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Acute exposure to sawdust does not alter airway calibre and responsiveness to histamine in asthmatic subjects

S. De Luca*, N. Caire **, Y. Cloutier ***, A. Cartier **, H. Ghezzo **, J-L. Malo **

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ABSTRACT: We investigated the effects of particles of sawdust delivered through a special device at known concentrations (close to the threshold limit value-short term exposure limit (TLV-STEL) of 10 mg·m^{-3}) on FEV_1 and PC_{20} in 12 asthmatic subjects free of clinical sensitization to this product. Subjects were studied over two days (day 1: exposure to sawdust; day 2: sham exposure) in random order with a maximum interval of 1 week. On each day, after the assessment of spirometry and PC_{20}, subjects underwent exposure to sawdust or sham exposure. Sawdust was inhaled for a total of 30 min at average concentrations varying from 8.0 to 19.3 mg·m^{-3} (mean=11.5 mg·m^{-3}). Twenty-five to 39.7% (mean=34.6%) of inhaled particles had a diameter < 10 μ (diameter allowing deposition in the trachea and lower respiratory tract). At the end of each period of exposure, FEV_1 was assessed. After recovery, the second PC_{20} was obtained. Serial measurements of FEV_1 were carried out every hour for up to 6 h after the end of exposure. At that time, PC_{20} was reassessed. Only one subject showed an acute bronchoconstriction immediately after exposure to sawdust (maximum fall of 14% in FEV_1) with complete recovery 10 min later. Overall, inhalation of sawdust did not modify PC_{20} by comparing the mean result of the first test with the second and the third assessments. Also, the mean changes in PC_{20} at each interval after exposure to sawdust were not significantly different from the variations in PC_{20} on the sham day. We conclude that acute exposure to particles of sawdust does not affect airway calibre and responsiveness to histamine in asthmatic subjects.

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* Divisione di Pneumologia, Ospedale Regionale di Torrette, Ancona, Italy.
** Department of Chest Medicine, Hôpital du Sacré-Coeur.
*** Institut de Recherche en Santé et Sécurité du Travail du Québec, Montreal, Canada. N. Caire was a research student of the Faculté de Médecine, Université de Montréal.

Correspondence: Dr Jean-Luc Malo, Department of Chest Medicine, Hôpital du Sacré-Coeur, Montreal, Canada H4J 1C5.

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Changes in airway calibre, either spontaneously or as a result of therapy, and bronchial hyperresponsiveness are the hallmark of asthma [1]. Several factors can diminish airway calibre and increase the level of bronchial responsiveness such as exposure to an antigen [2] and sensitizing occupational agents [3–5]. Gas pollutants, such as ozone, can also modify airway calibre and responsiveness, but this effect is small and transient [6]. It has been reported that respiratory infections can have a similar effect in normal subjects [7], although JENKINS and BRESLIN [8] generally failed to demonstrate any significant variation of airway responsiveness to histamine in normal and asthmatic individuals affected by upper respiratory tract infections. The effect of other factors such as exercise, inhalation of cold air and nebulization of distilled water on airway calibre and responsiveness has been investigated. Although these agents can alter airway calibre, conflicting results on bronchial responsiveness have been published [9–15]; when present, the effect on bronchial responsiveness seems to be small and transient.

In the investigation of occupational asthma, it is important to distinguish the effect of an irritant from a sensitizing agent. Indeed, the latter but not the former is included in the generally accepted definition of this condition [5]. The coupling of peak expiratory flow rates which assess airway calibre and bronchial responsiveness to histamine or methacholine has been proposed in the investigation of this condition [5]. The rationale for this approach is that changes in airway calibre and non-allergic bronchial responsiveness can be observed after exposure to sensitizing agents but not after exposure to irritant occupational agents. However, to our knowledge, the effect of an irritant occupational agent, administered as particles and at known concentrations, on airway calibre and non-allergic bronchial responsiveness has

not been investigated.

The aim of this study of twelve asthmatic subjects is to evaluate the early and late effects of an exposure for 30 min to an irritant agent as particles, such as sawdust. We selected sawdust as this product is frequently encountered in the work-place. Several types of wood dust can cause occupational asthma [5]. The asthmatic subjects were not sensitized to this product. Sawdust was aerosolized at concentrations close to the threshold limit value for the short term exposure limit (TLV-STEL) of 10 $mg \cdot m^{-3}$. Airway calibre and non-allergic bronchial responsiveness were respectively assessed by forced expiratory volume in one second (FEV_1) and the dose of histamine causing a 20% fall in FEV_1 (PC_{20} [16]) immediately and up to 6 h after the exposure.

