Hanns Moshammer*, Shifra Shahraki, Thomas Mondel and Patrik Gebhart

Lung function and dust in climbing halls: two pilot studies

DOI 10.1515/reveh-2016-0024
Received July 12, 2016; accepted September 27, 2016; previously published online November 12, 2016

Abstract: In climbing halls, high levels of dust are found because magnesia powder is used to dry hands. Concerns have been raised about possible health effects after reports from asthmatics experiencing worsening of symptoms while or after climbing. We investigated acute and sub-acute effects of climbing in dusty halls on lung function in two pilot studies. The first study examined 109 climbers before and after a climbing activity that lasted at least 1 h. In the second study, 25 climbers from different age classes participated in a 2-day climbing competition. Of these, 24 agreed to take part in our investigation, but only 22 provided valid lung function tests on both days. The climbers underwent lung function tests before the first round of the competition (in the morning), after the second round approximately 3 h later and in the morning of the second day before the competition started again. In the first study, we found acute effects, a decline in lung function immediately after the exposure, likely due to protective reflexes of the bronchial muscles and stronger declines in persons with higher exhaled nitric oxide (NO) pre-climbing. In the second study, we also expected sub-acute effects on the next day due to inflammation. On the first day of the competition (second study), dust levels at a central monitor increased over time in a linear manner. Most of the dust was in the size range between 2.5 and 10 μm and dust levels of particulate matter (PM10) reached 0.5 mg/m^3. There was a decline in lung function over 24 h in persons with higher exhaled NO levels pre-exposure. All spirometric parameters were affected though the effects were not statistically significant in all cases. Younger age classes started earlier in the morning. Because of the increasing trend in dust levels we expected stronger effects with higher numbers but for the acute effects the reverse was true, possibly because younger climbers use magnesia more or with less experience thus causing higher individual exposure. No differences by age or by time of the first climb were observed for the 24-h lung function change.

Keywords: asthma exacerbation; atopy; eosinophilic inflammation; exhaled NO; magnesia dust.

***Corresponding author: Doz. Dr. Hanns Moshammer,** Institute Environmental Health, ZPH, Medical University Vienna, Kinderspitalgasse 15, 1090 Vienna, Austria,
Phone: +43 1 40160 34935,
E-mail: hanns.moshammer@meduniwien.ac.at
Shifra Shahraki, Thomas Mondel and Patrik Gebhart: Institute Environmental Health, ZPH, Medical University Vienna, Austria

Introduction

In climbing gyms as well as in other indoor sports halls a high dust load is often observed. This is primarily due to the use of magnesia to keep the palms dry for a strong grip. The dust is mainly in the coarse size fraction, i.e. consisting of particles with a diameter greater than 2.5 microns. However, there is also a fine fraction. The measured concentrations reach or occasionally even exceed occupational exposure limits for general respirable dust (1–4).

Magnesia has probably no significant specific toxicological properties. Health effects, if any, are therefore most likely due to the irritative (mechanical) effect of the dust particles on the mucous membranes. It is worth mentioning that the magnesium particles do not dissolve even at a relative humidity of up to 100% because of their very low solubility in water (1).

The respiratory tract is covered with a thin layer of "epithelial lining fluid". This serves not only as protection but is mainly composed of surfactant, which has been well documented to reduce surface tension. However, the surfactant also has the ability to displace particulate matter (PM) of less than 6 microns and thereby facilitate mucociliary clearance (5, 6).

Massive health consequences of exposure to dust are not typically reported by the climbers. In some cases, especially with those suffering from asthma or from hyperreactivity of the respiratory system, symptoms like cough and increased mucous production are described. These symptoms could possibly indicate an asthma attack during and after climbing in halls.

When the operator of a climbing hall experienced an asthma attack himself and when high dust levels were found in that hall we were asked to investigate the issue. We suggested a first pilot study to see if climbers experienced a

decline in lung function as an acute (and likely reversible) effect of dust exposure. We expected the strongest signal of an early response, if any, about 2 h after exposure. When we realised that only a few climbers stayed that long we conceived a second study to observe climbers over 24 h. Because of the special setting of that second study, only a small number of climbers could be included.

