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2014

Tham, K. W., & Fadeyi, M. O. (2014). Towards whom should indoor environmental quality control be sympathetic : asthmatics or non-asthmatics? *Building and environment*, 88, 55-64.

<https://hdl.handle.net/10356/79422>

<https://doi.org/10.1016/j.buildenv.2014.10.014>

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Towards whom should indoor environmental quality control be sympathetic - asthmatics or non-asthmatics?

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Abstract

From two independent research studies conducted to understand effect of varying thermal and indoor air pollution exposures on non-asthmatics and asthmatics' responses and work performances, this paper attempts to answer the question: Towards whom should IEQ control be sympathetic - asthmatics or non-asthmatics? The studies were conducted in a 240m³ field environment chamber at the National University of Singapore. Subjects, between 20 and 30 years of age, were recruited from the university community to participate in these studies. In the first study, the interventions were two room air temperatures, i.e. 21.2±0.3°C and 25.0±0.2°C at constant ventilation. This study lasted for 8 hours. Data were collected for inhaled air thermal sensations and salivary α -amylase concentration before and after 8-h exposures. In the second study, subjects were exposed to limonene and ozone (of simulated outdoor origin) at realistic concentrations for 3 hours. Data for subjects' work performances, perceptual responses, and salivary α -amylase concentration at initial and after 3-h exposures were reported in this paper. The main findings suggest that: (i) temperature settings should be sympathetic towards asthmatic subjects because of their higher sensitivity to temperatures at the lower spectrum of thermal comfort conditions; (ii) IAQ settings should be sympathetic towards non-asthmatic subjects because of their higher sensitivity to perceived air quality acceptability in the same temperature range. Knowledge gained from this paper has practical implications towards creation of environmentally friendly indoor environment for asthmatic and non-asthmatic building occupants.

Keywords: Asthmatics; non-asthmatics; indoor air quality; thermal comfort, Human-environments interactions

1. Introduction

The Global Initiative for Asthma (GINA) dissemination committee report documented that around 300 million people worldwide are suffering from asthma [1-2]. The spread of asthmatic population across different regions of the world is presented in Figure 1. It was documented in the GINA report that the current asthmatic population may increase to around 400 million by 2025. This has manifested an increase in the proportion of asthmatics in the population of building occupants [3]. These trends reiterate the importance of considering asthmatics in standards development for indoor environmental quality (IEQ).

One drawback of existing thermal comfort and indoor air quality (IAQ) standards is that they focused primarily on healthy human population, and make no explicit provisions for populations with asthma. However, asthmatics are believed to be more vulnerable to, and thus exhibiting adverse health effects when exposed to unfavourable thermal and air quality conditions [4-7]. Vulnerability of asthmatics may be explained by their poorly protected C-fibres. C-fibres carries message towards the central nervous system and are responsible for mediating local axon reflexes. Stimulation of C-fibres by unfavourable thermal and indoor air pollution will cause various physiological problems [8-13]. The epithelial layer which is meant to protect the C-fibres are either denudated or damaged in asthmatics. However, the epithelial layer is relatively healthier in non-asthmatics [14]. Numerous evidence linked physiological like symptoms and measured physiological condition with poor work performance [15-18]. This causal relationship is another concern especially with increasing asthmatic population in indoor environment.

Despite this concern, very little has been done to compare perceptual and physiological responses and work performances of healthy building occupants and occupants with asthma

when exposed to varying thermal conditions and indoor air pollution. Design of IEQ control systems for optimum thermal and indoor air quality performance should have the capability to receive accurate feedbacks from building occupants and respond appropriately in due time. Design of high performance IEQ control is especially important for asthmatics. There are numerous evidences that suggested the importance of appropriate environmental controls in prevention and treatment of asthma [19]. Thus, omission of asthmatics from design of IEQ standards raises an important question; should IEQ controls be sympathetic towards asthmatics or non-asthmatics?

