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ORIGINAL ARTICLE

Military small arms fire in association with acute decrements in lung function

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ABSTRACT

Objective After introduction of unleaded ammunition, Norwegian Armed Forces received reports of acute respiratory symptoms in soldiers after exposure to fumes from firing the standard weapon, HK416. The aim of the present study was to examine lung function before and after exposure to fumes from HK416 in a double-blinded standardised study design using three different types of ammunition.

Methods Fifty-four healthy, non-smoking male volunteers (19–62 years) fired the weapons for 60 min with either leaded, unleaded or 'modified' unleaded ammunition. Gaseous and particulate emissions were monitored. Spirometry and exhaled nitric oxide (eNO) were performed within 14 days before (T0), shortly after (T1) and 24 hours after (T2) shooting. Methacholine provocation and diffusing capacity of carbon monoxide (DLCO) were carried out at T0 and T2.

Results The mean forced expiratory volume in 1 s on a group level was significantly reduced both at T1 and T2 compared with T0, with means and 95% CI of 226 mL (158 to 294 mL) and 285 mL (218 to 351 mL), respectively. The same significant pattern was seen for DLCO, forced vital capacity and eNO. The methacholine test indicated a slight increase in bronchial hyperreactivity. However, there were no significant differences between types of ammunition used.

Conclusion Exposure to fumes from military weapons might be a respiratory hazard for soldiers who do live-fire training regularly or are in a closed combat environment.

What this paper adds

- Worldwide, millions of soldiers do live-fire training regularly, and some soldiers experience acute respiratory symptoms afterwards.
- There have been several reports on acute respiratory symptoms and chronic lung diseases in soldiers returning from combat operations.
- The present study is to our knowledge the first that has studied the association between fumes from arms and respiratory health effects in an experimental setting.
- In the present study, exposure to fumes from military weapons results in acute decline in lung function lasting more than 24 hours.
- A precautionary initiative could be to include military personnel repeatedly exposed to fumes from arms in a surveillance programme of lung function testing.

INTRODUCTION

The Norwegian Armed Forces have received several reports from soldiers complaining of acute airway symptoms as well as general symptoms of malaise after firing small arms. These symptoms appeared after a new standard weapon (HK416; Heckler & Koch, Germany) in combination with new unleaded (UL) ammunition were introduced in 2008. Norwegian Armed Forces suspected that these health effects appeared after using the newly introduced UL ammunition but not when using the leaded (L) ammunition (original type). In 2011, the Norwegian Labour Inspection Authority stopped all use of the UL ammunition. Modified UL (MUL) ammunition was then developed to reduce emissions of copper and zinc by half, when fired.

We found previously that, when shooting with small arms, soldiers are exposed to emissions of CO, particulate matter (dust), combustion products, copper, zinc, bismuth, lead and tin from HK416 small arms using UL, MUL and L ammunition.[1] The concentrations of particulate matter and copper were high and exceeded their respective occupational exposure limits (8 hour/day, 5 days/week) for all three types of ammunition, although the lowest values were for L ammunition.

There has been increased interest concerning health effects in soldiers returning from combat operations in Iraq and Afghanistan, such as acute respiratory symptoms and chronic lung diseases.[2–6] Various respiratory hazards have been suggested, including exposure to geological dust, burn pits, vehicle exhaust emissions, industrial air pollution, isolated exposures and emissions after firing military weapons.[3 7] We have in a previous paper reported in detail the exposure from firing small arms and the association between exposure and general and respiratory symptoms.[1] General symptoms such as chills, headache and/or malaise were reported by 75% of the subjects and appeared 3–12 hour after exposure. Respiratory symptoms (shortness of breath, coughing, discomfort in the mouth/throat/chest) were experienced by 80% of the subjects and appeared during or shortly after firing lasting several hours.

We hypothesised that lung function might change after exposure to emissions from firing small arms, a risk that has hardly been studied and should be of interest worldwide. Therefore, the purpose of this experimental study was to examine

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lung function by spirometry, diffusing capacity of the lung for carbon monoxide (DLCO) and methacholine challenge before and after firing small arms. We also wanted to investigate whether L, UL and MUL ammunition would affect lung function differently.

