Airway responsiveness of firefighters after smoke exposure

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Abstract

Ventilatory function and airway reactivity of 20 firefighters were studied one hour before and one hour after exposure in a smoke chamber. None showed an increase in airway reactivity to inhaled histamine before exposure. Eight (80%) of the regular firefighters, however, had an increase in airway reactivity after exposure in the smoke chamber. After six hours, three of the firefighters still had increased airway reactivity. All were non-reactive after 24 hours but the ventilatory function of the three firefighters who were reactive after six hours did not return to baseline values. The duration of service as a firefighter is the major contributing factor to the change in airway reactivity. This increase in airway responsiveness among regular firefighters suggests that some form of chronic epithelial injury is needed before an increase in airway responsiveness is seen.

Firefighters are exposed to a wide variety of respiratory hazards including ozone, sulphur dioxide, carbon monoxide, oxides of nitrogen, aldehydes, hydrogen chloride, and particulates.¹⁻⁴ Acute reduction in pulmonary function after exposure to the products of combustion is well documented.⁵⁻⁷ The changes in airway responsiveness are, however, not.⁸ Determining the changes in airway responsiveness can help in understanding the mechanisms of airway obstruction and may identify persons with high risk of chronic obstructive pulmonary disease. This paper describes the changes in airway responsiveness in a group of firefighters after exposure in a smoke chamber and analyses the factors contributing to this change.

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Methods

STUDY POPULATION

Two groups of male Malay firefighters volunteered for exposure in a smoke chamber without protective respiratory equipment. The first group consisted of 10 recruits who had not been firefighting but had been inside the smoke chamber on two previous occasions. The second group were regular firefighters. From a questionnaire none of the subjects had a history of chronic cough, chronic phlegm production, wheezing, asthma, or atopy.

SPIROMETRY AND HISTAMINE CHALLENGE

Baseline spirometry and a histamine challenge test (HCT) were performed one hour before exposure in the smoke chamber. At the time of baseline spirometry and HCT no subject had fought a fire for the previous 72 hours nor had had any respiratory tract infections during the previous two weeks. All the volunteers were required to refrain from smoking and drinking caffeinated beverages for eight hours before the tests. Spirometry was performed using a Fleisch type pneumotach spirometer. A minimum of three acceptable tracings were obtained with the subjects in a standing position without nose clips. The tracing with the largest forced vital capacity (FVC) and forced expiratory volume in one second (FEV₁) was used for analysis. The FEV₁, FVC, and forced expiratory flow rates (FEF_{25-75%}, FEF_{75-85%}, FEF_{50%}, and FEF_{75%}) were obtained using standard techniques.^{9 10}

The HCT was performed using standard concentrations of histamine diphosphate solution delivered by a hand held De Vibliss No 40 glass nebuliser. The mouthpiece of the nebuliser was placed between the teeth of the subject who exhaled to slightly below functional residual capacity (FRC) and then inhaled slowly over one to two seconds towards total lung capacity (TLC), at which stage he was required to hold his breath for three seconds. At the beginning of inspiration the operator gave the bulb of the nebuliser one firm squeeze. The FEV₁ was measured 60 seconds after each dose and followed immediately by the next dose. The challenge was stopped when the FEV₁ fell by more than 20% from the postsaline value or when the maximum dose of 3.9 µmol was

delivered. The provocative dose of histamine causing a 20% fall in FEV₁ (PD₂₀) was determined by plotting the percentage change in FEV₁ from the postsaline value on a linear scale against the log dose of histamine.¹¹ If subjects did not respond by the end of the protocol, the largest dose of 3.9 µmol was assigned to the PD₂₀ for analysis.

The smoke chamber is a large enclosed building with a simple obstacle course within. The firefighters were to negotiate the obstacles using their helmet lights for illumination. Smoke was produced by controlled combustion of a mixture of diesel oil, petrol, wood dust, and plastics.

Spirometry and HCT were repeated one hour after exposure in the smoke chamber. Those subjects with increased airway responsiveness had the HCT repeated six and 24 hours after exposure.

