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RESEARCH ARTICLE

Pulmonary toxicity following exposure to a tile coating product containing alkylsiloxanes. A clinical and toxicological evaluation

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Context. Coating products are widely used for making surfaces water and dirt repellent. However, on several occasions the use of these products has been associated with lung toxicity. *Objective.* In the present study, we evaluated the toxic effects of an aerosolized tile-coating product. *Methods.* Thirty-nine persons, who reported respiratory and systemic symptoms following exposure to the tile-coating product, were clinically examined. The product was analysed chemically and furthermore, the exposure scenario was reconstructed using a climate chamber and the toxicological properties of the product were studied using *in vivo* and by *in vitro* surfactometry. *Results.* The symptoms developed within few hours and included coughing, tachypnoea, chest pain, general malaise and fever. The physical examination revealed perihilar lung infiltrates on chest radiograph and reduced blood oxygen saturation. The acute symptoms resolved gradually within 1–3 days and no delayed symptoms were observed. By means of mass spectrometry and X-ray spectroscopy, it was shown that the product contained non-fluorinated alkylsiloxanes. The exposure conditions in the supermarket were reconstructed under controlled conditions in a climate chamber and particle and gas exposure levels were monitored over time allowing estimation of human exposure levels.

Mice exposed to the product developed symptoms of acute pulmonary toxicity in a concentration-and time-dependent manner. The symptoms of acute pulmonary toxicity likely resulted from inhibition of the pulmonary surfactant function as demonstrated by *in vitro* surfactometry. Among these patients only a partial association between the level of exposure and the degree of respiratory symptoms was observed, which could be because of a high inter-individual difference in sensitivity and time-dependent changes in the chemical composition of the aerosol. *Conclusion.* Workers need to cautiously apply surface coating products because the contents can be highly toxic through inhalation, and the aerosols can disperse to locations remote from the worksite and affect bystanders.

Keywords   Aerosols; Respiratory disorder; Sealing product; Coating product; Occupational exposure

Introduction

Worldwide, numerous cases of pulmonary injury have been reported after application of spray products for surface coating of leather, textiles, glass and tiles.[1–4] Most cases occurred after indoor use of consumer products in spray cans, usually causing only one or few cases per exposure event, but larger outbreaks have been reported.[1,4–7] Cases are usually benign with spontaneous recovery in less than 48 h,[1,5–7] but severe and even lethal cases have also been reported.[8,9] The toxicological mechanisms are only partly understood; a pathophysiological mechanism including a reaction to lung surfactants causing alveolar collapse, interstitial inflammation and oedema has been suggested.[3,10,11]

In general, spray products for surface coating contain fluorinated acrylates or siloxanes as the active compound, one or more solvents, stabilizers and in some cases also a propellant. The often relatively complex composition of these products, may lead to a broad range of respiratory effects, including early upper airway irritation as well as delayed effects in the lower airways.[2,7] Outbreaks have been related to both types of the active compounds[2,7] but also the physico-chemical characteristics of the solvent applied have showed to play a crucial role for the toxicity of the product.[10] Outbreaks have

occurred in relation to changes in the composition of both solvents and active compounds of a product suggesting that both are important for the observed toxic effects.[7,12]

We describe an incident during renovation of a supermarket in Greenland where workers, supermarket employees and customers developed acute respiratory symptoms following exposure to an aerosolized tile-coating product. The physical and chemical characteristics of the generated aerosol were subsequently evaluated and its toxic effects assessed *in vivo* and *in vitro*. The laboratory work was carried out to gain insight into the toxicological mechanisms. Identification of the chemical composition of the product is necessary to generalize from the specific product used in the present case to other generic products.

Methods

Workplace exposure

$50 \, m^2$ of a $300 \, m^2$ tile-floor at the ground level of a supermarket was sprayed with 30 L (22.8 kg) of the tile-coating product Stain Repellent Super® (SRS, Akemi GmbH, Nürnberg, Germany) for 30 min by two workers, who used an airless spray gun (Graco Minimax®) equipped with a model 411 nozzle (diameter = 279.4 µm). The outdoor average humidity was 50%. The indoor temperature of the two levels in the store was not measured but was approximately 18–22°C. According to the manufacturer's technical instruction sheet (www.akemi.de), the product was supposed to be applied using a brush, paint roller, mop or a low pressure airless spray gun with a maximal pressure less than 1 bar. However, in this case the product was applied using a high pressure (135 bar) airless spray gun. The ground area of the two-storey building was $900 \, m^2$ with a ceiling height of 4 m yielding a total volume of $7200 \, m^3$. Upon spray application, a large fraction of the product will immediately be adsorbed to surfaces, but the concentration of aerosolized product is assumed to be high, especially near the spraying site. SRS aerosols were visible for hours and spread to the first floor containing the supermarket through open spaces, comprising approximately 10% of the floor separation, and further translocated to the office area at the same floor. The day after the episode, surfaces in most of the building were visibly contaminated by the product.

