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The Effect of Oleoresin Capsicum "Pepper" Spray Inhalation on Respiratory Function*

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ABSTRACT: We performed a randomized, cross-over controlled trial to assess the effect of Oleoresin capsicum (OC) spray inhalation on respiratory function by itself and combined with restraint. Thirty-five subjects were exposed to OC or placebo spray, followed by 10 min of sitting or prone maximal restraint position (PMRP). Spirometry, oximetry, and end-tidal CO_2 levels were collected at baseline and throughout the 10 min. Data were compared between groups (ANOVA) and with predefined normal values. In the sitting position, OC did not result in any significant changes in mean percent predicted forced vital capacity (%predFVC), percent predicted forced expiratory volume in 1 s (%predFEV1), oxygen, or CO_2 levels. In PMRP, mean %predFVC and %predFEV1 fell 14.4 and 16.5% for placebo and 16.2 and 19.1% for OC, but were not significantly different by exposure. There was no evidence of hypoxemia or hypercapnia in either groups. OC exposure did not result in abnormal spirometry, hypoxemia, or hypoventilation when compared to placebo in either sitting or PMRP.

KEYWORDS: forensic science, oleoresin capsicum, positional asphyxia, restraint physiology, respiratory function

Oleoresin Capsicum (OC) "pepper" spray has gained wide acceptance as a swift and effective force method to subdue violent, potentially dangerous individuals in the prehospital and law enforcement setting (1). Derived from the extract of the capsicum pepper plant, OC spray causes irritation over areas of contact (primarily the face, eyes, nose, and mouth), resulting in pain and discomfort. With widespread use, there is concern that OC spray may be associated with significant risk for injury because there have been a number of in-custody deaths in subjects exposed to OC (2,3). As symptoms of cough, gagging, and shortness of breath are common with exposure, concern has focused on the respiratory effects of OC (4,5).

In addition, individuals subdued with OC spray in the prehospital field often require physical restraint, including the prone maximal restraint or hobble position (PMRP), in which the individual is restrained prone with wrists and ankles secured behind the back. Some have argued OC in combination with physical restraint can lead to significant respiratory compromise and risk for asphyxiation and death (1).

While capsaicin, the active ingredient of OC, has been studied for its ability to induce cough, there have been few studies on OC spray or its physiologic effects, particularly on respiratory, pulmonary, and ventilatory function. In addition, no studies on the effects of OC in combination with positional restraint have been reported. We investigated the effects of OC spray on respiratory function by itself and in combination with PMRP to determine if exposure results in any significant compromise in respiratory or pulmonary function.

Methods

We conducted a randomized, cross-over, controlled trial on volunteer human subjects recruited from the local law enforcement training academy and enforcement detail. Potential subjects were advised that participation was completely voluntary and would in no way affect their training. No exclusion was made on the basis of race, ethnicity, gender, age, obesity, or history of asthma or pulmonary disease.

Subjects performed four experimental trials over two separate days in a university medical center pulmonary function laboratory. For each trial, subjects were exposed to either OC or placebo spray via inhalation in an isolation box followed by 10 min in sitting or PMRP. The four separate trials were: placebo exposure followed by sitting; placebo exposure followed by PMRP; OC exposure followed by sitting; and OC exposure followed by PMRP. The order of trials was randomized with the exception that no subject received two OC exposures on the same experimental day.

Prior to the study, weight, height, age, gender, and ethnicity were recorded. Before each trial, baseline spirometry, transcutaneous oxygen saturation, and expired end-tidal CO_2 levels were recorded. Spirometry measurements of forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) were obtained using a Medgraphics Cardiopulmonary Diagnostic System® in accordance with the American Thoracic Society's standards, including reproducibility criteria that the two largest FVCs and FEV1s of at least three acceptable measurements be within 0.200 L (6). Oxyhemoglobin % saturation (SpO_2) was monitored using a pulse oximeter sensor placed on the index finger (Ohmeda Biox 3740 Pulse Oximeter®). Expired end-tidal CO_2 levels were monitored by means of a quantitative CO_2 detector using a Medgraphics Cardiopulmonary Exercise System CPX/D®.