Methods

First pilot study

Dust concentration (PM10) was measured by a dust monitor (beta-gauge method, Thermo FH 62 C14) (Thermo Fisher Scientific, Waltham, MA USA) provided by the Carinthian Environmental Protection Agency. Although by this method a quasi-continuous measurement is possible, the instrument only stored results in 30-min intervals. The instrument was placed on a table in each hall at a central location on one wall. For each participating climber, the times were noted when he/she entered and left the hall. These times were equivalent with the spirometry taking. For the whole interval between the two time periods, the individual exposure was estimated by calculating a time-weighted average over all half-hour mean values included. Cumulative exposure was calculated by multiplying average exposure (in $\mu g/m^3$) with duration of exposure (in hours).

To better quantify the health effects of exposure to dust at first the respiratory physiology of climbers before and after climbing was assessed by spirometry using the portable instrument EasyOne (7). In principle, the examinations followed the ERS/ATS standards (8). The field conditions and also the lack of compliance by some of the climbers necessitated some relaxing of these standards. When after the three trials only one valid manoeuvre was produced, this was accepted after close inspection of the lung function curve. On the one hand, this procedure has likely increased non-differential measurement error. On the other hand, excluding all climbers that either before or after climbing could not produce two comparable and valid manoeuvres and would have substantially reduced the number of participants.

Because paired before-after differences were the outcome parameters of interest, inter-individual differences or pre-existing lung-function decrements did not influence the results. Thus, some source of variability could be reduced.

All climbers visiting each hall during the observation period were approached upon entering. The only exclusion criterion was visit duration of less than 1 h. The study protocol was explained (two lung function tests before and after the climbing session) and they were included in the study after they had provided informed and written consent.

However, the project ran into five serious problems as follows:

(a) It is not known how lung function is affected solely by the physical exertion while climbing. It was therefore planned to conduct a similar study of climbers outdoors whose dust exposure is negligible during climbing compared to that in the hall. However, the recruitment of climbers on the rock proved difficult, so this part of the project could not be completed.

(b) It had been expected that visitors of climbing gyms would usually stay for several hours per visit (9). In fact, the approached climbers often stayed for a much shorter time. Some finished their sports practice in less than an hour and therefore had to be excluded from the examination because this was deemed too short for an effect. Following an irritative stimulus, the airway response occurs in two distinct phases (10, 11): relatively quickly, the airways constrict by a contraction of the smooth muscle in the wall of the bronchi. This reaction is triggered by neural reflex arcs and corresponds to a physiological protective response. The relaxation of the muscles is caused mainly by the secretion of nitric oxide (NO) (12). In the second phase, there is an inflammatory swelling of the mucous membrane and increased mucus production. Inflammation is a result of mechanical and toxic damage to the mucosal epithelium, a reaction of the immune system and the nitrosative stress due to the secreted NO. This second phase of airway constriction begins a few hours after exposure and lasts after a single acute exposure for about 24 h. The second phase is pathophysiologically more relevant, but cannot be captured immediately after the (short-term) exposure.

(c) The acute reflectory constriction of the airways is likely not so much determined by the cumulative burden than the maximum peak exposure. The dust measurements in the climbing gyms were, however, recorded only as half-hourly average values and thus allow only a rough estimate of the peak exposure of each climber.

(d) In the first hall, the dust measurement failed on 2 days when climbers were examined. The exposures for these days, therefore, had to be estimated.

(e) This first hall is a boulder hall. It consists of contiguous low cellar rooms, all of which drain into a central larger hall. While most climbers use the niches in the individual cellars for climbing, the dust monitor was positioned in the central hall. Although it captured the average exposure throughout the climbing gym sufficiently well, it could not capture the larger spatial and temporal variability of the individuals' exposure.

In order to estimate the missing dust data, different relationships between measured dust per hall and the diurnal variation (modelled as a sine-cosine curve) and the number of climbers on each day was assessed in linear regression. The model with the logarithm of dust provided the best fit. The regression coefficients allowed the estimation of the average exposure in the hall during a particular half hour. But the short-term variation was likely underestimated by this approach. Because of the assumed higher spatial variability in hall 1 and because of the underestimation of temporal variability in the same hall a dummy variable for "hall" had to be included in the final regression models estimating the exposure effect on the change in lung function.

"Exposure" was assessed as "duration of exposure", "average concentration" and "cumulative exposure". The exposure was assumed to last from the end of the first to the start of the second lung function test (hours). Average concentration ($\mu g/m^3$) was the time-weighted exposure over all half-hour periods during which the exposure lasted. Cumulative exposure was average concentration time duration in hours.