The 39 persons exposed were present for 10–150 min during and/or after the spraying event in the building. At ground level, three men worked in close vicinity to two persons operating the spray gun and all five were present during the spraying. After the spray event, 25 persons were exposed for 30–150 min at ground level or at the above floor close to ground level apertures. The remaining nine persons were exposed for less than 30 min on the first floor. None of the 39 exposed were wearing eye or respiratory protection, and the mechanical ventilation system of the building was turned off.

Patient data

Upon arrival to the small hospital in the local town, 1–6 h after the spraying, the patients were clinically evaluated, but vital parameters and symptoms were not systematically registered. Upon arrival to the larger Dronning Ingrids Hospital (DIH) in Nuuk, 12 h after the spraying, all the patients were interviewed using a structured questionnaire and had a standardized clinical examination as well as an arterial blood gas and plain radiograph of the chest. The information about symptoms and clinical data were extracted from the hospital records.

Two months after the exposure, the patients were interviewed using a structured questionnaire, and all the patients had a standardized clinical follow-up including a pulmonary function test, a plain radiograph of the chest, and an exercise test on a stationary bicycle with a blood oxygen saturation measurement. The patients who had had an abnormal plain chest radiograph two months earlier and all patients that reported symptoms at the follow-up had a plain radiograph of the chest taken at the follow-up examination.

Chemical analyses

According to the material safety data sheet (MSDS), the SRS product contains hydrogenated naphtha (50–100%). Analyses of the product by mass spectrometry (MS) confirmed the content of C_9–C_{13} alkanes. Consequently, n-decane (C_{10}) was used as a reference solvent for the simulated workplace exposure scenario and *in vitro* surfactometry. The chemical analysis by MS showed presence of alkylsiloxanes, while fluorinated compounds were not detected in the product (WD-XRF, data not shown). For further experimental details, cf. supplementary material available online at http://informahealthcare.com/doi/abs/10.3109/15563650.2014.915412.

Animal study

Inbred BALB/cA male mice aged 5–7 weeks, were purchased from Taconic M&B (Ry, DK) and were housed as described.[13] Treatment of the animals followed the procedures approved by The Animal Experiment Inspectorate, Denmark (No. 2012-15-2934-00616-C1). Using a nose-only inhalation chamber,[14] the mice (n = 10/group) were exposed to an aerosol of the SRS product until effects on the respiratory parameters were observed (10–60 min). The generated concentration of the product was calculated by dividing the mass of aerosolized product by the volume of the dilution air. For each experiment, the mice were placed in body plethysmographs in the exposure chamber head-out-only.[15] Data acquisition software (Notocord Hem, Notocord Systems SA, Croissy-sur-Seine, FR) was used to collect respiratory parameters. Prior to exposure, a 15-min baseline period was recorded for each mouse. To assess exposure-related effects, the respiratory parameters during exposure were compared to baseline levels, that is, each mouse served as its own control. The acquisition software measured several breathing parameters including respiratory frequency, tidal volume and time of break, which is a specific marker of upper respiratory tract irritation. Comprehensive descriptions of the breathing parameters have been made elsewhere.[16–18] Data acquisition and calculations were performed as described previously.[14]

The mice were euthanized immediately after the experiments.

In vitro *surfactometry*

The pulmonary surfactant formulation HL10 (porcine) was dissolved in a phosphate buffer. The solution was mixed with either n-decane (a component in the solvent mixture used in the SRS product) or the SRS product. Following incubation, the pulmonary surfactant function was measured using a Langmuir-Wilhelmy film balance. For further experimental details, cf. supplementary material available online at http://informahealthcare.com/doi/abs/10.3109/15563650.2014.915412.

Simulated workplace exposure scenario

In order to simulate the occupational exposure scenario, experiments were conducted in a 20.3 m^3 chamber (air exchange rate, 1.0 h^{-1}) using a 1-compartment model. 2 m^2 of ceramic tiles placed on the chamber floor were sprayed with the SRS product using an airless spray gun identical to the one used in the supermarket. The working pressure was up to 135 bar, which is significantly higher than recommended by the producer of the SRS product (max 1 bar overpressure). The higher pressure was expected to result in generation of smaller particles. Emitted volatile organic compounds and particle concentrations were measured up to 19 h after the spraying. For further experimental details, cf. supplementary material available online at http://informahealthcare.com/doi/abs/10.3109/15563650.2014.915412.

