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Impact of asthma, exposure period and filters on human responses during exposures to ozone and its initiated chemistry products

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Abstract

The impact of asthma, exposure period and filter condition downstream of the mixing box of air-conditioning system on building occupants' perceptual response, work performance and salivary α-amylase secretion during exposures to ozone and its initiated chemistry products is studied. The experiments were conducted in a Field Environmental Chamber "FEC" (240 m^3) simulating an office environment. Experiments were conducted during periods when the air handling system operated with new or used pleated panel filters at constant recirculation (7 h^{-1}) and ventilation (1 h^{-1}) rates. Average ozone and secondary organic aerosols (ozone initiated chemistry products) measured during non-asthmatic and asthmatic subjects' 3-h exposures in the FEC were in the ranges ~20-37 ppb and ~1.6-3 μg/m^3, respectively. Asthmatic subjects' perceived odour intensity and sensory (eye, nose and throat) irritation ratings were generally lower than those of non-asthmatic subjects, possibly explaining why asthmatic subjects accept perceived air quality more than non-asthmatic subjects. However, asthmatic subjects' perceived physiological-like symptom ratings (flu, chest tightness and headache) and concentrations of secreted salivary α-amylase were generally higher than those of non-asthmatic subjects. Asthmatic subjects had significantly lower accuracy than non-asthmatic subjects in a task that required higher concentration though they had higher work speed. Filter condition did not make any significant difference for subjects' responses.

Keywords: Ozone initiated chemistry; Asthma; Perceptual responses; Work performance; Human-environment interactions; Indoor air pollution

Practical implications

There has been an increase in the prevalence of asthma in many populations. However, human-indoor air pollution interactions studies have focused mainly on non-asthmatic subjects. This study shows how non-asthmatic and asthmatic subjects' perceptual responses, work performance and physiological (salivary α-amylase) condition could differ during commonly experienced exposures to low and realistic concentrations of indoor air pollutants, represented by ozone and its initiated chemistry products in this study, for extended periods in air-conditioned environments having air filters in the air system to reduce exposure. Better understanding of this issue may provide clues to answer this important question: "Toward whom should indoor air quality controls be sympathetic: asthmatics or non-asthmatics?"

1. Introduction

Indoor air pollution is regarded to be among the top five environmental risks to public health (US EPA, 1993). Ozone, an air pollutant commonly present in indoor environments, is one contaminant of concern. Association between ozone exposures and human morbidity and mortality is well documented in the literature (The Royal Society, 2008). Ozone exposures in indoor environments are major contributors to human morbidity and mortality (Chen et al. 2012). Additionally, ozone exposures can degrade human work performance (Folinsbee et al. 1977). Ozone is a powerful oxidizing agent. Numerous ozone-reactive chemicals found in indoor environments produce major byproducts when reacted with ozone, which are potentially detrimental to health (Weschler, 2006).

According to Wolkoff et al. (2006, 2013), the stable products of ozone-terpene chemistry are more important to human perceptual responses than is ozone itself. Increased odour intensity, sensory (nose, eye, and throat) irritation and physiological like symptoms — flu like, chest tightness and headache — are examples of perceptual responses reported by human subjects when exposed to indoor air that contains both ozone and its reactive organic compounds (Kleno and Wolkoff, 2004; Nojgaard et al. 2004; Strom-Tejsen et al. 2008). Researchers have demonstrated that products of ozone-terpene chemistry are more irritating to the upper airways than are terpenes or ozone alone (Clausen et al. 2001; Rohr et al. 2002; Wilkins et al. 2003; Wolkoff et al. 1999, 2000, 2008).

Current understanding about the association between indoor ozone and its initiated chemistry products and human health, thus far has focused on healthy human subjects. Human populations with impaired health conditions, e.g. asthmatics, have been neglected. There is a knowledge gap regarding how asthmatic conditions could alter human responses during exposures to ozone and its initiated chemistry products. This gap in knowledge needs to be addressed. Bridging this gap in knowledge is important because of increasing asthma prevalence, which of course means increased proportions of asthmatic individuals living, studying and working in indoor environments (Masoli et al. 2004). This study focuses on subjects with mild asthma. They constitute the higher proportion of asthmatics occupying indoor environments (Asthma Foundation of South Australia, 2008).

In a realistic scenario, building occupants spend considerable time indoors. Studies addressing human interactions with ozone and its initiated chemistry products have usually focused on short-term effects associated with single exposures. The impact of extended period of exposures to ozone and its initiated chemistry products on human responses has been neglected. This study attempts to bridge this gap in knowledge.

Also, it is important to understand how filters (new and used), which are meant to reduce indoor air pollution, may influence human responses during exposures to ozone and the products of ozone initiated chemistry. Very few comparative studies have been done to understand how new and used filters could influence human responses during exposures to ozone and its initiated chemistry products (Beko et al. 2008). There are no studies that address this issue in air-conditioning systems that are designed and operated to recirculate indoor air at a high rate to conserve energy and avoid moisture problem — a common practice in air-conditioned buildings in tropical regions.

The primary objective of this study is to explore the effect of asthma status, exposure period and filter condition on human responses during exposure to commonly experienced concentrations of indoor air pollutants, represented here by ozone and its initiated chemistry products. Human responses examined in this study include perceptual responses — perceived odour intensity, sensory (ear, nose and throat) irritation, physiological symptoms, work performance and salivary α-amylase concentrations.

2. Materials and Methods

This section provides information on the recruitment of subjects, on ethical review and approval, and on experimental protocols.

Recruitment of subjects and ethical approval

Subjects were recruited according to health criteria relevant to the studies and participated on a paid voluntary basis. An Institutional Review Board in Singapore approved the use of human subjects for the present study (IRB approval number: NUS-384). Non-asthmatic subjects, between 18 and 25 years of age, and asthmatic subjects, between 20 and 26 years of age were recruited from the university community. Medical doctor diagnosed

mild asthmatic subjects were used in this study. The decision on asthma condition is based on Global Initiative for Asthma (GINA) classification of asthma (Yawn, 2008). Before commencing the experiments, subjects were given training sessions to familiarize them to the test procedures to be taken. The subjects wore typical clothing attire for office workers in the tropics. Subjects were blind to the experimental conditions.

Experimental protocol

Studies were conducted in a large field environmental chamber (FEC) simulating an office environment (11.5 × 7.9 × 2.6 m; 240 m^3). The FEC has polymeric tile flooring, sealed windows, acoustic ceiling tiles, and contains typical office furniture. The air-handling unit (AHU) that serves this space is located in a room above the FEC. Outside air is provided via an internal airshaft drawing air from the roof of the building. The outside air is psychrometrically mixed with the return air, filtered and conditioned by the cooling coil before being distributed to the FEC via ceiling-mounted diffusers. Return air is drawn from the FEC by way of grilles integrated into the suspended ceiling. The approximate volume of the recirculation loop is 30 m^3. The study was conducted at an outdoor-air ventilation rate of 1 h^{-1} and a recirculation rate of 7 h^{-1}. Details on measurement protocols for ventilation and recirculation rates can be found in Fadeyi (2009).