METHODS

Subjects

We recruited 54 healthy, non-smoking male volunteers employed in the Norwegian Armed Forces or the Norwegian Defence Research Establishment (mean 40 years, range 19–62) and divided them randomly into three groups: one group for each type of ammunition.[1] There were no significant differences in mean age among the three groups. The subjects had no reported medical history of chronic obstructive pulmonary disease, allergies, asthma or bronchial hyper-responsiveness. They had no sign of respiratory infections during the 4 weeks prior to the study and had not been exposed to fumes from firing weapons during the 2 weeks prior to the study. All subjects were interviewed on the day of shooting to ensure that they met the inclusion criteria. None of the subjects were excluded based on the findings from baseline pulmonary function tests or the eNO value. However, one subject was excluded on the day of the shooting because he had influenza.

Exposure

This study of the health effects caused by firing arms was carried out in a controlled, experimental environment.[1] The anticipated concentrations of fumes were believed to reflect the exposure levels soldiers can be exposed to during real firing exercises.

Firing of small arms was carried out in standardised semi-airtight plastic tents (measuring 1.2×1.2×3.5 m) placed on plywood equipped with insulating mats of good quality (thickness 15 mm) to lie on and sleeping bags (for −25°C), and both were of military standard used during winter practice. All subjects wore winter clothing and none complained they were cold during the shooting session. Air temperature was registered for all subjects on the day of shooting. The mean temperature was −6°C (range −10.0°C to +8.8°C). Fume exposure was monitored inside the tent and regulated to keep the CO level between 200 and 300 ppm by adjusting the number of rounds fired.[1] This level was based on knowledge about the effect of this level of CO during a regular firing exercise with exposure for 1 hour and was below the level known to produce mild symptoms in humans after such exposure.[8] The exposure time in this study was 1 hour. Exposure measurements were presented in a previous study.[1] In brief, there were no significant differences between the three ammunition groups with respect to the number of rounds fired with a mean±SD of 15±9 rounds (range 4–45) or in CO exposure (236±36 ppm). The concentrations of particulate matter (15.1 mg/m^3 ±5.0) and copper (5.3 mg/m^3 ±2.1) were high and exceeded their respective threshold limit value (TLV) of 10 mg/m^3 and 0.2 mg/m^3, respectively. The concentration of the combustion products, NH_3, CH_4, NO_2, NO, N_2O, HCN and SO_2, were generally low and did not exceed their respective TLV.

In the three groups, the first (n=17) used L ammunition (SS109, RUAG, Switzerland) for firing, the second (n=19) used UL ammunition (NM229; Nammo, Norway) and the third *(n=18)* used MUL ammunition (NM255; Nammo). The subjects were not informed of which type of ammunition they were allocated. All used HK416 small arm with a 16.5-inch barrel.

Lung function tests

All pre-tests were performed 2–13 days before the trial. Measures of lung function including forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and forced expiratory flow at 25%–75% of maximum ($FEF_{25–75}$) were carried out before exposure (T_0), shortly after shooting (90–150 min; T_1) and 24 hours after shooting (T_2). The DLCO and methacholine challenge test were carried out at T_0 and T_2. A 12% and at least 200 mL change in FEV_1 is suggested by the European Respiratory Society (ERS)/American Thoracic Society (ATS) guidelines as a criterion for a positive reversibility test.[9] Therefore, we used the same criteria as indicating a significant intraindividual change in FEV_1. The lung function tests were performed each time in the following order: spirometry, DLCO and methacholine challenge. All tests were blinded and conducted by three trained health workers, and a given subject was tested in the same indoor environment by the same health worker using the same spirometer at all three time points. All tests were conducted according to the equipment specified by the ERS/ATS guidelines (Spirotrac 2160, Vitalograph, Maids Moreton, UK).[10] DLCO was corrected for haemoglobin concentration. Methacholine bronchial provocation test results were expressed as the cumulative dose causing a 20% decrease in FEV_1 (PD20) compared with baseline; a PD20 of <7.8 mmol was considered as positive. The methacholine response was also expressed as the dose–response slope (DRS) between FEV_1 results after the first ($FEV_{1,pretest}$) and last ($FEV_{1,post\text{-}test}$) methacholine dose. DRS values were calculated for each subject as follows[11]: $DRS = (FEV_{1,pretest} - FEV_{1,post\text{-}test}) \times 100 / \text{maximum accumulated dose of methacholine} \times FEV_{1,pretest}$. Exhaled nitric oxide (eNO) was measured at a flow rate of 50 mL/s using a NIOX MINO (Aerocrine AB, Stockholm, Sweden) according to the manufacturer's protocol. The same NIOX MINO apparatus was used for all measurements. All lung function and eNO tests were done in the same laboratory separately from the shooting range.

