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RESEARCH ARTICLE

Air pollution inhalation during acute exercise is dependent of the body mass index and ventilation of young men

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Abstract

Obesity and physical inactivity threaten human health, and both could be solved with exercise. However, a higher amount of pollutants is inhaled during exercise. Exposure to air pollution increases the incidence and progression of diseases. Therefore, the aim of the study was to investigate the rate of pollution inhalation of lean, overweight, and obese individuals in a low and high-intensity hypothetical exercise session. Healthy sedentary men ($n = 135$) classified as lean, overweight, or obese were enrolled in our study. All participants performed a cardiopulmonary exercise testing (CPX) to collect ventilation rate (VE) data, which was used to predict total ventilation and pollutant inhalation of a 5-km running session. Air pollutant concentration of São Paulo City, Brazil was evaluated and the toxicological risk was estimated based on the potential intake dose. The concentrations of $PM_{2.5}$ were 29.57 μg/m^3 and 51.71 μg/m^3, PM_{10} were 45.85 μg/m^3 and 74 μg/m^3, NO_2 were 63.71 μg/m^3 and 66.65 μg/m^3, and O_3 were 69 μg/m^3 and 37 μg/m^3, respectively in the summer and winter. In the hypothetical exercise session, total VE and time in both the first and second threshold were increased in the obese group ($p < 0.001$) ($p < 0.001$). The inhalation of $PM_{2.5}$, PM_{10}, NO_2, and O_3, during the hypothetical session, was increased in obese individuals ($p < 0.001$). Obese individuals should be considered a susceptible population, once they are more exposed to air pollution during exercise.

Keywords Air pollution · Environmental pollution · Exercise · Body mass index · Obesity · Risk assessment

Introduction

Currently, obesity, physical inactivity, and environmental pollution are among the ten threats to global health, and the ambient air pollution has been recognized as the greatest environmental risk to human health. According to the World Health Organization, nine out of ten people breathe polluted air every day (WHO 2019). Obesity, physical inactivity, and pollution are important risk factors for the development of many chronic diseases, inducing morbidity and early mortality (Kampa and Castanas 2008; Burnett et al. 2014; Checa Vizcaíno et al. 2016; Pope et al. 2016). Worryingly, 91% of the world's population lives in places where air quality exceeds the WHO guideline limits. Regarding the main pollutants, particulate matter (PM), nitrogen dioxide (NO_2), and ozone (O_3) exposure cause serious risks to health (WHO 2016).

Physical activity promotes benefits on health and quality of life. Exercise training performs an immunoregulatory role responsible for the maintenance of low levels of inflammatory mediators and induces immunometabolic adaptations (Da Silva et al. 2019; Dorneles et al. 2019; Dorneles et al. 2020). In this sense, endurance exercise emerges as a low-cost non-pharmacological therapy for the treatment and prevention of obesity and related diseases (Kodama et al. 2009; Lee et al. 2012; Fiuza-Luces et al. 2013). The primary recommendations from American College of Sports Medicine (ACSM) include the practice of at least 30 min continuous moderate-

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intensity endurance exercise (in an intensity of ~ 65% of the maximal oxygen uptake, VO_{2max}) or 20 min of vigorous exercises (intensity up to 75–80% VO_{2max}) 3 days per week (Haskell et al. 2007).

However, environmental characteristics, such as air pollution, may modulate the exercise adaptations. The increased metabolic demand during exercise is related to higher oxygen uptake (VO_2) and carbon dioxide production (VCO_2), which elevates the minute ventilation (VE) and the amount of inhaled gas and particles during the bout. Furthermore, a transition of nasal respiratory pattern to an oronasal pattern is observed during the increase of exercise intensity (Niinimaa et al. 1980; Chlif et al. 2007; Sood 2009). Volume and intensity of the bout have a pivotal role on VE increasing, yet, some individual characteristics, as body composition might impact on respiratory mechanisms in response to acute exercise. In fact, several respiratory factors accompany obesity, such as decreased thoracic compliance and increased airway resistance (Sood 2009; Shoukri 2015).

Although regular physical activity is associated with reduced mortality rates by all causes, the benefits of exercise can be negated in polluted environments. Obese individuals may be more susceptible to the harmful effects of air pollution, as hyperventilation during exercise can lead to excessive inhalation of air pollutants and, consequently, additional health risk, overcoming benefits of exercise. To the author's knowledge, there is a lack in the literature regarding the impact of different intensities of endurance exercise on VE and the estimation of air pollutant inhalation by individuals with different body mass index (BMI). Therefore, the aim of this study was to investigate the pollution inhalation of lean, overweight, and obese individuals in a moderate and high-intensity exercise session.