Results

Patients

Within 1–6 h after the spraying, 43 persons contacted the hospital because of respiratory symptoms. All had been exposed to the SRS aerosols, none had worn eye- or respiratory protection and none had changed their working clothes after leaving the worksite.

Forty of these, including all 18 workers, were considered significantly clinically affected and evacuated by plane to the larger national hospital, DIH, because the hospital in Maniitsoq was equipped and staffed only for basic medical procedures. One declined; thus the case series comprised 39 persons; 29 males, median age 33 years (range: 15–59 years), all without previous medical records except for two patients, one with mild asthma and one with ischaemic heart disease (NYHA II). 27 were smokers, median pack years 7 (range: 1–40). 27 were Inuit, the rest were Danish Caucasians. The evacuation went well and the 39 patients arrived at DIH 12 h after the spraying event. All 39 patients had an onset of respiratory symptoms within the first hour from exposure and within 1–12 h they complained of coughing (39/39), shortness of breath (29/39), chest pain (8/39), general malaise (19/39) and headache (18/39). Many patients had tachycardia (15/39) and tachypnoea (17/33) and two-third of the patients had a temperature rise above 38°C. None of the patients had upper respiratory tract manifestations or eye irritation. Within the first 12 h after the spraying event the body temperature normalized on all patients. None of the patients had any worsening of symptoms after 12 h and there were seen no delayed effects in the following days. Additional clinical data are given in Table 1.

Three patients differed markedly from the rest by having severe respiratory symptoms. These patients were either high-dose and short-time exposed ($N = 1$) or medium-dose and long-time exposed ($N = 2$). Their respiratory frequencies were 18–40 breaths/min (normal value: 12–18). The oxygen saturation without supplemental oxygen was 76–85% (normal value: 95–100%). SaO$_2$ on arterial blood gas analyses with 5 L/min supplemental oxygen on nasal prong was 95–97% (normal value: 95–100%) and PaO$_2$ was 9.7–11.4 kPa. (normal value: 10–12 kPa). Two of the three patients showed bilateral perihilar infiltrates on plain radiograph of the chest. Two had leucocytosis at 14.8 and 15.1×10^9/L, respectively (normal value: $3–9 \times 10^9$/L) and elevated C-reactive protein at 76 and 87 mg/L, respectively (normal value: 0–10 mg/L). After 48 h, the patients could manage without oxygen supplements, and after 72 h, they were clinically unaffected. No mechanical ventilation was needed.

One of the three patients had operated the spray gun and was exposed for 30 min during this work. He had no recognized disease, but a history of 35 pack years of smoking. The other two worked at the same level of the building for 150 min after the spraying, but were not in the area during the spray

Table 1. Selected clinical data on 39 patients exposed to aerosols from a tile-coating product containing alkylsiloxanes.

Parameter	Median (Range)	Reference values
Respiratory symptoms	39/39	
Body temperature (°C)	38.1 (37.5–39.4)	36.5–37.5
Heart rate (min^{-1})	95 (61–120)	60–80
O$_2$ saturation (%) (pulse oximetry) (No supplemental oxygen)	97 (76–100)	95–100
Respiratory frequency (min^{-1})	19 (12–40) ($N = 33$)	12–18
PaO$_2$ (kPa) (with 0–5 L. Oxygen)	11.2 (8.8–21.7) ($N = 35$)	10–12
PaCO$_2$ (kPa)	5.2 (4.1–5.9) ($N = 35$)	4.7–6.0
Abnormal plain chest radiograph	7/39	

The most unusual findings within 1–12 h after initial exposure to aerosols from a tile-coating product containing alkylsiloxanes.

event. One of these had a history of 35 pack years of smoking and the other had ischemic heart disease (NYHA II).

The remaining 36 patients all had dry cough and most experienced shortness of breath. Their oxygen saturation was normal, median: 98% (range: 95–100%). Five had bilateral perihilar infiltrates on the plain chest radiography, all five had been working close to the spraying area, two of them as bystanders while spraying and three of them had been working at the ground level for 120–150 min. After 24 h, virtually all symptoms had subsided, the body temperature, respiratory frequency, pulse rate and blood gases had normalized.

Only one of five high-dose exposed subjects, who operated or worked in the vicinity of the spray gun during the spray application were among the most clinically affected group, while two of the large group of medium-dose exposed were severely affected.

