessitates the need for estimating the association between exposure and adverse health effects. The US Environmental Protection Agency (US EPA) human health risk assessment (HHRA) framework is a handy tool that has been used to estimate human health risk that can result from exposure to a given pollutant. In their studies, they 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 and has been previously used in few studies in South Africa. However, an HHRA framework on PM₁₀, CO, NO₂, SO₂ and O₃ has never been previously used in Pretoria West, South Africa. Hence, in view of the known health effects of exposure to sub-10 µm 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 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 ~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% men and 50.25% women) in 2011. This constitutes 23.2% young (0–14 years) persons, 71.9% of working age (15–64 years) and 4.9% of elderly (65+ years) persons.

Data collection procedure
The study was an ecological study that focused on the comparison of groups, rather than individuals. Ecological study makes biological inferences about the effects of exposure on individual risks or groups. This study used secondary data obtained from the South African Weather Service (SAWS) through the South African Air Quality Information System (SAAQIS) website (http://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 are managed, that is, captured, stored, validated, analysed and reported in South Africa.

The data originators obtained the data from a fixed ambient air quality monitoring station located at Pretoria West at longitude 28.146108, latitude −25.7555 and 1329 m above sea level. The monitoring station is routinely managed and maintained in order to achieve optimal results. The calibration of the monitoring unit is handled annually by the South African National Accreditation System. Moreover, at every quarter, the SAWS carries out a calibration certification of the monitoring unit using appropriate reference gases. Data requested by the researchers from the originators include hourly daily ambient level concentrations of PM₁₀, CO, NO₂, SO₂ and O₃ for the year 2014.

Data analysis
SPSS V20 was used for the statistical analyses of the data. Descriptive statistics such as mean and SD were 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.\(^3^9\) HHRA is predictive in nature and uses existing exposure data to measure the health effects of human exposure to a particular pollutant.\(^4^0\) 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 performed 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.\(^4^1\) The dose–response assessment was not performed in this study. Rather, we compared the measured ambient concentration of pollutants in the study area with the South African National Ambient Air Quality Standard (NAAQS) 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 the inhalation route as the major route of exposure to the monitored pollutants. As previously used in South Durban, South Africa,\(^3^5\) this study used a scenario assessment method. Normal (average exposure) and worst-case (continuous exposure) scenarios were computed for intermediate (24-hour) and chronic (annual) exposure periods. The normal acute (1-hour) exposure periods were also determined. These were determined among different age groups, namely infants (birth to a year), children (6–12 years) and adults (19–75 years).

For exposure to non-carcinogenic pollutants (PM\(_{10}\), CO, NO\(_2\), SO\(_2\), O\(_3\)), the acute exposure rate equation is given as:

\[
AHD = \frac{C}{IR/BW} \quad (1)
\]

where AHD is the average hourly dose for inhalation (µg/kg/hour), C is the concentration of the chemical (µg/m\(^3\)), IR is the inhalation rate (m\(^3\)/hour) and BW is the body weight (kg).\(^4^1\)

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:

\[
ADD = \left( \frac{C \times IR \times ED}{BW \times AT} \right) \quad (2)
\]

where ADD is the average daily dose of the chemical of interest (µg/kg/day), C is the amount of the chemical in ambient air (µg/m\(^3\)), IR is the inhalation rate (m\(^3\)/day), ED is the exposure duration (days), BW is the body weight of the exposed group (kg) and AT is the averaging time (days).\(^4^2\)

\[
ED \text{ (exposure duration)} = ET \times EF \times DE \quad (3)
\]
where ET is the exposure time (hour/day), EF is the exposure frequency (days/year) and DE is the duration of exposure (year). The standards for each age group are based on different assumptions, as shown in table 1.43 43

The EF value is calculated on the basis that a person will be absent from his place of abode (study area) for 14 days annually.43 The DE for an infant, child and adult was determined at 1, 12 and 30 years, respectively. The AT is estimated as the product of the duration of exposure and 365 days/year.

Table 2 shows the estimated ET values for each population group which was based on the average and continuous scenarios for acute, intermediate and chronic exposure periods.35 Default values were used for IR and BW43 and are given in table 3 for each exposure group.