For these experiments, the filter section in the AHU contained either new or used filters (10 cm deep AmAir 1300 extended-surface pleated panel filters consisting of synthetic, electrostatically charged media) with an efficiency rating of MERV 13 as defined in ASHRAE 52.2. The new filters were deployed as received from the manufacturer. The used filters were taken from the filter bank of an AHU, operating in recirculation mode, which serviced an office building at the National University of Singapore. Prior to their removal, they had been in service for 10 months (equivalent to about 6720 h). The new and used filters

were identical, aside from time in service, and came from the same supplier. For the pollutants of interest, single-pass efficiencies of new and used filters employed in this study were similar as reported in Fadeyi et al. (2009).

Two rounds of experiments were conducted for each of non-asthmatic and asthmatic subjects. This is due to limitation posed by FEC capacity. Studies for non-asthmatic and asthmatic subjects were conducted separately because recruitment of asthmatic subjects took longer. The first round of experiments involving non-asthmatic subjects were conducted on different days in the following order of filter placed in the AHU: new (1^{st} day) – used (2^{nd} day) – new (3^{rd} day) filter. The second round of experiments involving non-asthmatic subjects was conducted on different days and in this order: used (1^{st} day) – new (2^{nd} day) – used (3^{rd} day) filter. The order of subjects' exposure to filter condition was balanced to reduce errors that may arise from order-of-exposure bias. Different sets of subjects were used for the first and second rounds of experiments. Eighteen and 20 subjects participated in the first and second round of experiments, respectively. In all, 38 non-asthmatic subjects (14 and 24 male and female, respectively) participated in the study. The ratio of male to female subjects was 0.6: 1.

Asthmatic subjects' experiments followed the same of order as non-asthmatic experiments, i.e. new-used-new filter for the first round of experiments, and used-new-used filter for the second round of experiments. Fifteen and 18 subjects participated in the first and second rounds of experiments, respectively. In all, 33 asthmatic subjects (15 and 18 male and female, respectively) participated in the study. The ratio of male to female subjects is 0.8: 1. Thus, the ratios of male to female subjects in non-asthmatic and asthmatic subjects were similar.

In all the twelve experiments, subjects were exposed to ozone, limonene and products of their chemistry at realistic concentrations for a continuous 3-hour working session in the FEC – typical office hours (9 am to 12 pm) before lunch break. To modestly associate subjects' responses to ozone and its initiated chemistry products, ozone and limonene were the only pollutants deliberately injected into the FEC. Furthermore, the FEC was fairly well controlled to reduce the influence of outdoor air pollutants on indoor air pollutants. An activated carbon filter was placed at the outdoor air intake as a necessary precaution needed to reduce the influence of outdoor ozone and organics on the experiments. Prior to subjects entering the FEC, ozone and limonene was released within the FEC for 45 minutes. Figure 1 shows the timeline for the experiments.

Ozone was generated at delivery rate of 135 mg h^{-1} at the outdoor air duct to simulate outdoor to indoor transport of ozone via supply air. At this rate, the resulting ozone concentrations in the chamber were in the range often experienced in tropical office environments (Zuraimi et al. 2007). Average ozone measured during subjects' 3-h exposures in the FEC were in the range ~20-37 ppb. Limonene (98% purity) was emitted in the FEC at a constant rate of ~200 mg h^{-1} using modified emission vials; the resulting chamber steady state concentrations (35-36 ppb) with subjects present were lower than what building occupants would be exposed to following floor cleaning with a limonene-scented agent. Limonene was generated directly in the FEC to simulate a common scenario in which highly ozone-reactive terpenes are emitted indoors. Detailed information on ozone and limonene generation, and how ozone and its initiated chemistry products – secondary organic aerosols (SOA) – were measured can be found in Fadeyi et al. (2013). Temperature (^{o}C) and relative humidity (RH %) were fairly well controlled at 22.9±0.7 and 58.3±1.4, respectively, for the twelve experiments. Questionnaire elicitations regarding the intensity of perceptions of the

indoor environment were carried out at various times. Subjects filled the questionnaire immediately (~ 5 mins) after entering the FEC (P1), midway through the experiment, at ~90 minutes after entry (P2), and at ~5mins before leaving the FEC (P3). Subjects assessed the acceptability of air in the FEC using the continuous acceptability scale (Wargocki et al, 2000), which ranges from 'Clearly unacceptable' (-1) to 'Clearly acceptable' (+1). There is a break in the scale in the middle to clearly distinguish between acceptable and unacceptable air quality.

Odour intensity was evaluated on the continuous intensity scale, which ranges from "No odour" (0) to "Overpowering odour" (100). Other points marked on the scale are "Slight odour" (20), "Moderate odour" (40), "Strong odour" (60) and "Very strong odour" (80). Eyes, nose and throat irritation were evaluated on the continuous irritation scale which ranges from "No irritation" (0) to "Overpowering irritation" (100). Other points marked on the scale are "Slight irritation" (20), "Moderate irritation" (40), "Strong irritation" (60) and "Very strong irritation" (80). Perceived flu, chest pain and headache symptoms resulting from exposure to ozone and its initiated chemistry pollutants were evaluated using visual analogue scales with labelled endpoints: left end-point = 0, right end-point = 100 (Wargocki et al. 2000). Percentage of subjects dissatisfied with perceived air quality was calculated from subjects' acceptability rating of perceived air quality using the equation developed by Gunnarsen and Fanger (1992) (Equation 1).

$$PD\ (\%) = [\{EXP\ (-0.18 + (-5.28 \times ACC_{mean}))\} / \{1 + \{EXP\ (-0.18 + (-5.28 \times ACC_{mean}))\}\}] \times 100 \qquad (1)$$

Here, PD = percentage of subjects dissatisfied; ACC_{mean}= mean of acceptability rating of perceived air quality.

A passive drool salivary sampling procedure was applied for saliva collection at initial occupancy and after 3-h exposures. Subjects expelled saliva into a labeled sampling tube. Kinetic immunoassay measurement was employed to determine amount of subjects' salivary α-amylase (a stress indicator) secreted during their 3-h exposures to ozone and its chemistry products, taking into account each subject's health status and also the applied filter condition.

Additionally, simulated office tests were administered to explore how asthma and filter condition might influence human work performance during exposure to ozone and its initiated chemistry products. Subjects were required to complete concentration endurance (task 1), numerical reasoning (task 2), and arousal test (task 3) within a stipulated period. Details about these tasks can be found in Willem (2006).

3. Results

This section provides information on (i) ozone and SOA concentrations during occupied periods, (ii) perceptual responses and α-amylase concentrations, and (iii) work performance.

Ozone and SOA concentrations during occupied period

Figure 2 shows ozone and SOA concentrations as functions of time for the different experimental conditions. Non-asthmatic and asthmatic subjects were exposed to nearly the same amount of ozone and SOA during their occupancy of the FEC. Similarly, subjects were exposed to approximately the same amount of ozone and SOA with either new or used filters placed in the AHU.

Perceptual responses and α-amylase concentrations

Figure 3 shows comparisons between non-asthmatic and asthmatic subjects' perceptual responses, irrespective of filter condition. There was no significant difference (p>0.05)

between asthmatics and non-asthmatic subjects' perceived odour intensity and eye irritation. However, asthmatic subjects' perceived nose and throat irritation were significantly less (p<0.0001) than those of non-asthmatic subjects. The asthmatic subjects' lower perceived sensory irritations may have caused them to be more satisfied with their perceived air quality than non-asthmatic subjects. Details of the p-values of comparisons between subjects' perceptual responses as a function of their health status, exposure period and filter condition can be found in the supplementary material (Table S1).