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OC and placebo spray exposure was delivered in a 5 by 3 by 3 foot plastic exposure box. On one end of the exposure box, a hood for the subject was attached. On the opposite end, a small opening was created from which OC or placebo spray was delivered. This method allowed for a uniform and reproducible delivery from a standard distance of 5 ft (currently recommended by manufacturer) without contamination of the laboratory. OC was delivered by a commercially available standard duty aerosol spray canister widely used by law enforcement agencies nationwide (Cap-stun 5.5% OC spray, Zarc International®). This aerosol contains 5.5% OC (0.92% capsaicinoids), 64% isopropyl alcohol carrier agent, and 30.5% isobutane/propane propellant. Placebo spray was delivered by a similar canister containing only carrier and propellant agents (68% isopropyl alcohol and 31.5% isobutane/propane).

The canister was used to deliver a 1 s OC or placebo spray from one end of the exposure box. The subject's head remained in the hood of the exposure box for 5 s after the spray. Transthoracic impedance monitoring using an Edentec Sleep Recorder System® was performed to assess for inhalation or breathholding during the exposure.

After exposure, subjects were immediately placed in sitting or PMRP. In the sitting position, the subject sat in a chair with feet flat on the floor and back upright against the back of the chair. In the restraint position, the subject lay prone on their stomach on a medical examination table with head turned to the side. The subject's wrists were bound together behind the back by means of police handcuffs and ankles bound together and secured to the handcuffs by means of the maximal restraint cuff currently used by local law enforcement agencies.

During the 10 min period, repeat spirometry were performed at 1.5 and 10 min into the period. Continuous pulse oximetry and end-tidal CO_2 levels were recorded at 1, 5, and 9 min (avoiding the potential influence of spirometry testing), and arterial blood was sampled from the radial artery at the wrist to determine arterial pO_2, and pCO_2 levels at 8 min into the period. After the initial 10 min postspray period, the subject had a 1 h washout period to allow for resolution of any residual effects prior to starting the second experimental trial.

Data from trials in which subjects did not inhale during exposure were excluded from analysis. Spirometry data that did not meet criteria for acceptability and reproducibility as outlined above were also excluded. Raw spirometry data (FVC and FEV1) were converted to a percentage of predicted %predFVC and %predFEV1 for each subject to normalize for height, gender, age, and race as per standard practice (6,7).

Statistical analysis was performed using an analysis of variance (ANOVA) for repeated measures with position (sitting or restraint) and exposure (OC or placebo) as factors. A probability value of less than 0.05 was considered statistically significant and 95% confidence intervals are reported. Data analysis was performed by means of a computerized statistical software package (Stata 6.0 for Windows, Stata Corporation®).

Clinically, data were also analyzed as absolute values in comparison with normal values defined prior to the start of the study. Hypoxemia was defined as a pO_2 less than 85 mmHg or oxygen saturation less than 95%. Hypercapnia was defined as pCO_2 or end-tidal CO_2 levels greater than 45 mmHg. Spirometric measurements were considered abnormal if they fell below 1.65 standard deviations of established predicted values (6).

Power analysis determined that 32 subjects would be needed to detect a 10% difference in spirometry parameters if such differences existed. The research design and methods of this study were approved by the Human Subjects Committee and Institutional Review Board.

Results

Thirty-four out of 37 subjects completed the study, performing 136 trials. Of the three subjects who did not complete the study, two were excluded due to acute injuries prior to the study that prevented participation (rib and wrist fractures). One subject was excluded after a vasovagal syncopal episode. The incident occurred during arterial blood draw of the subject's first trial, in which he had been randomized to placebo followed by sitting. He was never exposed to OC or restrained at any time, and he recovered uneventfully.

