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Research Article

Smoke Exposure Has Transient Pulmonary and Systemic Effects in Wildland Firefighters

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Respiratory exposure to air pollutants is associated with cardiovascular morbidity and mortality and firefighters have been shown to be at an increased risk of work-related cardiovascular events. Wildland firefighters experience intermittent, intense exposure to biomass smoke. The aim of this study was to characterize the respiratory and systemic effects of smoke exposure in wildland firefighters. Seventeen seasonal firefighters from a northeastern Ontario community were recruited at the beginning of the fire season and baseline measurements obtained; postexposure measurements were made at various times within 16 d of firefighting. Spirometric measurements showed a transient decline in forced vital capacity within 7 d of fire exposure, not evident by 8–16 d. Induced sputum showed a significant increase in macrophages and epithelial cells within 7 d, with evidence that macrophages had internalized particles; such changes were not evident in the second week following exposure. Likewise, peripheral blood analysis revealed significant increases in erythrocytes, hemoglobin, monocytes, and platelets within the first week after fire exposure, which were diminished 8–16 d in postexposure group. We conclude that acute exposure to forest-fire smoke elicits transient inflammatory responses, both in the airways and systemically. Whether these changes contribute to the observed increased risk of cardiovascular events requires further study.

1. Background

Air pollution is known to be associated with a variety of adverse health effects in humans. Epidemiological evidence clearly documents that air pollution exposure is associated with increased morbidity and mortality due to cardiovascular and respiratory causes [1]. Although the relationship between episodes of poor air quality and acute cardiovascular effects has been recognized for decades, the mechanistic relationships between respiratory exposure to air pollutants and the development of systemic sequelae have only come to be understood more recently. Seaton et al. [2] are widely credited with introducing the hypothesis that the cardiovascular effects of pollution exposure are mediated through the induction of pulmonary inflammation, the mediators from which have systemic effects including the stimulation of an acute phase response [3], increases in blood coagulability [3], and disruption of normal heart rate regulation [4].

Ambient air pollution arises from both natural and anthropogenic sources, and the contribution of forest fires to ambient air pollution has substantial public health relevance. The products of biomass combustion are numerous and can vary considerably depending on the nature of the fuel and burning conditions [5]. Forest firefighters and those living in communities proximal to wildfire can experience extremely high exposure to gaseous and particulate combustion products, with documented health effects [6, 7]. In addition to the threats to individuals exposed locally, pollutants released from forest fires can travel thousands of kilometers to heavily populated urban areas [8].

Given the high smoke exposures experienced by wildland firefighters and the epidemiological evidence supporting an association between cardiopulmonary effects and poor air quality, we hypothesized that inhalation exposure to smoke from forest fires would provoke an inflammatory response in the airways and stimulate changes in the peripheral blood

consistent with increased cardiovascular risk. Swiston et al. have recently examined this issue in forest firefighters within the first day after exposure to biomass smoke [9]; in the present study, we aimed to investigate the extent to which such changes persisted over the first 2 weeks after firefighting. We recruited seasonal forest firefighters working in northeastern Ontario and measured airway and systemic parameters before and at various time points between 1 and 16 d after firefighting.

2. Methods

2.1. Subject Recruitment. Subjects were recruited from among wildland firefighters ("Fire Rangers") employed by the Ontario Ministry of Natural Resources and based out of the Sudbury Fire Management Headquarters during the summers of 2006 and 2007; subject characteristics are summarized in Table 1. All subjects had baseline forced expiratory volume in 1 second (FEV_1) greater than 70% predicted, were not taking any corticosteroid therapy or immunomodulatory medication, and were free from respiratory infection within the previous month. Subjects were asked to refrain from nonsteroidal anti-inflammatory drug use beginning 3 d before baseline measures and for the duration of the study.

This study protocol was approved by the Laurentian University Ethics Board and all subjects provided written, informed consent to participate in the study.

2.2. Study Design. Baseline (BL) measurements were made either before any exposure during the firefighting season or with at least 3 weeks since the previous fire exposure. During the active fire season, Fire Rangers returning from fire exposure contacted the research group for the postexposure (PE) measurements. Because the Fire Rangers were often flown to remote sites, the postexposure measurements were made between 1 and 16 d after exposure, depending on travel constraints. Pre- and postexposure measures were taken in the morning and included blood samples, induced sputum, and lung function, including forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC). A questionnaire was administered at the postexposure visit to obtain a qualitative determination of smoke exposure (including type, duration, and intensity of exposure), physical exertion during firefighting, and symptoms.