Material and methods

Subjects

Twelve asthmatic subjects who met the criteria for asthma of the American Thoracic Society [1] were included (table 1). Exposure to relevant allergens (*i.e.* those for which subjects showed immediate skin reactivity on prick skin testing with a battery of fifteen common inhaled allergens) was avoided for at least two weeks before the study period, except for house dust. The three atopic subjects showed immediate skin reactivity to house dust and *D. farinae* only. No subject had reported respiratory tract infections in the previous two months. Each subject was in a clinically stable condition (absence of nocturnal awakenings due to asthma, no overuse of bronchodilators) and changes in FEV_1 did not exceed 10% at the baseline assessments of each visit. The subjects had no relevant history of sensitization to wood dust, had never worked in places where wood was processed and did not report having hobbies using wood. Medications were withheld before the test for the intervals proposed by the special committee of the American Academy of Allergy [17]. Oral theophylline derivatives and inhaled beta-2 adrenergic agents were stopped for 48 h and 8 h respectively before each visit. There was no change in the regular use of inhaled beclomethasone. The study protocol was approved by the Hospital Ethics Committee and a consent form was signed by each subject.

Methods

Spirometric assessments of FEV_1, forced vital capacity (FVC), and maximum mid-expiratory flow rates ($FEF_{25-75\%}$) were obtained according to the proposed standards of the American Thoracic Society [18]. The best of three reproducible (±20 $l \cdot min^{-1}$) peak expiratory flow rates (PEFR) was also recorded.

The histamine inhalation test was performed according to a standardized procedure [16] using a Wright nebulizer (output=0.14 $ml \cdot min^{-1}$; mean diameter of particles =1.3 μ) at tidal volume breathing for 2 min.

Spirometric assessments were carried out on a Collins 9 *l* water-spirometer (Collins, Braintree, Mass). PEFR was assessed with a mini-Wright peak-flow meter (Clement Clark International, London, England).

The device for nebulization of sawdust was made of three parts: a generator of particles, an exposure room and an exhaust (fig. 1). The generator consisted of a vacuum which collected the sawdust with an endless screw from a small reservoir. The rotation speed of the screw could be regulated to obtain the desired concentration in the exposure room. The vacuum was supplied by a source of compressed air. A vibrator on the reservoir allowed the alimentation of sawdust to the endless screw. The air which went out from

Table 1. – Baseline anthropometric, clinical and functional results

n.	Sex	Age (yr)	Height (cm)	Atopy *	Duration of asthma (yr)	Medication	FEV_1 *l*	FEV_1 (%pred)	PC_{20} ($mg \cdot ml^{-1}$)
1	M	67	165	+	4	T; B2	2.50	76.2	2.70
2	M	54	159	–	2	B2; Be 100	1.99	81.9	0.76
3	M	53	173	–	3	T; B2	3.05	88.7	1.70
4	F	57	163	+	22	B2; Be 400	1.58	63.0	0.44
5	F	22	168	+	5	nil	2.05	61.4	1.35
6	F	18	166	–	2	B2	3.45	105.2	1.30
7	F	61	180	–	2	B2; T	2.71	93.4	3.13
8	M	66	175	–	3	B2; Be 300	2.40	75.2	0.96
9	M	39	164	–	6	B2; Be 200	2.32	82.0	1.33
10	F	62	162	–	8	B2; Be 200	2.31	97.1	2.12
11	F	62	172	–	10	B2; Be 100	1.96	76.9	2.07
12	M	60	177	–	4	B2; T	2.77	78.9	2.17
Mean		51.8	168.7		5.9		2.43	81.7	1.47
SD		16.6	6.6		5.6		0.52	12.9	1.75

*: see text for definition; B2: inhaled beta-2 adrenergic agent; T: oral theophylline derivatives; Be: inhaled beclomethasone and daily dose in μg; arithmetic means and SD except for PC_{20} for which the geometric means and SD are listed.

