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Research Article

EXPOSURE TO $PM_{4.0}$ FROM THE COMBUSTION OF CASHEW NUTS SHELL IN THE RESPIRATORY SYSTEM OF MICE PREVIOUSLY EXPOSED TO CIGARETTE SMOKE

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ABSTRACT

The use of residual biomass as a source for energy production reveals a potential for global growth in the coming years. The energy utilization of residual products from the processing of cashew nut, such as cashew nuts shell (CNS), is a reality in industries and artisanal centers. However, such combustion can promote the release of pollutants, such as particulate matter (PM), with great capacity to cause or aggravate respiratory diseases. In the present work, we analyze the harmful effects on the respiratory system of mice exposed to $PM_{4.0}$present in CNS combustion exhaust gases associated with cigarette smoke (CS). C57black/6 mice were exposed to cigarette smoke or ambient air (Air group) for 60 days, and after this period the animals were submitted to a single nasal instillation containing $MP_{4.0}$ (CS+PM) or saline solution (CS). 24 h later, the animals were tracheostomized, cannulated and connected to a ventilator for small animals (Scirec©-flexVent®) to perform the analyzes referring to the variables of the respiratory system mechanics. Our results show statistically significant changes in some variables analyzed (R_N,G,H, C_{ST},CI, e PV loop area) of the CS and CS+PM groups in relation to the Air group.CNS as a biofuel can be feasible, but our results reinforce the urgent need to seek control methods for the exhaustion of these gases in the atmosphere. Further investigation is necessary in order to know safe parameters for individuals who are continuously exposed to CNS combustion exhaust gases.

INTRODUCTION

With the beginning of the first industrial revolution, characterized by the use of mineral and vegetal coal as the main energy matrix, the first concerns about the damages caused by the air pollution were born. With the increasing demand for energy, fossil fuels presented themselves as a quick and cheap solution for industries. However, the attempt to reduce emissions of environmental pollutants and global warming has created the need to use alternative sources of clean energy, which provide lower emissions of pollutants into the atmosphere. Following this trend, the renewable energy sector grew between 15% and 55% per year between 2005 and 2012 (REN, 2013), with emphasis on the use of biomass as a source for energy production, with the highest Growth potential in the coming years (WEC, 2010).

Brazil is one of the largest agricultural producers in the world, with great potential for the production of residual biomass. Some biomass wastes contribute to the growth of alternative energy production in the industrial sector, such as coffee grounds (Silva *et al.*,1998), rice husks (Maffioletti and Mota, 2013), sugarcane bagasse (Alcarde, 2015), eucalyptus (Nogueira *et al.*, 2014) and cashew nuts shell (CNS) (Paiva and

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Silva-Neto, 2013), the latter being the object of study of this article.

Obtaining the CNS begins through the withdrawal process almond cashew stalk (decorating), held in tanks with cardol, composed of 10% of the liquid extracted from cashew nut itself. This is heated in boilers at a temperature of ±800 °C. The by-product of this stage is the almond, of great commercial value, and the CNS, drenched in cardol, which hold great potential fuel (Lima, 2008).

The energy utilization of residual products from the processing of cashew nut, such as CNS, is already a reality in industries and craft centers. However, combustion of CNS, used in industries and craft centers, promotes the release of pollutants such as: carbon dioxide (CO_2), carbon monoxide (CO), ozone (O_3), nitrogen dioxide (NO_2), dioxide Sulfur (SO_2), polycyclic aromatic hydrocarbons (PAHs), and fine particles such as particulate matter with aerodynamic diameter equal to or less than 4 μm ($PM_{4.0}$), capable of penetrating the respiratory system with ease and reaching the pulmonary alveoli , Causing serious respiratory diseases (WHO, 2005; Lewné *et al*., 2007; Xiao *et al*., 2011). Particulate matter (PM) is one of the pollutants most associated with negative health outcomes. These effects are noted mainly by more vulnerable individuals, such as children, the elderly and people diagnosed with cardiorespiratory diseases (Dockery, 2009).