The "change in lung function" was defined as the difference between the value before and the value after climbing. A positive value, therefore, indicated a decline in lung function. Because only the difference was assessed, individual factors (like body size and age) that affect lung function were accounted for. Because of

the coarse nature of the dust, mostly effects on the larger airways were expected, which are best represented by changes in the peak flow (PEF) and the total forced volume in the first second (FEV1). Resistance in the smaller airways is rather represented by the mid- and end-expiratory flows (MEF50, MEF25 and MMEF). A subgroup of climbers with hyperreactive airways (e.g. asthmatics) might also react to the fewer smaller particles and also to the mechanical irritation of the higher ventilation rate during exertion. Therefore, we also expected a stronger decline in the latter parameters in persons with higher exhaled NO (as a sign of eosinophilic inflammation or atopy) before climbing (13, 14). Exhaled NO was measured in both studies with NObreath from Bedfont (Bedfont Scientific Ltd, Harrietsham, Maidstone, Kent, UK) (15) following the guidelines in the user manual and the ATS/ERS standards (16).

Statistical analysis was performed in STATA SE 13.1 (StataCorp LP, College Station, TX, USA). Besides descriptive statistics differences in parameters (before-after) were regressed against each exposure parameter separately. "Hall" was included as a dummy variable in each model. Also in additional models, the effect of NO concentration before climbing was examined.

The examination was approved by the Ethics Committee of the Medical University of Vienna. All climbers provided signed informed consent.

The second pilot study

To study climbers approximately 24 h after their climbing experience, a 2-day climbing championship (a marathon climbing competition) was organised in another climbing hall. Twenty-five climbers from different age-classes participated in the competition. Of these, 24 agreed to take part in our investigation, but only 21 provided valid lung function tests on both days. The climbers underwent lung function tests before the first round of the competition (in the morning), after the second round approximately 3 h later and in the morning of the second day before the competition started again. While during the first pilot study, only one dust monitor was operated that only measured the dust concentration every 30 min; in the second study also another monitor was used (Grimm Spektrometer 1.108, laser-scatter technique) (GRIMM Aerosol Technik GmbH & Co. KG, Ainring, Germany) (17). This allowed for a comparison between the two methods, a higher temporal resolution, and the separate estimation of coarse (2.5–10 μm) and fine particles (< 2.5 μm diameter). The monitors were placed on an observation platform at the middle height of the hall and near the right-hand side of the climbing wall.

In each round of climbing, the younger climbers started first. Every 5 min, a new climber entered the competition that consisted of four climbing courses that had to be mastered consecutively within 3 min each. Three rounds had to be performed on the first day and two more on the second.

The competition started with the youngest age group. So starting order was a proxy for age and, because of increasing dust concentration over time, also of exposure during climbing. Because of the small number of participants, multivariate regression models were not feasible. Instead, the impacts of starting number and of NO concentration before climbing on the before-after difference were assessed in separate models.

The second study was approved by the Ethics Committee of the Carinthian Hospital Association, and all climbers provided signed informed consent.

Table 1: Characteristics of the halls and climbers (of the first study).

	Hall 1[a]	Hall 2	Hall 3
Number of climbers, male	46 (45)	20 (18)	43 (40)
Dust concentration, μg/m³[b]	245.1 (85.0)	92.5 (47.1)	334.5 (191.0)
Exposure duration, h[b]	1.5 (0.5)	2.2 (0.8)	1.8 (0.5)
Cumulative exposure, μg h/m³[b]	373.1 (199.2)	204.8 (111.9)	628.2 (476.8)
Age, years[b]	25.9 (7.6)	27.9 (6.3)	27.5 (6.6)
Height, cm[b]	180.7 (7.5)	178.6 (7.4)	178.7 (5.2)
Weight, kg[b]	75.8 (9.1)	72.0 (9.2)	73.0 (7.1)
FVC before, l[b]	5.7 (0.9)	5.3 (1.1)	5.7 (0.7)
FEV1 before, l[b]	4.6 (0.7)	4.3 (1.0)	4.5 (0.7)
PEF before, l/s	10.4 (1.9)	10 (2.1)	9.9 (1.5)
MEF25 before, l/s	2.3 (1.0)	2.2 (0.9)	2.1 (0.9)
MEF50 before, l/s	5.4 (1.6)	4.8 (1.6)	5.0 (1.1)

[a]Hall 1 was the boulder hall where some dust data were missing and had to be estimated. [b]Mean (standard deviation).