Materials and methods

Subjects

This cross-sectional study was approved by the Ethic's Research Committee of the UFCSPA (63282416.6.0000.5345), and all experimental procedures were performed according to the Declaration of Helsinki. All participants were informed about the study and signed the informed consent. A total of 135 apparently healthy sedentary young men were recruited from local universities via advertisements and oral communications.

According to the criteria established by the World Health Organization, participants were assigned to three experimental groups according to their body mass index (BMI): lean (n = 45), overweight (n = 45), and obesity grade 1 (n = 45). Participants were healthy, no smokers, age ranging from 20 to 40 years, and free of illness and injury, and they were not engaged in physical training programs for a period of 6 months prior to the experimental trial. Exclusion criteria included excessive alcohol intake (> 10 drinks/week), orthopedic limitations, neuromuscular or joint injury, autoimmune or cardiac diseases, or type 1 and type 2 diabetes, and acute and chronic infections. Individuals who presented very low VO_{2peak} (< 25.00 mL kg min) or high values of VO_{2peak} (> 50.0 mL kg min) were also excluded. Body mass (kg) and height (m) were determined by a semi-analytical scale with a stadiometer attached (Welmy, Santa Barbara D'Oeste, Brazil) before the cardiopulmonary exercise testing (CPX).

Cardiopulmonary exercise testing

The CPX was performed on an electric treadmill (Centurion 300) using a ramp protocol (Balady et al. 2010). The laboratory temperature control (18–22 °C) and relative humidity (40–60%) were continuously monitored. Briefly, exercise test started at 4.5 km/h and no slope for all study participants. Then, the load (speed and slope) was individually increased for each participant considering their physical condition and tolerance. The test was terminated after verification of at least two of the following circumstances: (a) request of the participant due to extreme tiredness and/or perception of the intense dyspnea; (b) reached the maximum heart rate (HR) predicted by age (HRmax) which was ≥ 85%; (c) peak respiratory exchange ratio RER > 1.1; (d) VO_2 plateau which was reached even with increasing workload.

Ventilatory and metabolic parameters were collected by respiration using Metalyzer 3B (Germany) and were analyzed after the mean of the data in eight respiratory cycles. The CPX system was calibrated before each test concerning both airflow and O_2 and CO_2 analyzers. The average of the last test 30 s was used to determine the VO_{2peak} (Balady et al. 2010). The first ventilatory threshold (VT_1) was identified as the minimum workload at which the ventilatory equivalent ratio for oxygen (VE/VO_2) systematically increased without an increase in the ventilatory equivalent ratio for carbon dioxide (VE/VCO_2), and the second ventilatory threshold (VT_2) as the lowest workload where both VE/VO_2 and VE/VCO_2 increased (Balady et al. 2010). VT_1, VT_2, and VO_{2max} were obtained by visual inspection of graphs by 3 independent observers.

Air pollution concentration

The megacity São Paulo, with a population of 21 million, is the biggest city of Brazil, representing more than 11% of the total population of the country and covering an area of

7947 km^2 (UN 2014; IBGE 2016). The concentration of air pollutants was provided by the Environment Company of São Paulo State (CETESB - Companhia Ambiental do Estado de São Paulo). The pollutants selected for analyses were particulate matter $_{2.5}$ and $_{10}$ ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), and ozone (O_3).

We evaluate air pollution concentration in the hottest day of the summer and coldest day of the winter in the year 2019, in order to have a panorama of air pollution exposure during seasonal variations. The meteorological data of temperature, humidity, precipitation, and solar radiation were accessed in the National Institute of Meteorology (INMET).

Prediction of total ventilation and pollutant inhalation during exercise

The minute ventilation (VE, L/min) was extracted at VT_1 and VT_2 identified in CPX each participant and used to predict VE_{total} in two hypothetical 5-km endurance exercise sessions. The intensities were used based on moderate-intensity and high-intensity endurance exercise recommendations (Garber et al. 2011) but with equalized volume (5 km). The volume of exercise was equalized considering the time required to complete a 5-km exercise with the speed (km/h) identified in each VT_1 and VT_2. Thus, the VE corresponding to VT_1 and VT_2 of the VO_{2max} was extracted from incremental test and used in Eq. 1:

$$\mathrm{VE_{total}\ (L) = VE\ (L\ min^{-1}) \times time\ spent\ to\ complete\ 5\ km\ at\ each\ ventilatory\ threshold\ intensity} \quad (1)$$

Further, the VE_{total} values were used to calculate the inhaled dose of $PM_{2.5}$, PM_{10}, NO_2, and O_3 during the two hypothetical exercise sessions, according to Eq. 2:

$$\mathrm{Pollutant\ inhaled\ (\mu g) = VE_{total} \times pollutant\ concentration\ (\mu g/m^3)/1000} \quad (2)$$

In which, pollutant inhaled is the total amount of pollutant inhaled during the exercise, VE_{total} is the total ventilation during the two hypothetical endurance exercise sessions, and pollutant concentration is the concentration of air pollutant measured in São Paulo City.