Twenty-four subjects were men and 10 were women. The mean age was 31.7 years (range 22 to 46 years), mean weight 79.1 kg (range 52 to 107 kg), and mean body mass index 25.9 kg/m^2 (range 19.2 to 31.6 kg/m^2). Of the 136 trials, 8 were excluded from analysis because the subject did not adequately inhale (as measured by impedance monitoring) when exposed to OC. Four sets of spirometry data were excluded as testing did not meet American Thoracic Society (ATS) criteria for reproducibility and variability (6), and two sets of arterial blood gas (ABG) data were excluded because of venous rather than arterial sampling.

In the sitting position, OC exposure did not result in a statistically significant change in spirometry. At baseline prior to exposure, mean %predFVC were similar: 102.8% for placebo and 103.1% for OC. After exposure, there were no changes in mean %predFVC at 1.5 min (102.0% versus 102.4%) or at 10 min (101.8% versus 102.3%) for placebo and OC, respectively (Table 1). Similarly, there were no differences in %predFEV1 between placebo and OC in the sitting position. Mean baseline %predFEV1 was 100.1% and 100.3%, respectively. After exposure, there were no changes in mean %predFEV1 at 1.5 min (98.9% versus 98.9%) or at 10 min (99.2 versus 99.0%), respectively (Table 2).

OC spray exposure did not result in any significant differences in oxygenation or any hypoxemia when compared to placebo in the sitting position. For placebo, mean SpO^2 was 99.2% ± 0.9% (SD) at baseline, 99.0% ± 1.1% at 1 min, 98.6% ± 1.4% at 5 min, and

TABLE 1—*Mean % predicted forced vital capacity (% predFVC) at baseline, 1.5 and 10 min after exposure.*

	%predFVC* ± SD†		
Exposure/ Position	Baseline	1.5 Min After Exposure	10 Min After Exposure
Placebo/ sitting	102.8% ± 9.2% (CI‡: 99.5–106.1%)	102.0% ± 9.0% (98.8–105.1%)	101.8% ± 9.1% (98.6–105.1%)
OC/sitting	103.1% ± 8.7% (99.9–106.3%)	102.4% ± 7.9% (99.5–105.3%)	102.3% ± 8.6% (99.2–105.5%)
Placebo/ PMRP§	101.9% ± 10.0% (98.4–105.4%)	87.5% ± 8.3% (84.5–90.4%)	87.9% ± 8.3 % (84.9–90.8%)
OC/PMRP	103.4% ± 8.1% (100.3–106.5%)	87.5% ± 7.3% (84.7–90.3%)	87.2% ± 7.3% (84.3–90.0%)

* Mean % predicted forced vital capacity.
† Standard deviation.
‡ 95% confidence interval.
§ Prone maximal restraint position.

99.5% ± 0.6% at 9 min after exposure. For OC, SpO^2 was 99.2% ± 0.9% at baseline, 99.1% ± 1.1% at 1 min, 99.0% ± 1.2% at 5 min, and 98.0% ± 3.6% at 9 min after exposure. Arterial pO^2 at 8 min was 96.8 ± 10.8 mmHg for placebo, and 99.4 ± 11.8 mmHg for OC (Fig. 1).

Arterial carbon dioxide (CO^2) levels decreased after OC exposure compared to placebo in the sitting position. Mean end-tidal CO^2 levels were similar at 38.0 ± 3.5 mmHg for placebo and 38.2 ± 4.22 mmHg for OC at baseline. At 1 min after exposure, mean end-tidal CO^2 was 36.8 mmHg ± 4.35 mmHg for placebo, but dropped to 32.4 mmHg ± 5.1 mmHg ($p < 0.01$) for OC. At 5 min, mean levels were 36.5 ± 5.1 mmHg and 32.9 ± 5.8 mmHg ($p < 0.01$) for placebo and OC, respectively. At 9 min, mean levels were 37.0 ± 4.6 mmHg and 35.2 ± 5.9 mmHg ($p < 0.01$), respectively. Mean arterial pCO_2 at 8 min was 39.4 ± 3.9 mmHg for placebo and 36.4 ± 5.1 mmHg ($p < 0.01$) for OC (Fig. 2).