2.3. Spirometry. Spirometry was performed with a MicroPlus handheld spirometer (MicroDirect, Lewiston, ME) according to the American Thoracic Society standards [10]. FEV_1 and FVC were repeated three times and the best effort was used for each.

2.4. Sputum Induction and Processing. Sputum was induced by inhalation of a hypertonic saline mist and processed according to Pin et al. [11] and modified according to Pizzichini et al. [12]. Approximately 80% of the population is able to produce adequate sputum using this method [13]. Sputum plugs were selected from the expectorate and if sufficient quantity was obtained (approximately 200 mg), cell smears were prepared and stained with DiffQuik (Fisher Scientific). Differential cell counts were performed by counting 400 nonsquamous cells from duplicate slides. In a subsequent reading of the same slides, macrophages were subdivided into those with no visible particle inclusions (negative), those with fewer than 20 inclusions (low-positive), and those with more than 20 inclusions (high-positive), as described by Mukae et al. [14]. All counts were performed by a technician blinded to the subject and exposure status.

Table 1: Subject characteristics.

Subject	Gender	Smoker	Age	Sampling days 1–7	Sampling days 8–16
1	F		26	√	
2	F		28	√	
3	M	√	30	√	
4	F	√	19	√	
5	M		24	√	
6	M		31	√	√
7	M		30	√	√
8	M		30	√	√
9	M		25	√	√
10	M		20	√	√
11	M		20	√	
12	M		26	√	
13	M		27	√	
14	M		27	√	
15	F	√	24		√
16	M		29		√
17	F		30		√

Note: 1–7 or 8–16 day samples provided by the same subject were not after the same fire exposure.

2.5. Peripheral Blood Analysis. Complete blood counts (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) measurements were performed by LifeLabs Medical Laboratory Services. Erythropoietin concentrations and CC16 levels were measured in serum in our laboratory using commercially available ELISA assays (R&D Systems, Minneapolis, USA, and ALPCO Diagnostics Salem, USA, resp.); the limits of detection of the assays were 2.5 ng/mL and 0.4 ng/mL, respectively. All samples were processed the same day. After processing, samples were sent to LifeLabs for analysis on the same day, and in-house samples were frozen at −80°C until all samples were collected and then analyzed together. None of the measurements of the samples were below the assay detection limits.

2.6. Statistical Analysis. Data are expressed as mean ± SEM. Differences between baseline and postexposure firefighting values were evaluated using a paired t-test. A P value of 0.05 or less was considered to be statistically significant. Pearson correlation analysis was used to examine the relationship between spirometric measurements and macrophage particle inclusions in the lung.

3. Results

3.1. Subject Characteristics. A total of 17 firefighters (12 male, 5 female) were enrolled in the study (Table 1). The average age of the subjects was 27 y with a range from 19 to 30 y. Three subjects smoked: 2 smoked less than 5 cigarettes per day, and 1 smoked approximately 1/2 pack per day. Firefighter experience ranged from 2 to 11 y, with an average of 5 y. All subjects were healthy, free from acute or chronic disease, had normal measures for blood pressure and heart rate, and had a forced expiratory volume greater than 70% predicted for age, gender, and height. None of the subjects used protective gear to prevent smoke inhalation during the study. Some subjects provided multiple samples, such that a total of 14 samples were collected before and after 7 days of fire exposure, and 8 samples were collected before and after 8–16 days of exposure.

3.2. Self-Reported Exposure, Exertion, and Symptoms. Firefighters perceived a relatively low level of smoke exposure: 80% of firefighters reported heavy smoke exposure for only a few seconds at a time, and less than 10% reported heavy smoke exposure for 10 min or longer (data not shown). When fighting a fire, subjects reported levels of exertion comparable to running 44% of the time and comparable to walking 56% of the time. All firefighters reported noticing gray/black-coloured sputum and nasal mucous after exposure. None reported injury or requirement for medical attention.

3.3. Spirometry. There were no significant changes in measured FEV_1 within the first or the second week after exposure (PE) to firefighting (Figures 1(a) and 1(b)). Compared to baseline (BL), FVC was significantly decreased when measured within the first week after return from firefighting (Figure 1(c)) (BL: 5.6 ± 0.36; 1–7 d PE: 5.3 ± 0.37; $P = 0.049$). In contrast, FVC measured during the second week after return from firefighting was not significantly different from baseline (Figure 1(d)) (BL: 5.6 ± 0.48; 8–16 d PE: 5.9 ± 0.49). There were no significant changes in the ratio of FEV_1/FVC within the first or second week after return from firefighting (data not shown).