Toxicological risk

The toxicological risk was estimated based on the potential intake dose of the pollutants and the reference dose. We considered the calculation of toxicological risk of performing 30 min of running, 5 days/week with the second threshold intensity and the risk of incremental time of exercise in an environment with $PM_{2.5}$ concentration equal to the city of São Paulo in our study. Equation 3 was used to calculate the potential intake dose for both situations (I, μg/kg day) considering:

$$I = C_A \times (IP \times FR \times FA \times ET \times EF \times ED)/BW \times 1AT \quad (3)$$

C_A = average $PM_{2.5}$ concentrations of monitored day (μg/m^3)

IP = inhalation rate during exercise, representing total ventilation of the second threshold (m^3)

FR = retention factor—we assumed a retention factor of FR = 1, which represents the highest exposure and the highest potential impact on subjects' health.

FA = absorption factor—we assumed an absorption factor of FA = 1, which represents the highest exposure and the highest potential impact on subjects' health.

ET = exposure time (h/day)—exercise's duration

EF = exposure frequency (day/year): 5 times/week—20 time/month—260 days/year

ED = duration of exposure (year): 260 days of exposure/365 days = 0.71

BW = body weight (kg)

AT = average time—period of exposure in which the dose was measured (day): 260

Toxicological risk:

$$RQ = I/RfD \quad (4)$$

In which:

RQ = risk quotient;

Risk quotients are classified as follows: RQ ≤ 1: unlikely risk, even in population groups that are sensitive to adverse health effects; RQ > 1: there is a risk of non-carcinogenic adverse effects on human health.

I = potential intake dose (μg/kg day);

RfD: reference dose for $PM_{2.5}$ = 1.14, according to Silva et al. (2016).

Statistical analysis

The normality of the data was measured using Kolmogorov-Smirnov test. Unpaired Student t test was used to compare variation of the air pollutant concentration and seasonal variation. A 1-way analysis of variance (ANOVA) followed by Bonferroni's post hoc test was applied to compare age, BMI, waist circumference, ventilation rate, VO_2, respiratory exchange ratio, speed, and time during different exercise conditions and air pollutant concentrations. A 2-way analysis of variance (ANOVA) followed by Bonferroni's post hoc test was applied to compare the inhaled dose of $PM_{2.5}$, PM_{10}, NO_2, and O_3 between exercise intensity and pollution concentration. All analyses were performed using SPSS 20.0 (SPSS Inc., EUA). The significance level was set at $p \geq 0.05$.

Results

Sample characterization

Sample characterization showed no difference in age among the groups ($p = 0.0529$), and BMI and waist circumference were increased in obese and overweight compared with lean individuals, as well as in obese compared with overweight men ($p < 0.001$). In the first ventilatory threshold, the VE and VO_2 of the obese group were higher than in the lean participants ($p = 0.0114$) ($p = 0.0462$). Also, the obese group showed decreased speed ($p < 0.001$) compared with the lean and overweight groups. In the second ventilatory threshold, no difference in VE was observed among groups ($p = 0.5250$). While, VO_2 and speed were increased in the lean and overweight subjects compared with the obese group ($p < 0.001$). In the maximal oxygen capacity (VO_{2max}), VE of the lean group was increased compared with obese subjects ($p = 0.0490$). The lean and overweight groups demonstrated increased VO_2 and speed compare with the obese group ($p < 0.001$) and respiratory exchange ratio was not different among groups ($p = 0.9120$). Regarding the hypothetical exercise session, the total VE and duration of session in both the first and second threshold were increased in the obese group compared with the lean and overweight groups ($p < 0.001$) (Table 1).

Pollutant concentration

$PM_{2.5}$ and PM_{10} concentrations were higher in the winter compared with the summer ($p < 0.001$). NO_2 concentrations demonstrated no difference ($p > 0.05$). O_3 levels in the summer demonstrated increased concentration when compared with the winter ($p < 0.001$) (Table 2).

Meteorological measures

The summer day showed increased temperature and UV radiation compared with the winter day ($p < 0.05$). There was no significant difference in humidity, wind speed, and precipitation between summer and winter days ($p > 0.05$) (Table 3).