It is evident in Figure 4 that asthmatic subjects' percentages of dissatisfaction data were in the lower region of that of non-asthmatic subjects. Furthermore, data presented in Table 1 suggest that non-asthmatic subjects' perceived odour intensity and sensory irritation can modestly better be used to predict acceptability of perceived air quality than would be the case for asthmatic subjects. This observation is more evident at P1 (5 minutes after entering the chamber) and P2 (90 minutes after entering the chamber). Details of the correlation analysis can be found in the supplementary material (Figures S1 to S4).

Asthmatic subjects' lower perceived sensory irritation does not necessarily mean that they were less affected physiologically. As evident in Figure 3, asthmatic subjects were more significantly (p<0.05) affected physiologically than non-asthmatic subjects, judging from asthmatic subjects' elevated perceived physiological related symptoms (flu, chest tightness, and headache). Asthmatic subjects' secreted α-amylase (stress indicator) concentration (84.2 unit/ml) also was more than that of non-asthmatic subjects (68.8 unit/ml), albeit the difference was not significant (p>0.05) (see Figure 5), perhaps due to the use of mild asthmatic subjects for these experiments. Figure 5 also shows that subjects' exposure to low and realistic concentrations of ozone and its initiated chemistry products is associated with a significant increase (p<0.0001) in their α-amylase secretion (stress indicator). Further

investigations are required to test whether the association is a result of causality, i.e. that the exposure to ozone and its initiated chemistry products is the reason for an increase in α-amylase secretion.

Figure 6 shows subjects' perceptual responses with either a new or a used filter placed in the AHU. There was no significant difference (p>0.05) between new and used filters. As evident in Figure 7, there was also no significant difference (p>0.05) between new and used filter impacts on the amounts of α-amylase secreted.

Figure 8 shows subjects' perceptual responses for each of the three periods of assessment, irrespective of subjects' health status and filter condition in the AHU. Neither an increase nor a decreasing trend in subjects' perceptual responses over time was evident in perceptual responses for nose and throat irritation and flu and chest tightness like symptoms. It was evident in the case of odour intensity (decrease), eye irritation (increase), and headache like symptom (increase) only for perceptions during initial period (P1) as compared with the time midway through the experiment (P2).

Work performance

As is evident in Figure 9, the time it takes asthmatic subjects to complete their concentration task was significantly (p<0.05) less than that of non-asthmatic subjects. However, asthmatic subjects' accuracy in the concentration task was very significantly lower (p<0.0001) than that of non-asthmatic subjects. Figure 10 shows that the change in filter condition did not make any difference in subjects' performance for all the tasks performed.

4. Discussion

Despite observed increasing trends in the measured indoor air pollutant concentrations during subjects' exposures, perceived odour intensity ratings decreased significantly with

increased exposure time. This is not surprising. According to Ekman et al. (1967), perceptual odour intensity decreases exponentially with time of chemical stimulation of olfactory organ through a process known as adaptation. Subjects' adaptation caused the observed difference in subjects' perceived air quality during initial exposure (P1) and extended period of occupancy (either P2 or P3) to be significant, whereas, there was no significant difference between P2 and P3.

Human irritation sensations can result from a range of reactions of the subjects' nose, eyes, or throat with air pollutants - dominated by ozone and its initiated chemistry products in this study (Wolkoff et al. 2012). This study suggests that nose irritation is the most sensitive, among sensory organs, to period of exposure. In terms of perceived physiological like symptoms, headache was the most sensitive to period of exposure. The series of work tasks performed by the subjects during the exposure period may have contributed to the perceived headache like symptoms shown in Figure 8.

It is not clear why asthmatic subjects' perceived sensory irritation ratings were lower than those of non-asthmatic subjects. There is need for further investigation of this observation. However, asthmatics' relatively damaged or denudated epithelial layer (Oortgiesen et al. 1998) may provide an explanation to the asthmatic subjects' higher perceived physiological and measured physiological conditions. The epithelial layer is a barrier protecting the C-fibres, a nerve carrying messages to the central nervous system. The C-fibres are believed to be responsible for mediating local axon reflexes, the release of neuropeptides and neurogenic inflammation (Belvisi, 2003). Asthmatics subjects' relatively damaged or denudated epithelial barrier will enhance direct contact of inhaled indoor air pollutants with the C-fibres more easily than would be the case for non-asthmatics' relatively healthy epithelial layer. According to Belvisi (2003), stimulation of C-fibres causes an

increase of neuropeptides. Tachykinins substance P (SP) and neurokinin A (NKA) are examples of neuropeptides believed to be released when C-fibre sensory nerves are stimulated by inhaled air pollutants. The released neuropeptides are believed to be responsible for various inflammatory diseases because they are strong activators of airway smooth-muscle contraction, vasodilatation, bronchial edema and mucus hypersecretion. Neuropeptides released from C-fibres have been proposed to be important in the pathology of diseases such as asthma (Barnes 1995, 1996; Karlsson 1993). Inflammation effects and pulmonary defense reflex responses caused by stimulation of C-fibres can lead to chest-tightness, cough and headache (Waeber and Moskowitz, 2005).

The stimulation of C-fibers can also cause stress in human (Koskela, 2007). Stress will cause an increase in salivary α-amylase secretion. While evidence of a significant increase in the subjects' stress level, as indicated by α-amylase concentrations (Figures 5 and 7), can be associated with inhaled ozone and its initiated chemistry products (Paz, 1997), work tasks performed by the subjects may also have contributed to their stress level. The non-significant difference between the impacts of the filter condition on all the human responses examined can be explained by the similarity on the impact of new and used filters on ozone and SOA concentrations.

An implication of the findings from this study is that asthmatic subjects' relatively lower perceived sensory irritations and higher acceptability feedback ratings of perceived air quality, despite having higher perceived physiological symptoms, will delay relaying actual adverse indoor air pollution conditions to control systems in time for prompt mitigation strategies. By the time asthmatic subjects perceive air pollution, their physiological health condition may be adversely affected. Such delay may increase asthmatics' health risk, relative to the risk for non-asthmatic subjects. Thus, indoor air quality controls that are

sympathetic towards non-asthmatics, instead of asthmatics, may be appropriate in creating healthy indoor air quality condition for both asthmatics and non-asthmatics.

According to Ho et al. (2014), air pollution can increase human arousal. Association between increased α-amylase secretion and human arousal has been reported in the literature (Sapolsky 1992, 2003). Relatively higher stress level observed in asthmatic subjects, as indicated by the measured α-amylase concentrations, may have caused them to be more aroused. The higher arousal may have contributed to asthmatic subjects completing more tasks in shorter time than non-asthmatic subjects (see Figure 9). The asthmatic subjects' higher speed of task completion may have contributed to their reduced accuracy of tasks performed, especially for tasks requiring a high level of concentration. Asthmatic subjects' relative lack of concentration can also be attributed to their higher manifestation of physiological like symptoms (flu, chest tightness, and headache). There is considerable evidence linking physiological like symptoms with poor work performance (Nishihara et al. 2014; Wargocki et al. 2007; Tham and Willem 2004).

The authors acknowledge that there may be other explanations to the observed differences between asthmatic and non-asthmatic subjects' work performance speed and accuracy. The differences in performance between asthmatics and non-asthmatics may not necessary be a consequence of the exposures in this study or the asthmatic condition. Further investigations are necessary for better understanding of this issue.