3.4. Sputum Analysis. Of the 14 subjects that provided measurements at baseline and within 1–7 d of return from fire, 10 fulfilled the criteria for the production of adequate sputum samples. Post-fire exposure, macrophage cell numbers increased significantly in the sputum (Figure 2(a)) (BL: 74.4 ± 22.70 × 10^4 cells/mg; 1–7 d PE: 160.3 ± 69.70 × 10^4 cells/mg; $P = 0.025$). There were no significant changes in neutrophil, lymphocyte, eosinophil, or basophil cell numbers. Bronchial epithelial cells significantly increased within 1–7 d of return from fire (Figure 2(b)) (BL: 3.1 ± 1.33 × 10^4 cells/mg; 1–7 d PE: 11.3 ± 5.31 × 10^4 cells/mg; $P = 0.05$).

The number of inclusion low-positive macrophages increased from 23 ± 5.3% at baseline to 35 ± 4.1% after exposure ($P = 0.02$) with a concurrent decrease in the fraction of inclusion negative macrophages (BL: 68 ± 8.5%; PE: 50 ± 4.4%; $P = 0.02$) within 1–7 d following fire exposure (Figure 3). No significant changes were measured in the inclusion high-positive macrophages. Pearson correlation analysis of FEV_1 and the proportion of inclusion high-positive macrophages produced a correlation coefficient of −0.61 ($P = 0.01$), while FVC versus inclusion high-positive macrophages was −0.569 ($P = 0.02$).

Of the 8 subjects that provided measurements at baseline and within 8–16 d of return from fire, 7 fulfilled the criteria for the production of adequate sputum samples. There were no measureable changes in macrophage, neutrophil, lymphocyte, eosinophil, or basophil cell. numbers nor in bronchial epithelial cells within 8–16 d of return from fire (data not shown). Likewise, there were no measureable changes in the inclusion negative, inclusion low-positive, or inclusion high-positive macrophages within 8–16 d following fire exposure (data not shown).

3.5. Peripheral Blood. Circulating total red blood cells (RBC), total white blood cells (WBC) and leukocyte subsets, platelets, erythropoietin, ESR, and CRP levels were compared at baseline and at 1–7 d and at BL and within 8–16 d of return from fire exposure.

Total RBC measured at baseline and 1–7 d after return from firefighting significantly increased after fire exposure (Figure 4(a)) (BL: 4.9 ± 0.13; 1–7 d PE: 5.0 ± 0.12 × 10^{12}/L; $P = 0.018$), with a concomitant increase in haemoglobin levels (BL: 149.6 ± 3.41; 1–7 d PE: 153.1 ± 2.98 g/L; $P = 0.014$). There was no significant change in the total WBC (data not shown). However, differential cell counts showed a significant increase in monocytes (Figure 4(c)) (BL: 0.5 ± 0.04; 1–7 d PE: 0.6 ± 0.07 × 10^9/L; $P = 0.05$). There were no differences between neutrophils, lymphocytes, eosinophils, or basophils (data not shown). Platelet counts significantly increased 1–7 d after fire exposure (Figure 4(e)) (BL: 237.0 ± 11.15; 1–7 d PE: 248.7 ± 13.2 × 10^9/L; $P = 0.027$); platelets were also elevated in 6 of the 8 subjects 8–16 d after exposure, but this did not reach statistical significance. There were no significant changes in the erythropoietin levels, ESR, CRP, or CC16 (data not shown).

No significant changes were observed in any of the peripheral blood measures in the second week after exposure (Figures 4(b), 4(d), and 4(f)).

4. Discussion

Inhalation of air pollution not only induces airway inflammation but has systemic effects as well, which are thought to be responsible for the increased risk of adverse cardiovascular events associated with air pollution exposure observed epidemiologically [1]. Exposure to smoke from forest fires can initiate similar inflammatory responses: various epidemiological studies have shown associations between wildland fires and emergency room visits for both upper and lower respiratory tract illnesses (including asthma), respiratory symptoms, and decreased lung function as well as increased cardiopulmonary mortality [6, 7]. Occupational exposure to smoke in the context of urban firefighting has been investigated by Kales et al., who examined firefighter deaths in association with work tasks and reported an increased risk