Ethics

The study was registered at https://clinicaltrials.gov (NCT01477645). All subjects provided a written informed consent. The study was approved by the Regional Ethical Committee, South East (RECno.2011/1335b).

Statistics

Paired Student's t-tests for parametric data or related samples Wilcoxon signed rank test for non-parametric data were used to identify differences in mean biological variables within each group before and after exposure. The Pearson's correlation test was used to analyse associations between air temperature and lung function changes. One-way analysis of variance and Tukey's honest significant difference (HSD test) were then used to analyse differences among the ammunition-use groups. All analyses were performed using IBM SPSS Statistics V.22.

RESULTS

Lung function

No significant differences in lung function were found between the three different exposure groups at inclusion (T_0; table 1). Results from the lung function tests are summarised in table 2 and figure 1. The decline in mean FEV_1 from before to immediately after shooting for the whole group was 226 mL (95% CI 158 to 294 mL) or 6%. There was still a significant deterioration in FEV_1 between prior to and at 24 hours after shooting, and the decline in mean FEV_1 for the whole group was 285 mL

Table 1 Lung function of the study population before exposure (T_0)

		Ammunition		
	Total (n=54)	L (n=17)	UL (n=19)	MUL (n=18)
FEV1 % p.	105 (102 to 109)	106 (99 to 114)	106 (100 to 112)	104 (98 to 109)
FVC % p.	112 (109 to 116)	115 (108 to 122)	113 (107 to 118)	110 (104 to 115)
FEV1/FVC %	77 (75 to 78)	76 (72 to 79)	77 (74 to 81)	77 (75 to 80)
FEF25%–75% p.	86 (79 to 92)	84 (70 to 97)	89 (76 to 102)	83 (74 to 93)
DLCO % p.	109 (104 to 114)	108 (100 to 117)	113 (102 to 124)	105 (100 to 110)

Data are presented as means (95% CI).
DLCO, diffusing capacity of carbon monoxide; FEV1, forced expiratory volume in 1 s; FEF, forced expiratory flow; FVC, forced vital capacity; L, leaded; MUL, modified unleaded; UL, unleaded;p,predicted.

(218 to 351 mL) or 7%. The decline in FEV_1 within all three groups of ammunition was significant at both times. Thirty-seven subjects had a decline in FEV1 of more than 150 mL, and 19 of these subjects had a decline of more than 400 mL 24 hours after exposure. When we used a cut-off value for a positive change in FEV_1 of 12%, six subjects had such a categorical decline at 24 hours after shooting compared with before shooting. These subjects were re-tested within 10 days and five of the six subjects showed a return to normal lung function values. One subject still had a change in FEV_1 of 12% but had normal FVC 5 months after shooting, and clinically, there was no suspicion of permanent airway disorder. No significant differences were found in lung function changes between the different ammunition-using groups.

An overall group decline in mean FVC was found when comparing before and shortly after shooting ($p<0.01$) as well as after 24 hours ($p<0.001$): 143 mL (95% CI 46 to 240) and 126 mL (56–195), respectively. This was also seen for the MUL group at both time points ($p<0.01$), but significant declines in FVC were not seen for those using L or UL ammunition. Changes in FEV_1/FVC showed the same pattern as FEV_1. $FEF_{25–75}$, and the mean DLCO values had significant declines in all groups (table 2 and figure 1). The total declines in mean $FEF_{25–75}$ and DLCO were 636 mL/s and 10%, respectively, 24 hours after shooting compared with the values at T_0 ($p<0.001$).