Inhalation of air pollutants

The inhalation of $PM_{2.5}$, PM_{10}, NO_2, and O_3, during the hypothetical session, was increased in the obese group compared with lean and overweight groups ($p < 0.001$) (Fig. 1). In the

Table 1 Sample characteristics of total capacity test and hypothetical exercise session

	Lean ($n = 45$)	Overweight ($n = 45$)	Obese ($n = 45$)	p value
Age (years)	27.58 ± 6.764	31.18 ± 9.796	29.44 ± 8.215	0.0529
BMI (kg/m^2)	22.73 ± 1.939*	27.49 ± 1.137*	33.29 ± 2.996	< 0.001
Waist circumference	68.44 ± 5.684*	82.46 ± 6.705*	100 ± 8.651	< 0.001
VE 1st threshold (L/min)	38.98 ± 13.07*	40.38 ± 12.11	46.41 ± 11.72	0.0114
VO_2 1st threshold (mL/kg min)	24.99 ± 6.553*	23.91 ± 5.51	21.85 ± 5.746	0.0426
Speed 1st threshold (km/h)	7.911 ± 1.043*	7.851 ± 1.059*	6.776 ± 1.118	< 0.001
VE 2nd threshold (L/min)	75.15 ± 18.57	76.48 ± 17.59	79.39 ± 18.00	0.5250
VO_2 2nd threshold (mL/kg min)	39.83 ± 7.655*	37.05 ± 7.651*	31.84 ± 5.529	< 0.001
Speed 2nd threshold (km/h)	11.30 ± 1.811*	10.91 ± 2.053*	9.194 ± 1.334	< 0.001
VE of VO_{2max} (L/min)	133.4 ± 19.94*	128.3 ± 17.54	123.5 ± 19.01	0.0490
VO_{2max} (mL/kg min)	50.09 ± 8.172*	46.42 ± 9.203*	39.10 ± 6.561	< 0.001
Speed VO_{2max} (km/h)	14.06 ± 1.754*	13.58 ± 1.896*	11.58 ± 1.687	< 0.001
RER of VO_{2max}	1.089 ± 0.026	1.086 ± 0.028	1.087 ± 0.026	0.9120
Hypothetical exercise bout performed at the 1st threshold intensity				
VE total (L)	1469 ± 443.7*	1542 ± 411.3*	2090 ± 535	< 0.001
Exercise duration (min)	38.60 ± 5.306*	38.96 ± 5.872*	46.23 ± 13.54	< 0.001
Hypothetical exercise bout performed at the 1st threshold intensity				
VE total (L)	2016 ± 536.2*	2114 ± 618.9*	2609 ± 571.7	< 0.001
Exercise duration (min)	27.24 ± 4.437*	28.44 ± 5.236*	33.59 ± 7.354	< 0.001

Data are presented as mean ± SD.

Between groups, differences were verified by one-way ANOVA followed by Bonferroni's post hoc ($p < 0.05$)

BMI body mass index; *RER* respiratory exchange ratio; *VE* minute ventilation; *VO_{2max}* maximal oxygen capacity

* Denotes statistical difference compared with the obese group ($p < 0.05$)

Table 2 Air pollution concentration in the hottest day of the summer and coldest day of the winter in the year 2019

	$PM_{2.5}$ (μg/m^3)	PM_{10} (μg/m^3)	NO_2 (μg/m^3)	O_3 (μg/m^3)
Summer	29.57	45.85	63.71	69
Winter	51.71	74	66.65	37

Data provided by the Environment Company of São Paulo State (CETESB - Companhia Ambiental do Estado de São Paulo)

$PM_{2.5}$ particulate matter 2.5; *PM_{10}* particulate matter 10; *NO_2* nitrogen dioxide; *O_3* ozone

same way, all pollutants had higher inhalation levels in high-intensity than in moderate-intensity exercise in all groups ($p < 0.001$). Regarding the pollutant inhalation in each season, $PM_{2.5}$ ($p < 0.001$), PM_{10} ($p < 0.001$), and NO_2 ($p < 0.049$) inhalation were increased in the winter compared with the summer, and O_3 inhalation was increased in the summer in relation to the winter ($p < 0.001$).

Inhalation of $PM_{2.5}$, PM_{10}, and O_3 showed an interaction between group and season ($p < 0.007$) ($p = 0.023$) ($p = 0.002$), in which obese individuals inhaled more $PM_{2.5}$ and PM_{10} in the winter compared with all groups in both seasons. Also, obese individuals inhaled more O_3 in the summer compared with all groups in both seasons (Fig. 2). The obese group had increased inhalation of $PM_{2.5}$ ($p < 0.001$), PM_{10} ($p = 0.004$), and O_3 ($p < 0.001$), in high-intensity exercise compared with all groups in the winter for $PM_{2.5}$ and PM_{10}, and during the summer for O_3. The inhalation of NO_2 showed no difference in the interaction of group with intensity ($p = 0.708$), group with season ($p = 0.966$), intensity with season ($p = 0.781$), and group with intensity and season ($p = 1.00$). No difference was observed between group with intensity in inhalation of $PM_{2.5}$ ($p = 0.725$), PM_{10} ($p = 0.721$), and O_3 ($p = 0.729$), and among group, intensity and season in inhalation of $PM_{2.5}$ ($p = 0.976$), PM_{10} ($p = 0.982$), and O_3 ($p = 0.972$).