In parallel, smoking is one of the main risk factors for a number of chronic diseases, being the main cause of chronic obstructive pulmonary disease (COPD) (GOLD, 2016). As with smoking, air pollution is also related to reduced life expectancy with regard to COPD, concluding that individuals with chronic diseases are one of the population groups most susceptible to the toxic effects of air pollution (Rodrigues *et al*., 2015). Galvão and collaborators (2014) have identified, through gravimetric analysis, a high concentration of fine particles in regions where CNS is burned.

In view of the above, there is an urgent need to evaluate the effects of CNS's combustion emissions on health. This information may provide greater security in its use as biofuel. Considering that an individual with pre-established pulmonary disease living in the adjacent regions from where this vegetable biomass burns, in this work we analyze the deleterious effects on the respiratory system of mice exposed to $PM_{4.0}$ present in the combustion exhaust gases of CNS associated with cigarette smoke.

MATERIALS AND METHODS

CNS combustion reactor

A CNS combustion system was developed to collect $PM_{4.0}$ from combustion exhaust gases from CNS (Figure 1). For this collection, the CNS (500 g) was first placed in a cylindrical stainless steel burner (Figure 1A). Then, the initial combustion ignition of the CNS was accomplished by supplying liquefied petroleum gas (GLP-Figure 1B) and ambient air from an air compressor (Figure 1C). The combustion process of the CNS was accompanied by thermocouples (Figure 1D) and flow transducers (Figure 1E) connected to a data acquisition system (FieldLogger-Figure 1F) for the analysis and control of temperature and LPG fluxes and (Unpublished data), directing the information to a notebook (Figure 1G).

The exhaust gases generated by the combustion of the CNS were directed by a chimney (Figure 1H) to a chamber containing a coupled cyclone (Aluminum Cyclone 37 mm SKC Figure 1I), in which it will select for the glass fiber filter (0, 8 μm porosity and 37 mm diameter), only particles below 4 μm ($PM_{4.0}$). The cyclone system was fed with a suction pump (AirChek XR5000 SKC Figure 1J) using a flow of 2.5 L/min, in order to allow the collection of only $PM_{4.0}$.

System of combustion and collection of $PM_{4.0}$ from the combustion of CNS

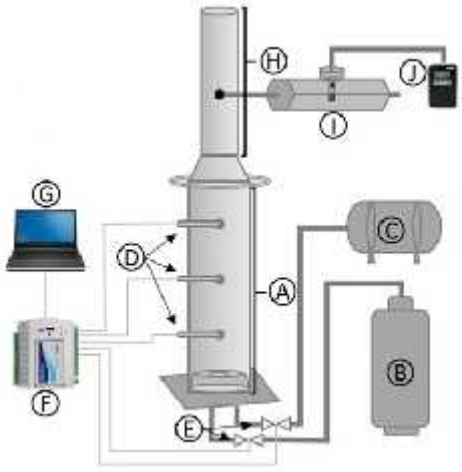

Figure 1 CNS combustion system for $PM_{4.0}$ collection. A - Biomass combustion reactor; B - LGP; C - Air compressor; D - Thermocouples; E - Flow transducers; F - Data acquisition system (Fieldlogger); G - Notebook, H - Chimney; I - Cyclone chamber for collecting filters with $PM_{4.0}$; J - Suction pump.

Preparation of the aqueous suspensions for intranasal instillation of $PM_{4.0}$

After collecting the $PM_{4.0}$ filters from the combustion exhaust gases of the CNS, the cleaned filters were placed in an oven at 50°C for 24 hours and weighed on analytical balance (FA-2104N). Then, the CNS combustion process was carried out to collect $PM_{4.0}$ (aluminum cyclone). Subsequently, the $PM_{4.0}$ containing filters were put back into the oven at 50 °C for 24 hours, and again weighed.

The filters were then packed in a Becker containing saline solution and sonicated for 8 hours in an ultrasonic sonicator (Q3350-QUIMIS®). After sonication, the filters were again placed in the oven at 50ºC for 24 hours and weighed. The efficiency of the extraction of the particles ($PM_{4.0}$) is calculated by the difference between the masses of the filters before and after the collection process (Maatz *et al*., 2009). The final particle: volume ratio was 1:1 (1 μg:1 μL), where we used 30 μg of $PM_{4.0}$ mass in 30 μL of the final solution.