the generator was directed to the exposure room which was a vertical cylinder of plexiglass (127x30 cm). A hole in the wall of this cylinder allowed the subject to breathe the aerosol through a face-mask. The aerosol was sucked out of the exposure room by a pump (HFS-113UT, Gilian, Wayne, NJ). During the aerosolization, sawdust concentration was measured by a photometer located at 4 cm from the mouth and recorded on paper. Average values at each minute of exposure were obtained from the paper graph, summated for the 30 min of exposure and divided by the total duration of exposure (30 min) to derive the average exposure. The diameter of the aerosolized particles was assessed for six subjects by a cascade impactor (Sierra Instruments Inc, Carmel Valley, CA), placed 14 cm from the mouth of the subject. The percentage of inhaled particles, according to their size, was determined by weighing the different filters of the cascade impactor. The aerosol was inhaled by the subject at tidal volume breathing through the mouth. The device was regulated to obtain concentrations close to 10 $mg \cdot m^{-3}$, which is the threshold limit value-short term exposure limit (TLV-STEL) for sawdust originating from soft woods (black spruces (*Picea mariana*), balsam firs (*Abies balsamea*) and jack pines (*Pinus banksiana*)).

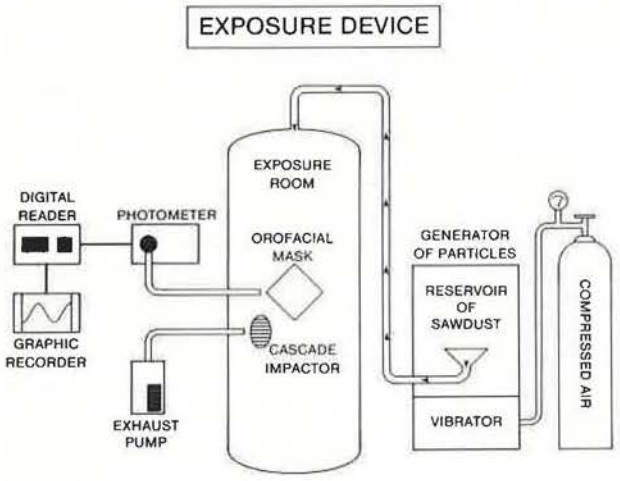

Fig. 1. – See text for description of the exposure device

Study design

Subjects were studied on two days (day 1=exposure to sawdust; day 2=sham exposure) allocated in random sequence within one week. On each study day, subjects came at the same time in the morning. After spirometric measurements, they underwent the first histamine challenge. After functional recovery (FEV_1 returning to ±5 % of baseline), subjects were asked to inhale sawdust for increasing periods of time (1, 4, 10, 15 min) for a total of 30 min. After complete recovery, the second histamine challenge was performed. Serial measurements of FEV_1 and PEFR were done every hour for 6 h after the beginning of exposure. Then, the third histamine test was performed. PEFR was monitored for the rest of the day until going to bed. A similar procedure was followed on the sham day except that no sawdust was aerosolized.

Analysis of results

The best FEV_1 and PEFR at each time interval was kept for analysis. Predicted values for spirometric indices were obtained from KNUDSON *et al.* [19]. The daily percentage change in PEFR was calculated from the following formula: (highest–lowest value/ highest value) x $100 \cdot PC_{20}$ and was interpolated from the dose-response curve drawn on a semi-logarithmic noncumulative scale. Logarithmic transformation (log_{10}) of PC_{20} was used before testing the data. Student's paired t-test was used to compare the means. Selecting twelve subjects and assuming that and ß levels were 0.05 and 0.10 respectively meant that we could detect a significant

Table 2. – Features of the exposure

n.	Concentration of particles ($mg \cdot m^{-3}$)			Diameter of particles (% total)		
	highest	lowest	mean	>10 µ	10–2 µ	<2 µ
1	15	4	9	-	-	-
2	13	9	11	-	-	-
3	13	4	10	-	-	-
4	18	3	8	-	-	-
5	16	4	8	-	-	-
6	19	5	10	64.3	23.3	12.4
7	14	6	10	60.3	20.5	19.2
8	19	5	12	-	-	-
9	24	4	9	70.2	24.6	5.2
10	24	7	17	65.8	16.7	17.5
11	26	4	15	61.1	23.9	15.0
12	32	5	19	75.0	16.6	8.4
Mean	19.4	5.0	11.5	66.1	20.9	13.0
SD	5.9	1.7	2.7	5.6	3.6	5.4