TABLE 2—*Mean % predicted forced expiratory volume in 1 s (% predFEV1) at baseline, 1.5 and 10 min after exposure.*

Exposure/ Position	%predFEV1* ± SD†		
	Baseline	1.5 Min After Exposure	10 Min After Exposure
Placebo/ sitting	100.1% ± 9.3% (CI‡: 96.7–103.4%)	98.9% ± 9.6% (95.6–102.3%)	99.2% ± 10.1% (95.6–102.8%)
OC/sitting	100.3% ± 9.1% (97.0–103.6%)	98.9% ± 9.4% (95.5–102.4%)	99.0% ± 9.5% % (95.6–102.5%)
Placebo/ PMRP§	99.7% ± 9.4% (96.4–102.9%)	83.2% ± 9.7% (79.7–86.7%)	83.7% ± 10.3% (80.0–87.3%)
OC/PMRP	101.1% ± 8.0% (98.0–104.2%)	82.5% ± 10.0% (78.7–86.3%)	82.0% ± 8.2% (78.89–85.2%)

* Mean % predicted forced vital capacity.
† Standard deviation.
‡ 95% confidence interval.
§ Prone maximal restraint position.

The restraint position resulted in a statistically significant decrease in FVC and FEV1. For placebo, mean %predFVC fell from a baseline of 101.9 to 87.5% ($p < 0.01$) at 1.5 min, and 87.9% ($p < 0.01$) at 10 min during PMRP (Table 1). Mean %predFEV1 fell from a baseline of 99.7 to 83.2% ($p < 0.01$) at 1.5 min, to 83.7% ($p < 0.01$) at 10 min during PMRP (Table 2). Exposure to OC made no statistical or clinical impact on pulmonary function in PMRP. For OC, mean %predFVC fell from a baseline of 103.4 to 87.5% ($p < 0.01$) at 1.5 min and 87.2% ($p < 0.01$) at 10 min (Table 1). Similarly, mean %predFEV1 fell from a baseline of 101.1 to 82.5% ($p < 0.01$) at 1.5 min and 82.0% ($p < 0.01$) at 10 min after exposure in PMRP (Table 2).

Similar to the results in the sitting position, OC exposure followed by PMRP did not result in abnormalities in oxygenation or hypoxemia. At baseline, mean oxygen saturation was 99.4 ± 1.0% and 99.3 ± 1.1%; at 1 min, 97.9 ± 2.3% and 98.1 ± 2.8%; at 5 min, 98.3 ± 1.9% and 98.8 ± 1.3%; and at 9 min, 97.4 ± 3.7% and 98.4 ± 2.0% for placebo and OC, respectively. Similarly, mean arterial pO_2 levels at 8 min were 90.2 ± 10.2 mmHg for placebo and 90.0 ± 15.2 mmHg for OC (Fig. 1).

Again similar to the results from the sitting trials, CO_2 levels decreased slightly after OC. At baseline prior to exposure, mean CO_2 levels were 37.7 ± 4.0 mmHg and 36.3 ± 7.7 mmHg for placebo and OC, respectively. At 1 min, levels were 38.8 ± 4.1 mmHg, but decreased to 36.7 ± 6.4 mmHg ($p < 0.01$) for OC. At 5 min, levels were 39.1 ± 4.0 mmHg and 36.6 ± 5.4 mmHg ($p < 0.01$) and at 9 min, 39.5 ± 3.8 mmHg and 37.7 ± 4.7 mmHg ($p < 0.01$), respectively. Similarly, mean arterial pCO_2 levels at 8 min were 40.9 ± 4.3 mmHg for placebo, and 39.1 ± 5.2 mmHg ($p < 0.01$) for OC (Fig. 2).