5. Conclusion

The following are the main conclusions from this study:

- The asthmatic subjects' perceived sensory irritations were generally lower than that of non-asthmatic subjects. However, asthmatic subjects perceived physiological like

symptoms were significantly higher than those of non-asthmatic subjects, irrespective of filter conditions and exposure periods.

- Asthmatic subjects' percentage dissatisfied data were in the lower region of that of non-asthmatic subjects.
- Non-asthmatic subjects' perceived odour intensity and sensory irritations can be used to predict acceptability of their perceived air quality modestly better than for asthmatic subjects.
- The amount of α-amylase secreted in asthmatic subjects was higher than that of non-asthmatic subjects. However, the difference was not statistically significant, irrespective of filter condition.
- Asthmatic subjects' performance in the concentration task was very significantly lower than that of non-asthmatic subjects during exposure to ozone and its initiated chemistry products, irrespective of filter condition.
- New versus used filters has no significant impacts on the human responses examined.
- The effect of exposure period was evident in the case of subjects' perceived odour intensity (decreased trend), eye irritation (increased trend) and headache like symptoms (increased trend).
- Association between 3-h exposures to low and realistic concentrations of ozone and its initiated chemistry products and significant increase in subjects' α-amylase concentration was observed. Further investigations providing evidence to test casualty are encouraged.

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Table 1. Correlations (r)* of non-asthmatic and asthmatic subjects' perceived odour intensity and sensory (eye, nose, and throat) irritations ratings with their perceived air quality acceptability ratings

	Exposure period	Non-asthmatic	Asthmatic
Odour Intensity	P1	0.71	0.51
	P2	0.62	0.63
	P3	0.61	0.48
Eye Irritation	P1	0.51	0.39
	P2	0.53	0.50
	P3	0.59	0.63
Nose Irritation	P1	0.67	0.10
	P2	0.63	0.52
	P3	0.67	0.56
Throat Irritation	P1	0.38	0.35
	P2	0.49	0.37
	P3	0.62	0.38

*Correlations (r) was computed using each of the non-asthmatic and asthmatic subjects' perceived odour intensity, sensory (eye, nose, and throat) irritations and perceived air quality acceptability ratings (see Figures S1-S4 for details)

Figures Captions

Figure 1. Timeline for experiments with chamber occupied by non-asthmatic and asthmatic subjects. Note: P1- Initial exposure assessment; P2- Exposure assessment: mid-way through the experiment; P3- Exposure assessment just before subject left the chamber

Figure 2. Ozone and SOA concentrations as functions of time for the different experimental conditions. Note: Each data point is an average of data from six experiments.

Figure 3. Comparisons between non-asthmatic and asthmatic subjects' perceptual responses, irrespective of filter condition. Note: *$p<0.0001$; **$p<0.05$; ***$p>0.05$; OI= odour intensity, EI=Eye irritation, NI=Nose irritation, TI=Throat irritation; CT=Chest tightness; PD= percentage of dissatisfaction. Each perceptual response data is an average of data from of 3 perceptual (P1, P2 and P3) responses

Figure 4. Correlations of non-asthmatic and asthmatic subjects' percentage of dissatisfaction data with their acceptability of air quality data.

Figure 5. Non-asthmatic and asthmatic subjects' α-amylase concentrations before and after 3-h exposures to ozone and its initiated chemistry products. Note: Comparison of non-asthmatics versus asthmatics: $p>0.05$.

Figure 6. Subjects' perceptual responses with either new or used filter placed in the AHU. Note: *$p<0.0001$; **$p<0.05$; ***$p>0.05$; OI= odour intensity, EI=Eye irritation, NI=Nose irritation, TI=Throat irritation; CT=Chest tightness; PD= percentage of dissatisfaction. Each perceptual response data is an average of data from of 3 perceptual (P1, P2 and P3) responses

Figure 7. Subjects' α-amylase concentrations before and after 3-h exposures to ozone and its initiated chemistry products when new or used filter was placed in the AHU. Note: Comparison of New versus Used filter: $p>0.05$

Figure 8. Subjects' perceptual responses for each of the three periods of assessments, irrespective of subjects' health status and filter condition in the AHU. Note: *p<0.0001; **p<0.05; ***p>0.05; OI= odour intensity, EI=Eye irritation, NI=Nose irritation, TI=Throat irritation; CT=Chest tightness; PD= percentage of dissatisfaction.

Figure 9. Comparisons between asthmatic and non-asthmatic subjects' work performance during exposures to ozone and its initiated chemistry products, irrespective filter condition. Note: *p<0.0001; **p<0.05; ***p>0.05; Task 1= Concentration; Task 2= Numerical reasoning; Task 3: Arousal.

Figure 10. Subjects' work performance during exposure to ozone and its initiated chemistry products, with either new or used filter placed in the air handling unit (AHU). Note: *p<0.0001; **p<0.05; ***p>0.05; Task 1= Concentration; Task 2= Numerical reasoning; Task 3: Arousal.

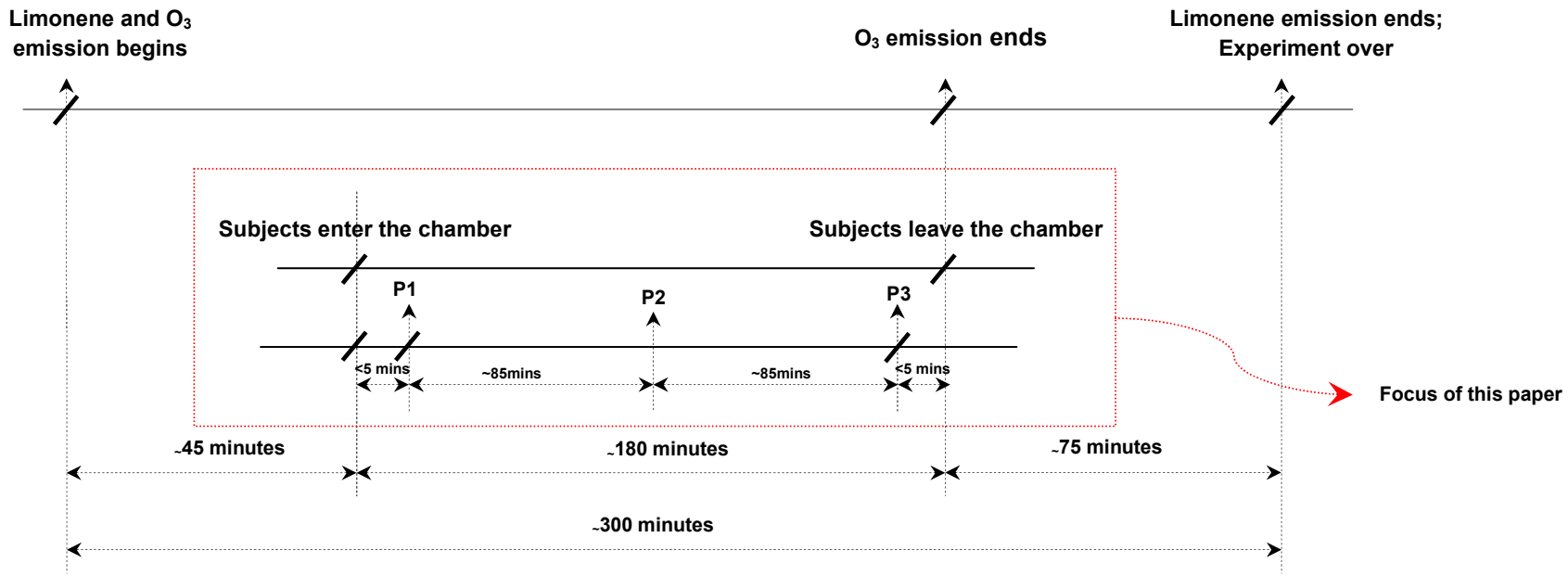
Limonene and O_3 emission begins
O_3 emission ends
Limonene emission ends; Experiment over
Subjects enter the chamber
Subjects leave the chamber
P1
P2
P3
<5 mins
~85mins
~85mins
<5 mins
Focus of this paper
~45 minutes
~180 minutes
~75 minutes
~300 minutes

Figure 1.