The Pearson's correlation coefficient between $FEV1\Delta T_0\text{-}T_1$ and air temperature was $r=-0.17$ ($p=0.2$). Neither of the other lung function variables was significantly correlated with air temperature.

Only one subject had a positive methacholine challenge test after shooting, but there was a significant change in the DRS for the whole group when comparing this measure before and after shooting: it changed from 0.54 to 0.98 ($p=0.03$) (table 2). The eNO levels decreased significantly at T_1 and T_2 (table 2)

DISCUSSION

To our knowledge, this is the first study to examine the effects on lung function after exposure to fumes from firing small arms. The most noticeable findings were the significant declines in lung

Table 2 Lung function before (T_0), 90–150 min after exposure (T_1) and 24 hours after exposure (T_2)

			Ammunition					
	Total (n=54)	p Value	L (n=17)	p Value	UL (n=19)	p Value	MUL (n=18)	p Value
FEV_1T_0 (L)	4.4 (4.2 to 4.6)		4.4 (4.1 to 4.8)		4.4 (4.0 to 4.7)		4.3 (4.0 to 4.6)	
$\Delta T_0\text{-}T_1$ (mL)	226 (158 to 294)	<0.001	211 (85 to 338)	0.003	184 (49 to 319)	0.01	284 (175 to 33)	<0.001
$\Delta T_0\text{-}T_2$ (mL)	285 (218 to 351)	<0.001	268 (140 to 397)	<0.001	240 (125 to 355)	<0.001	347 (225 to 470)	<0.001
FVC T_0 (L)	5.7 (5.5 to 5.9)		5.9 (5.4 to 6.3)		5.7 (5.4 to 6.0)		5.6 (5.2 to 6.0)	
ΔT_0-T1(mL)	143 (46 to 240)	0.005	139 (–98 to 376)	0.23	113 (–54 to 280)	0.17	179 (54 to 305)	0.008
$\Delta T_0\text{-}T_2$ (mL)	126 (56 to 195)	0.001	127 (–20 to 274)	0.08	74 (–37 to 184)	0.18	179 (53 to 305)	0.008
FEV1/FVC T_0 (%)	77 (75 to 78)		75.8 (72.4 to 79.1)		77.2 (73.6 to 80.8)		77.2 (74.6 to 79.7)	
$\Delta T_0\text{-}T_1$	2.0 (1.0 to 3.1)	<0.001	1.7 (–0.2 to 3.6)	0.07	1.6 (–1.0 to 4.1)	0.22	2.8 (1.8 to 3.8)	<0.001
$\Delta T_0\text{-}T_2$	3.5 (2.7 to 4.2)	<0.001	3.2 (2.2 to 4.2)	<0.001	3.2 (1.6 to 4.8)	0.001	4.0 (2.5 to 5.5)	<0.001
$FEF_{25\text{-}75}T_0$ (L/s)	3.9 (3.5 to 4.2)		3.8 (3.2 to 4.4)		4.0 (3.4 to 4.7)		3.7 (3.3 to 4.2)	
$\Delta T_0\text{-}T_1$ (mL/s)	499 (364 to 635)	<0.001	440 (267 to 613)	<0.001	493 (177 to 808)	0.004	563 (341 to 784)	<0.001
$\Delta T_0\text{-}T_2$ (mL/s)	636 (502 to 770)	<0.001	562 (324 to 801)	<0.001	642 (355 to 928)	<0.001	700 (496 to 904)	<0.001
DLCO T_0 (mmol/(min*kPa))	12.3 (11.8 to 12.8)		12.5 (11.2 to 13.8)		12.3 (11.7 to 13.0)		12.1 (11.3 to 12.9)	
$\Delta T_0\text{-}T_2$	1.1 (0.8 to 1.4)	<0.001	0.9 (0.4 to 1.4)	0.001	1.3 (0.7 to 1.8)	<0.001	1.1 (0.7 to 1.6)	<0.001
eNO T_0 (ppb)	26.0 (23.4 to 29.2)		26.4 (21.3 to 31.5)		23.8 (19.3 to 28.3)		28.9 (22.7 to 35.1)	
$\Delta T_0\text{-}T_1$	5.6 (3.3 to 7.8)	<0.001	4.7 (1.8 to 7.6)	0.004	4.1 (0.5 to 7.6)	0.029	8.0 (2.7 to 13.3)	0.005
$\Delta T_0\text{-}T_2$	5.6 (3.2 to 7.9)	<0.001	3.5 (–0.5 to 7.6)	0.08	5.6 (2.7 to 8.6)	0.001	7.4 (1.9 to 12.9)	0.01
Methacholine T_0 (DRS)	0.54 (0.3 to 0.8)		0.39 (0.2 to 0.5)		0.48 (0.3 to 0.6)		0.73 (0.1 to 1.4)	
$\Delta T_0\text{-}T_2$	0.44 (0.0 to 0.8)	0.03	0.18 (0 to 0.3)	0.12	0.25 (0 to 0.76)	0.10	0.83 (0 to 1.9)	0.05

