Acute Upper and Lower Respiratory Effects in Wildland Firefighters

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Objectives: *To assess acute respiratory effects experienced by wildland firefighters.* ***Methods:*** *We studied two Interagency Hotshot Crews with questionnaires, spirometry, and measurement of albumin, eosinophilic cationic protein (ECP), and myeloperoxidase (MPO) as indicators of inflammation in sputum and nasal lavage fluid. Assessments were made preseason, postfire, and postseason.* ***Results:*** *Fifty-eight members of the two crews had at least two assessments. Mean upper and lower respiratory symptom scores were higher postfire compared to preseason ($P < 0.001$). The mean forced expiratory volume in 1 second was lower postfire compared to preseason ($P < 0.001$) and then recovered by postseason. Individual increases in sputum and nasal ECP and MPO from preseason to postfire were all significantly associated with postfire respiratory symptom scores.* ***Conclusions:*** *Wildland firefighting was associated with upper and lower respiratory symptoms and reduced forced expiratory volume in 1 second. Within individuals, symptoms were associated with increased ECP and MPO in sputum and nasal lavage fluid. The long-term respiratory health impact of wildland firefighting, especially over multiple fire seasons, remains an important concern.* (J Occup Environ Med. *2008;50:1019–1028*)

Municipal and wildland firefighters have an increased prevalence of respiratory problems.[1,2] The chief inhalation hazards associated with wildland firefighting have been identified as carbon monoxide (CO), aldehydes, and respirable particulate matter.[3] Much research has documented deleterious effects of smoke exposure in municipal firefighters.[4–7] Those results, however, may not be generalizable to wildland firefighters, given differences in smoke composition, generally longer duration of fires fought by wildland firefighters, and the fact that respiratory personal protective equipment is routinely, though not always, worn by municipal firefighters but is not generally worn by or even recommended for wildland firefighters.[8]

The Federal government employs about 15,000 seasonal and permanent wildland firefighters each year.[9] Many additional wildland firefighters are employed by State and private agencies. There are four types of wildland firefighter crews: engine crew, hand crew, helicopter crew, and smokejumpers. Type 1 Interagency Hotshot Crews (IHCs) are elite 20-member hand crews that construct fire lines using hand tools during the most dangerous phases of fire suppression.

At a 1997 conference on wildland firefighter health and safety, attendees acknowledged that respiratory problems were common in wildland firefighters and accounted for 30% to 50% of visits to fire incident medical aid stations.[10] Studies examining respiratory symptoms and pulmonary function in wildland firefighters have

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found increases in symptoms, airways hyperresponsiveness, and declines in lung function cross-shift and cross-season.[11–16]

These previously observed increases in subjective symptoms and declines in objective measures of lung function suggest that wildland firefighting is associated with upper and lower airways inflammation and raise concern about potential risk of long-term respiratory effects, including asthma, chronic obstructive pulmonary disease (COPD), and upper airways conditions such as sinusitis. To our knowledge, examination of induced sputum or nasal lavage fluid for objective measures of eosinophilic and neutrophilic inflammation during wildland firefighting has not previously been done.

The question addressed by the present study was whether wildland firefighting is associated with acute and sub-chronic respiratory effects. To address this question, we serially assessed symptoms, spirometry, and markers of inflammation in sputum and nasal lavage fluid in members of two type 1 IHCs of wildland firefighters.

Materials and Methods

Study Population

From 2004 through 2006, we attempted to collect medical and exposure data preseason (in May), in a wildfire setting, and postseason (in October, a minimum of 2 weeks postfire exposure) on all members of the only two type 1 IHCs employed by the National Park Service. We studied the Alpine IHC of Rocky Mountain National Park for 7 days while fighting the Boundary Fire (Fox, AK, July 2004), a very large and intense wildfire, for 7 days while working the Tuolumne Grove Fire (Yosemite National Park, CA, October 2005), a less intense prescribed burn, and for 6 days while fighting the Red Eagle Fire (Glacier National Park, MT, August 2006), a large wildfire. We studied the Arrowhead IHC of Sequoia and Kings Canyon National Parks for 3 days while fighting the South Sundance Fire Complex (Sundance, WY, July 2005), a smaller wildfire that was nearly completely contained during our testing. Preseason participation was 100% for both crews in both years, but crew turnover within seasons and the demobilization of the Alpine IHC shortly after the late-season fire in California resulted in incomplete data for some preseason participants on each crew. Also, a fire-related death of an Arrowhead crew member in the 2004 season led to a decision to cancel studies of this crew for the remainder of that season.