Treatment

Patients were administered supportive therapy, including supplemental oxygen and inhaled glucocorticoid.

Follow-up

At a clinical follow-up two months after the incident, 15 out of 36 examined patients still complained of shortness of breath during hard physical work. All had normal clinical examination and lung function test, normal O_2 saturation and all were able to increase the O_2 saturation during exercise on a stationary bicycle. All plain radiographs of the chests were normal, and the perihilar infiltrates observed on the abnormal plain chest radiographs seven weeks earlier had resolved. The two patients with known comorbidity (mild asthma and ischemic heart disease [NYHA II]) did not have any need to increase their usual medication. Three patients had moved to Denmark; one was extensively evaluated because of a continuous feeling of dyspnoea, however, with normal lung function, normal carbon monoxide diffusion capacity test (DLCO) test and normal plain radiograph of the chest. The other two were lost for follow-up.

Toxicity of inhaled SRS aerosols in mice

Exposure to aerosolized SRS induced a concentration- and time-dependent decrease in the tidal volume. No further effect was seen on the tidal volume after 60 min of exposure to 59 mg/m^3, a 35% reduction was observed after exposure to 76 mg/m^3 for 60 min and a 50% reduction was seen after a 30-min exposure to 103 mg/m^3 (Fig. 1a). Further increase in exposure concentration markedly reduced the exposure time needed to reach a 50% reduction in the tidal volume (Fig. 1a). The reduction in tidal volume was associated with a reduction in expiratory flow rate (data not shown). Also, the reduction in the tidal volume was associated with an increase in the respiratory frequency (Fig. 1b).

After 60 min of exposure, or when a 50% reduction in tidal volume was reached, the mice were exposed to laboratory air for 30 min in order to assess the reversibility of the response. The reduction in tidal volume did not resolve

within the 30-min recovery period (Fig. 1a). In contrast, the increased respiratory frequency gradually approached the pre-exposure baseline level during the recovery period (Fig. 1b). Based on breathing pattern analyses (time of break), no irritation of the upper airways was detected even at the highest concentration of SRS, 5700 mg/m^3 (data not shown). After end of the recovery period, mice exposed to concentrations 76 mg/m^3 or more showed general signs of intoxication including piloerection and reduced mobility.

Assessment of pulmonary surfactant inhibition in vitro

Compression–expansion isotherms were obtained using a Langmuir-Wilhelmy film balance. The pulmonary surfactant formulation incubated with the control solvent (n-decane) reached a mean surface pressure of 71 mN/m at maximum compression (Fig. 2). This corresponds to a surface tension of 1 mN/m, as the surface tension of pure water at 25°C is 72 mN/m. Thus, the surfactant was able to reduce the surface tension to the near-zero value necessary for a normal lung function. In contrast, pulmonary surfactant incubated with the SRS product reached a surface pressure of only 52 mN/m at maximum compression, which corresponds to a surface tension of 20 mN/m, that is the SRS product significantly inhibited the surfactant function.

Exposure assessment from chamber simulation

A total ion chromatogram of an air sample collected on Tenax TA in the time interval from 1 to 11 min after spraying is shown in (Fig. 3). The broad peak at the retention time interval from approximately 9 min to approximately 21 min was assigned as a mixture of hydrocarbons ranging from C_9 (e.g. 3-methyl octane) to C_{13} (e.g. 3,4-dimethyl undecane). The peak observed at 22.2 min. was assigned as isooctyl trimethoxy silane, while the peak at 29.1 min was an unknown alkyl silane.

The total concentration of airborne hydrocarbons (n-decane equivalents) decreased over time. The concentration was 563 mg/m^3 immediately after the spraying, 438 mg/m^3 1 h later and 184 and 34 mg/m^3 after 3 and 19 h, respectively.

Figures 4a and 4b show the temporal evolution of the particle size distribution and the total concentration, respectively. The majority of the observed particles were formed during the actual spraying process, and were in two main size modes; a smaller mode located around 60 nm and a larger mode at 750 nm. Just after spraying, the maximum particle concentration was reached, approximately 1.4×10^5 particles per cm^3, and afterwards it exponentially decayed because of air exchange, wall deposition and sedimentation.