In an attempt to answer this important question, two studies have been conducted at the National University of Singapore (NUS) to compare the perceptual responses, work performance, and physiological conditions of asthmatics and non-asthmatics when exposed to varying thermal conditions and indoor air pollution (ozone and its initiated chemistry products). This paper reflects on emerging evidences from the two studies conducted at NUS to answer the question.

2. Methods

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

2.1 Subject recruitment and ethical approval

Subjects, between 20 and 30 years of age and non-smokers, were recruited from the university community according to health criteria relevant to the studies and participated on a paid voluntary basis. Medical doctor diagnosed mild asthmatic subjects were used in this study, and the selected asthmatic subjects were not experiencing any other illness and not on

medication apart from that used for asthma management. An Institutional Review Board in Singapore approved (IRB approval number: NUS-384) the use of human subjects for the present study.

The first study (thermal study) involved sixty-four subjects with equal number of cases-asthmatics (n=32) and controls- non-asthmatics (n=32). The ratio of male to female subjects for asthmatics and non-asthmatics subjects was 1:1. The second study (indoor air pollution - exposure to ozone and its initiated chemistry products) involved seventy-one subjects comprising thirty-eight non-asthmatics (14 and 24 male and female, respectively) and thirty-three asthmatics (15 and 18 male and female, respectively). In the case of non-asthmatic subjects, the ratio of male to female subjects was 0.6: 1. In the case of asthmatic subjects, 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 for the second study that addressed indoor air pollution. Experiments for asthmatic and non-asthmatic subjects were conducted on different days, due to delay in recruiting asthmatic subjects, but with the same experimental conditions.

Before commencing the experiments, subjects were given training sessions to familiarize them with the test procedures that they would be subject to. The subjects wore typical clothing attires for office workers in the tropics. In both thermal and IAQ study, subjects were blind to the experimental conditions.

2.2 Experimental protocols

Both studies were conducted in a large field environmental chamber (FEC) simulating an office environment (11.5 x 7.9 x 2.6 m; 240 m³). The FEC has polymeric tile flooring, sealed windows, acoustic ceiling tiles, and contains typical office furniture - see Figure 2. The air handling unit (AHU) that serves this space is located in a room above the FEC. Outdoor air is

drawn from the roof of the building via an internal airshaft and filtered before being mixed with the return air. The mixed air is subsequently filtered and then 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 m³.

In the first study, the interventions were two room air temperatures, i.e. 21.2±0.3°C and 25.0±0.2°C (referred to as T1 and T2 respectively in this paper) at constant ventilation of 9 L/s/person (or the equivalent of 2 air change per hour). The recirculation rate was approximately 7h⁻¹. This study lasted for 8 hours. In this study, subjects stayed in the simulated office and completed surveys pertaining to thermal sensation. Subjects' saliva was collected for α -amylase analysis. This is because relatively high α -amylase concentration can be found in saliva [8]. Variation in salivary α -amylase concentrations was used as an indicator for physical and psychological stress [20-22] caused by IEQ exposures. A passive drool salivary sampling procedure was applied for saliva collection at initial occupancy and after 8-hr thermal exposures. Subjects expelled saliva into a labeled sampling tube. Kinetic immunoassay measurement was employed to determine salivary α -amylase concentration.

In the second study subjects were exposed to ozone and limonene and by-products from their reactions at realistic concentrations for a continuous 3-h working session in the FEC. The FEC had been configured to simulate an office environment in the same way as in the first study. The study was conducted at ventilation and recirculation rates of 1 and 7, respectively. Temperature setting was kept constant at 23±0.3°C. Prior to subjects entering the FEC, ozone and limonene had been released within the FEC for 45 minutes. Ozone was generated at a rate of 135 mg h⁻¹ (250 ppb/h) at the outdoor air duct; at this rate the resulting ozone concentrations in the chamber were in the range often experienced in tropical office

environments [23]. At steady state, ozone concentration in the chamber was around 35-39ppb when occupants were present in the chamber. Limonene (98% purity) was emitted in the FEC at a constant rate of approximately 200 mg h^{-1} using modified emission vials; the resulting chamber concentrations (35-36ppb) when subjects were present were lower than what building occupants would be exposed to following floor cleaning with a limonene scented agent. Detailed information on ozone and limonene generations can be found in Fadeyi et al. [24].