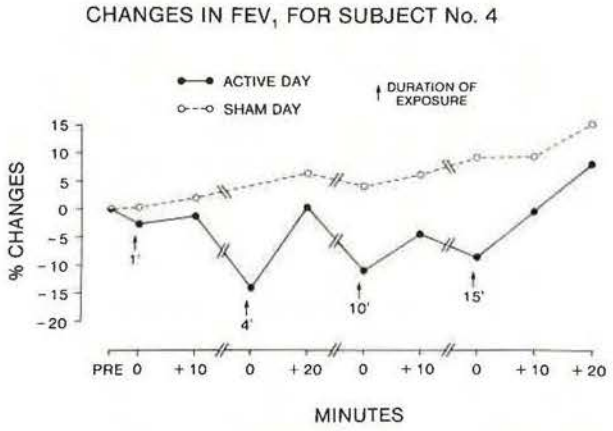

Fig. 2. – Changes in FEV_1 after exposure to sawdust and on the sham day in subject n. 4. Durations of exposure are indicated by the arrows.

difference of less than half a doubling dose of histamine in the differences in PC_{20} at each interval after exposure to sawdust as compared with the variations in PC_{20} observed on the sham day.

Results

Table 1 shows that four subjects had a baseline FEV_1 value <80% predicted. Baseline PC_{20} results demonstrated mild to severe bronchial hyperresponsiveness.

Average concentrations of sawdust during each minute of exposure varied from 8–19.3 $mg \cdot m^{-3}$ (table 2). In the six subjects for whom the recording was made, 25–39.7% of inhaled particles had a diameter <10 μ, allowing deposition in the trachea and lower respiratory tract [20].

Baseline FEV_1 and PC_{20} were not significantly different on the two study days (t=1.77 and t=0.76 respectively; p>0.1). Only one subject (n. 4) had a small acute bronchoconstriction after exposure to sawdust (fig. 2), with functional recovery 10 min after each period of exposure. Overall, there were no significant changes in FEV_1 in the hours after the exposure on the active and sham days (fig. 3). Also, there were no significant (<20%) changes in PEFR in the evenings of the active and placebo days.

On the active day, FEV_1, FVC and $FEF_{25-75\%}$ values assessed before the first histamine challenge were not different from the results before the second (t=0.86, t=0.36, t=0.11 respectively; p>0.1) and the third (t=1.05, t=0.33, t=0.48 respectively; p>0.1) tests. Similar results were obtained on the sham day. Results of PC_{20} on the exposure and sham days are shown in figure 4. In one subject (no. 3), we observed significant (>3.2-fold difference [21]) changes from the first to the second and third PC_{20} results on the active day. However, a similar behaviour was noted on the sham day. Overall, on the active day, we did not find any significant modification comparing the mean PC_{20} results of the first with the second (t=1.7; p>0.1) and third (t=0.11; p>0.1) assessments. Moreover, the mean changes in PC_{20} at each of the two intervals after exposure to sawdust as compared Bwith baseline were not significantly different from the variations in PC_{20} observed on the sham day (t=1.15 and 1.12 respectively; p>0.1). Comparison of daily percentage changes of PEFR on the active and sham days for every subject did not show any significant differences (t=0.79; p>0.1).

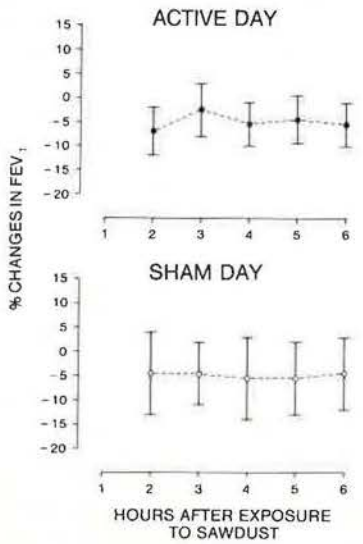

Fig. 3. – Mean (±1 SD) changes in FEV_1 after the end of exposure to sawdust or on sham day.