Animals

This study followed all the rules in force for the maintenance of animal welfare. All the protocols used were previously approved by the ethics committee for the use of animals of the State University of Ceará.

C57black/6 mice with body mass of 25±5g and access to water and food *ad libitum*, were used in this study. The animals were exposed to cigarette smoke for 60 days. To study the effects of cigarrete smoke, mice were exposed to 12 commercial cigarettes per day for 60 days using an inhalation chamber (40 cm long, 30 cm wide, and 25 cm high). The animals were placed in the inhalation chamber, housed inside an exhaust hood. The cigarettes were coupled to a 60 mL plastic syringe, and the cigarette smoke was sucked into the syringe and then immediately expelled into the inhalation chamber. The animals were kept in this condition, with presence of cigarette smoke in this environment, for 6 min. Then the inhalation chamber cap was removed, and the exhaust fan connected to evacuate the smoke for 1 min. Exposure to cigarrete smoke was repeated four times (4 x 6 min) with a 1 min escape interval after each exposure. This procedure was repeated three times a day (8h am, 12h am and 4h pm) (Valença *et al*., 2008).

We used 16 animals randomly divided into three groups. In the first group (n=8), the animals were exposed for 60 days to ambient air, and subsequently received intranasal instillation of 30 µL of solution from filter sonication in clean glass fiber in saline solution (0.9% NaCl) (Air group). In the second group (n=4), the animals were submitted to the protocol of exposure to cigarette smoke for 60 days and received intranasal instillation of 30 µL of solution from sonication of the clean glass fiber filter (CS group). In the third group (n=4), the animals were submitted to the protocol of exposure to cigarette smoke for 60 days, and received intranasal instillation of 30 µg $PM_{4.0}$ from the CNS combustion exhaust gases diluted in 30 µL of saline solution (CS+PM group).

Intranasal instillation

Exposure of the animals to the solution containing $PM_{4.0}$ (CS+PM group) or saline solution (Air and CS groups) was performed via intranasal instillation 24 hours after the last exposure to cigarette smoke or ambiente air. Prior to intranasal instillation, the animals were sedated with sevofluorane (1 alveolar minimum concentration-AMC). The instillation causes a reflex of apnea followed by deep inspiration that leads the fluid into the lung. The animals received instillations containing 30 µg of $PM_{4.0}$ from the CNS combustion exhaust gases, diluted in 30 µL of saline solution (CS+PM group), or 30 µL of clean filter fiber filter sonication solution (Air and CS groups). The technique used was effective to avoid wastage of the material. All analyzes were performed 24 hours after instillation.

Experimental Protocol

24 h after intranasal instillation of saline solution (Air and CS groups) or particulate matter (CS+PM), the animals were anesthetized with sodium pentobarbital (50 mg/kg, i.p., Hypnol® 3%, Syntect, Brazil) and tracheotomized. The animals were intubated with a 18-gauge cannula (Eastern Medikit, Delhi, India) that was then connected to a computer-controlled ventilator for small animals (Scirec©-*flexVent*®, Montreal, QC, Canada). The animals were ventilated at baseline settings: respiratory frequency of 120 breaths/min, tidal volume of 10 mL/kg, limiting pressure of 30 cmH_2O, and positive end-expiratory pressure (PEEP) of 3 cmH_2O. Animals were then paralyzed with pancuronium bromide (0.5 mL/kg, i.p., Cristália, Lindoia, MG, Brazil). Initially we standardized the mechanical history of the respiratory system with two deep inflations (DI, 6-s long, peak pressure: 30 cmH_2O). Followed by 5 minutes of ventilation at baseline. Soon after, the impedance of the respiratory system (Z_{rs}) was measured with the forced oscillation technique (Hantos *et al*., 1992), 12 sequential 30 s sampling intervals, for a total of 6 minutes (Bates, 2009).

The experimental Z_{rs} was fitted to the constant phase model as previously described (Hirai *et al*., 1999):

$$Z_{rs} = R_N + I\,2\pi f\,i + \frac{G - Hi}{(2\pi f)^{\alpha}} \quad \text{Eq. (1)}$$

$$\alpha = \frac{2}{\pi}\tan^{-1}\frac{H}{G} \quad \text{Eq. (2)}$$

where R_N is the Newtonian resistance, which represents the central airways resistance, $i = \sqrt{-1}$, f is the frequency (Hz), I represents airway inertance, and G and H are respectively the dissipative and elastic properties of lung tissue (Hantos *et al*., 1992).