The study protocol was approved by the National Institute for Occupational Safety and Health (NIOSH) Human Subjects Review Board and informed consent was obtained from each research participant.

Questionnaire

A preseason questionnaire, a modification of the standardized American Thoracic Society (ATS) Adult Respiratory Questionnaire,[17] ascertained: lifetime chronic respiratory conditions, history of tobacco use, history of symptoms over the past week, volunteer firefighter status, and lifetime occupational history. A separate postfire/postseason questionnaire ascertained: exposures, new diagnoses, work histories, and changes in symptoms since the last interview. A validated symptom scale, with Likert scoring where 0 = none, 1 = trivial, 2 = mild, 3 = moderate, and 4 = severe for upper and lower airways symptoms, was used to derive overall symptom scores by summing the responses to questions on 19 symptoms.[18] Symptoms ascertained included cough, wheeze, sputum production, shortness of breath or chest tightness, and shortness of breath while walking, as well as various eye, nose, and throat symptoms. At each wildfire studied, participants were also asked daily to rate the severity (none, mild, moderate, or severe) of his or her smoke exposure for the preceding shift.

Spirometry

Spirometry was conducted preseason, daily cross-shift during each studied wildfire, at the conclusion of each studied wildfire, and postseason. Technicians who had completed a NIOSH-approved spirometry course followed ATS guidelines using an ultrasonic flow spirometer (EasyOne Diagnostic Spirometry System 2001, ndd Medical Technologies, Zurich, Switzerland).

We used equations for predicted values and lower limits of normal (LLN) derived from National Health and Nutrition Examination Survey (III) data.[19] We defined obstruction as a ratio of the forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) $<$ LLN with FEV_1 $<$ LLN; borderline obstruction as an FEV_1/FVC ratio $<$ LLN with normal FEV_1 and normal FVC; and restriction as a normal FEV_1/FVC ratio with FVC $<$ LLN. We used several criteria to define FEV_1 changes in an individual as potentially significant: two criteria, a decline of 12% or greater[20] and a decline of 8% or greater,[21] for cross-season decline; and one criterion of 10% or greater for cross-shift FEV_1 decline.[22]

We followed ATS procedure by requesting medications and asking participants to abstain from these medications for 1 hour before performing spirometry at the preseason, postfire, and postseason assessments. Nevertheless, we did not ask a participant to abstain from his or her medications during cross-shift testing at a wildfire, as we felt this may have jeopardized his or her safety.

Induced Sputum and Nasal Lavage Analyses

Whole induced sputum was collected using a well-validated technique that minimizes salivary contamination of the sample.[23] Nasal lavage was collected using normal saline as previously described.[24]

Due to the challenges of handling specimens in the setting of active forest fires, we used a simplified sample processing and analytical approach modified from Metso et al.[25] In this approach, cells within the sample are lysed and total intracellular and extracellular myeloperoxidase (MPO) and eosinophilic cationic protein (ECP) are assessed, providing measures of neutrophilic and eosinophilic inflammation, respectively. Albumin was also examined as a marker of inflammatory-associated transudation. After collection, samples were frozen on dry ice in the field and held at −80°C until analyzed. In the laboratory, samples were thawed, volumes measured, and mucus liquefied by addition of two volumes 1% dithiothreitol (Sputolysin Stat-Pack; Caldon Biotech) to one volume of sample. In addition, the protease inhibitor phenylmethanesulfonyl fluoride (Sigma Chemical Co) was added to a final concentration of 1 mM and proteolytic inhibitor cocktail (Cat. # P-8340, Sigma Chemical Co) was added to a final concentration of 0.05%. After shaking for 15 minutes at room temperature, aliquots were removed and complete cell lysis facilitated by addition of an equal volume of 0.4% hexadecyltrimethylammonium bromide, followed by vigorous shaking for 1 hour at room temperature.[26] The samples were centrifuged (800 × g for 10 minutes) and supernatant fluids stored frozen in aliquots at −80°C until analyzed for albumin, ECP, and MPO.