Risk characterisation

Risk characterisation is the quantitative estimation of the health risk of exposure to a pollutant. Here, an estimate of possible non-carcinogenic effects from exposure to a known pollutant is determined using the hazard quotient (HQ).36 43 It reflects the probability of an adverse health outcome occurring among healthy and/or sensitive individuals. Non-cancer risks were calculated for acute and chronic exposure scenarios as:

\[
HQ = \frac{ADD}{REL} \quad (\text{chronic exposure}) \quad \text{or} \quad HQ = \frac{AHD}{REL} \quad (\text{acute exposure})
\]

where REL is the dose at which significant adverse health effects will occur in exposed groups compared with the 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 are used are 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, that is, 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

RESULTS

PM10 concentration

The mean hourly, daily and annual concentration of PM10 in Pretoria West are 67.74, 52.01 and 48.26 µg/m3, respectively (table 5). Although, the daily (24 hours)
guideline limit of 75 µg/m³ set by the NAAQS was not exceeded, the annual recommended mean limit of 45 µg/m³ that should not be exceeded was surpassed. The 1-hour (acute) scenario was not considered since a 1-hour REL value for PM₁₀ was not found in the literature. The HQ from the health risk characterisation from exposure to PM₁₀ 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 an HQ<1.0 indicates that PM₁₀ is not likely to induce adverse health outcomes. However, infants (2.0×10⁻² vs 4.2×10⁻¹) followed by children (1.1×10⁻¹ vs 4.2×10⁻¹) are likely to be affected from exposure to PM₁₀ compared with adults (3.0×10⁻² vs 2.7×10⁻¹) under the normal and worst-case scenario, respectively, for intermediate exposure. For the chronic (annual) exposure scenario for normal and worst-case exposures, HQ >1.0 for the whole study population. This indicates that there may be some risks for sensitive population to develop health-related problems from chronic exposure to PM₁₀. 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.

**SO₂ concentration**

The measured average concentration of SO₂ for 1 hour, 24 hours and annual averages in the study area were 29.63, 21.48 and 18.68 µg/m³, respectively (table 5). These values are far less than the mean values of 350, 125 and 50 µg/m³ as provided by NAAQS for 1 hour, 24 hours and annual averages, respectively, that should not be exceeded (table 4). Estimation of risk for acute and intermediate (normal and worst-case) exposures to SO₂ revealed that HQ<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 (2.0×10⁻³) are likely to be affected the same way from exposure to SO₂ compared with adults (1.4×10⁻³). Under the normal and worst-case scenarios for chronic exposure, HQ>1.0 for the whole study population. This indicates that there may be some risks for sensitive

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### Table 4 Reference exposure levels for different pollutants

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>1 hour (µg/m³)</th>
<th>8 hours (µg/m³)</th>
<th>24 hours (µg/m³)</th>
<th>Annual mean (µg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM₁₀</td>
<td>–</td>
<td>75*</td>
<td>40*</td>
<td></td>
</tr>
<tr>
<td>NO₂</td>
<td>200*</td>
<td>188†</td>
<td>40*</td>
<td></td>
</tr>
<tr>
<td>SO₂</td>
<td>350†</td>
<td>125*</td>
<td>50*</td>
<td></td>
</tr>
<tr>
<td>CO</td>
<td>29 770‡</td>
<td>10 305‡</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>O₃</td>
<td>226†</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>

Source: Department of Environmental Affairs. *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 µg/m³.

CO, carbon monoxide; NO₂, nitrogen dioxide; PM₁₀, particulate matter; SO₂, sulphur dioxide; O₃, ozone.

### Table 5 Summary statistics of ambient concentrations of pollutants

<table>
<thead>
<tr>
<th>Averaging period</th>
<th>PM₁₀ (µg/m³)</th>
<th>NO₂ (µg/m³)</th>
<th>SO₂ (µg/m³)</th>
<th>CO (µg/m³)</th>
<th>O₃ (µg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td>Mean±SD</td>
</tr>
<tr>
<td>1 hour</td>
<td>67.74±61.63</td>
<td>17.44±17.26</td>
<td>29.63±33.64</td>
<td>1442.6±1248.05</td>
<td>29.78±8.69</td>
</tr>
<tr>
<td>8 hours</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>618.30±618.30</td>
<td>22.15±7.96</td>
</tr>
<tr>
<td>24 hours</td>
<td>52.01±50.58</td>
<td>13.13±13.21</td>
<td>21.48±27.71</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Annual</td>
<td>48.26±43.41</td>
<td>11.50±11.61</td>
<td>18.68±25.36</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

CO, carbon monoxide; NO₂, nitrogen dioxide; PM₁₀, particulate matter; SO₂, sulphur dioxide; O₃, ozone.