Results

The first study

A total of 120 climbers were recruited in three climbing gyms (Table 1). Of these, 109 provided valid lung function tests before and after climbing for at least 1 h. The interval between the two spirometric tests was on average 1.8 h (standard deviation 0.6 h). Their average exposure ranged from 38 to 722 μg/m³ (mean ± standard deviation: 252 ± 158) and the cumulative exposure was 443 ± 365 μg h/m³.

After controlling for "hall", most of the lung function changes displayed a positive association with all three exposure variables, although this association only rarely reached significance. Surprisingly, this association was often strengthened upon additionally controlling for the change in exhaled NO (Table 2). The effect was most pronounced for FEV1. Exhaled NO before climbing predicted decline in some lung function parameters indicative of increased resistance in the smaller airways.

The second study

This study was organised in collaboration with a climbing competition and so only a small number of climbers participated and could be recruited. Only 21 climbers provided valid lung function test on all three occasions. Dust levels increased, beginning in the morning of the first day

Table 2: Results of linear regression of exposure metrics on the differences in lung function values (before-after exposure).

	Diff FVC (l)	Diff FEV1 (l)	Diff MEF25 (l/s)	Diff MEF50 (l/s)
After controlling for "hall"				
Average concentration[a]	0.02 (0.16)	0.03 (0.07)	−0.04 (0.25)	0.03 (0.49)
Duration	1.5 (0.15)	**1.88 (0.02)**	**5.8 (0.004)**	2.2 (0.46)
Cumulative exposure[a]	0.01 (0.16)	**0.01 (0.05)**	−0.004 (0.78)	0.01 (0.56)
Additionally controlling for Diff NO				
Average concentration[a]	0.03 (0.12)	**0.03 (0.04)**	−0.04 (0.3)	0.04 (0.47)
Duration	1.6 (0.14)	1.92 (0.18)	**5.8 (0.003)**	2.2 (0.46)
Cumulative exposure[a]	0.01 (0.12)	**0.01 (0.03)**	−0.002 (0.85)	0.01 (0.55)
Without controlling				
NO before	−0.005 (0.12)	0.002 (0.42)	**0.014 (0.03)**	**0.018 (0.05)**

[a]per 100 µg/m³ (and hour for the cumulative exposure). Beta coefficient of linear regression (p-value). **Bold**: $p < 0.05$.