p Value showing paired sample t-test comparing before and after exposure.
Data are presented as means (95% CI).
Δ, difference in measured values (mL); DLCO, diffusing capacity of carbon monoxide; DRS, dose–response slope; eNO, exhaled nitric oxide; FEF25-75, forced expiratory flow at 25%–75%; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; L, leaded; MUL, modified unleaded; UL, unleaded. tT_0, baseline measurements; T_1,measurements 90–150 min after exposure; tT_2, measurements within 24 hours after exposure.

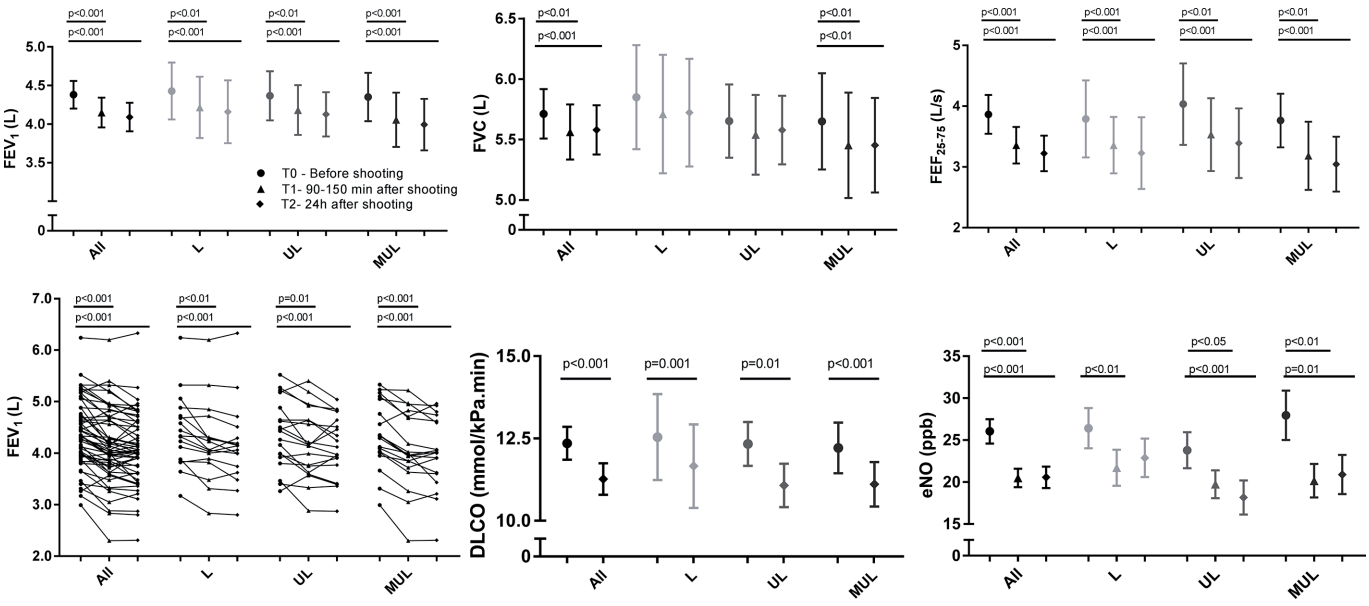

Figure 1 Lung function and exhaled nitric oxide before (T0), 90–150 min after exposure (T1) and 24 hours after exposure (T2) (mean (95% CI). FEV1 is illustrated as mean (95% CI) and individual changes. DLCO: diffusing capacity of carbon monoxide; eNO: exhaled nitric oxide; FEF25-75, forced expiratory flow at 25%–75%; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity. L, Leaded Ammunition; UL, Unleaded Ammunition; MUL, Modified Unleaded Ammuntion.

functions measured by spirometry and gas diffusion after firing small arms with three types of ammunition (L, UL and MUL). Furthermore, this reduction in lung function lasted 24 hours, and in a few cases even longer. Bronchial hyper-responsiveness increased as measured by the DRS. No significant differences were found between the three types of ammunition with respect to declines in lung function. The lack of difference between the ammunition types was rather unexpected as, to our knowledge, there are no previous reports of lung function decline from firing small arms with the use of L ammunition. On the contrary, our initial hypothesis was that UL ammunition would induce a decline in lung functions, as respiratory symptoms have been reported from soldiers exposed to such ammunition.

After shooting, all physiological pulmonary measures had a significant decline or a declining trend considering both the total results and the results within each group. Declines in the lung function variables FEV_1, FEV_1/FVC, $FEF_{25–75}$ and DLCO indicate that both central and distal airways were affected by pollutants in the fumes after firing. To our knowledge, there are no studies of lung function measurements after firing arms. Most studies from firing ranges focuses on health effects after lead exposure, which includes noise issues, lung cancer, cardiovascular and neurotoxic effects.[12] In addition, studies of deployed military personnel have suggested higher prevalence of respiratory symptoms and respiratory illness consistent with airway obstruction.[3 7] These were observational studies based on retrospective cohort studies and case reports. However, the association between chronic lung disease and airborne-hazards exposure requires further longitudinal research studies with objective pulmonary assessments.[3]