Questionnaire on the intensity of sick building syndrome (SBS) symptoms and perceptions towards the indoor environment were carried out at various times. Subjects filled the questionnaire immediately (~ 5 mins) after entering the FEC and ~ 5 mins before leaving the FEC. Subjects assessed the acceptability of air in the FEC using the continuous acceptability scale [25] 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, eyes, nose and throat irritations, and flu, chest pain and headache were 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 irritations 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). Flu, chest pain and headache resulting from exposure to FEC air were evaluated using visual analogue scales with labelled endpoints- left end-point = 0, right end-point = 100 [25]. Percentage of subjects dissatisfied with perceived air quality was

calculated from subjects' acceptability rating of perceived air quality using the equation Gunnarsen and Fanger [26] developed – see Equation 1.

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

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

Subjects' salivary α -amylase concentrations were measured before and after exposure, adopting the same techniques in the first study, to objectively obtain a surrogate measure of the impact of ozone, limonene and their by-products on subjects' physiological conditions. Additionally, simulated office tests were administered to measure the effect of ozone, limonene and their by-products on asthmatic and non-asthmatic subjects' work performance. The purpose of these tests is to establish the relationship between indoor air pollution exposure and work performance of asthmatic and non-asthmatic subjects. Subjects were required to complete concentration endurance (task 1) numerical reasoning (task 2), and arousal test (task 3) within stipulated period of time. Details about these tasks can be found in Willem [27].

3. Results

This section provides findings from the first (thermal) and second (IAQ) studies.

3.1 First study: impact of temperature settings on asthmatics and non-asthmatics

Figure 3 shows subjects' salivary α -amylase concentration and inhaled air thermal sensations at initial and after 8-h exposures for the asthmatic and non-asthmatic subjects. At the start of exposure, the geometric mean (GM) of salivary α -amylase concentration were

consistently higher level at T1 ($P<0.05$). After 8-hour exposure, this effect persisted and there was a higher salivary α -amylase secretion in the asthmatics. More importantly, the increase of salivary α -amylase concentration in the asthmatics after the 8-hour exposure at T1 was significantly higher than that of the non-asthmatics ($P<0.05$). This suggests that whilst non-asthmatics had been able to adapt to an exposure at 21°C, asthmatics are less able to do so. After an 8-hour exposure at a higher air temperature of 25°C increases in the α -amylase concentration in non-asthmatics and asthmatics were not statistically significant.

In both groups, the inhaled air thermal sensation was significantly lower at T1 than that at T2 at the start ($P<0.001$) as well as at the end of the session ($P<0.001$). Asthmatics tended to rate their inhaled air thermal sensation slightly lower (cooler) at T1 than the non-asthmatics ($P<0.05$, one-tailed) although this relative sensation decreased from 0.37 (being the difference between -2.18 for asthmatics and -1.81 for non-asthmatics initially) to 0.25 after the 8 hour exposure. At T2 the relatively low difference initially (0.05) increased to 0.10, with the asthmatics perceiving the inhaled air temperature to be warmer than the non-asthmatics. More interestingly, the difference in perceptions between the two temperatures was higher for the asthmatics both at initial exposure and at the end of an 8 hour exposure. On initial exposure, the difference of 1.91 (between -2.18 at T1 and -0.27 at T2) reduced to 1.59 (between -1.45 at T1 and 0.08 at T2) at the end of the 8 hour exposure for asthmatics. This magnitude is larger than the corresponding difference of 1.59 (between -1.81 at T1 and -0.22 at T2) being reduced to 1.02 (between -1.2 at T1 and -0.18 at T2) at the end of the 8 hour exposure for non-asthmatics.