Thereafter, starting at the functional residual capacity (FRC) defined by the PEEP, the flexiVent delivered 7 inspiratory pressure steps for a total pressure of 30 cmH_2O, followed by 7 expiratory steps, pausing at each step for 1 s. At each step plateau pressure (P) was recorded and related to the total volume (V) delivered to produce a quasi-static PV (pressure-volume) curve. Static compliance (C_{ST}) was calculated as the slope of the curve (Salazar and Knowles, 1964). Two quasi-static PV curves were obtained to measure C_{ST}, an estimate of inspiratory capacity (IC), and PV loop area. Another forced oscillation technique ensued to determine respiratory system mechanics.

Statistical analysis

Results are presented as mean ± SD, where *n* represents the number of samples. Data normal distribution and homogeneities of variances were tested with Kolmogorov-Smirnov (with Lilliefors's correction) and Levene median tests, respectively. If both conditions were satisfied, Student's t-test was used. If any condition was refused, Mann-Whitney non-parametric test was used instead. A difference was considered significant if $p < 0.05$.

RESULTS

Our results concerning the analysis of respiratory system impedance (Z_{rs}), calculated from the forced oscillation technique, are presented in Figure 2 (A-C). Some comparisons between the variables related to airway resistance (R_N), tissue resistance (G) and tissue elastance (H) of Air, CS and CS+PM groups presented statistically significant differences.

Values of the variables of the constant phase model

The results concerning the analysis of the volume pressure curve are presented in Figure 3 (A-C). Some comparisons between the static complacency (C_{ST}), estimation of inspiratory capacity (IC) and PV loop area of the Air, CS and CS+PM groups, presented statistically significant differences.