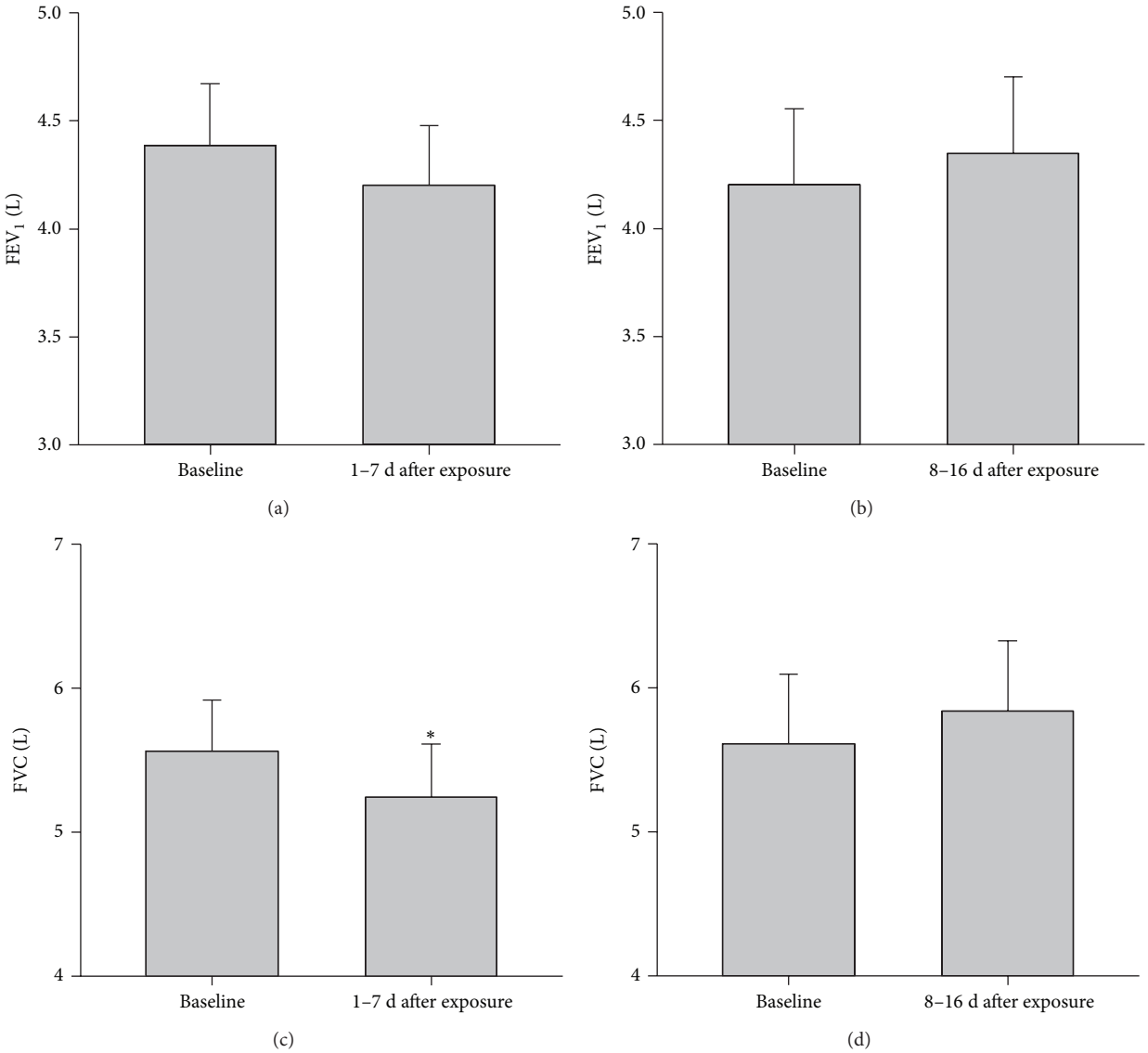

Figure 1: Spirometric measures were measured at baseline and at 1–7 d after exposure and at baseline and 8–16 d after exposure. FEV_1 is shown in panels (a) (1–7 d) and (b) (8–16 d); FVC is shown in panels (c) (1–7 d) and (d) (8–16 d). Measures are expressed in liters (L) as mean ± SEM. There were no significant changes in FEV_1 after fire exposure at either time point. There was a significant decrease in FVC in firefighters 1–7 d after fire exposure ($^*P = 0.049$) but not at 8–16 d after fire exposure.

of acute cardiovascular death shortly after fire exposure [15]. This is in line with work done by Peters et al. (2004) who reported that elevated concentrations of traffic-derived fine particles transiently raised the risk of myocardial infarction within a few hours after exposure [16]. Smoke inhalation by wildland firefighters provides another real-life situation of exposure to particulate matter, allowing for further exploration of these airway and systemic interactions.