3.2 Second study: impact of ozone and its initiated chemistry products on asthmatics and non-asthmatics

Perceptual responses of asthmatic and non-asthmatic subjects both at initial and after 3-h exposures are shown in Figure 4. Continuous exposure to chemical stimuli-ozone and its initiated chemistry products caused the perceived odour intensity of both the asthmatics and non-asthmatics to decrease. This observation corroborates with evidence in the literature [28] that perceptual odour intensity decrease exponentially with increase of time of chemical stimulation of the olfactory organ through a process known as “adaptation”. Adaptation of perceived odour increases their odour detection threshold. Perceived odour intensities (OI) for asthmatic and non-asthmatic subjects were almost the same both at initial ($p=0.8047$) and after 3-h exposure ($p=0.8953$). These observations suggest that, at realistic and low concentration of ozone initiated chemistry there would probably be no difference in the sensitivity of asthmatic and non-asthmatic subjects to odour intensity perception.

As shown in Figure 4, exposure to ozone and its initiated chemistry products (irritants) caused both group of subjects to develop perceived sensory (eye, nose, and throats) irritations (indicated as EI, NI, and TI). Unlike perceived odour intensity, there were significant differences in both group of subjects’ sensitivity to nose and throats irritations (indicated as NI and TI respectively) at initial exposure to ozone and its initial chemistry products. The difference between asthmatic and non-asthmatic subjects’ perceptual responses was significant in the case of NI ($p=0.0029$) and TI ($p=0.0439$). However, the difference was not significant ($p=0.2726$) in the case of eye irritation (EI).

After 3-h exposure the difference between asthmatic and non-asthmatic subjects’ perceptual responses reduced for EI and NI to the extent that the difference is statistically insignificant (EI: $p=0.6235$ and NI: $p=0.4378$). The closeness was due to noticeable increase in asthmatic subjects’ EI and NI perceptions while non-asthmatics perception increases slightly for the same period of exposure. Unlike EI and NI, the difference for TI was even

more significantly different ($p=0.0058$) largely due to noticeable increase in non-asthmatic subjects' TI perceptions and the slight increase of the asthmatic subjects.

Asthmatic subjects' lesser sensitivity may have caused them to accept their exposed IAQ condition significantly more than non-asthmatic subjects both at initial ($p=0.0366$) and 3-h exposures ($p=0.0334$) - see Figure 5. This translates (based on Equation 1) to asthmatic subjects being significantly less dissatisfied than non-asthmatic subjects both at initial ($p=0.0414$) and 3-h exposures ($p=0.0329$) - see Figure 4. Although asthmatic subjects were less dissatisfied with their exposed IAQ, this does not mean asthmatic subjects were lesser affected physiologically. As evident in Figure 4, asthmatic subjects' are be more affected physiologically judging from their elevated physiological related symptoms (flu, chest tightness, and headache) ratings both at initial and after 3-h exposures. The difference was significant for flu symptom ($p=0.0364$) at initial exposure, and for chest tightness symptom ($p=0.0165$) after 3-h exposure.

There was significant increase ($p<0.001$) in both asthmatic and non-asthmatic subjects' salivary α -amylase concentrations after the 3-h exposure (Figure 6). As expected, due to asthmatics' damaged or denudated epithelial barrier, the effect of ozone and its initial chemistry products on salivary α -amylase concentration secretion was more for asthmatic than non-asthmatic subjects. Exposure to ozone and its initial chemistry products caused a net 68.8 and 84.2 unit/ml increase in non-asthmatic and asthmatic subjects' salivary α -amylase concentrations, respectively. Exposure to ozone and its initial chemistry products tended to cause more arousal in asthmatic than non-asthmatic subjects. This presumption is strongly supported by higher salivary α -amylase secretion in asthmatic subjects.