Human albumin was quantified using enzyme-linked immunosorbent assay (ELISA) (Cat. # E80-129, Bethyl Laboratories, Inc., Montgomery, TX), following manufacturer's instructions. Assays were performed using reagents included in the ELISA kit, as well as MaxiSorp 96-well ELISA plates (Nunc A/S, Denmark), and TMB Microwell Peroxidase Substrate (KPL Inc, Gaithersburg, MD). ECP was measured in duplicate by fluoroimmunoassay (Pharmacia CAP System; Phadia, Uppsala, Sweden) as per the manufacturer's instructions. MPO was measured by ELISA in duplicate and at several sample dilutions for extrapolation from the MPO ELISA standard curve, as instructed by the manufacturer (Assay Designs Inc, Ann Arbor, MI). For our analyses, we used the resulting concentrations (reflecting intracellular plus extracellular content) of each analyte in sputum and nasal lavage fluid[24] and total recovered amounts of albumin, ECP, and MPO. Total recovered amounts were calculated by multiplying the analyte concentrations by the recovered volumes.

Exhaled Breath Carbon Monoxide

Exhaled breath CO was collected daily cross-shift on each participant during each studied wildfire using a breath CO monitor according to the manufacturer's instructions (Micro 4 Smokerlyzer, Bedfont Scientific, Medford, NJ).

Statistical Methods

We restricted our analyses to data from: 1) all three assessments (ie, preseason, at fire, and postseason) from the first complete fire season of data for participants with all three assessments during at least one fire season ($n = 32$); and 2) the preseason or fire assessment and one other assessment from the first season for which a preseason or fire and only one other assessment was done for other participants ($n = 26$). Data from participants with only a preseason survey ($n = 11$) were excluded from analyses of health effects.

Cross-season predictors of several outcome variables (FEV_1, FVC, upper and lower respiratory symptom scores, and sputum and nasal lavage fluid albumin, ECP, MPO, and volume) were examined using the SAS MIXED procedure for repeated measures with a first-order autoregressive correlation structure.[27] In multifactor models, FEV_1 values were adjusted for age, sex, height, and race/ethnicity. Sputum and nasal lavage fluid results were log-transformed for inclusion in the models. The following time-varying predictor variables were examined: cumulative months spent fighting fires (throughout career), days spent fighting fires (current season), fire assignment, asthma, allergies, upper respiratory infections, upper and lower respiratory symptom scores, and smoking status. Similar models were examined to assess preseason to postfire differences and postfire to postseason differences in outcome variables. To investigate the influence of the preseason values of each of the outcome variables on subsequent postfire and postseason values of the same variable, we ran models where the outcome variables were restricted to postfire or postseason observations.

We also examined postfire associations between respiratory symptom scores and change in inflammatory markers from preseason within individuals using ordinary least squares techniques. These models were adjusted for preseason respiratory symptom scores.

Cross-shift mean changes in FEV_1 and exhaled breath CO at a fire were investigated using paired difference *t* tests. We moreover examined associations between individual participants' mean cross-shift change in FEV_1 and several predictor variables (age, gender, height, race/ethnicity, asthma, allergies, fire assignment, self-reported smoke exposure rating, and postshift exhaled breath CO) using multiple regression models comparable to those detailed above.

Results

Characteristics for the entire group of preseason participants ($n = 69$) and the group of participants included in the health effects analyses ($n = 58$) are detailed in Table 1. Based on these characteristics, the group analyzed was very similar to the entire population of these two crews. They had a median age of 26 years, had similar firefighting experience, and were comprised primarily of White, non-Hispanic males. Ap-

TABLE 1
Characteristics of Interagency Hotshot Crew (IHC) Members at Preseason Assessment

Variable	Participants at Preseason Testing $n = 69$	Participants Included in Analyses $n = 58$
Median age, yr	26 (22, 33)*	26 (22, 33)*
Median time spent as a firefighter, mo	1 (1, 85)	1 (1, 85)
Male *n* (%)	61, 88%	52, 90%
White, non-Hispanic *n* (%)	64, 93%	55, 95%
Current smoker *n* (%)	5, 7%	3, 5%
Former smoker *n* (%)	17, 25%	15, 26%
Allergies (ever) *n* (%)	24, 35%	20, 34%
Asthma (ever) *n* (%)	13, 19%	10, 17%
Median forced expiratory volume in 1 s (FEV_1) % predicted	101 (87, 116)	102 (87, 116)
Median forced vital capacity (FVC) % predicted	102 (89, 116)	102 (89, 118)
Median FEV_1/FVC (%)	83 (73, 88)	83 (73, 89)

*Tenth, 90th percentiles.

proximately 5% were current smokers and about 26% were former smokers. Nearly 35% reported having allergies and about 17% reported having been diagnosed with asthma. Median pulmonary function values were about 100% of predicted.