Discussion

Oleoresin capsicum, the active component of OC spray, is the oily extract of the pepper plant of genus capsicum, consisting of a complex mixture of capsacinoids, including capsaicin and a variety of its closely related analogues (8). Biochemically, capsacinoids

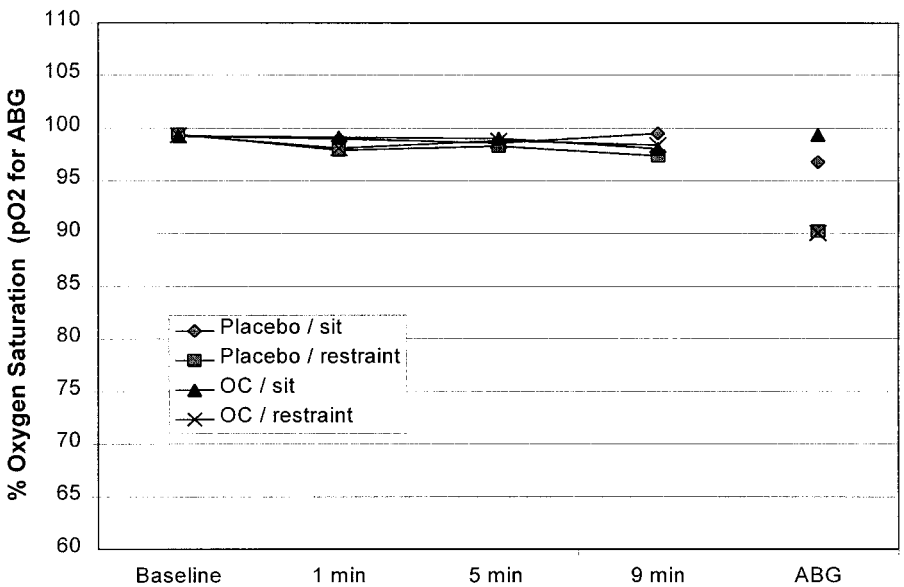

FIG. 1—*Oxygenation by exposure and position.*

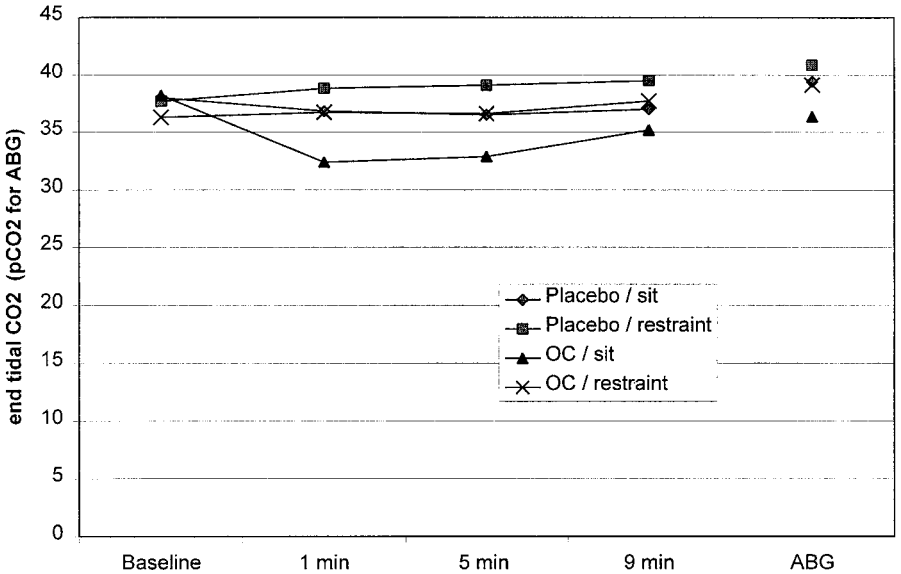

FIG. 2—*Ventilation (CO_2 levels) by exposure and position.*

stimulate chemo-nociceptors in primary afferent nerve endings, resulting in immediate pain and burning sensation over exposed areas of the skin, ocular, nasal, and oropharyngeal tissues. In addition, they cause the release of peripheral neuropeptides, including substance P, which can lead to neurogenic inflammation (3,9). With inhalation or oropharynx exposure, OC causes a variety of symptoms, including cough, gagging, inability to vocalize, and subjective shortness of breath (10).