The arousal probably caused asthmatic subjects to attempt more tasks in shorter time than non-asthmatic subjects (see Figure 7). The difference is significant ($p=0.0086$) in the

concentration task (task 1). Asthmatic subjects' lack of concentration is apparent in "task 1" that required high level of concentration but not in those that required lesser concentration (see Figure 8). Asthmatic subjects' performance in concentration task (task 1) was highly significantly lower ($p < 0.0001$) than that of non-asthmatic subjects. The difference was also significant ($p = 0.0364$) in numerical reasoning task. There is no statistically significant difference ($p = 0.9815$) between asthmatic and non-asthmatic subjects' performance for arousal task (task 3). Table 1 shows p-values (2-tailed, 1 tailed- left and right) of comparisons between non-asthmatic and asthmatic subjects during human subjects' exposures to ozone and its initiated chemistry products

4. Discussion

Human salivary α -amylase concentrations increase in the event of physical and psychological stress exposure [20-22]. For example, there are emerging evidences suggesting strong positive association between autonomic (sympathetic) nervous system (SNS) and increase in salivary α -amylase concentrations [8]. Sympathetic nervous systems regulate the function of cells within the respiratory tract. When C-fibers sensory nerves, which are efferent neurons of the sympathetic nervous systems, are stimulated by inhaled air, a number of pulmonary defense reflex responses can occur, including cough and bronchoconstriction [9-10].

The degree of stimulation will depend on the temperature of inhaled air. Low air temperature is known to stimulate various airways more than higher temperature [11]. The stimulation of various airways will cause activation and recovery of SNS. The rapid

activation and recovery of SNS will cause stress [8]. The stress will cause increase in saliva α -amylase secretion.

As a stress indicator salivary α -amylase concentration increased after exposure to inhaled air temperature lower than the subjects' natural environment (in the tropics). In the first study, 8-hr nasal breathing of air at 21°C and 25°C increased subjects' stress. However, salivary α -amylase concentration was more when subjects were exposed to lower (21°C) air temperature than when they were exposed to higher (25°C) air temperature. This is because the effect of lower (21°C) air temperature in stimulating (cooling and drying) the airways is relatively greater than at the relatively higher (25°C) air temperature [11].

The effect of lower air temperature in cooling and drying airway is known to be greater in human population with impaired health (e.g. asthma, chronic allergen exposure, and viral respiratory infections) than healthy population [29-30]. The effect (of 8h exposure to lower air temperature) is more in asthmatic subjects because of asthmatics' damaged or denuded epithelial barrier while that of non-asthmatics is healthy [14]. It is important to note that the observed non-asthmatic subjects' α -amylase concentration that was higher than that of asthmatic subjects during initial occupancy of the experimental chamber could probably be due to their thermal exposure and activity history before coming for the study as we have no control over these prior to subjects presenting themselves on the day of their participation. The focus of this paper is how the effects of an 8h exposure to 21°C manifest as differences between asthmatic and non-asthmatic subjects' amylase concentrations. As evident from Figure 3, an 8h exposure to 21°C caused significant increase in asthmatic subjects' amylase concentration, while there was no increase for non-asthmatic subjects.

Epithelial tissues help protect C-fibers. The damaged or denuded epithelial barrier brings the low air temperature in direct contact with the C-fibers relatively easier in

asthmatics than non-asthmatics with healthier epithelial barrier. Perhaps, this made asthmatics to be more sensitive in their nasal perception of air temperature. Our findings support this understanding. There was significant increase ($p < 0.05$) in asthmatic subjects' salivary α -amylase secretion after 8-h exposure to lower (21°C) air temperature while there was no increase in the healthy (non-asthmatics) subjects' salivary α -amylase secretion during the same 8-h exposure period.

This study suggest that asthmatics' stress (as indicted by α -amylase concentration), and even that of non-asthmatics, may be reduced by maintaining a higher air temperature of 25°C which, apart from not compromising inhaled air thermal sensation (near neutrality for both asthmatics and non-asthmatics), also has the advantage of a lower cooling energy consumption for the tropics. However, operating at a higher temperature may have implications on IAQ. Increase in temperature can increase VOC emissions from building materials [31-32]. When this occurs, ozone generated from the indoor environment or transported indoors via outdoor air exchange, will initiate relatively increased chemical reactions with emitted unsaturated VOCs from building materials. Such reactions would lead to oxidation products and secondary organic aerosols [33-34]. Ozone initiated chemistry products can have adverse impact on human health and comfort [35-37].