STATISTICAL ANALYSIS

Statistical analysis of changes from baseline spirometry was undertaken using a paired *t* test. Comparison of the changes in ventilatory function between recruits and regulars were adjusted for age, height, length of service, duration of exposure in the smoke chamber, pack-years of smoking, and the pre-exposure level using analysis of covariance (J Lee, personal communication). The factors contributing to the change in PD₂₀ were analysed by comparing the standardised partial regression coefficients through multiple linear regression analysis. The post-exposure FEV₁ was entered as one of the independent variables to assess the effect of a change in baseline airway calibre.¹²

Results

GENERAL CHARACTERISTICS OF STUDY POPULATION

The recruits were slightly younger (age range 17–22) than the regular firefighters (age range 21–29). The regulars had been in firefighting for an average of 4.6 years (range 1–9 years). All 20 firefighters were smokers with the regulars having a slightly higher

Table 1 General description of study population of firefighters

	Recruits Mean (SD)	Regulars Mean (SD)
Age (y)	19.5 (1.58)	25.1 (3.21)
Height (cm)	170.5 (4.79)	170.0 (6.86)
Length of service (y)	—	4.6 (2.55)
Pack-years of smoking	2.9 (1.29)	4.2 (1.93)

pack-years of smoking (table 1). The recruits spent 10.8 minutes and the regulars 11.8 minutes in the smoke chamber.

CHANGES IN VENTILATORY FUNCTION

Small changes in ventilatory function were seen among the recruits after exposure in the smoke chamber (–1.8% to 3.0%). These were not statistically significant. Changes in the regular firefighters were greater in magnitude and were statistically significant. The largest change was in FEF_{75%} (table 2). The differences in the changes in ventilatory function between recruits and regulars were not statistically significant (table 3).

Ventilatory indices for recruits returned to pre-exposure levels within six hours. In four of the regular firefighters, ventilatory function did not return to pre-exposure levels even after 24 hours despite administration of a bronchodilator.

CHANGES IN AIRWAY RESPONSIVENESS

The 20 subjects showed no increased airway responsiveness before exposure in the smoke chamber. None of the recruits was responsive after exposure but eight (80%) of the regulars became responsive after exposure. Of these, three continued to be responsive after six hours and all were non-responsive after 24 hours. Ventilatory function of the three regulars who were responsive after six hours, however, did not return to pre-exposure levels despite administration of a bronchodilator.

The duration of service as a firefighter seems to

Table 2 Mean and percentage changes in ventilatory function and airway responsiveness after exposure in a smoke chamber

	Recruits			Regulars		
	Before	After	Difference (%)	Before	After	Difference (%)
FEV ₁	3.54	3.52	–0.02 (–0.6)	3.59	3.55	–0.04 (–1.1)*
FVC	4.01	4.08	0.07 (1.7)	4.05	4.05	0.00 (0.0)
FEF _{25–75%}	4.25	4.20	–0.05 (–1.2)	4.21	4.05	–0.16 (–3.8)*
FEF _{75–85%}	1.80	1.78	–0.02 (–1.1)	1.84	1.67	–0.18 (–9.8)*
FEF _{50%}	4.56	4.48	–0.08 (–1.8)	4.40	4.03	–0.37 (–8.4)*
FEF _{75%}	2.20	2.27	0.07 (3.2)	2.23	2.00	–0.23 (–10.3)*
PD ₂₀	3.91	3.91	0.00 (0.0)	3.91	2.15	–1.76 (–45.0)

*p < 0.01 (paired *t* test).

Difference = after – before.

% = Difference/before × 100.

Table 3 Changes in ventilatory function between recruits and regulars adjusted for age, height, duration of service, time in smoke chamber, and pre-exposure indices.*

	Recruits		Regulars		<i>p</i> Value
	Difference (after-before)				
	Un-adjusted	Adjusted	Un-adjusted	Adjusted	
FEV ₁	-0.02	0.01	-0.04	0.07	0.23
FVC	0.07	0.07	0.00	0.00	0.34
FEF _{25-75%}	-0.05	-0.05	-0.16	-0.16	0.66
FEF _{75-85%}	-0.02	0.01	-0.18	-0.20	0.31
FEF ₅₀	-0.08	-0.07	-0.37	-0.39	0.11
FEF ₇₅	0.07	0.03	-0.23	-0.20	0.19

*By analysis of covariance (ANCOVA).

provide the greatest contribution to the change in PD₂₀ (table 4). The postexposure FEV₁ is not a major contributory factor when compared with the duration of service.

Discussion

Firefighters can be expected to show an increase in airway responsiveness after exposure to products of pyrolysis. Products such as ozone,^{13,14} sulphur dioxide,¹⁵ and oxides of nitrogen¹⁶ have been shown to increase airway responsiveness in some subjects. Changes in airway responsiveness after fire exposure, however, have not been adequately described.⁸ This study showed that changes in airway responsiveness are confined to those subjects with a fairly long history of firefighting activities.