The alveolar deposited mass was calculated, under the assumption of unit density and spherical shape, for three different periods of exposure (Table 2). During the first 15 min after the spraying, the calculated alveolar deposited dose was 1.64 mg. An additional 45 min of exposure almost doubled the alveolar deposition (Table 2). A prolonged exposure scenario of 150 min only increased the deposited mass slightly
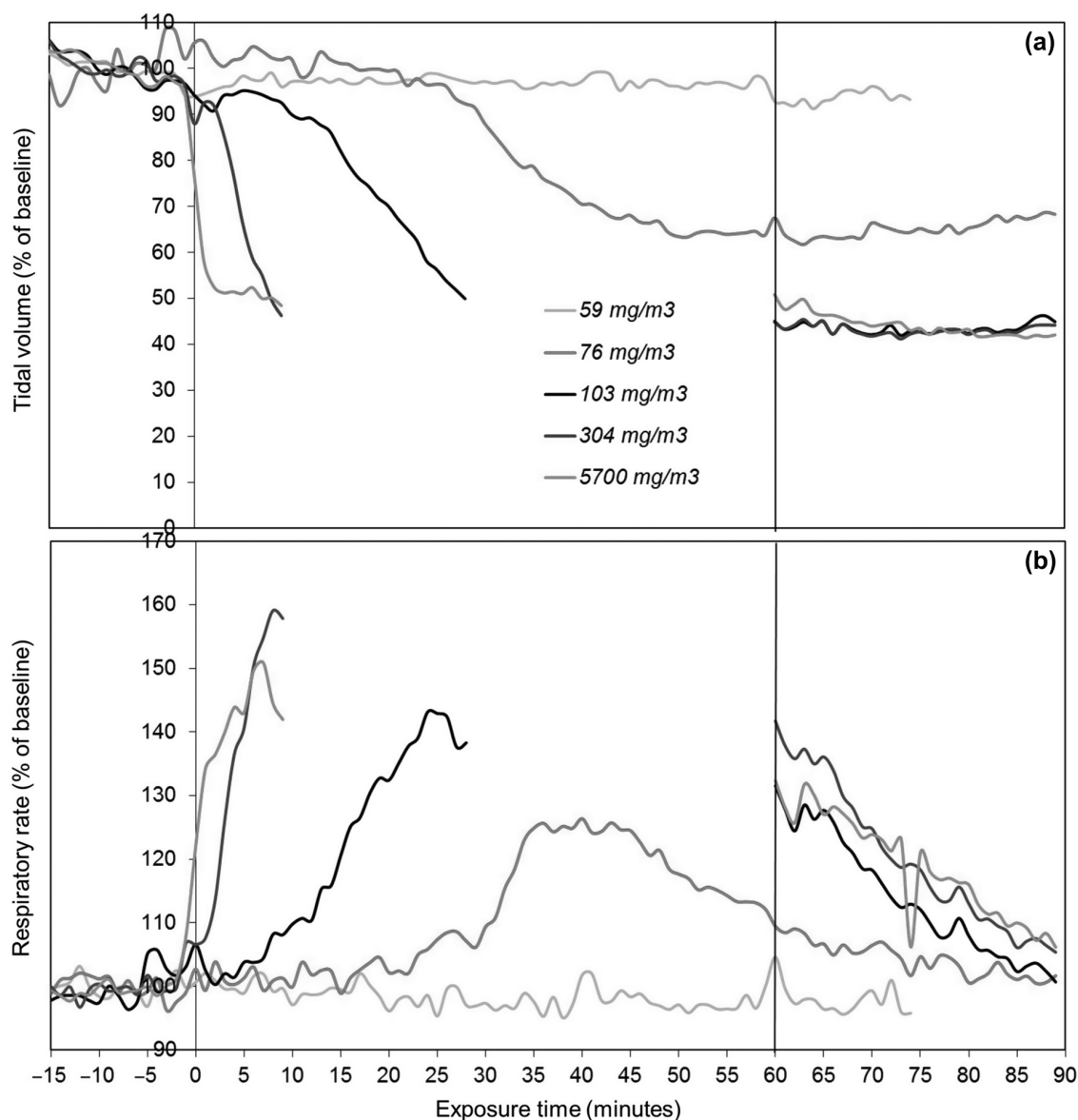
Fig. 1. Concentration- and time-dependent effects of inhaled SRS product on the tidal volume (a) and respiratory rate (b) in mice ($n = 10$ per exposure). Each study started with a 15-min pre-exposure baseline period, followed by a 10- to 60-min exposure period. The exposure period is followed by a 30-min recovery period. Generated concentrations are given (colour version of this figure can be found in the online version at www. informahealthcare.com/ctx).

because of the low mass concentrations remaining 60 min after the spraying event (Fig. 4).

Discussion

We report an outbreak of acute respiratory distress in bystanders and workers from the spray application of a surface coating product without the use of respiratory protective equipment. According to the manufacturer's instruction, the SRS product could be applied using a brush, paint roller, mop or a low pressure airless spray gun. However, the product was incorrectly handled and was sprayed on by using a high-pressure airless spray gun, which formed a high concentration of small droplets.