### Table 6 Hazard quotients for normal and worst-case exposure scenarios to particulate matter (PM₁₀)

<table>
<thead>
<tr>
<th>Exposed group</th>
<th>Exposure</th>
<th>Intermediate</th>
<th>Normal</th>
<th>Chronic</th>
<th>Worst case</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (birth to 1 year)</td>
<td>2.0×10⁻²</td>
<td>4.2×10⁻¹</td>
<td>1.0×10¹</td>
<td>2.44×10²</td>
<td></td>
</tr>
<tr>
<td>Child (6–12 years)</td>
<td>1.1×10⁻¹</td>
<td>4.2×10⁻¹</td>
<td>3.62×10²</td>
<td>1.45×10³</td>
<td></td>
</tr>
<tr>
<td>Adult (19–75 years)</td>
<td>3.0×10⁻²</td>
<td>2.7×10⁻¹</td>
<td>2.81×10²</td>
<td>2.25×10³</td>
<td></td>
</tr>
</tbody>
</table>

The 1-hour (acute) scenario was not considered since a 1-hour reference exposure level value for PM₁₀ was not found in the literature.

individuals as a result of exposure to SO₂. The severity of exposure differs for different age groups.

NO₂ concentration
The monitored 1-hour, 24-hour and annual concentrations of NO₂ shown in Table 5 were 17.44, 13.13 and 11.50 µg/m³. The NAAQS 1-hour, 24-hour and annual guidelines of 200, 188 and 40 µg/m³, 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 (2.3×10⁻³) might be affected by acute exposure to NO₂ than adults (1.5×10⁻³). Moreover, having an adverse health outcome from normal and worst-case chronic exposure to NO₂ was found to be higher (HQ>1.0) for all age groups. Children (3.05×10²) appear more likely to be affected by normal chronic exposure than infants (8.6×10¹) and adults (2.37×10²), whereas for worst-case chronic exposure, adults (1.893×10³) are more likely to be affected.

CO concentration
CO concentrations of 1442.6 µg/m³ (1-hour average) and 618.30 µg/m³ (8-hour average) (Table 5) were not exceeded in comparison with the NAAQS guideline of 29 770 µg/m³ for 1-hour and 10 305 µg/m³ for 8-hour exposure limits. Estimation of risk for acute exposure to CO revealed that HQ <1.0 for infants, children and adults (Table 9). This implies a negligible risk, even for sensitive infants, children and adults. However, infants and children (1.3×10⁻³) may suffer the effects compared with adults (8.0×10⁻⁴). 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-hour CO (HQ<1.0).

O₃ concentration
The monitored concentration of O₃ for 1-hour and 8-hour averages in the study area were 29.78 and 22.15 µg/m³, respectively (Table 5). The NAAQS and annual guideline of 226 and 120 µg/m³, respectively, were not exceeded at Pretoria West (Table 4). The HQ calculated for the acute and intermediate (normal and worst-case) exposure scenarios showed no likelihood of adverse health effects being experienced by any individuals (HQ<1.0) (Table 10). During acute exposure, adults (2.2×10⁻²) are less likely to be affected than infants and children (3.0×10⁻³), while the reverse is the case for continuous exposure to O₃ for 8 hours.

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-hour PM₁₀ ambient quality standard of 75 µg/m³ was not exceeded on any of the days during the monitoring period. This is in contrast to other studies conducted elsewhere in South Africa. A 24-hour PM₁₀ of 157.37 µg/m³ (highest peak) and 110 µg/m³ was reported by Thabethe et al. and Matooane and Diab, respectively. The average annual concentration of PM₁₀ recorded in our study was slightly above the guideline limit of 45 µg/m³ set by the

<table>
<thead>
<tr>
<th>Exposed group</th>
<th>Acute</th>
<th>Intermediate Normal</th>
<th>Intermediate Worst case</th>
<th>Chronic Normal</th>
<th>Chronic Worst case</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (birth to 1 year)</td>
<td>2.5×10⁻³</td>
<td>6.0×10⁻³</td>
<td>1.5×10⁻¹</td>
<td>8.5×10¹</td>
<td>2.05×10²</td>
</tr>
<tr>
<td>Child (6–12 years)</td>
<td>2.3×10⁻³</td>
<td>4×10⁻²</td>
<td>1.5×10⁻¹</td>
<td>3.05×10²</td>
<td>1.218×10³</td>
</tr>
<tr>
<td>Adult (19–75 years)</td>
<td>1.5×10⁻³</td>
<td>1.0×10⁻²</td>
<td>9.0×10⁻²</td>
<td>2.37×10²</td>
<td>1.893×10³</td>
</tr>
</tbody>
</table>
Table 9 Hazard quotients for normal and worst-case exposure scenarios to carbon monoxide (CO) at different levels of exposures

<table>
<thead>
<tr>
<th>Exposed group</th>
<th>Exposure</th>
<th>Intermediate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (birth to 1 year)</td>
<td>Acute</td>
<td>1.3×10−3</td>
</tr>
<tr>
<td>Child (6–12 years)</td>
<td>Intermediate*</td>
<td>9.0×10−3</td>
</tr>
<tr>
<td>Adult (19–75 years)</td>
<td>Normal</td>
<td>3.0×10−3</td>
</tr>
</tbody>
</table>

*Intermediate—8-hour exposure period.