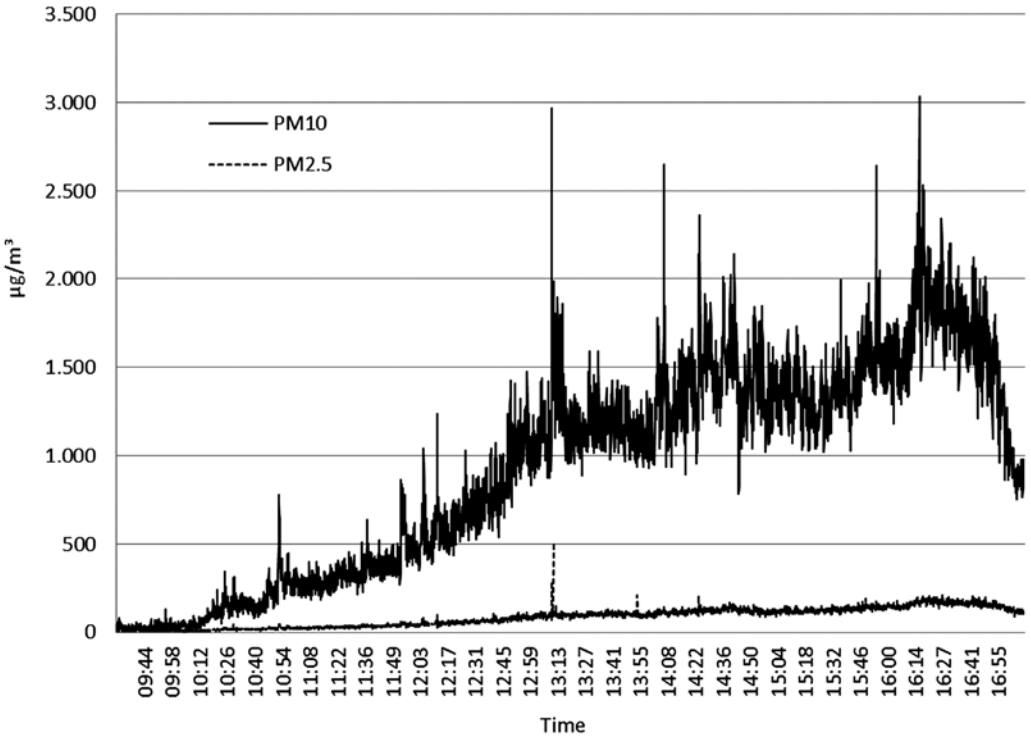

Figure 1: PM10 and PM2.5 according to the laser-scatter technique (Grimm, in µg/m³) on the first day of the second study from 9:30 in the morning until 5 o'clock in the evening (x-axis).

until mid-afternoon when the first-day parts of the competition ended (Figure 1). According to the Grimm measurements, the dust mainly consisted of coarse particles. Half-hour mean values of the two monitors during that period were highly correlated with each other ($R^2 = 0.81$), but the Grimm monitor reported substantially higher values. For PM10 the ratio was roughly 1:4 so that according to the Grimm PM10 reached about 2 mg/m³ in the afternoon while according to the beta-gauge method the highest half-hour mean was about 0.5 mg/m³ only.

Twenty-four climbers participated on the first day and five of these had high (>40 ppm) NO concentrations in the exhaled air before climbing. Three of these climbers did not show up on the next day. The five climbers displayed the strongest declines in the same-day comparisons although the findings were far from any statistical significance. There was also a decline in lung function over the 24 h in persons with higher exhaled NO levels pre-exposure. All spirometric parameters were affected though not in all cases the effects were statistically significant. The effect was mostly driven by the few persons with exhaled NO above a cut-off value of 40 ppb (Figure 2). They also reported being asthmatic. The effect was most pronounced for PEF.

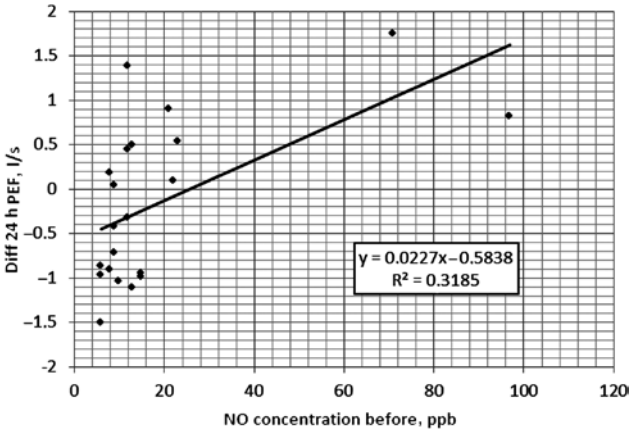

Figure 2: Association between exhaled NO before climbing and the difference in PEF after 24 h.

Table 3: Results of linear regression of the starting order in the competition of the second study on the differences in lung function values (before-after exposure).

Parameter	Coefficient	p-Value
Diff FVC (l)	−0.004	0.931
Diff FEV1 (l)	−0.009	0.073
Diff MEF75 (l/s)	−0.036	0.206
Diff MEF50 (l/s)	−0.022	0.234
Diff MEF25 (l/s)	**−0.030**	**0.045**
Diff MMEF (l/s)	**−0.033**	**0.039**

Bold: $p < 0.05$.

Over 3 h a stronger decline was observed in those starting earlier or who were the youngest (Table 3). This decline was significant for MEF25 and MMEF only. Controlling for exhaled NO before climbing did not substantially alter that result. No differences by age or by time of the first climb were observed for the 24-hour lung function change.

Discussion

Dust in climbing halls has a very special composition very different from other (environmental) respirable dust exposure (1–4). Although in the case of these studies no physicochemical analysis of the dust was performed, there is no doubt that the studies are relevant for this special kind of dust. The laser-scatter technique clearly indicated the predominance of coarse (2.5–10 μm) dust in our second study.