Large amounts of the SRS product were aerosolized and spread into the indoor environment, contaminating the air

of a two-level building. Air concentrations reached levels high enough to cause pulmonary symptoms even in persons far from the primary source of contamination. The symptoms reported included fever, tachycardia and tachypnoea, reduced blood oxygenation and acute changes on plain radiograph of the chest. The symptoms are comparable to what has been reported in other case series with aerosolized coating products.[6,7]

The dose–response relation between exposure and symptoms in these patients was not straight forward. For example, there was only one out of five high-dose exposed subjects in the most clinically affected group, while two of the large group of medium-dose exposed were severely affected. This may partly be because of variations in individual vulnerability of the subjects. This is suggested by increased age and cardiac comorbidity in one of the most severely affected
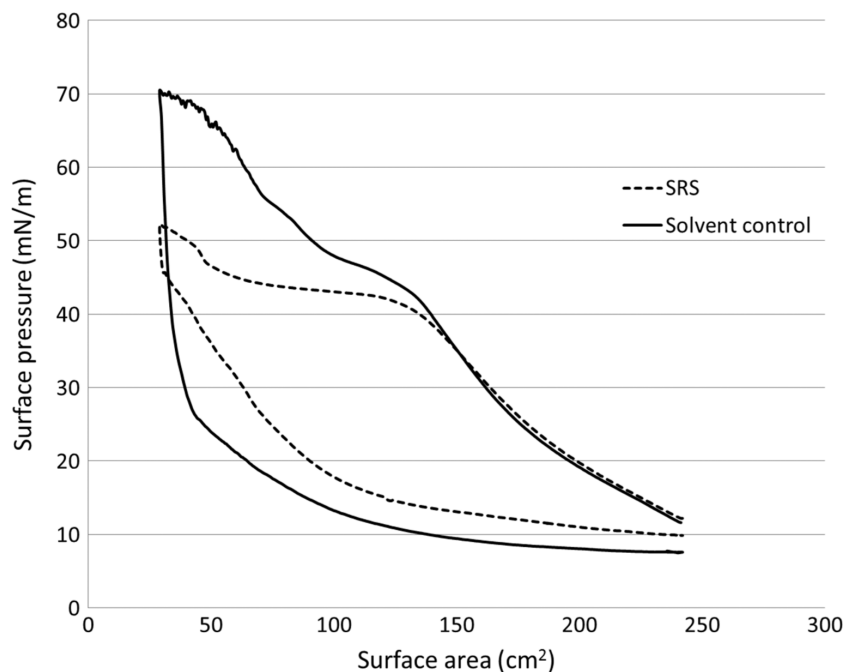
Fig. 2. Langmuir isotherms. Pulmonary surfactant incubated with either SRS product or n-decane (solvent control) is transferred to a KSV mini trough with an area of 243 cm^2. A reduction of the trough area (compression of the surfactant film) results in an increase in the surface pressure (equal to a reduction of the surface tension). For simplicity and because of loss of surfactant to the subphase during the compression cycles, only the second of three compression isotherms of solvent- or SRS-incubated HL10 are presented.

patients and increased age and 35 pack years of cigarette smoking in another. Both were not present during the spray event, but worked in the fog of aerosols for 3 h afterwards along with 16 other workers who were presumably equally exposed in relation to both exposure time and concentration/distance to source.
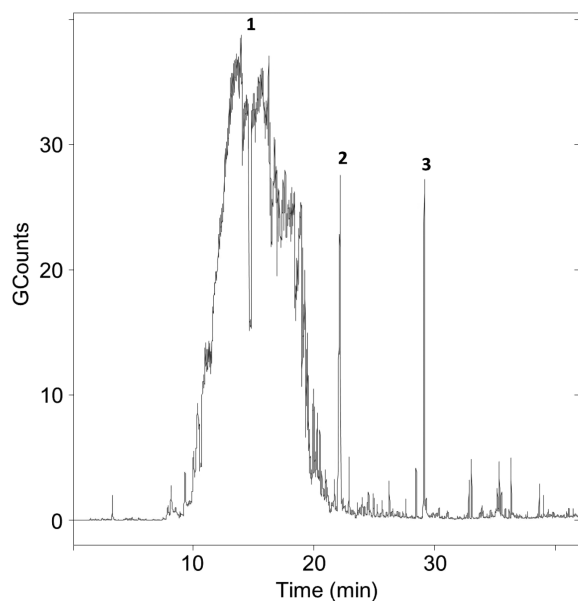

Fig. 3. Total ion chromatogram showing VOCs emitted after spraying of 83.6 g SRS product at a 2 m^2 surface of ceramic tiles inside a 20.3 m^3 chamber. The major peaks 1 and 2 are assigned as: C_9–C_{13} hydrocarbons (solvent) and isooctyltrimethoxy silane, respectively. Peak 3 is an unknown organosilane (colour version of this figure can be found in the online version at www.informahealthcare.com/ctx).