Consequences of outdoor to indoor transport of ozone via outdoor air air-exchange (ventilation) rate [38] may explain the observation by Willem [27] that increased ventilation rate can adversely compromise building occupants' physiology, perceptual responses, and actual and self-reported work performances. Thus, it is important to understand how ozone (of outdoor origin) initiated chemistry, which would be enhanced by increasing indoor air temperature, could influence asthmatics and non-asthmatics. It is also important to understand who is more sensitive to indoor air pollution, asthmatics or non-asthmatics. Such

understanding is important in the design, operations and management of indoor environment that consist of asthmatics and non-asthmatics.

The increase in both asthmatic and non-asthmatic subjects' perceived sensory irritations from their initial exposure to after their 3-h exposure is due to their continuous exposure to ozone and its initiated chemistry products. According to Oortgiesen et al. [14], human sensitivity to chemical stimuli or even to new chemical stimuli will increase after continuous exposure. It remains unknown if asthmatic subjects' perceived sensory irritations ratings, especially in the case of EI and NI, would exceed that of non-asthmatics if they were to be exposed to the chemical stimuli-ozone and its initiated chemistry products- for much longer duration. Nevertheless, our findings showed asthmatic subjects' perceived sensory irritations ratings generally increased with increase in exposure time.

Additionally, stimulation of C-fibers sensory nerves by irritants – indoor air pollution- can facilitate inflammatory effects and a number of pulmonary defense reflex responses, including chest-tightness, cough and bronchoconstriction. Inflammation effects and pulmonary defense reflex responses can lead to headache [12-13]. This explains reported perceived physiological (flu, chest-tightness, and headache) like symptoms by both groups of subjects. Of the two groups, asthmatic subjects perceived the physiological like symptoms more during exposures to ozone and its initiated chemistry products. This observation can be attributed to asthmatics subjects' relatively damaged or denudated epithelial barrier which brings the irritants into direct contact with the C-fibers more easily than it would have been in a healthy epithelia layer of healthy subjects (non-asthmatics).

The rapid activation and recovery of SNS by ozone and its initiated chemistry products caused stress in both stress, as indicated by increased by salivary α -amylase secretions, in both non-asthmatic and asthmatic subjects. As expected, increased saliva α -amylase secretion

was more in asthmatic subjects. It is important to note the higher salivary α -amylase secretions observed in both non-asthmatic and asthmatic subjects when exposed to ozone and its initiated chemistry products than the effect of lowered air temperature exposure at realistic thermal conditions and chemical concentration levels.

The ventilation rates in the thermal study and indoor air pollution studies were 2 and 1 air change rate per hour, respectively. At higher ventilation rate, higher salivary α -amylase secretions will be expected [39]. Additionally, lower temperature (21°C) is expected to cause higher salivary α -amylase secretions, judging from thermal study reported in this paper, than that of 23°C temperature used in the ozone initiated chemistry study. Despite the potential effects of increased higher ventilation rate and lower temperature, subjects' exposures to ozone and its initiated chemistry products caused higher salivary α -amylase secretions than that of exposure at 21°C temperature. The increased salivary α -amylase secretions ranged from 68.8 to 84.2 units/ml while in the case of 21°C temperature it ranged from 'no increase' to 12.64 units/ml. This observation shows the relatively higher impact of ozone and its initiated chemistry product, even at low and realistic concentrations, on objectively measured physiologic condition.

Asthmatic subjects' relatively lack of concentration during exposure to ozone and its initiated chemistry products can be attributed to their higher manifestations of physiological like symptoms (flu, chest tightness, and headache) and higher salivary α -amylase secretion. There are numerous evidences linking physiological like symptoms with poor work performance [16-18]. Since subjects were exposed to realistic, low concentrations of ozone and limonene, the competency (as a plausible confounder) in performing simple numerical reasoning (task 2) and arousal (task 3) tasks have apparently dominated possible adverse effect of being exposed to ozone initiated chemistry pollutants.