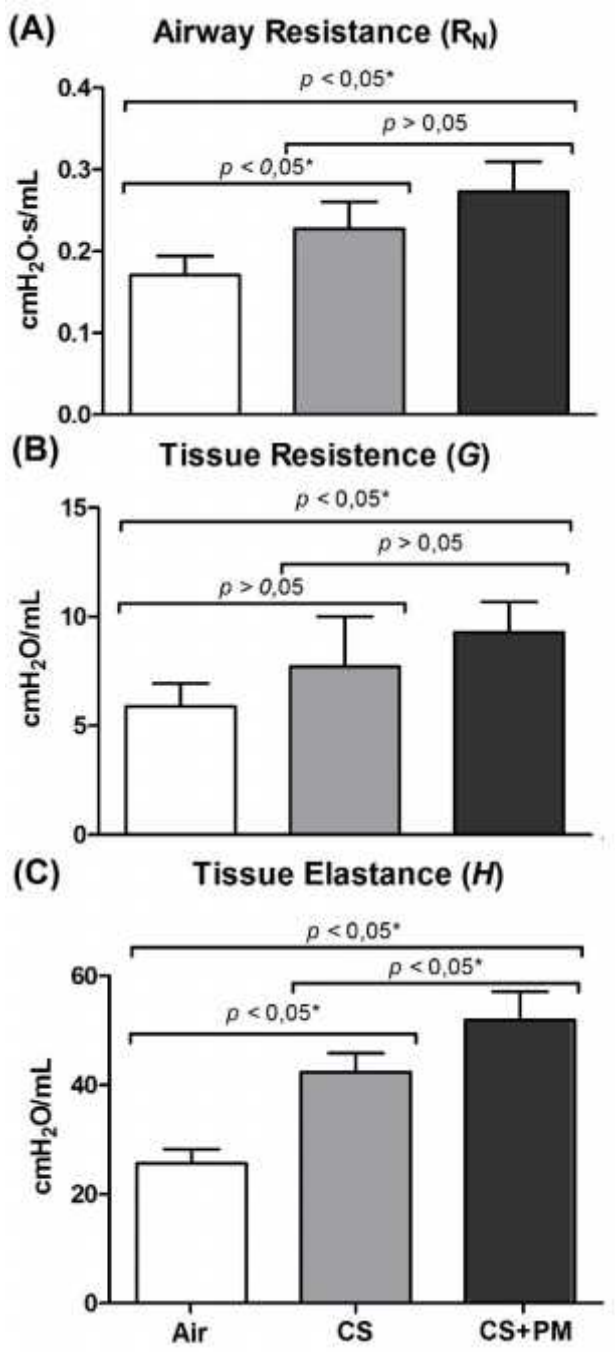

Figure 2 Values for airway resistance(R_N), tissue resistance (G) and tissue elastance (H), of the animals exposed to ambient air, Air group (white column), and to the cigarrete smoke, CS group (gray column), and to cigarrete smoke and $PM_{4.0}$ from the combustion of cashew nuts shells, CS+PM group (black column). Values are represented by mean±standard deviation of the mean. * Represents statistically significant values in comparison to the control group (p <0,05).

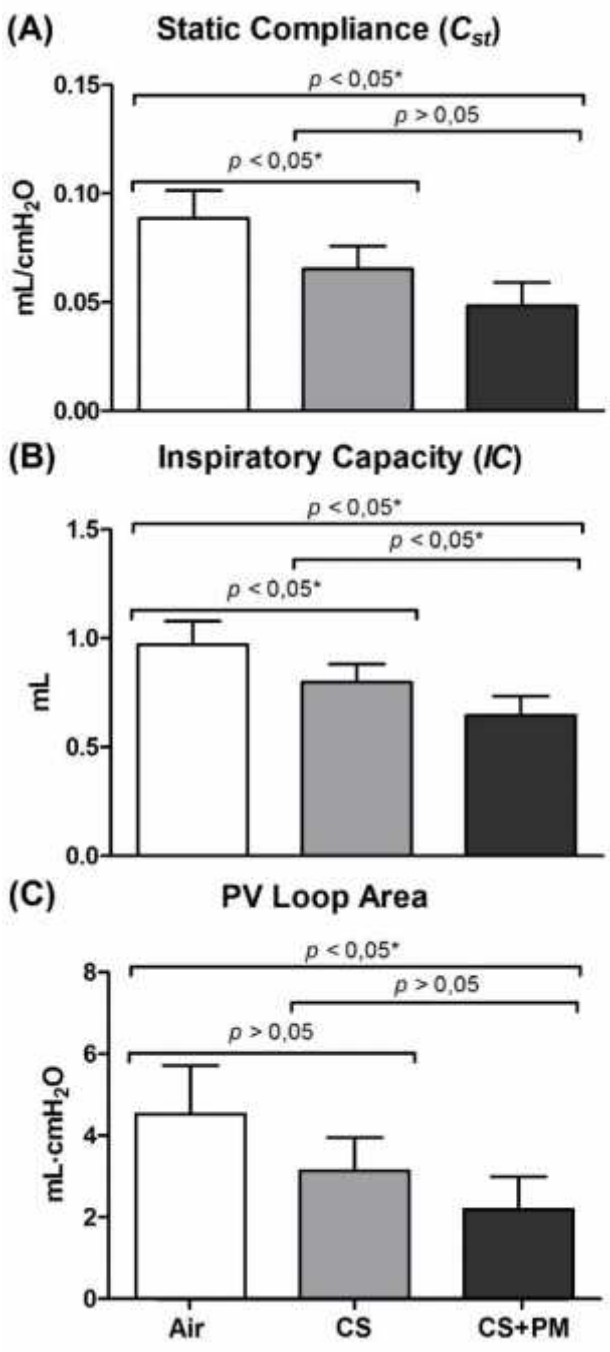

Figure 3 Values referring to the variables collected from the PV curve. The static complacency (C_{ST}), estimation of inspiratory capacity (IC) and PV loop area, of the animals exposed to ambient air, Air group (white column), and to the cigarrete smoke, CS group (gray column), and to cigarrete smoke and $PM_{4.0}$ from the combustion of cashew nuts shells, CS+PM group (black column). Values are represented by mean±standard deviation of the mean. * Represents statistically significant values in comparison to the control group (p <0,05).