One limitation of the present study is the lack of accurate exposure information from the workplace. Estimation of actual exposure of the individual workers and customers is associated with a significant degree of uncertainty because of unpredictable spreading and drying of the aerosol.

Another factor that might have influenced the concentration–response relationship is the changes in aerosol composition over time. Our simulated occupational exposure scenario revealed that both fine and ultrafine particles were formed. While the ultrafine particles will remain airborne for some time, evaporation of the solvent will lead to even smaller particles with higher concentrations of the active agents. These particles do not only contain a higher concentration of active (and toxic) agent, they also have a higher mobility and may be present far from the emission source. Furthermore, upon inhalation, small particles (ca. 5–100 nm) may reach lower and more susceptible parts of the airways compared to larger particles. This indicates that the toxic potency of the aerosols could increase with time because of a higher concentration of active substance and increased alveolar deposition, explaining why persons were affected even when entering the building after cessation of the spray event.

In the mouse exposure study, acute toxic effects were observed as a persistent reduction in the tidal volume and expiratory flow rate and associated with a transient increase in the respiratory frequency. In contrast to the change in tidal volume, the effect on the respiratory frequency was reversible. This suggests that two independent mechanisms are involved; first, a reflexively rapid shallow breathing response is initiated because of vagal nerve stimulation and after a period of exposure, a more persistent depression of tidal volume
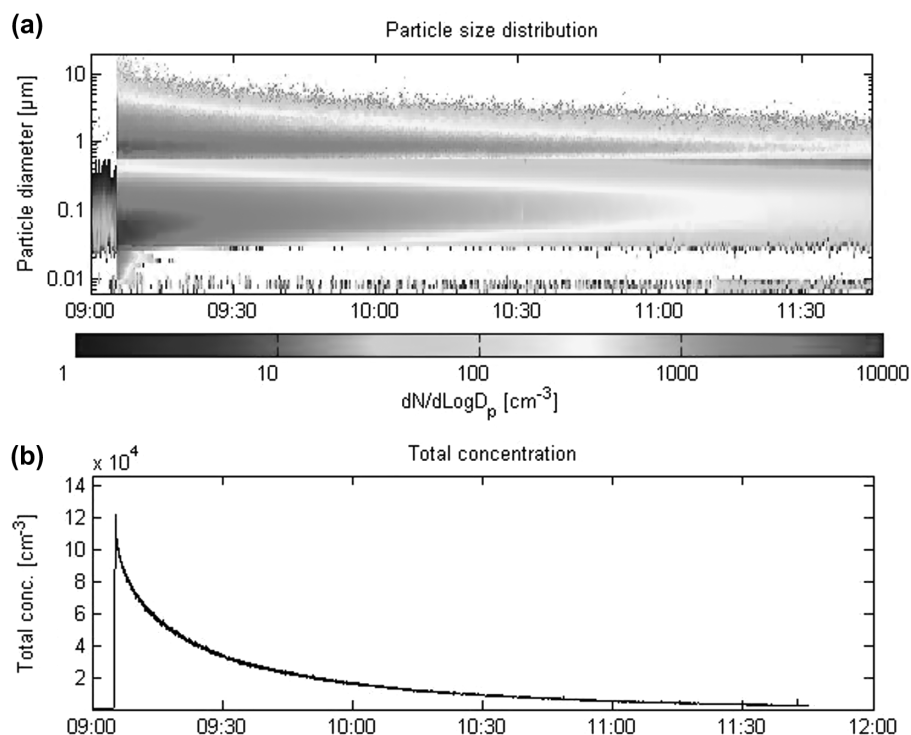
Fig. 4. Temporal evolution of particle size distributions (a) and the total particle concentration (n/cm³) over time (b) (colour version of this figure can be found in the online version at www.informahealthcare.com/ctx).