Bronchial responsiveness (BR) expressed as individual DRS values increased for the whole study group, implying that the exposure in this study caused a significant increase in BR. However, the FEV_1 was significantly lower when BR was measured 24 hours after exposure compared with FEV_1 at baseline. This alone might have changed the DRS.[11]

Statistical analysis performed in the present study did not identify any single pollutant that was more associated with lung function decline than any other pollutant, nor any pollutant for which there was a dose–response relationship with lung function decline. Dust, Cu and CO were uniformly measured above American Conference of Governmental Industrial Hygienists TLV (ACGIH, 2013), and the majority of subjects in all groups showed lung function decline. Accordingly, the lack of an exposure gradient may explain the lack of associations between agent(s) that may be responsible for the lung function decline. The uniform level of exposure in the present study implies that the design of the barrel of HK416 was more important for the level of exposure than type of bullet. More than two-thirds of the metal present in the emission was copper with a concentration of 5.3 mg/m^3, which is above the TLV. The TLV for copper fumes is 0.2 mg/m^3, which means that the exposure to our subjects was 27 times above recommended value, and might be an explanation for the lung function changes in some of them. Copper as a proportion of airborne dust was also similar between the three ammunition types (approximately 35%). Zinc was measured at concentrations below TLV. However, because zinc possesses some of the same toxic properties as copper, it could have contributed to the observed effects.[13] The CO level, as predicted, was between 200 and 300 ppm, which is a concentration exceeding TLV by a factor of 5. This significantly increased the levels of carboxyhaemoglobin (COHb; data not shown) which might explain the reduced DLCO after 24 hours. The reduced DLCO could arise from a combination of an increased circulating level of COHb and/or an inflammatory response in the respiratory alveoli with secondary oedema of the pulmonary interstitium leading to reduced gas diffusion, although of a much milder form than that seen involved in silo-filler's disease.[14]