Toxicological risk

A session of aerobic exercise performed for 30 min with the intensity detected in the second threshold in individuals from our study would cause toxicological risk with exposure to $PM_{2.5}$ at 115 μg/m^3 for lean individuals and 125 μg/m^3 for overweight and obese individuals. Considering the $PM_{2.5}$ concentration used in our study, the time to achieve the risk by performing a running was 1 h and 25 min in the lean group and 1 h and 30 min in the overweight and obese groups, as illustrated in Fig. 3.

Discussion

Our study points out an increased ventilation and VO_2, and reduced speed of obese participants in the first threshold of the cardiopulmonary exercise testing, while in the second threshold, VO_2 and speed were decreased in this group. Considering the hypothetical aerobic exercise session, obese individuals would had increased total ventilation during the session and spend more time to perform the exercise, consequently inhaling more air pollutants.

In our study, obese individuals showed increased total ventilation during exercise, which might occur due to alterations on respiratory physiology caused by obesity, including changes in lung volumes, ventilatory behavior, and decreased respiratory compliance. In the same way, obesity could cause a fatigue of inspiratory muscle causing inefficiency of respiratory muscles (Chlif et al. 2005; Chlif et al. 2007). So, obese subjects demonstrate a pattern of breathing fast and shallow, increasing the oxygen cost of breathing (Rochester 1998; Kress et al. 1999; Chlif et al. 2009). Also, during a peak exercise, obese individuals may hyperinflate and increase ventilation rate to avoid significant levels of expiratory flow limitation, contributing to our result of increased respiratory rate during exercise (Sakamoto et al. 1993).

The concentrations of $PM_{2.5}$, PM_{10}, and NO_2 pollutants were higher in the winter, while the O_3 concentration was higher in the summer, which were expected results once levels of particulate matter and NO_2 are elevated in the winter due to thermal inversion phenomenon, and increased production of O_3 is detected in the summer due to higher solar radiation (Cichowicz et al. 2017; Trinh et al. 2018; Marmett et al. 2019). Consequently, the inhalation of particulates and NO_2 was higher in the winter and O_3 in the summer in lean, overweight, and obese individuals. As well as, the inhalation of pollutants was augmented in high-intensity exercise compared with moderate intensity, since the VE was increased, and the inhalation of pollutants increased as well. Similarly, Pasqua et al. (2018) hypothesized an aerobic exercise with 65% intensity for 30 min in the ten cities with the highest and lowest levels of $PM_{2.5}$ and PM_{10}, and demonstrated that the

Table 3 Meteorological parameters during the hottest day of the summer and coldest day of the winter in the year 2019

Season	Temperature (°C)	Humidity (%)	Precipitation (mm)	Wind speed (m/s)	UV radiation (h)
Summer	27.76	67.33	0	1458	2700
Winter	18.08	68.46	0.008	1.05	1013

Data were accessed from the National Institute of Meteorology (INMET)