The exact mechanism of increased airway responsiveness is uncertain. No single mechanism could explain the increase in airway responsiveness following exposure to products of combustion. Ozone,^{13,14} sulphur dioxide,¹⁵ and cigarette smoke¹⁷ are known to cause increases in airway reactivity through reflex bronchoconstriction and could explain the findings for the regular firefighters. For at least four of the

regular firefighters, however, the reduction in ventilatory indices could not be fully explained by reflex bronchoconstriction, as they failed to return to pre-exposure levels after 24 hours and administration of bronchodilator. This may be because of ongoing inflammatory response in the airways. There is also evidence that in airway inflammation the vagal reflex is more sensitive or more active. Inhalation of 0.6 ppm ozone for two hours caused an increased response to histamine and metacholine. This enhanced responsiveness to histamine was diminished greatly by large doses of atropine, which suggests that postganglionic vagal endings become more active after bronchial irritation of inflammation caused by ozone.^{13,14} Furthermore, airway inflammation causes bronchoconstriction and this may cause increased proximal bronchial deposition of inhaled substances, thus contributing to an increase in central airway resistance. This has been shown to shift the dose response curve for histamine to the left.¹⁹

The greater magnitude of change in ventilatory function and increase in airway responsiveness among the regular firefighters compared with recruits suggests that some form of chronic epithelial injury is needed before an increase in airway responsiveness is seen. Such injury may increase the sensitivity of airway receptors or increase the permeability of the respiratory epithelium.

The lack of power in this study may have precluded the finding of statistically significant factors contributing to the change in PD₂₀. The magnitude of the standardised partial regression coefficients, however, clearly showed the duration of service as firefighter to be the major factor contributing to the increase in airway reactivity after exposure. The change in baseline FEV₁ could be a confounding factor¹² but its contribution is small in relation to the duration of service.

The degree of physical exercise in negotiating the obstacle course inside the smoke chamber may contribute to an increase in airway responsiveness. Furthermore, a less fit firefighter may spend more time in the smoke chamber as well as having a higher rate of respiration. The effect of physical exercise, however, was reduced by performing the HCT one hour after exposure in the smoke chamber and the effect of variable dose of exposure was partially adjusted for by using the time spent in the smoke chamber in the analysis.

This study does not allow any further conclusions to be drawn as to whether repeated exposures to fire will cause persistent airway hyperresponsiveness or rapid deterioration in lung function. Airway responsiveness, however, has been postulated to be a risk factor for the subsequent development of chronic obstructive pulmonary disease.¹⁹ The increase in airway responsiveness among regular firefighters suggests therefore, that these firefighters are at an

Table 4 Factors influencing change in airway responsiveness among regular firefighters (*n* = 10)

Independent variables	Partial regression coefficients	Standardised partial regression coefficients	<i>p</i> Value	<i>R</i> ²
Age (y)	0.049	0.119	0.11	
Height (cm)	0.052	0.275	0.28	
Duration*	-0.436	-0.843	0.84	
Pack-years	-0.123	-0.180	0.18	
Exposure†	-0.194	-0.258	0.26	
Post FEV ₁ ‡	-1.323	-0.221	0.46	0.73

*Duration of service as a firefighter (y).

†Exposure time in smoke chamber (minutes).

‡Postexposure FEV₁ (l/s).

increased risk of accelerated loss in pulmonary function.

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Coincidence

Coincidence is a potent mechanism for the propagation and maintenance of myths. Many a time the juxtaposition of an unconnected event and the appearance of symptoms of an illness convinces the person concerned (not to mention his doctor) that the two are causally related.

A young man was taken on to help excavate the site of an 18th century infirmary cemetery. He had no previous experience of work of this type and needed constant reassurance that he would not "catch anything from the skeletons." All went well until he and a companion unearthed a lead coffin that was in poor condition and holed in a number of places. Both thought that they could smell an emanation from the coffin although later examination showed that the coffin contained nothing but bare bones. The young man was extremely anxious, and was sent home

by the site director. The next day he presented with an eczematous rash on both cheeks, which he was sure was the result of exposure to some effluvium from the coffin. He refused to work on the site any further and the director advised him to see his own doctor and ask for referral to a dermatologist. In the meantime, the director sought some information herself but met with a conspicuous lack of success from the sources she contacted.

Eventually the report came back from the dermatologist that the young man had indeed acquired an allergic skin reaction, not to noxious vapours from the coffin, but from his girlfriend's new hair spray. He, meanwhile, had found himself more congenial work, and the site director eventually managed to find a source of advice to whom she relayed this tale.

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