5. Pragmatic Implications

Should IEQ controls be more sympathetic towards asthmatics or non-asthmatics? A reflection of emerging evidence suggests the following:

- Asthmatics are more sensitive in their nasal perception of air temperature than non-asthmatics. Asthmatics' perception was cooler at lower (21°C) and warmer (and closer to neutrality) at higher temperature (25°C). 8-hour exposure to lower temperature of 21°C significantly increases asthmatics' stress (salivary α -amylase concentration), while there was no increase in non-asthmatics' stress. After an 8-hour exposure at a higher air temperature of 25°C increases in the α -Amylase concentration in non-asthmatics and asthmatics were not statistically significant. Temperature controls sympathetic towards asthmatics, instead of non-asthmatics, will improve liveable indoor environment experience for both asthmatics and non-asthmatics. This study suggests that temperature should NOT be controlled at 21°C but raised. The increased temperature may cause increase in organic emissions from building surfaces though.
- There is an emerging trend for subjective information to be incorporated as feedback to the control system [40]. However, asthmatic subjects' relatively lesser sensitivity to perceived air quality acceptability and sensory irritation will delay their relaying of adverse IAQ conditions to control systems for prompt action. By the time asthmatic subjects can correctly perceive air pollution (to the same extent as non-asthmatics) they are being exposed to, their physiological health condition may have been adversely affected. Such delay will increase asthmatics' exposure risk and also that of non-asthmatic subjects. Increase in exposure risk will compromise asthmatics and non-

asthmatics work performance, especially those requiring high concentration level and arousal tasks. Thus, IAQ controls that are sympathetic towards non-asthmatics, instead of asthmatics, will improve liveable indoor environment experience for both asthmatics and non-asthmatics.

- These observations -asthmatics subjects' lower perceived sensory irritations, higher reported physiological like symptoms and higher salivary α -amylase concentrations- have potential practical implications on how IEQ control systems should be designed. These observations suggest that if control systems needed to improve IAQ is sympathetic towards asthmatic subjects, the control systems may be misinformed in due time because of the asthmatic subjects' lower perceived air quality acceptability and sensory irritations. The delay in appropriate action to improve IAQ will have adverse physiological health implications not only on asthmatics, but also on non-asthmatics. However, if control systems are sympathetic towards air-acceptability feedbacks from non-asthmatics, adverse physiological health effects will be minimized in both groups. This is because control systems would have been given the appropriate information in time to initiate appropriate action to reduce indoor pollution rather than later should control action be based on asthmatics' perceptual feedback.

6. Conclusion

Towards whom should IEQ controls be sympathetic - asthmatics or non-asthmatics? To answer this question, findings from two independent research studies conducted to understand effect of varying thermal and indoor air pollution exposures on non-asthmatics and asthmatics' responses and work performances were reported in this paper. The main

findings suggest that: (i) temperature settings should be sympathetic towards asthmatic subjects because of their higher sensitivity to temperatures at the lower spectrum of thermal comfort conditions; (ii) IAQ settings should be sympathetic towards non-asthmatic subjects because of their higher sensitivity to perceived air quality acceptability in the same temperature range.

It is important to note that understanding on this subject is not conclusive. Further research efforts are still needed to better understand how exposure to IEQ conditions can influence perceptual and physiological responses and work performances of healthy and impaired health populations. For example, we do not have knowledge about impact of long term exposure to realistic concentrations of ozone and its initiated chemistry products on asthmatics and non-asthmatics. We do not know how impact of short term exposure to the higher end realistic concentrations of ozone and its initiated chemistry products would affect asthmatics and non-asthmatics. Asthmatic subjects used in this study had mild asthma condition. We do not know how asthmatic subjects with chronic asthma condition would response to studied IEQ conditions in this present study. We also do not know how IEQ (IAQ and thermal) conditions could influence human population with other forms of impaired health condition.