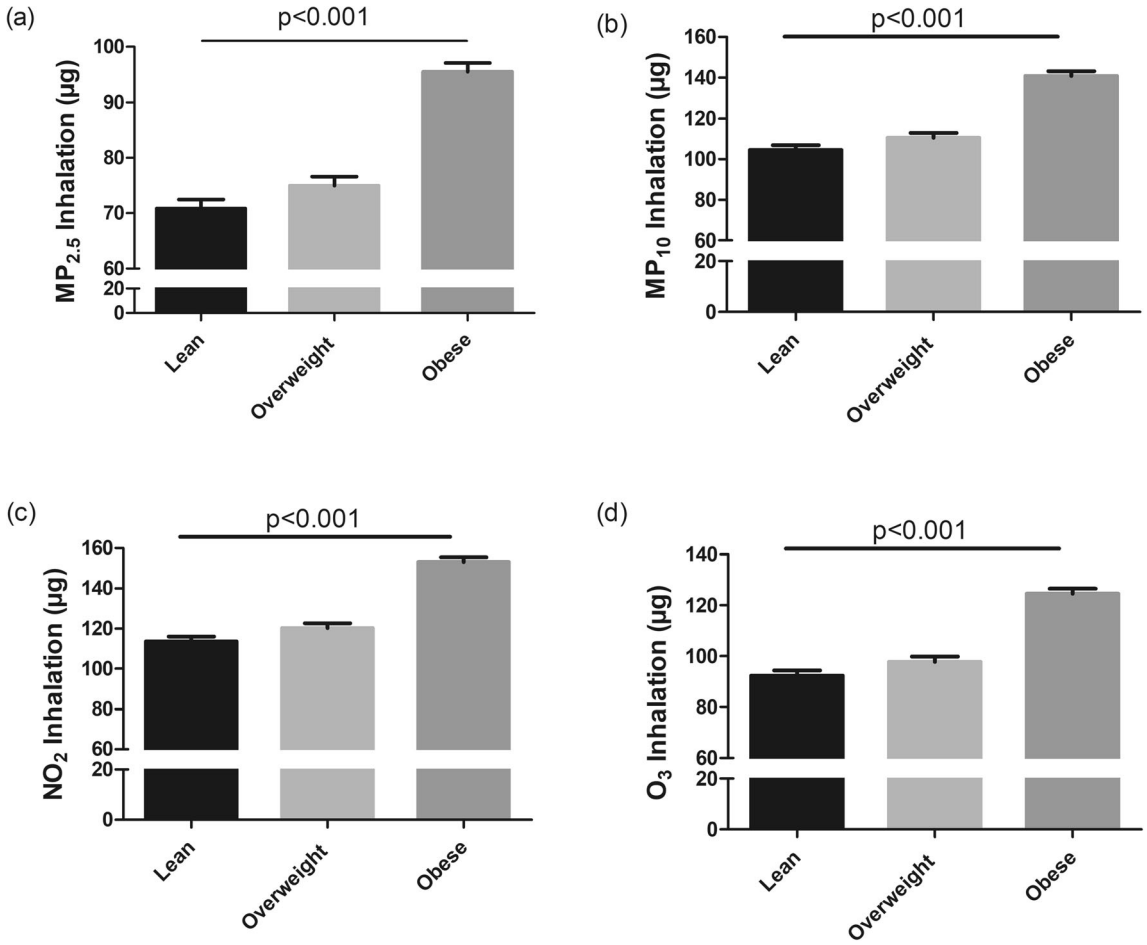

Fig. 1 Air pollutant inhalation during a hypothetical session of exercise of lean, overweight, and obese individuals. **a** $PM_{2.5}$ inhalation. **b** PM_{10} inhalation. **c** NO_2 inhalation. **d** O_3 inhalation. Data are presented as mean ± SD. Between groups, differences were verified by one-way ANOVA followed by Bonferroni's post hoc ($p < 0.05$). Statistical difference compared with the obese group ($p < 0.05$)

inhalation of pollution was higher during exercise and in the ten more polluted cities.

Obese individuals inhaled more $PM_{2.5}$, PM_{10}, NO_2, and O_3. The inhalation of $PM_{2.5}$, PM_{10}, and O_3 demonstrated interaction of group with season, and intensity with season, indicating that a higher inhalation could depend on BMI and intensity of exercise. Obese subjects that perform exercise in the winter would inhale higher amount of $PM_{2.5}$ and PM_{10}, while in the summer, it would result in higher inhalation of O_3. Besides, it was already demonstrated that the deposition of particles in airways increases 6 to 10-fold during high-intensity exercise due to an increase on that ventilation switch nasal breathing to oral overcoming the nasal filtration and increasing pollution dose inhaled (Niinimaa et al. 1980; Chalupa et al. 2004; Giles and Koehle 2014).

Regarding the influence of body mass on pollutant inhalation, obese participants may face additional health risks by being exposed to higher levels of pollutants, as Mazidi and Speakman (2018) demonstrated by indicating that the effects of $PM_{2.5}$ were more deleterious in areas with higher rates of obesity, especially on cardiovascular diseases and stroke mortality. The increase vulnerability of obese individuals was also observed in studies demonstrating that obesity may enhance the effects of air pollution on the prevalence of cardiovascular diseases and stroke (Chen et al. 2007; Qin et al. 2015).

In our study, high-intensity exercise led to higher inhalation of air pollutants, a result more prominent in obese individuals. Despite benefits of exercise, even performed in a polluted environment, obese individuals might have increased susceptibility to pulmonary infections (Misumi et al. 2019; Marmett et al. 2020). It is well-known that air pollution exposure enhances the severity of respiratory infections and exercise mobilizes higher volumes of air that increase the contact of the respiratory tract with pollutants (Kurt et al. 2016). Air pollutants trigger oxidative and proinflammatory processes and, together with intense exercise, increase the vulnerability to respiratory infection (Puett et al. 2014; Hart et al. 2015). An experimental study demonstrated that obesity predisposes to a unique form of pathogenesis during systemic viral infection (Misumi et al. 2019). Furthermore, obese individuals had compromised pulmonary function, impairing ventilation,