Recently, Swiston et al. [9] examined the acute changes in airway and systemic inflammatory responses in wildland firefighters, 4 hours after completing a firefighting shift [15]. In the present study we performed a similar analysis on wildland firefighters, but over a 16 d period after fire exposure. We grouped the data into two time periods: within 1–7 days and within 8–16 days after exposure. We demonstrate that smoke inhalation in healthy firefighters induces measureable inflammatory response during the first week, which was not apparent in the second week after exposure; this response was characterized by an increase in sputum macrophages, airway particulate, and associated bronchial epithelial cell loss. In addition, we measured systemic changes characterized by increases in red blood cells, platelets, and monocytes, all of which are associated with an increased risk for cardiovascular disease.

A small decline in forced vital capacity with exposure to forest fire within 7 days was detected. This was not accompanied by a decline in FEV_1. Several other studies have shown a similar decline in pulmonary function, but in

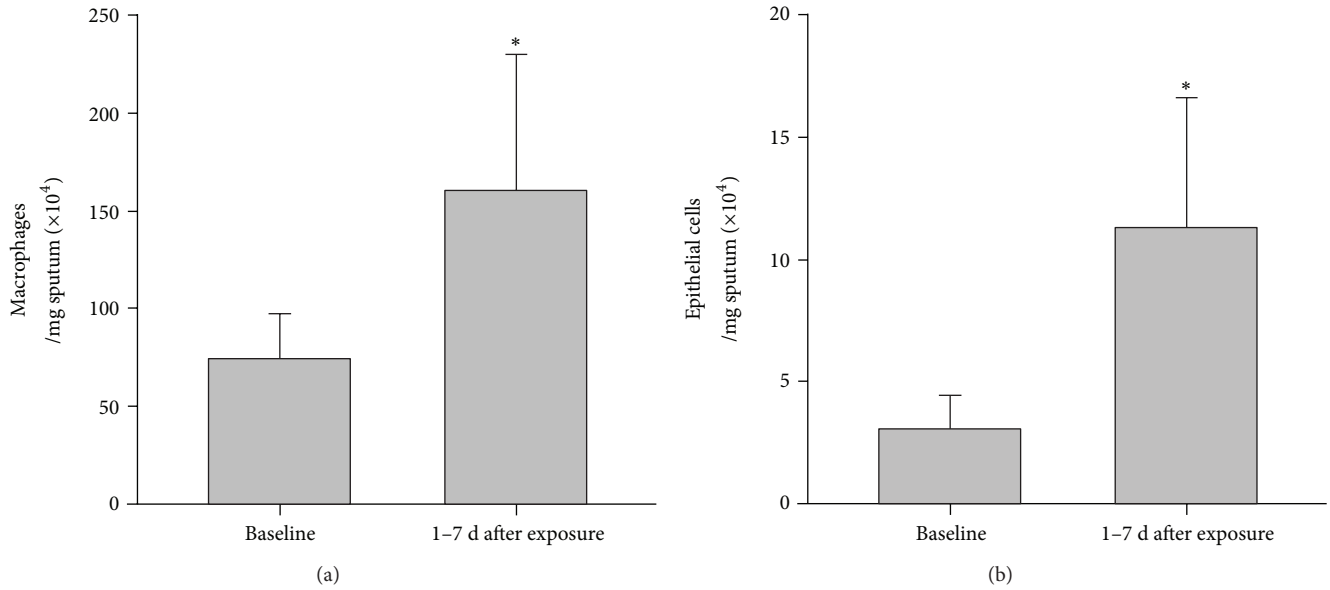

Figure 2: Induced sputum macrophage (a) and epithelial (b) cell counts at baseline and 1–7 days after fire exposure. There was a significant increase in macrophages ($^*P = 0.025$) and epithelial cells ($^*P = 0.05$) after 1–7 days after fire exposure.