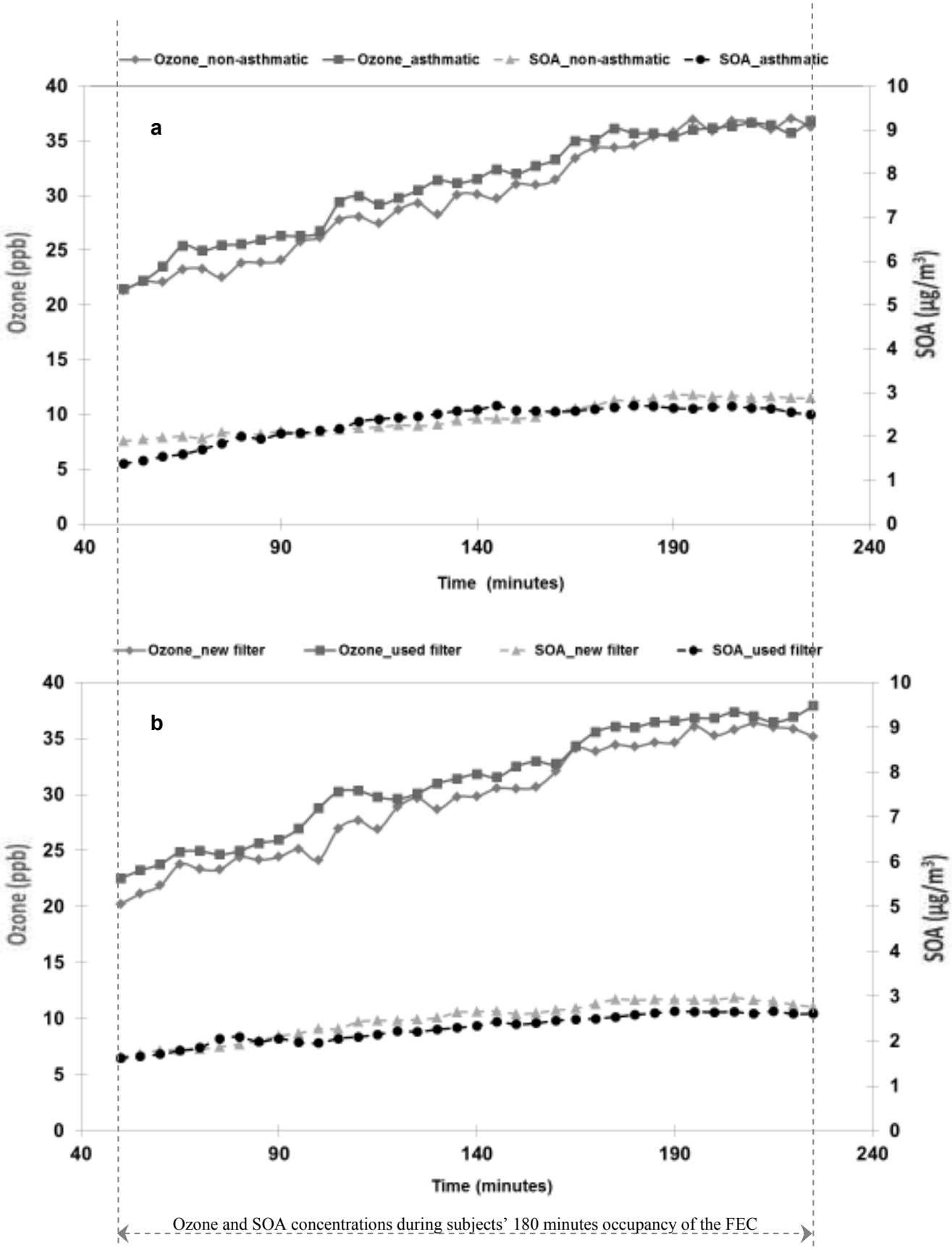
Ozone_non-asthmatic
Ozone_asthmatic
SOA_non-asthmatic
SOA_asthmatic
a
Ozone (ppb)
SOA (µg/m³)
Time (minutes)
Ozone_new filter
Ozone_used filter
SOA_new filter
SOA_used filter
b
Ozone (ppb)
SOA (µg/m³)
Time (minutes)
Ozone and SOA concentrations during subjects' 180 minutes occupancy of the FEC

Figure 2.