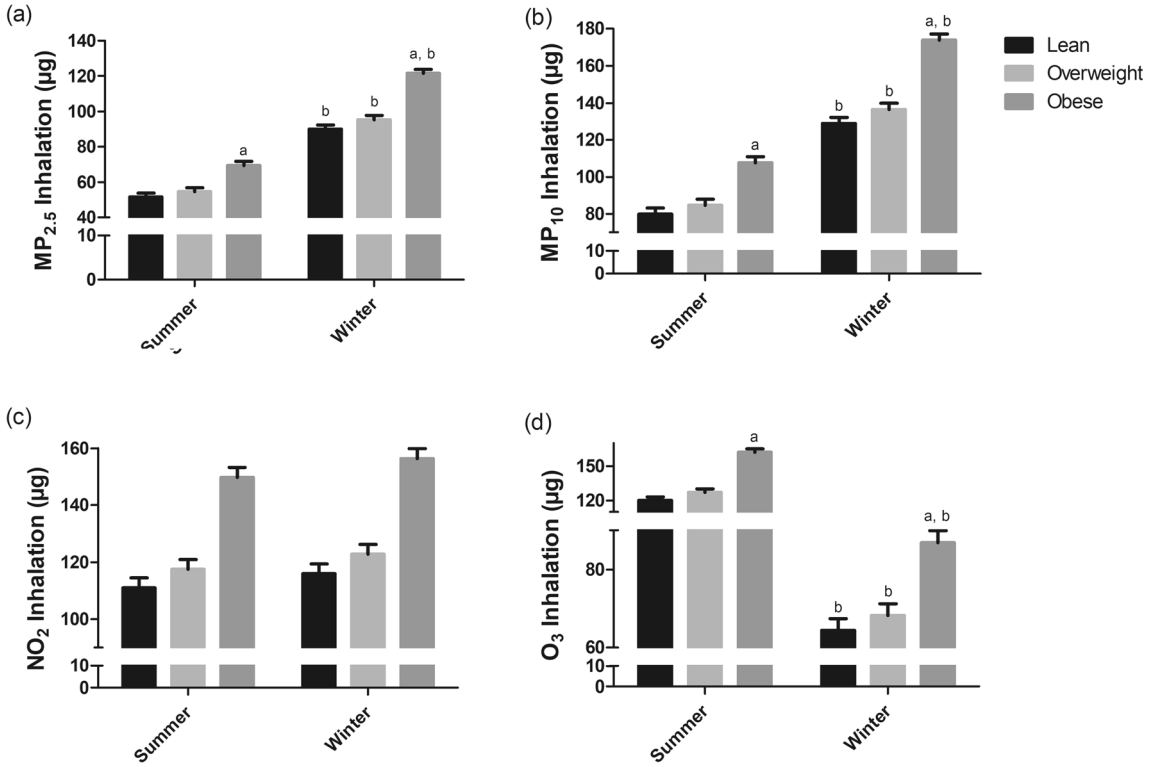

Fig. 2 Interaction of air pollutant inhalation during a hypothetical session of exercise of lean, overweight, and obese individuals in the summer and winter seasons. **a** $PM_{2.5}$ inhalation. **b** PM_{10} inhalation. **c** NO_2 inhalation. **d** O_3 inhalation. Data are presented as mean ± SD. Between groups, differences were verified by two-way ANOVA followed by Bonferroni's post hoc ($p < 0.05$). [a] Denotes statistical difference compared with the lean and overweight groups ($p < 0.05$). [b] Denotes statistical difference compared with all groups in the summer season ($p < 0.05$)