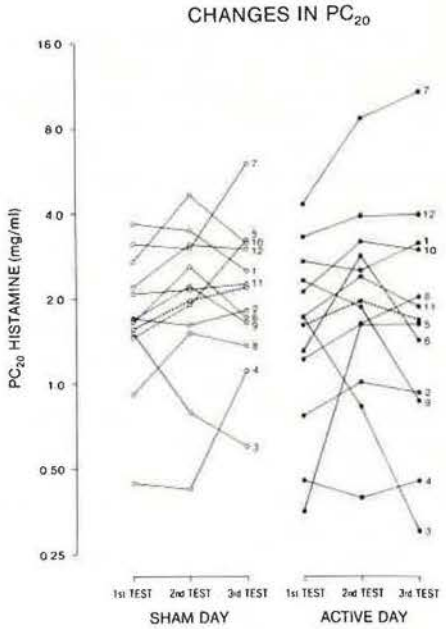

Fig. 4. – Values of PC_{20} for the first, second and third histamine tests on the sham and active days. The number of each subject is indicated and is the same as in table 1. Mean changes are represented by the dotted lines.

Discussion

It is generally assumed that acute exposure to an irritant material can cause a bronchospastic reaction in subjects with heightened bronchial responsiveness. However, to our knowledge, investigation of the acute effect of inhaled particles such as sawdust, at known concentrations, has not been carried out in asthmatic subjects. In the present study, we were able to observe a bronchospastic reaction in only one subject and the bronchoconstriction was rather mild (maximum fall of 14% in FEV_1) and transient. Moreover, for this subject, we did not find any significant modification of PC_{20} after this bronchospastic reaction. The absence of an acute bronchospastic effect after exposure to particles of sawdust in subjects with heightened bronchial responsiveness is interesting. Indeed, in the present study, the concentration of sawdust was generally close to the threshold limit value-short term expo-sure limit (TLV-STEL) and the diameter of particles allowed a significant deposition in the trachea and lower respiratory tract.

Even if we showed that sawdust did not generally change airway calibre, we also wanted to investigate the possible effects on bronchial hyperresponsiveness. Indeed, for occupational sensitizing agents, such changes can be detected even in the absence of significant alteration in airway calibre [4]. Our results indicate that acute inhalation of sawdust does not affect the level of bronchial responsiveness to histamine in asthmatic subjects. To detect clinically significant changes in bronchial responsiveness, we used a widely accepted index, the PC_{20}. PC_{20} results were obtained immediately and 6 h after the exposure to disclose any possible late changes. Indeed, it is known that besides antigens or occupational sensitizing agents, other stimuli such as exercise [22] and hypotonic solutions [12] can cause late asthmatic reactions, although these reactions are rare according to a recent report [23]. We thought that this possibility ought also to be investigated in the case of the irritant material, sawdust, which was used in the present study. Our results apply to asthmatic subjects with mild bronchial hyperresponsiveness as they were more likely than subjects with severe baseline bronchial hyperresponsiveness to demonstrate changes in bronchial hyperresponsiveness. Before concluding that sawdust does not alter bronchial hyperresponsiveness, our study would need to be extended. Firstly, a similar study would have to be carried out in normal subjects using methacholine which allows nebulization of larger doses than histamine. Secondly, the same protocol would have to be applied in a dose-response manner, *i.e.* by increasing the levels of ventilation. Thirdly, longer exposures should be considered. Finally, other types of wood dust, including those originating from hardwoods, should be used.

We think it unlikely that the use of inhaled beclomethasone had any significant blocking effect. Firstly, only half of our subjects were taking this medication at the time of the study and their behaviour was not different from the other subjects. Secondly, these subjects were on low dosages of beclomethasone and had only taken 50–100 μg of this preparation approximately one hour before the challenges. A blocking effect of inhaled beclomethasone on immediate and late asthmatic reactions to common allergens has been noticed in only approximately half of the subjects and with a higher dose [24]. Inhaled steroids have not been documented as blocking the non-immunologically-mediated bronchospastic reactions, such as exercise-induced asthma [25].

Some authors [6] have demonstrated that normal subjects have a significant alteration in airway calibre and responsiveness to methacholine and histamine after exposure to ozone. This effect was detected by using airway's resistance, a functional index which is more sensitive but less specific than FEV_1. Ozone causes an inflammatory reaction which is present at the level of the bronchial mucosa [26]. As we found no significant changes in PC_{20} after exposure to sawdust, we think that such inflammatory alterations did not occur in our subjects as the inert material which we used, sawdust, is probably removed by lung clearance before producing any significant modification of the bronchial mucosa.