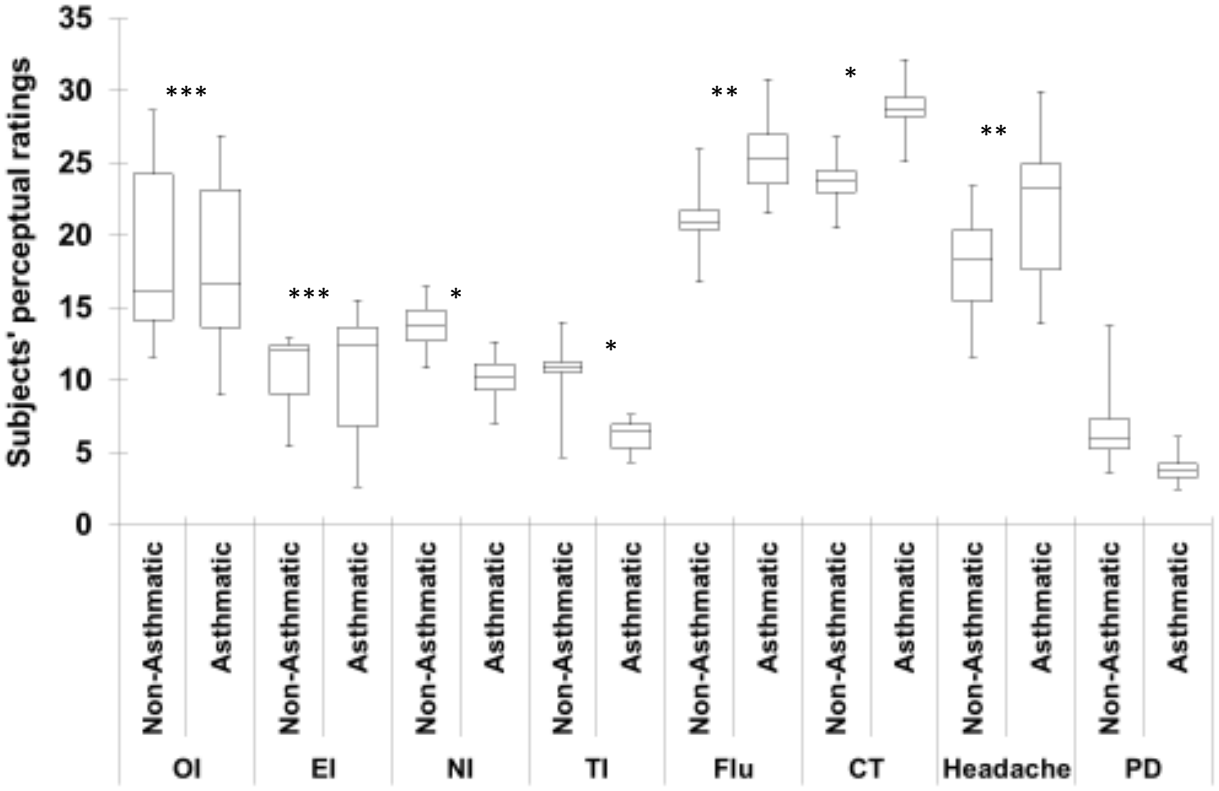

Figure 3

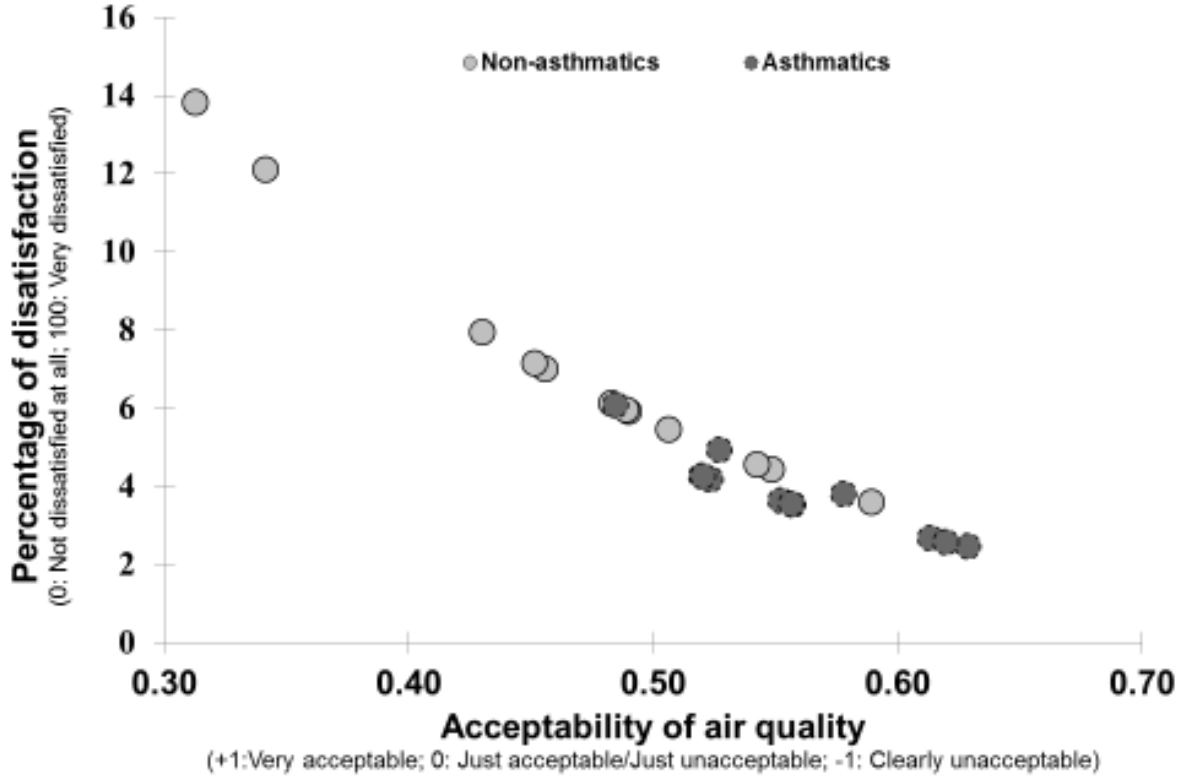

Figure 4

Figure 5

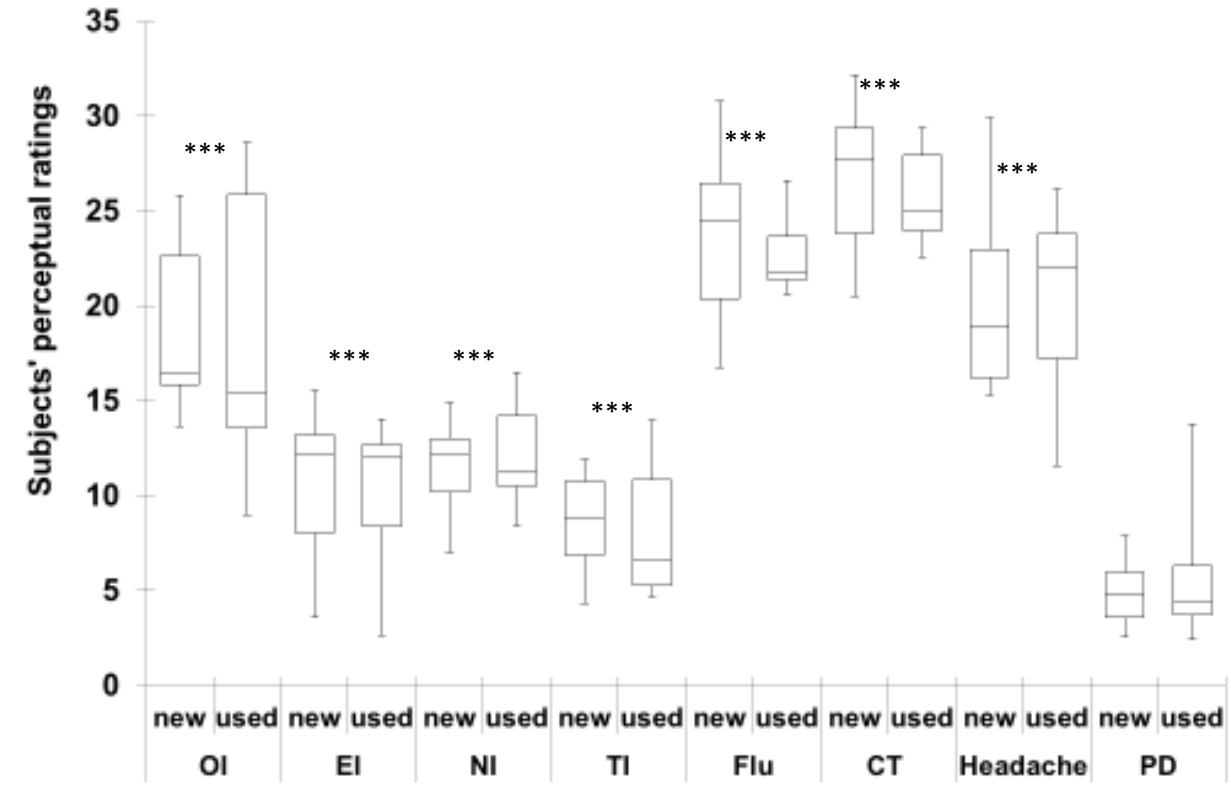

Figure 6

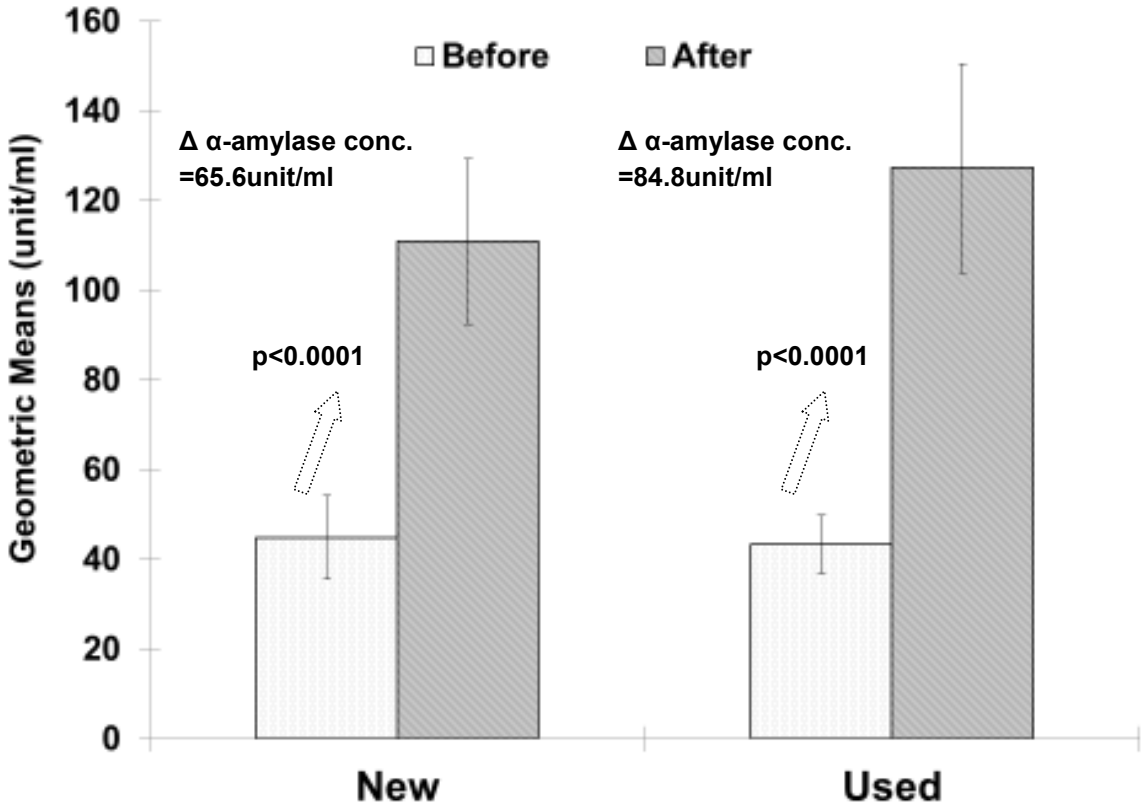

Figure 7

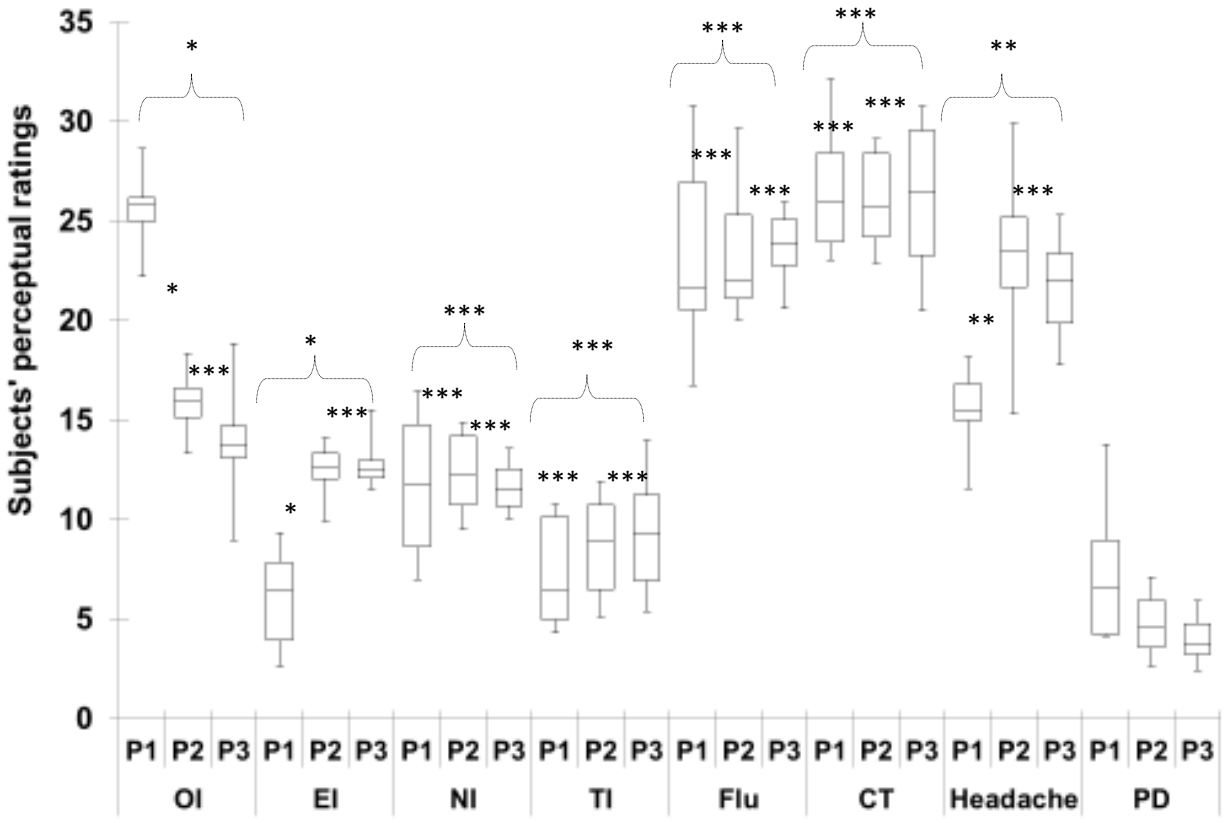