Values of the variables obtained through the PV curve

The absolute values, referring to the variables calculated from the analysis of the respiratory system impedance (Z_{rs}) and the PV curve of the groups exposed to the anbiemte air (Air group), the cigarette smoke (CS group) and the cigarette smoke associated to the $PM_{4.0}$ from combustion exhaust gases from the CNS (CS+PM group) are shown in Table 1.

DISCUSSION

Several studies have demonstrated the deleterious effects of PM from burning biomass and cigarette smoke to health (Mainali *et al*., 2015; Lee *et al*., 2015; Nakamura *et al*., 2015). However, few investigated the association of these two factors. It is known that the environment in which the individual is inserted is relevant with regard to worsening of a previous lung disease (Hansel *et al*., 2013).

Differences between lung function parameters

Table 1 Values are mean ± SD of animals exposed to ambient air (n=8, Air group), cigarrete smoke (n=4, CS group), and cigarrete smoke and $PM_{4.0}$ from the combustion of cashew nuts shells (n=4, CS+PM group). *p<0.05, statistically significant difference

Measure		Group	Value	*P* value Student´s test t	
forced oscillation technique	**Airway resistance (R_N)**	**Air**	**0.170 ± 0.023**	**Air x CS**	**0.0056**
	($cmH_2O.s/mL$)	CS	0.227 ± 0.032	Air x CS+PM	0.0001
		CS+PM	0.272 ± 0.036	CS x CS+PM	0.1151
	Tissue damping (G)	Air	5.89 ± 1.03	Air x CS	0.0818
	(cmH_2O/mL)	CS	7.70 ± 2.30	Air x CS+PM	0.0008
		CS+PM	9.27 ± 1.40	CS x CS+PM	0.2900
	Tissue elasticity (H)	Air	25.60 ± 2.67	Air x CS	0.0001
	(cmH_2O/mL)	CS	42.37 ± 3.44	Air x PM	0.0001
		CS+PM	51.93 ± 5.17	CS x CS+PM	0.0218
PV-curve	C_{ST}	Air	0.088 ± 0.012	Air x CS	0.0109
	(mL/cmH_2O)	CS	0.065 ± 0.010	Air x CS+PM	0.0003
		CS+PM	0.048 ± 0.010	CS x CS+PM	0.0635
	CI	Air	0.970 ± 0.107	Air x CS	0.0188
	(mL)	CS	0.797 ± 0.082	Air x CS+PM	0.0004
		CS+PM	0.647 ± 0.086	CS x CS+PM	0.0454
	Hysteresis	Air	4.53 ± 1.18	Air x CS	0.0632
	(area)	CS	3.13 ± 0.82	Air x CS+PM	0.0055
		CS+PM	2.19 ± 0.79	CS x CS+PM	0.1510

In general, unless the concentration is above that suggested by regulators for the emission of environmental pollutants, acute effects secondary to PM exposure are found mainly in susceptible groups with pre-existing lung disease (Donaldson and Macnee, 2001).

Chronic obstructive pulmonary disease (COPD) is one of the major lung pathologies developed through the use of cigarettes. The characteristics of human COPD can be induced in mice by the administration of proteases, particles and exposure to cigarette smoke. An ideal pathological condition would allow to reproduce all the different anatomical lesions associated with the disease. The murine model used in this work presents anatomical characteristics such as the concentration of mucous glands in the proximal trachea, which make difficult the reproduction of chronic bronchitis, and although these animals do not present respiratory bronchioles, initial focus of destruction in emphysema, prolonged exposure to cigarette smoke Produces lesions compatible with the mild form of centrilobular emphysema observed in humans (Wright and Churg *et al*., 2008).

Previous studies (Valença *et al*., 2004; Bezerra *et al*., 2011; Lanzetti*et al*., 2011; Valença *et al*., 2011; Pires *et al*., 2011;Nesi*et al*., 2016), using the same protocol and time of exposure to cigarette smoke, have observed the development of pulmonary emphysema capable of promoting morphological and morphometric alterations in the lungs, evidenced in the alveolar diameter analysis, a reliable parameter for the characterization of pulmonary emphysema (COPD) since it is associated with tissue remodeling, which results in the destruction of the extracellular matrix (Groneberg and Chung, 2004).