Table 10 Hazard quotients for normal and worst-case exposure scenarios to ozone (O3) at different levels of exposures

<table>
<thead>
<tr>
<th>Exposed group</th>
<th>Exposure</th>
<th>Intermediate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (birth to 1 year)</td>
<td>Acute</td>
<td>3.5×10−3</td>
</tr>
<tr>
<td>Child (6–12 years)</td>
<td>Intermediate*</td>
<td>3.0×10−3</td>
</tr>
<tr>
<td>Adult (19–75 years)</td>
<td>Normal</td>
<td>2.2×10−2</td>
</tr>
</tbody>
</table>

*Intermediate—8-hour exposure period.

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 PM10. 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. A review of 12 previous studies in the year 2001 affirmed that a 10 μg/m3 increase in PM10 causes an increase in hospital admissions for congestive heart failure and ischaemic heart disease. Among the vulnerable population (older people and those with a previous medical history of respiratory and cardiovascular diseases), long-term exposure to PM10 has been linked to an increase in morbidity and mortality from respiratory and cardiovascular diseases. Also for adults, large population studies have shown an association between respiratory (admissions for asthma, COPD and pneumonia) hospitalisation and ambient PM10. However, the effects seem to be stronger for older patients with even short-term exposures.

This study further revealed that the 1-hour, 24-hour and annual mean concentration for NO2 are below the national standard. Evidence from the risk characterisation assessment shows a negligible risk for acute and intermediate exposure to ambient levels of NO2.

However, 1-year exposure to ambient levels of NO2 could pose some risks to the sensitive individual. Recent epidemiological studies have revealed that exposure to low levels of NO2 could increase emergency room hospitalisation for acute and obstructive lung diseases in the general population. Studies conducted in Canada, Denmark and Italy found a significant association between exposures to levels of NO2 and acute ischaemic stroke. However, some studies did not find significant associations between exposure to ambient and personal levels of NO2 and health effects.

Our study further shows low ambient value (compared with national standard) for SO2 in Pretoria West. Similarly, there is no likelihood of health risk (HQ<1) associated with 1-hour and 24-hour exposure to SO2. However, some levels of risk for sensitive individuals were found for chronic (annual) exposure to SO2 in the study area. The possibility of SO2 worsening childhood asthma at fairly modest concentration, that is, well below the US EPA standards and WHO guidelines, has been reported. Multicity studies conducted in Europe and Asia offer further proof supporting the short-term association of SO2 with adverse health outcomes, including mortality and morbidity.

In this study, low ambient concentrations of CO and O3 were recorded. Researchers are of the opinion that exposure to ambient levels of CO is often not recognised; its toxicity is mostly under-reported and misdiagnosed due to its non-irritation and imperceptibility in the air we inhale. Exposure to CO has been linked to poison-correlated mortality in the USA. However, O3 is a strong oxidant that weakens biological tissues, thus resulting in increased use of medication, ailment and death. It has even been previously established that no level of exposure to O3 is safe since health risk has been found to be associated with O3 even at concentrations below the recommended standards.

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 NO2 and SO2. 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 relatively higher amount of air inhalation (the air intake per weight unit of a resting infant is twice that of an adult), and their immune system and lungs not being fully developed.

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 includes 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.

The findings in our study should be interpreted in the light of the following limitations. The ecological nature of this study used populations or groups of people as the unit of analysis rather than individuals. The ecological 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 sociodemographic 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 as it was the first conducted in the industrial area of Pretoria in South Africa that described the health risk associated with human exposure to PM and other gaseous pollutants. In addition, the study uses hourly ambient pollution data, with the method of data collection having undergone a validated process, and the study outcome is generalisable. 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 suspended particulates and gaseous pollutants, with the gaseous components comprising O₃, CO, NO₂ and SO₂. The acute, intermediate and chronic ambient concentrations of PM₁₀ and the gaseous pollutants recorded in Pretoria West were within the South African NAAQS. No health risk was found to be associated with acute and intermediate exposure to the pollutants, though infants and children, rather 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 for 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 policymakers 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. ASA, 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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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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