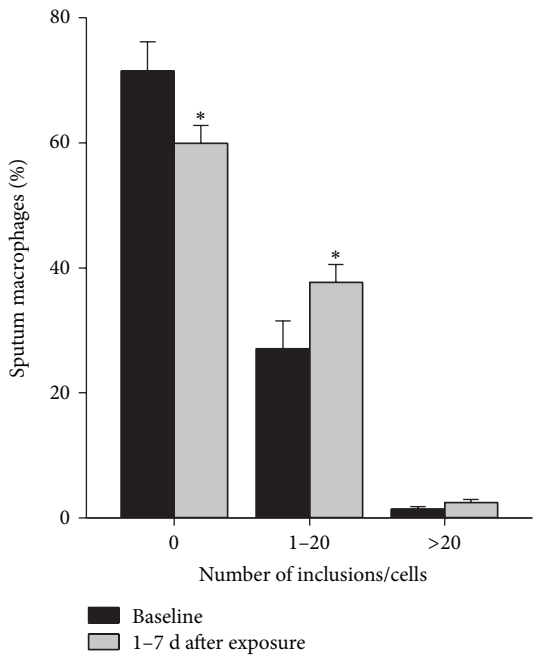

Figure 3: Induced sputum and macrophage particle inclusion cell counts, at baseline and after 1–7 days after fire exposure. 200 macrophage cells were counted per slide and percentage of having zero, less than 20, or more than 20 particle inclusions. Solid bars represent cell counts for each category at baseline. Grey bars represent cell counts for each category after fire exposure. Inclusion negative macrophages significantly decreased 1–7 days after fire exposure ($^*P = 0.01$). Low inclusion positive cells (<20/macrophage) significantly increased 1–7 days after fire exposure ($^*P = 0.02$). There were no significant changes in inclusion high-positive cells (>20/macrophage).

conjunction with a decline in FEV_1 [17, 18]. Although our observations suggest that the change in lung function resolves within a 2-week period after a single exposure to smoke, it is possible that repeated exposures over a longer period of time would result in a progressive decline in lung function. Indeed, the literature indicates that although acute exposure results in small decrements in spirometric measures that resolve, urban firefighters have been shown to experience an increased rate of lung function decline [18–22], and one study suggests that this is true in the case of wildland firefighters as well [23]. Additional studies examining decrements in lung function over time in forest firefighters compared to unexposed subjects would be necessary to establish whether there are chronic effects of occupational exposure to forest fire smoke on lung function.

Analysis of induced sputum samples indicated clearly that occupational exposure to biomass smoke elicits airway inflammatory responses in this population, with an increase in the fraction of airway macrophages, an increase in macrophages with evidence of particle assimilation, and an increase in airway epithelial cell shedding within 7 days following smoke exposure. Epithelial desquamation is commonly detected in airway inflammatory diseases [24, 25]. These airway changes were seen in conjunction with the decline in FVC and with increases in blood monocytes over the same time periods. This suggests a relationship between the ongoing airway inflammation and epithelial damage with the changes in lung function, concomitant with an active recruitment of monocytes from the bone marrow to the blood and airways (where they mature into tissue macrophages). This is in contrast to the results of Swiston et al. [9] who found a rapid increase in granulocytic cells (primarily neutrophils) in the airways within 4 h of return from firefighting, with