At the preseason evaluation, 13 participants reported ever having had physician-diagnosed asthma (Table 2). Three participants reported initial asthma diagnosis in adulthood after becoming a firefighter; all three reported current asthma and current asthma medication, but had normal spirometry. Of the 10 participants reporting an asthma diagnosis in childhood, two reported current asthma. One of these two, a current smoker, had abnormal (obstructive) spirometry and was taking asthma medication. None of the others reporting childhood asthma had abnormal spirometry. Among participants with no reported history of respiratory disease, four (three never smokers; one smoker) had borderline obstruction and one (a never smoker) had mild restriction at the preseason assessment.

At the postseason evaluation, firefighters reported an average of 16 fire assignments over the season. Crew assignments between preseason assessment and the studied fire averaged nine fires: firefighting averaged 4 days and shift length averaged 14 hours. Crew assignments between the studied fire and the postseason assessment averaged six fires: firefighting averaged 4 days and shift length averaged 14 hours.

Questionnaire

Upper and lower respiratory symptom scores were both higher postfire compared to preseason and postseason (Table 3). The mean upper respiratory symptom score was 5.0 preseason, compared to 14.1 postfire ($P < 0.001$) and 8.6 postseason ($P < 0.05$). The postseason score was also significantly lower than the postfire score ($P < 0.001$). The mean lower respiratory symptom score was 1.7 preseason, compared to 4.1 postfire, ($P < 0.001$) and 2.5 postseason ($P = 0.27$). The postseason score was significantly lower than the postfire score ($P < 0.05$).

TABLE 2
Preseason Characteristics of the 13 Participants who Reported Ever Having Asthma

Current Asthma	Age at Asthma Diagnosis (Yrs)	Smoking History	Forced Expiratory Volume in 1 Second (FEV_1) (% Predicted)	Forced Vital Capacity (FVC) (% Predicted)	FEV_1/ FVC (%)	Current Medication for Asthma
Yes	5	Yes	73*	98	61*	Yes
Yes	6	No	115	118	82	No
Yes	23	Yes	102	102	83	Yes
Yes	30	No	117	119	82	Yes
Yes	32	No	111	103	87	Yes
No	5	No	116	123	81	—
No	8	Yes	93	91	85	—
No	9	Yes	103	105	81	—
No	10	No	98	99	81	—
No	11	Yes	95	92	85	—
No	11	No	118	113	86	—
No	12	No	88	86	85	—
No	12	No	99	100	86	—

*Spirometry value abnormal.
–, not applicable.

TABLE 3
Unadjusted Mean Values for Symptoms, Spirometry, and Inflammatory Marker Concentrations

	Preseason		Postfire		Postseason		P*		
	Score or Value	n	Score or Value	n	Score or Value	n	Preseason to Postfire	Preseason to Postseason	Postfire to Postseason
Symptoms									
Upper respiratory symptoms	5.0	56	14.1	50	8.6	42	$P < 0.001$	$P < 0.05$	$P < 0.001$
Lower respiratory symptoms	1.7	56	4.1	50	2.5	42	$P < 0.001$	NS	$P < 0.05$
Spirometry									
FEV_1 (L)	4.57	56	4.35	50	4.54	42	$P < 0.001$	NS	$P < 0.001$
FVC (L)	5.58	56	5.53	50	5.62	42	NS	NS	NS
Sputum									
ECP (μg/L)	1457	56	1537	50	1128	42	NS	NS	NS
MPO (ng/mL)	10,457	56	6464	50	8075	42	NS	NS	NS
Albumin (μg/mL)	177	56	233	50	134	42	NS	NS	NS
Volume (mL)	4.3	56	5.8	50	4.9	42	$P < 0.05$	NS	NS
Nasal Lavage Fluid									
ECP (μg/L)	154	56	651	50	584	42	$P < 0.01$	$P < 0.05$	NS
MPO (ng/mL)	1468	56	3642	50	8745	42	NS	NS	NS
Albumin (μg/mL)	106	56	48	50	88	42	$P < 0.05$	NS	$P < 0.05$
Volume (mL)	6.4	56	6.4	50	5.8	42	NS	$P < 0.01$	$P < 0.01$

*P-values are from univariate models in which sputum and nasal lavage values were log-transformed.