Results of the present study have practical implications in the investigation of occupational asthma. Combined monitoring of airway calibre through FEV_1 and/or PEFR and bronchial responsiveness has been proposed in the investigation of this condition [3–5]. From our results, we can conclude that the demonstration of significant acute changes in airway calibre and/or PC_{20} in a worker exposed to sawdust is not due to an "irritant" effect caused by this material but rather to sensitization. Besides ozone, other agents have been investigated for their possible effect on bronchial responsiveness. A recent study [10] in which the level of bronchial responsiveness was assessed before and after two periods of exercise showed that exercise had no effect. Two other studies [13, 15] published in an abstract form reached the same conclusion. Different results were obtained by SUZUKI *et al.* [14] who documented a significant increase in responsiveness to methacholine after exercise. One can hypothesize that the different indices used to assess the response, airway's resistance in the latter study as opposed to FEV_1 in the former, can affect the results. Inhalation of ultrasonically distilled water increases the level of bronchial hyperresponsiveness but this effect is small as the reported changes are in the range of the reproducibility of PC_{20} [17]. More recently, MATTOLI *et al.* [12] have demonstrated modification of PC_{20} soon after inhalation of distilled water in ten asthmatic subjects. However, the time course of changes was short as eight subjects had their PC_{20} within a twofold difference from baseline as soon as 2 h after the challenge. Inhalation of cold air has no significant physiological effect on bronchial responsiveness [11].

For the present study, we selected sawdust as this product is frequently met in the workplace. However, we do not think that our results can be extended to all kinds of wood dust, originating from hard or soft species, or every "irritant" agent, especially chemicals. The "irritating" properties of different dusts might well be highly variable depending on other factors. Also, gases might be more likely to cause irritation than particles. Following an acute exposure to high levels of fumes, smokes or vapours, an asthmatic-like syndrome has recently been described [27] in healthy subjects without previous history of respiratory diseases. Such subjects can show an increased responsiveness to methacholine which can last for several years. Other studies are necessary to characterize the effects of other "irritant" materials, particularly those encountered in the workplace.

Development of exposure rooms such as the one described in the present study might well be justified in the investigation of the "irritating" properties of different types of dusts. Such exposure rooms might also be useful for specific inhalation challenges in the medicolegal assessment of occupational asthma. Information can be obtained on the concentration and diameter of inhaled particles.

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RÉSUMÉ: Nous avons investigué les effets aigus de particules de bois aérosolisées à des concentrations connues, *i.e.* voisines du seuil de 10 $mg \cdot m^{-3}$ pour une exposition aiguë (seuil "TLV-STEL"), sur le VEMS et la concentration d'histamine causant une chûte de 20% du VEMS (CP_{20}) chez 12 sujets asthmatiques ne présentant pas de sensibilisation clinique à ce produit. Nous avons étudié les sujets lors de deux journées (première journée: exposition à la poussière de bois; deuxième journée: journée témoin) dont l'ordre était au hasard dans un intervalle maximum d'une semaine. Lors de chaque journée, après la mesure de la spirométrie et de la CP_{20} de base, les sujets ont été soit exposés à la poussière de bois ou n'ont pas été exposés à ce produit. La poussière de bois fut inhalée durant 30 minutes au total à des concentrations moyennes variant de 8.0 à 19.3 $mg \cdot m^{-3}$ (moyenne=11.5 $mg \cdot m^{-3}$). Vingt-cinq à 39.7% (moyenne=34.6%) des particules aérosolisées avaient un diamètre < 10 µ (diamètre permettant la déposition dans la trachée et l'arbre respiratoire inférieure). A la fin de chaque période d'exposition, le VEMS fut mesuré. Après récupération, la seconde CP_{20} fut obtenue. Des mesures sériées de VEMS furent effectuées à chaque heure jusqu à 6 heures après la fin de l'exposition. A ce moment, la CP_{20} fut remesurée. Un seul sujet a démontré une bronchoconstriction minime après la fin de l'exposition à la poussière de bois (changement maximum du VEMS de 14%), avec récupération complète après 10 minutes. L'inhalation de poussière de bois n'a pas modifié significativement la CP_{20} en comparant les résultats moyens du premier test avec le second et le troisième tests. Aussi, les changements moyens de la CP_{20} à chaque intervalle après l'exposition à la poussière de bois n'ont pas été significativement différents des variations spontanées de la CP_{20} lors de la journée témoin. Nous concluons que l'exposition aiguë à des particules de bois n'affecte pas le calibre ni l'hyperexcitabilité bronchiques de sujets asthmatiques.