In a previous paper, we reported that 67% of the subjects exposed to small arms firing showed symptoms similar to those reported in metal fume fever.[1] Here, a significant reduction in FEV_1 was measured as early as within 90 min after the exposure session, while metal fume fever is a condition that typically appears 3–10 hours after exposure.[15] As lung function is usually unaffected in persons suffering metal fume fever,[16] the large

FEV_1 decline might have had other explanations. High levels of particulate matter or gases have the potential to cause an irritant effect in the airways, and the fumes from the use of small arms consist of a mixture of many compounds and particles with toxic properties.[1]

The exposure measurements in the present study are similar to the levels soldiers can be exposed to during training for combat. Statistically, there were no significant differences in the numbers of rounds fired (range 4–45), or the levels of exposure observed in the three study groups.[1] We did not expect that exposure after firing only a few shots could impair lung function when the ventilation was poor. This indicates that ventilation in combat training is of vital importance to reduce exposure to potential harmful agents and minimise adverse health effects.

The eNO values were decreased after firing arms. An increase in eNO may have been expected, since this is often seen after exposure to different agents in occupational settings.[17] However, a similar response with cross-shift reduction in FEV_1 and eNO was seen in a study in Norwegian cement production workers.[18] There might be several different explanations why eNO is reduced. Gun smoke could have triggered some of the same mechanisms as smoking. Smoking is known to down-regulate the NO synthases, resulting in lower eNO values.[19 20] Airway obstruction can also result in lower levels of eNO.[21 22] It has been suggested that the occlusion of small airways might trap the NO produced, or that a reduction in the volume of conducting airways might lead to an increase of luminal airflow and thereby lower the eNO value.[22] To our knowledge, there are no reports on measurements of eNO levels after gun smoke exposure. Here, the eNO level did decrease significantly, but only by a few ppb (mean 5.5). Thus, eNO is apparently not an appropriate variable to detect airway effects after firing small arms.

The optimal design of such a study is to perform a control sham exposure to control for possible confounding factors. This is not feasible in the present experimental design, as bullets without potentially harmful emissions are not available. In addition, the present design could not fully account for the effects of temperature; indeed, one possible hypothesis is that the spirometric changes after shooting were induced by inhalation of cold air during shooting practice. However, we have registered air temperature for all subjects the day of shooting and could find no significant correlation between ambient temperature and any of the lung function variables. Moreover, the subjects were active duty military personnel and accustomed to combat training during such temperatures, and had never previously experienced respiratory problems while inhaling cold air. Finally, none of the subjects reported feeling cold during shooting. Thus, it seems less likely that low temperatures influenced lung function decline.

One reasonable question is whether our experimental design is comparable with the real world of soldiers deployed to recent conflict zones. We imagine that combat inside buildings or in narrow alleys is comparable with our experimental design, while combat outdoor in the field is less comparable. Our experience is that shooting outdoors rarely produces respiratory symptoms, but it can occur in particularly still air conditions. However, most reports on respiratory and general symptoms that the Norwegian Armed Forces receives are after shooting indoors and outdoors at roofed shooting ranges.

CONCLUSION

Exposure to fumes from the use of small arms in this study resulted in acute declines in lung functions and might have impaired central and peripheral airways and probably alveoli and/or lung parenchyma. No differences in the degree of lung function decline were found between the different types of ammunition used. These findings with uniform levels of exposure imply that the design of the barrel of the weapon was more important than type of bullet, because all three ammunition types were associated with deteriorations in lung function. Future longitudinal research studies are necessary to better understand the potential long-term health consequences of shooting with such small arms. However, a precautionary initiative could be to include military personnel who are repeatedly exposed to small arms fumes and/or different irritating agents in a surveillance programme of lung function testing.

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Contributors All authors contributed to the design of the study, to interpretation of the results and in revising the manuscript. AKB, LIBS and AV participated in data collection. LS is a statistician and did the statistical analysis. AKB drafted the manuscript in collaboration with JK, AV and LIBS. The final version has been approved by all authors.

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Military small arms fire in association with acute decrements in lung function

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