Figure 8

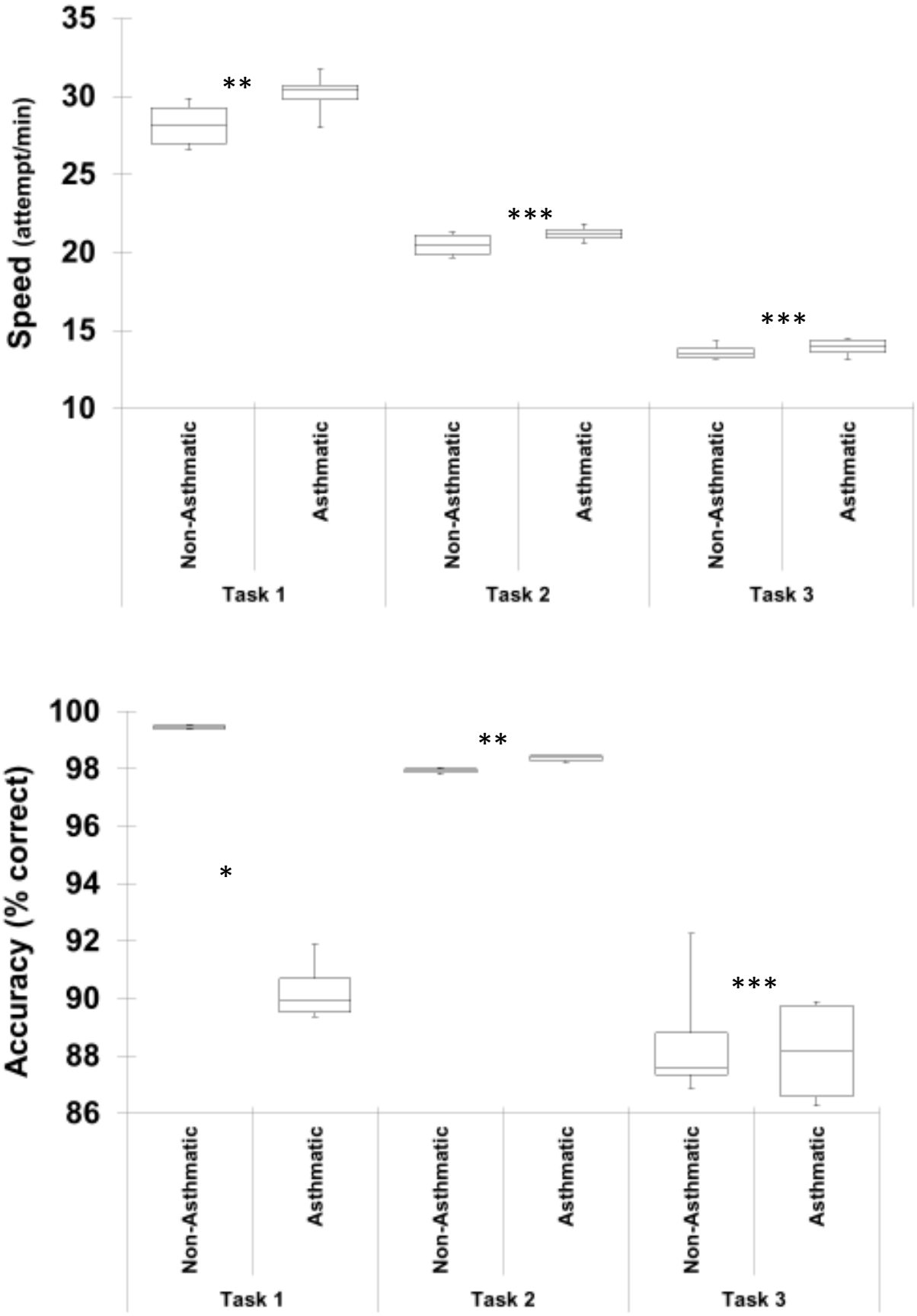
Speed (attempt/min)
35
30
25
20
15
10
**
***
***
Non-Asthmatic
Asthmatic
Task 1
Task 2
Task 3
Accuracy (% correct)
100
98
96
94
92
90
88
86
*
**
***

Figure 9:

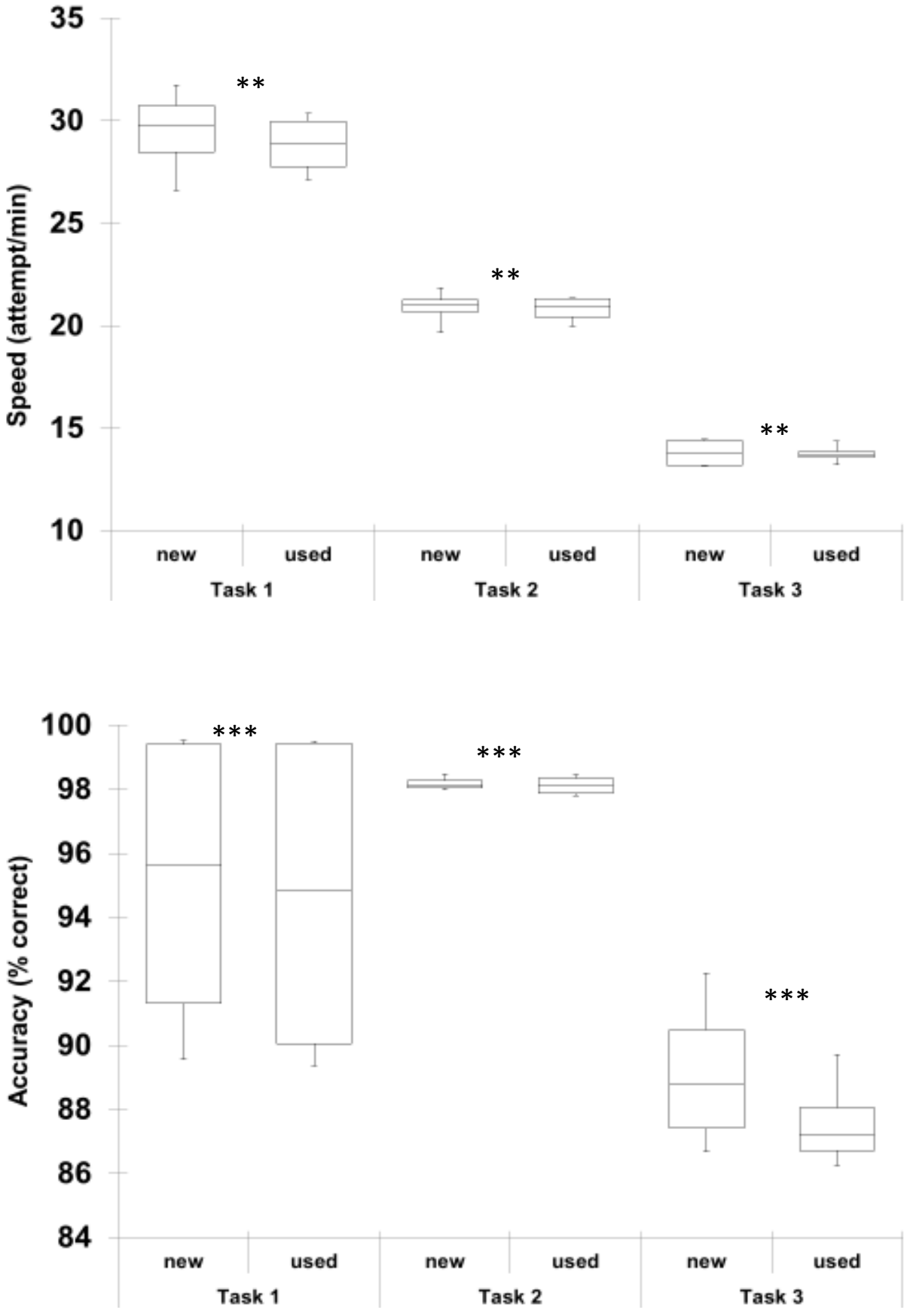
Speed (attempt/min)
35
30
25
20
15
10
**
**
**
new
used
new
used
new
used
Task 1
Task 2
Task 3
Accuracy (% correct)
100
98
96
94
92
90
88
86
84
***
***
***
new
used
new
used
new
used
Task 1
Task 2
Task 3

Figure 10