Furthermore, only air temperature was used to address thermal condition in this present study. We do not know how other environmental factors that influence thermal condition would influence healthy and impaired health human populations. Nevertheless, this present study provides initial understanding to highlight the importance of not neglecting human population with impaired health condition or assuming whatever happens with healthy population applies to population with impaired health condition. As evident in this study, human-environments interactions research effort addressing both healthy and impaired health

populations' responses to IEQ conditions is relevant towards determining whom IEQ controls should be sympathetic towards.

Acknowledgement

The financial assistance from the National University of Singapore is gratefully acknowledged through grant R296-000-117-112. Dr. Moshood Olawale Fadeyi participation in this research is partly funded by the Republic of Singapore's National Research Foundation through a grant to the Berkeley Education Alliance for Research in Singapore (BEARS) for the Singapore-Berkeley Building Efficiency and Sustainability in the Tropics (SinBerBEST) Program. We gratefully acknowledge the assistance provided by Dr. Jovan Pantelic and Tan Bing Qin during conduction of the experiments. We gratefully acknowledge assistance provided by Dr Gerald Koh and his research team at Saw Swee Hock School of Public Health, and Eileen Tan and Dr. Chew Fook Tim of the Department of Biological sciences at the National University of Singapore in collecting and processing α -amylase collected from subjects' saliva. The authors thank Dr. Henry Cahyadi Willem for helpful discussions during experimental design.

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Table 1: *p*-values of comparisons between non-asthmatic and asthmatic subjects during human subjects' exposures to ozone and its initiated chemistry products

	Initial exposure	After 3-hour exposure
	Non-Asthmatics vs Asthmatics	Non-Asthmatics vs Asthmatics
Odour Intensity	0.8047	0.8953
Eye Irritation	0.2726	0.6235
Nose Irritation	0.0029 *(0.0015)	0.4378
Throat Irritation	0.0439 *(0.022)	0.0058 *(0.0029)
Flu	0.0364 ** (0.0182)	0.7
Chest- tightness	0.1083	0.0165 ** (0.0082)
Headache	0.7342	0.1358
Acceptability	0.0366 ** (0.0183)	0.0329 ** (0.0167)

Bolded number shows significant difference ($p < 0.05$)

p-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test

*One-tailed *p*-value (right) -- (non-asthmatic > asthmatic)

**One-tailed *p*-value (left) -- (asthmatic > non-asthmatic)

Figure captions

Figure 1. Burden of Asthma in different regions of the world: (a) number of asthmatic population in each region; (b) % of asthmatic population in each region. Adapted from data presented in Global Initiative for Asthma (GINA) dissemination committee report [1]

Figure 2. General room set-up of the field environmental chamber

Figure 3. Subjects' salivary α -amylase concentration (a) and perceive thermal sensation before and after 8-hr exposures for the asthmatics (cases) and healthy young adults (controls)

Figure 4. Perceptual responses of asthmatic and non-asthmatic subjects both at initial and after 3-hr exposures (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test).

Figure 5. Perceived air acceptability of asthmatic and non-asthmatic subjects both at initial and after 3-hr exposures (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test)

Figure 6. Asthmatic and non-asthmatic subjects' salivary α -amylase concentration before and after 3-hr exposure to ozone and its initiated chemistry products

Figure 7. Asthmatic and non-asthmatic subjects' speed in performing tasks (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test)

Figure 8. Asthmatic and non-asthmatic subjects' accuracy in performing tasks (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test)

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Note: % of asthmatic population in each region = (Number of asthmatic population in the region/Total number of human population in the region)*100

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Note: OI= odour intensity, EI=Eye irritation, NI=Nose irritation, TI=Throat irritation; CT=Chest tightness; PD= percentage dissatisfied

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Figure 7. Asthmatic and non-asthmatic subjects' speed in performing tasks (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test)

Note: Task 1 = Concentration task; Task 2 = Numerical reasoning task; Task 3: Arousal task

Figure 8. Asthmatic and non-asthmatic subjects' accuracy in performing tasks (P-values indicate the significance of the difference in mean values between the asthmatic and non-asthmatic subjects; 2-tailed test)

Note: Task 1 = Concentration task; Task 2 = Numerical reasoning task; Task 3: Arousal task