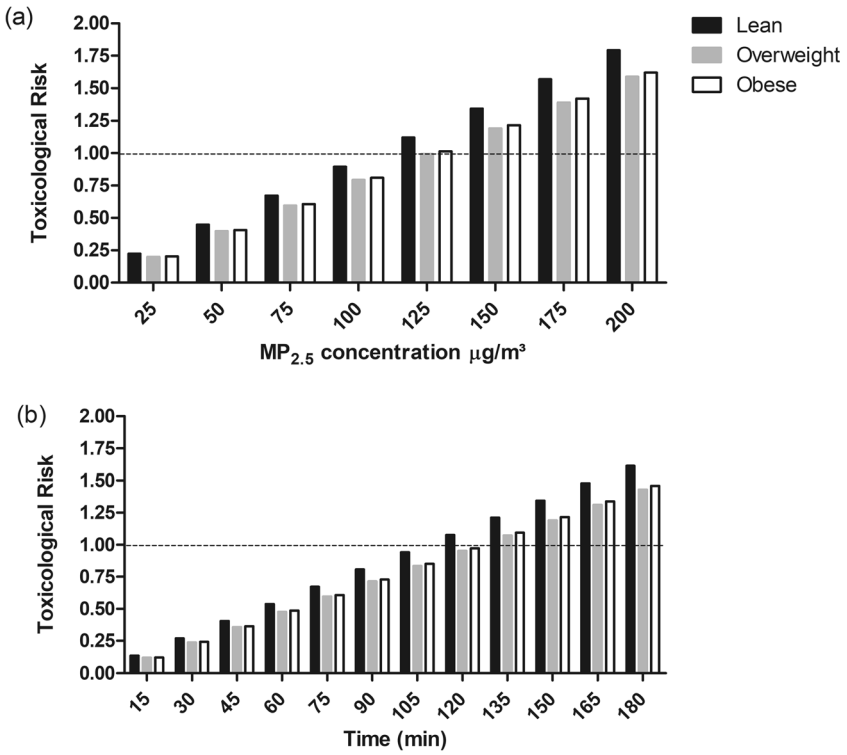

Fig. 3 Toxicological risk of lean, overweight, and obese individuals during exercise. **a** Toxicological risk of considering incremental time of exposure in a $PM_{2.5}$ concentration of 40.64 µg/m^3 during high-intensity exercise. **b** Toxicological risk considering incremental concentration of $PM_{2.5}$ during 30 min of high-intensity exercise

decreased immune responses to infections, increased inflammatory cytokines, and prolonged inflammation, which may contribute to the morbidity associated with infections (O'brien et al. 2012; Dietz and Santos-Burgoa 2020).

A study of SAPALDIA cohort demonstrated that the improvement of air quality attenuates lung function decline in normal weight and underweight participants; however, in obese individuals, no improvement was observed, highlighting the importance of contemplate health effects of air pollution exposure and obesity together (Schikowski et al. 2013). An increased susceptibility of obese people to air pollution exposure may be justified by oxidative stress and chronic low-grade inflammation that follows this condition; therefore, both air pollution and obesity are associated with increased incidence of disease and all-cause mortality (Galili et al. 2007; Pope et al. 2016; Iliodromiti et al. 2018; Khan et al. 2018).

Regarding the toxicological risk of exposure to $PM_{2.5}$, a session of aerobic exercise performed for 30 min would cause risk when pollution concentration overcome 115 $\mu g/m^3$ for lean individuals, and 125 $\mu g/m^3$ for overweight and obese individuals. However, when considering the pollution levels of São Paulo, from 1 h, 25 min of running would already represent a risk. Pasqua et al. (2018) estimated a relative risk of all-cause mortality for running, indicating that 15 min of running with moderate intensity in cities with high levels of $PM_{2.5}$ would no longer be beneficial and after 75 min, the risk of air pollution would overcome the benefits of exercise. Additionally, Tainio et al. (2016) established that in 30 min of cycling, the maximal benefits of the exercise (tipping point) would reach a $PM_{2.5}$ concentration of 95 $\mu g/m^3$ and the risk from air pollution would overweight the benefits of physical activity (break-even point) at 160 $\mu g/m^3$. Considering these studies together with our results, toxicological risk and relative risk of all-cause mortality demonstrated similar point in which exercising in a polluted environment could overcome the benefits of exercise.

It is important to highlight that even though obese individuals did not show additional risk compared with lean and overweight individuals, it does not mean that they are not a sensitive population. A great number of evidence cited above reported the susceptibility of the obese group to incidence and prevalence of diseases, as well as mortality, so the same dose of $PM_{2.5}$ could be more detrimental to health of obese than lean subjects (Schikowski et al. 2013; Iliodromiti et al. 2018; Khan et al. 2018).

The present study had some limitations. First, we only evaluated São Paulo air pollution, so our results may not be generalized to other world's cities. Second, our study was performed with healthy sedentary individuals; therefore, it is not possible to assume the data to another population. Third, the equation used to calculate the toxicological risk showed a result relativized to body mass, and it is possible that if body mass was not included, a difference would be detected. As strengths, we highlight the comparison of lean, overweight, and obese individuals, as well as the equalization of the volume of exercise considering the time required to complete a 5-km exercise to estimate the air pollution inhalation. Nevertheless, we observed the inhalation of four different pollutants, which are all included in the last global update of the WHO Air Quality Guidelines, demonstrating a complete scenario of air pollution.

Conclusion

Obese individuals had higher total ventilation and time during the hypothetical running leading to an augment in pollutant inhalation. So, this group should be considered a susceptible population, once they are more exposed to air pollution during exercise. Hence, exercise recommendations must consider the personal characteristics of individuals and environment particularities, and it may be necessary to modulate the exercise prescription according to the body mass index and the daily air pollution levels.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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