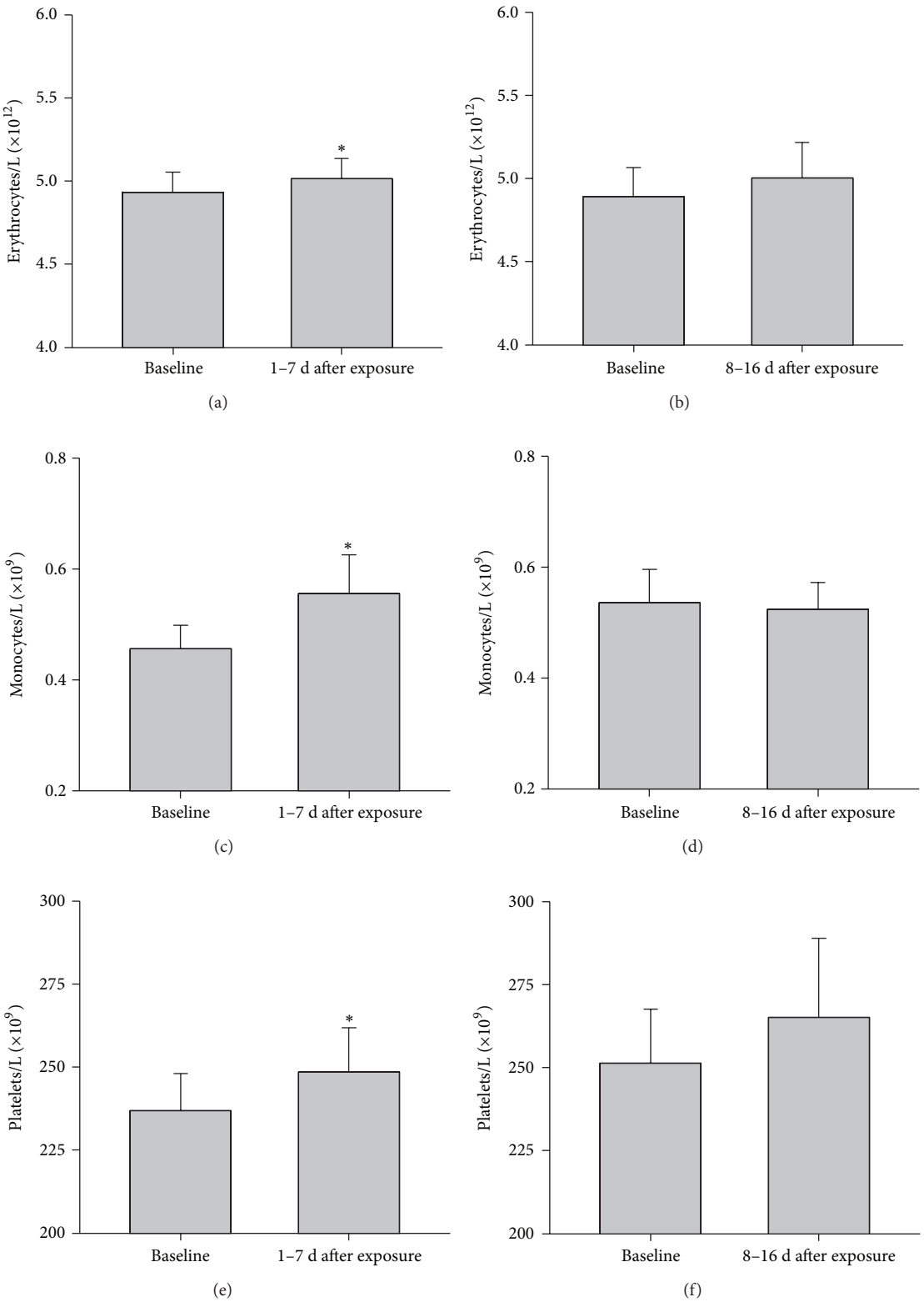

Figure 4: Blood cell counts measured at baseline and 1–7 d after exposure and baseline and 8–16 d after exposure. Erythrocytes are shown in panels (a) (1–7 d) and (b) (8–16 d); monocytes in panels (c) (1–7 d) and (d) (8–16 d); and platelets in panels (e) (1–7 d) and (f) (8–16 d). Measures are expressed in cells/L as mean ± SEM. There were statistically significant increases in all 3 cell types at 1–7 d after exposure ($P = 0.018$ for erythrocytes; $P = 0.05$ for monocytes; and $P = 0.027$ for platelets) but not at 8–16 d after fire exposure.

no measureable increases in macrophages at that time [15]. Together these findings suggest that there is a two-phase inflammatory response to particulate matter: an initial infiltration of granulocytes inducing lung damage, followed by a delayed infiltration of macrophages. The late infiltration of macrophages probably reflects their roles in the removal of inhaled particles and damaged cells and also in orchestrating inflammatory responses. That we observed increased macrophage numbers in the lung through the first week after exposure suggests a sustained increase in inflammatory responses. Ishii et al. have used an *in vitro* coculture system to elicit some of the mechanisms involved in the response to atmospheric particles and concluded that the interaction of the airway epithelium and alveolar macrophages can enhance monocyte production and recruitment, consistent with our findings [26]. This could hint at a mechanism underlying the observed relationship between particulate exposure and cardiovascular events: monocyte production in the bone marrow driven by pulmonary inflammatory responses raises blood monocyte levels, which have been implicated as a risk factor for coronary heart disease [27].

In addition to increased circulating monocytes, we also measured increased numbers of circulating platelets within 7 days of firefighting, which remained elevated in most subjects between 8 and 16 d. This is a novel finding and highlights the link between smoke inhalation and the possibility of enhancing hemostasis. Platelets have an important role in the progression of atherosclerosis and the pathophysiology of cardiovascular events [28]. This increase shortly after inhalation of smoke may explain in part the increased number of cardiovascular events reported after acute exposures to air pollution and suggests that people repeatedly exposed to biomass smoke (firefighters in particular) will also have regular increases in circulating platelet numbers, possibly leading to an overall increased individual risk for cardiovascular events [28]. It would be of interest to examine whether exposure to biomass smoke affects platelet activation as well. Of additional interest are findings indicating that interactions between monocytes and platelets are important in the pathophysiology of cardiovascular events [27, 29]; our observations of increased platelets and monocytes in forest firefighters within the first week after exposure would be consistent with the possibility that these individuals may be at increased risk for a cardiovascular event.