These were two pilot studies with inherent shortcomings. In the first study, only short-term changes in lung function could be investigated and it was not possible to repeat the lung function tests later, e.g. 24 h after the exposure. Because of the nature of a field study, lung function tests had to be performed under less than the optimal conditions. Nevertheless, there was an indication of a stronger reduction in lung function (especially for FEV1) with higher exposure, no matter how exposure was defined. Surprisingly, the model fit improved and the point estimates of the effects of exposure grew stronger and more precise when controlling for the change in exhaled NO. This change by itself negatively correlated with the change in lung function values (although only rarely significantly so). It seems a faster increase in exhaled NO in response to an irritative stimulus protects against (prolonged) lung function decline. This would indicate some systematic individual variation in response to irritation not covered by exhaled NO pre-exposure.

Some damage to the epithelium of the airways because of exposure to PM is expected and is mainly triggered by oxidative stress (18, 19). Acute effects of particles of different origins on lung function and respiratory symptoms have been demonstrated in several occupational settings, but most notably in cement workers (20–23).

Even more noteworthy was the observation of increased resistance in the smaller airways if the persons already had higher exhaled NO before they started climbing. Higher exhaled NO can signify eosinophilic inflammation which is a hallmark of atopic asthma. It is not so surprising that persons with asthma or hyperreactive airways display a stronger response. Nevertheless, we cannot discern if this was a response to the high dust exposure or to the bare physical stimulus of an increased ventilation rate. It is well known that asthmatics can even experience an attack during or after physical exercise.

The second study tried to remediate the shortcoming of the first as it was organised in a way that enabled us to collect lung function data 24 h later as well. Due to the special circumstances of the competition, only a small group of participants could be included though. Two different dust monitors were operated during the competition that reported quite different mass concentrations, although with high temporal correlation. Both monitoring methods do not measure the mass of particles directly but make use of optical properties of the dust (light scattering) or of the attenuation of beta rays from a C14 source. Both monitoring results might not be accurate for such a special composition of the dust. Nevertheless, even the lower concentration reported by the beta-gauge technique is rather high compared to environmental standards.

In this second study, an increase in airway resistance was again observed in persons with higher exhaled NO pre-exposure although this time the effect was more

pronounced after 24 h and in the larger airways (PEF). After 24 h such a response cannot be interpreted as reflectory or protective but would indicate some adverse inflammatory processes. On the other hand, the decline in lung function was not large even in the few persons with the highest exhaled NO that also had reported to be asthmatic and atopic. Past experience of climbers in climbing halls do not indicate a massively adverse effect, although some asthmatic climbers reported increased symptoms like coughing or even asthma attacks during or after climbing.

Dust levels during the second study increased over the course of the first day. But contrary to the hypothesis, not those who started the climbing competition later (when average dust levels in the hall where highest) experienced the strongest lung function declines. On the contrary, those who started first displayed stronger declines. We might put forward the hypothesis that this was not due to their early start, but due to their lower age and thus poorer climbing experience. The younger children indeed were seen to use more magnesia powder and to exert themselves more while climbing maybe out of nervousness or of sheer inexperience. Unfortunately, the design of the whole competition does not allow differentiating between the effect of starting time and age and experience. But again the observed effects were small and in this case did not last over 24 h but were visible only 3 h after the start of the climbing. An alternative explanation of the stronger effects in the younger climbers is also possible. For the first examination, the climbers first queued up before the nurse's room where we did our spirometry tests. Then they went into the climbing hall and they usually stayed there also after they had completed their first climbing round. So indeed the earlier or younger climbers could have been exposed for a somewhat longer time. For our second examination (between the 2nd and the 3rd round) we had to go and call the climbers from the hall for the spirometry examination.

Nevertheless, the observed effects indicate a need for further research. Climbers should be observed after a longer or more intensive exposure. Longer-term or chronic effects in some high-risk groups like asthmatics should be evaluated. The athletes should also be equipped with a personal monitor to better detect individual (cumulative as well as peak) exposure.

Although the observed effects appear not very pronounced on average, for a few sensitive individuals the dust exposure could be relevant for their future health. Therefore, there is a need for further research how the dust can be reduced in climbing gyms or in sports practice. In particular, a better ventilation regime, more efficient cleaning of the halls (to avoid resuspension of older dust) and a modified formulation of the adhesive seem feasible paths to consider (24).

Acknowledgments: The dust measurements in both studies were performed by the Carinthian Environmental Protection Agency. Also, their financial support for the instrumentation (EasyOne) is gratefully acknowledged. The authors report no conflicts of interest.

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