is induced. In a previous study, we have observed irreversible tidal volume (and expiratory flow rate) depression after inhalation of an aerosolized nanofilm product which induced alveolar collapse. Further, 24 h after exposure, oedema and pulmonary haemorrhage were observed.[19] A steep concentration–effect curve of the SRS product was observed. Thus, 30 min of exposure to 76 mg/m³ gave rise to a 35% reduction in tidal volume, whereas no effect was observed on this breathing parameter after 60 min of exposure to 59 mg/m³. This suggests that the *dose rate* rather than the *total inhaled dose* of substance is critical for the toxic effect. Upon inhalation, the SRS product may react with, for example, components in the pulmonary surfactants leading to inactivation of these as further explained below. It could be speculated that the higher dose rate exceeds the production of new surfactant components which therefore gives rise to toxic effects. A similar critical dose rate point was observed for a previously studied nanofilm product for non-absorbing flooring materials.[19]

Because of the similarities in respiratory effect, it is likely that the SRS product may possess similar mode of action as the nanofilm product albeit different chemical compositions, that is fluorinated silanes in 2-propanol and alkylsiloxanes in naphtha for the nanofilm product and SRS product, respectively.

Table 2. Calculated values for alveolar deposited mass for three different exposure times.

Exposure time	15 minutes	60 minutes	150 minutes
Alveolar deposited mass [mg]	1.64	3.11	3.55

Data are from the simulated workplace exposure scenario.

Development of toxic responses in the mice was monitored over a maximum of 90 min, which may be considered a limitation of the study. However, because several of the mice were in a moribund state after inhalation of the SRS product, the mice were euthanized before delayed effect could be assessed.

It has previously been hypothesized that certain waterproofing sprays may interact with the pulmonary surfactant system.[11] Pulmonary surfactants are vital for a normal lung function as they counteract alveolar collapse at the end of expiration. In the present study, a clear inhibition of a porcine pulmonary surfactant formulation was seen following incubation with the SRS product compared to incubation with the solvent control. Inhibition of surfactant properties was apparent from an increased surface tension at maximum compression (from 1 mN/m in the control group to 20 mN/m in the SRS group). This increased surface tension at simulated end-expiratory conditions is associated with a higher risk for alveolar collapse,[20] which is in agreement with the progressive decrease in tidal volume observed in the mice during the SRS aerosol exposure.

The quantity of SRS used for spray application in the simulated occupational exposure scenario was approximately 4.1 g/m³ (83.6 g in 20.3 m³) and thus, relatively close to the estimated approximately 3.2 g/m³ in the supermarket (22,800 g in 7200 m³). The concentration of gaseous hydrocarbon immediately after the simulated occupational exposure scenario was 563 mg/m³, which is above the Danish occupational 8-h exposure limit for decane (250 mg/m³),[21] one of the solvents used in the SRS product. The Danish occupational exposure limits are also valid in Greenland. In order to compare the chamber experiment with the supermarket incident, it is assumed that: (1) the airborne fraction of the sprayed SRS is the same, (2)

complete mixing of the air inside the supermarket within 1 h after initiation of the spraying process occurred and (3) that there was a maximum air exchange of 1 h^{-1}. Under these circumstances, the gas phase concentration of hydrocarbons in the supermarket 1 h after initiation of the spraying process is estimated to approximately 340 mg/m^3. Similar calculations for the particle phase renders a concentration 6.4 mg/m^3 after 1 h. This is considered fairly conservative because the ventilation system was turned off. In addition, the duration of the spraying events differed markedly. In the supermarket spraying was on-going for approximately 30 min, while the duration of the spray was only 10 s in the chamber experiment in order to avoid saturation of the analytical instruments. Thus, there may have been much higher local concentrations of gases and particles near the supermarket application site compared to the conditions in the exposure chamber.

Both observations in humans and in mice indicate that the SRS product is a very weak nose irritant. In mice, not even the highest concentration of SRS, 5700 mg/m^3 induced any signs of sensory irritation in the upper airways. Also, none of the workers reported any sensory irritation in the eyes or airways related to the spraying event. This is important in relation to risk assessment, because workers may be exposed to even very high concentrations without feeling discomfort, that is the product does not provide a 'warning signal'.

Conclusion

Application of a surface coating product using an inappropriately high-pressure spray gun caused acute respiratory distress, not only in workers operating the spray gun, but also in persons located elsewhere in the building. This outbreak indicates that these products are still not handled with sufficient caution. The severe toxicity and the persistence of the aerosol as demonstrated in the simulation study call for more vigorous safety precautions in the use of these products.

Declaration of interest

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Supplementary material available online

Supplementary: Detailed materials and methods section, Results from LTP-MS analysis of the SRS product, Supplementary Figures 1 and 2, and References; available online at http://informahealthcare.com/doi/abs/10.3109/15563650.2014.915412