Because of these immediate symptoms, many subjects lose their capacity to fight and resist. During the 1980s, OC spray was widely adopted by law enforcement agencies and also became available for general public use as deterrent devices (5). A number of custody deaths following OC spray have raised concern regarding its safety. In 1994, Granfield reported on 30 custody deaths associated with exposure to OC spray from 1990 through 1993 (1). Additional deaths have been reported since that time (5). Concern has focused on the respiratory effects of OC spray as playing a potential causal role in these deaths. Some suggest that when inhaled, the spray causes laryngospasm, bronchoconstriction, airway and pulmonary inflammation, and edema, placing subjects at risk for respiratory compromise (3,11,12).

Because of its ability to induce cough, capsaicin has been studied extensively as a model for understanding the cough reflex. There has also been interest in capsaicin because of its ability to block pain sensation and pruritis, presumably by depletion of substance P and other neurotransmitters. While animal and in-vitro human tissue studies suggest capsaicin induces significant increases in airway resistance and bronchoconstriction (13,14), clinical studies with nebulized capsaicin are less clear. In 1985, Fuller reported inhaled nebulized capsaicin resulted in a transient dose-dependent increase in airway resistance, maximal at 20 s and lasting less than 60 s (15). Collier and Blanc both reported no significant decrease in FEV1 in subjects who inhaled nebulized capsaicin at concentrations sufficient to induce cough (16,17). Both cough and deep inhalation, however, have bronchodilatory effects, which may mask direct bronchoconstriction caused by capsaicin (18). There is evidence that subtussive doses of inhaled capsaicin leads to marked changes in airway resistance and pulmonary function (19–21).

Unlike capsaicin, research on the human effects of OC spray are limited (5). A two-year joint study by the FBI and U.S. Army determined that no long-term health risks were associated with OC spray (22). Other studies have been limited to retrospective reviews of law enforcement experience in the field. In 1996, the California State Attorney General reported that no fatal consequences occurred in over 23 000 OC exposures (23). Watson et al. reviewed 908 OC spray exposures in their local jurisdiction and found less than 10% of subjects exposed required any medical attention. Moreover, less than 1% complained of respiratory symptoms requiring medical attention, and none were determined to have any significant injury (24).

In this study, we found no evidence that OC spray inhalation resulted in any respiratory compromise in the sitting position. There was no significant difference in %predFVC or %predFEV1 after exposure between the OC and placebo. These spirometric parameters remained within normal limits and there was no evidence of hypoxemia or hypoventilation.

Had significant laryngospasm or upper AW compromise occurred, we would expect a fall in FEV1 and possibly FVC. Had significant bronchoconstriction occurred, we would expect a significant fall in FEV1, but not necessarily FVC (a decrease in the FEV1/FVC ratio similar to that seen in asthmatics). Had significant pulmonary edema and inflammation occurred, we would expect a fall in FEV1 and FVC without reduction in the FEV1/FVC ratio. Moreover, had any of these findings significantly impacted respiratory function, we would expect evidence of CO_2 retention or hypoxia.

Some have suggested that OC exposure, in combination with physical restraint that can occur in the field setting, may result in significant respiratory compromise. In their review, Granfield suggested these deaths may be related to a "positional asphyxia" from restraint body position following OC spray exposure (1). This theory focused on the PMRP in which subjects lay prone with wrists and ankles bound together behind the back. Some have argued this position prevents adequate chest and abdominal movement for ventilation, potentially placing individuals at risk for hypoventilatory respiratory compromise and asphyxiation (25–27).

Previously, our group studied the respiratory and ventilatory effects of restraint on human subjects and found a progressive restrictive pulmonary function pattern from sitting to supine, prone, and restraint positions. These spirometry findings, however, remained within predicted normal limits and there was no evidence of hypoxemia or hypoventilation on serial ABG determinations, suggesting restraint position had no detrimental impact on respiratory function (28). Others studies have reported similar findings on respiratory function while restrained (29).