Blood analysis also showed increases in red blood cell numbers and hemoglobin levels within 7 days of firefighting. This is most likely due to exposure to carbon monoxide in firefighters and likely reflects an erythrogenic response to the formation of carboxyhemoglobin and subsequent mild hypoxia. We investigated this possibility by measuring erythropoietin in our serum sample but did not observe any changes in serum erythropoietin; since the half-life of this cytokine is only 3.5 h however, this is not unexpected. Like monocytes and platelet numbers, increased red blood cells are also independently associated with risk of cardiovascular events [30].

We would note three limitations with this study. First, we were unable to directly measure the amount of smoke inhaled by our subjects, so we relied upon the firefighters' qualitative report of smoke exposure and inhalation and an analysis of macrophage particle inclusion in the sputum. Other investigators have examined the relationships between subjective self-reports of smoke exposure and measured levels of PM2.5 [31] and carbon monoxide (CO) [32], giving us the opportunity to make rough estimates of exposure based on our subjects' self-reports. Most of our subjects reported "very low" or "low" levels of smoke exposure; based on the findings of Dunn et al. and Adetona et al., we can estimate that average exposure to PM2.5 was in the range of 50–300 ug/m^3, with CO levels averaging 0.4–2 ppm across the shift [23, 31]. Although these represent estimated average exposures over the course of an entire shift, instantaneous exposures could have been many times higher [31, 32]. While the observed effects in the present study may therefore be attributed to exposures other than inhaled smoke, the increase in sputum macrophages with inclusions confirms an increased inhalation of airborne particulate matter compared to baseline sampling. The largest increase was seen in macrophages with few particle inclusions, suggesting mild to moderate exposure, although given the time frame, this may also be attributed to impaired macrophage phagocytic ability [33]. Perhaps not surprisingly, we found a moderately strong inverse correlation between the spirometric measures and the presence of macrophages with high degrees of particle inclusion, suggesting that more intense exposure to smoke (or impaired clearance mechanisms) is responsible for decrements in lung function. Interestingly, although we did not observe a statistically significant decrease in FEV_1 after fire exposure, the correlation analysis did show a significant inverse correlation between FEV_1 and high particle inclusions.

Second, we did not exclude participants who smoked. Given that only 3 of the 17 subjects were smokers it was not feasible to do a subgroup analysis for the smokers, and indeed we acknowledge that smoking could have influenced measures of relevance. That being said, when excluding the 3 smokers from the data, we found that for most measures significant statistical difference remained. For three measures, statistical significance was lost, specifically, FVC ($P = 0.07$), blood monocytes ($P = 0.09$), and increases in low-particle-positive macrophages ($P = 0.06$). Given the prevalence of smoking in firefighters and in communities exposed to fire smoke we believe it is important to examine health consequences for all people. However, in a future study, it would be important to compare nonsmokers and smokers, as it is important to understand the health effects of occupational exposures on both smoking and nonsmoking subjects.

Lastly, although male and female subjects were both included in this study, the majority of our subjects were male, and the relatively small number of subjects means that subgroup analysis by sex/gender was not feasible. As expected, the FEV_1, FVC, and red blood cell indices in female subjects were lower than in male subjects at baseline (data not shown), but it should be noted that these were all within the predicted normal ranges. Previous studies have suggested that sex/gender is not a significant covariate for

changes in spirometric measures associated with exposure to smoke from wildland fires [17] and within our data there were no discernible differences between the responses of male and female subjects after fire exposure. Although the present study was not designed to detect sex/gender differences, future investigations could address these issues by ensuring sufficient statistical power within subgroups by sex/gender.

In summary, the present study has shown that healthy, seasonal, wildland firefighters exposed to biomass smoke mount a pulmonary and systemic inflammatory response that is sustained through the first week following exposure but diminishes within the second week. Examining wildland firefighters' physiological responses to smoke inhalation provides an opportunity to better understand the effects of biomass air pollution on humans as well as the occupational health risks of firefighters in general. The present results provide a plausible mechanism implicating exposure to inhaled pollutants in the increased risk of cardiovascular events seen in firefighters, which Kales et al. suggested was attributable to poor cardiovascular fitness [15]. In addition, these results support a growing body of research linking the inhalation of particulate matter with a local inflammatory response in the lungs, stimulating a systemic response [34, 35]. It is likely that repeated exposure to wildfire smoke will induce more chronic changes in the airways and circulatory system, inducing long-term consequences for firefighters. Further studies implementing strategies to attenuate the local and systemic effects of inhaled smoke need to be explored.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Acknowledgments

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