In relation to the variables related to respiratory system mechanics analyzed in this study, Newtonian resistance (R_N) (Figure 2-A) has been used as a good estimate of total airway resistance (Bates, 2009).

Thus, we can assume that statistically higher values in the CS and CS+PM groups compared to the Air group may represent a greater narrowing or increase in the airway smooth muscle stiffness (Bates, 2009).

The significant increase inR_N, may be provided by the inhalation of the PM present in the exhaust fumes from the CNS combustion. These particles, when inhaled induce the expression of pro-inflammatory mediators and Ca^{2+} dependent intracellular signaling pathways (Ermak and Davies, 2002). The biological functions of the Ca^{2+} ion in the lungs play a role in the regulation of various functions, such as mucus secretion, surfactant secretion and ciliary agitation frequency (Conway *et al*., 2003). In addition, in vivo studies report increased levels of SOD and CAT after inhalation of PM (Gurgueira *et al*., 2002; Pereira *et al*., 2007), which may indicate oxidative stress in the airway epithelium. As a result, several proinflammatory cytokines are expressed and the inflammation develops, with consequent airway hyperreactivity (Aanseth *et al*., 2005).

Changes in tissue resistance variables (G) and tissue elastance (H) (Figure 2 B-C), may be related to the intrinsic properties of the tissue, causing alterations in the rheology of the pulmonary tissue, due to alterations of the extra cellular matrix, tissue remodeling and of other constituents (Bates, 2009). Narrowing of the airways exerts an influence on H. This results in a distortion of the pulmonary parenchyma with closure of small airways, constituting an effectively smaller lung with proportionally greater tissue elastance. Another hypothesis suggests a modification in the properties of alveolar surfactant and/or loss of lung capacity due to the presence of liquid on the alveolar surface (Bates, 2009).

In addition, exposure to environmental pollutants causes accumulation of fluid in the interstitium and air spaces of the lung, leading to hypoxemia, decreased lung compliance and increased respiratory work (Rocco *et al*., 2003). The organic compounds from PM can interact with components of the surfactant, impairing the secretion of the same or modifying its

composition, increasing the surface tension, thus generating areas of collapse with consequent increase of H. In this sense, our results demonstrated a statistically increase Significant in the H group of the CS+PM group when compared to the CS and Air groups, suggesting that $PM_{4.0}$ from CNS was able to promote a worsening of this parameter in animals with pre-established pulmonary pathology.

Regarding the parameters obtained through the realization of the volume pressure curve (Figure 3), we observed significant alterations in some parameters of static complacency (C_{ST}), estimation of inspiratory capacity (IC) and PV loop area of the animals CS and CS+PM groups in relation to the Air group. The decrease in the C_{ST} parameter may be a reflection of the already discussed increase in tissue elastance (H).In experimental studies, inhalation of particles from combustion processes led to decreased lung compliance and an inflammatory response characterized by influx of polymorphonuclear cells and release of cytokines (Laks *et al*., 2008; Mazzoli-Rocha *et al*., 2008).

The decrease in the inspiratory capacity (IC), indicating stiffening of the lung tissue, observed by the increase in H, assuming that the animals of the CS and CS+PM groups presented a greater effort in the inspiration. On the other hand, the increase of PV loop area can be explained by possible alterations in the distribution of surfactant on the alveolar surface, associated with the presence of alveolar edema in the lung of the CS+PM group. The PV loop area (Hysteresis) is determined by four processes: recruitment/de-recruitment, surface tension, stress relaxation, and gas absorption during PV assays.

CONCLUSION

It is important to use residual biomass as an alternative to fossil fuels. However, one should have greater control over the use of CNS as biofuel in industries and artisan processing arrangements of cashew nuts. Exposure to $PM_{4.0}$ generated in this process, since it can generate changes in the respiratory system of animals, these results can be extrapolated to the population that is constantly exposed to these pollutants, especially those with pre-existing lung diseases, such as COPD caused by snuffing the cigarette smoke. The characterization of the human health effects of the combustion of residual biomass resumes the concern with biomonitoring measures and public policies to regulate its emission levels, or alternatives to avoid it. Further investigation is necessary in order to know safe parameters for individuals who are continually exposed to $PM_{4.0}$ produced from CNS combustion.

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