In this study, we found no evidence that OC exposure resulted in any additional change in respiratory function in the restraint position. In both the OC and placebo restraint groups, we saw declines in %predFVC and %predFEV1 consistent with our prior work (28). While these declines indicate a restrictive pulmonary function pattern, mean spirometric measurements remained within normal range. Moreover, there were no statistical differences between the OC and placebo groups relative to these declines in %predFVC and %predFEV1. In addition, there was no evidence of hypoxemia or hypoventilation after OC exposure in the restraint group. Accordingly, OC inhalation had no additional effect on the pulmonary function changes, oxygenation, or ventilation in combination with restraint.

There are a number of limitations to our study. First, this study was performed in a clinical laboratory environment. Field subjects are often in a state of extreme agitation and "excited delirium" as a result of underlying psychiatric disease or recreational drug intoxication. Individuals are often involved in violent, physical struggles prior to, during, and after the use of OC spray or positional restraint. These individuals undergo levels of exertion and psychological stress that can lead to exhaustion. Moreover, as this study focused on inhalation exposure, all subjects wore goggles to reduce ocular exposure. Eye irritation and pain from OC may exacerbate the physiologic stress of field subjects, which was not assessed by this study.

Some of the concern regarding OC has focused on individuals, such as asthmatics, who may have an increased risk for bronchospasm from exposure (3). We did not exclude subjects based on pulmonary disease, smoking history, or baseline spirometry. In fact, a small number (eight) of our volunteers either had a history of asthma, lung disease, smoking, or were currently on prescription respiratory inhaler medications. We found no significant differences in a subgroup analysis of these subjects. However, the small numbers of these volunteers limit the power of any conclusion regarding this population. Moreover, while none occurred with our subjects, we cannot exclude the possibility of a rare immune, anaphylactic reaction or drug interaction that might complicate OC exposure in the field.

We attempted to replicate OC exposure in the field as much as possible in the laboratory setting. In doing so, capsaicin dosing was not precisely standardized. Rather, subjects were exposed to a 1 s spray directed from 5 ft away as they might in the field setting (as recommended by both manufacturer and local police policies). In the real-life field setting, closer, longer, and repeated exposures often occur.

However, spray distances less than 5 ft generally do not allow for adequate aerosolization of OC and are likely to reduce the amount inhaled. Exposure in the box was limited to 5 s while in the laboratory. While this may seem a short period of time, spray in the field usually occurs in an open setting where OC dissipates rapidly. Moreover, by containing the spray within the exposure box, it is likely that subjects were exposed to a much higher concentration of capsaicin than might have occurred in the open air. The concentration of active capsacinoids (approximately 26 mg delivered per spray into a 2×10^6 cm^3 space) in our study was similar if not higher than other clinical studies on capsaicin inhalation.

We did not study repeated OC spray exposures that commonly occur in the real-life field setting. We also used an aerosol form of OC spray, rather than liquid or foam forms that are also available. We believe the aerosol form was more likely to be inhaled and thus more appropriate for our study. In order to standardize the exposure, we studied only one type of commercially available OC aerosol spray. Sprays differ in terms of carrier solvents, propellents, as well as capsacinoid concentrations (30). The impact of these differences was not investigated in this study.

As for restraint, we utilized a restraint device currently in use by our local law enforcement agency in the field to place subjects in the PMRP. Our spray exposure occurred prior to placement in the restraint position, rather than while the subject was restrained. It is likely that in the field setting, OC exposure occurs before or during struggle, rather than after the subject has been restrained.

Finally, we limited our observation period to 10 min. If significant changes in respiratory function occurred after 10 min of exposure, our study would not have detected these findings. Reports of fatalities following OC have reported a variable amount of time between exposure and sudden death event (4). However, we had no reports of adverse events occurring after our study was completed in any of our subjects.

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