In multifactor analyses, greater cumulative time spent fighting fires (throughout career) was significantly associated with higher upper respiratory score at each time point ($P < 0.05$) after adjusting for significant associations of preseason respiratory symptom score and with recent upper respiratory infection. Days spent fighting fires (current season), history of asthma, allergies, age, and smoking status were not significantly associated with respiratory symptom scores.

Spirometry

Univariate analysis showed a mean FEV_1 decline of 224 mL ($P < 0.001$) from preseason to postfire, followed by an increase of 190 mL from postfire to postseason ($P < 0.001$) (Table 3). The postseason mean FEV_1 was not statistically different from the preseason mean FEV_1 ($P = 0.60$). Mean FVC values did not change significantly over these same three time points.

One participant's FEV_1 fell 12% across the season. The next largest cross-season decline was 8%, observed in three participants. All four had lung function that remained within the predicted normal range at both preseason and postseason assessments. None reported having been diagnosed with asthma; three were former smokers. Their median age was 23.

The overall mean cross-shift change in FEV_1 was a 30 mL decline ($P = 0.12$). However, cross-shift change varied by fire incident: an 80 mL mean decline at the wildfire in Alaska ($P < 0.001$); a 64 mL mean decline at the prescribed fire in California ($P = 0.12$); a 6 mL mean decline at the wildfire in Wyoming ($P = 0.99$); and a 40 mL mean decline at the wildfire in Montana ($P = 0.08$). Figure 1 details the results from the wildfire in Alaska, the fire associated with the greatest cross-shift changes in FEV_1.

Four of the 19 participants at the Alaska fire each experienced a single cross-shift FEV_1 decline greater than 10% (range: 10% to 11%). All four had normal lung function at both preseason and postseason assessments. One reported having been diagnosed with asthma (resolved); two were former smokers. Their median age was 27. No other cross-shift declines of that magnitude were observed for any participants at the other three fires.

In multifactor analysis, after adjusting for a significant association between an individual's preseason FEV_1 and that individual's subsequent FEV_1 values, lower FEV_1 values were associated with greater upper respiratory symptom scores ($P < 0.05$), with higher sputum ECP recovered values ($P < 0.05$), and with higher sputum MPO recovered values ($P < 0.01$). Similar associations were observed when we examined concentration values of these inflammatory markers. Cumulative time spent fighting fires (throughout career), days spent fighting fires (that season), allergies, asthma, upper respiratory infection in the week preceding testing, and smoking status were not significantly associated with FEV_1 at any time point.

In multifactor analysis, cross-shift FEV_1 change was not significantly associated with age, gender, height, race/ethnicity, asthma, allergies, fire assignment, postshift exhaled breath CO, or self-reported smoke exposure rating.

Induced Sputum and Nasal Lavage Analyses

Induced Sputum. Mean sputum concentrations (reflecting intracellular

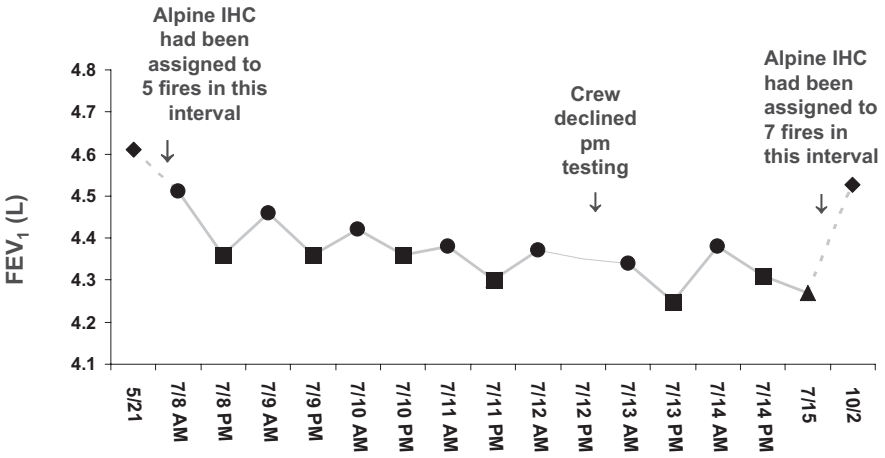

Fig. 1. Unadjusted mean FEV_1 values relating to the Boundary Fire, Fox, AK (n = 19 participants). IHC: Interagency Hotshot Crew. Preseason and postseason testing (◆); preshift testing during fire (●); postshift testing during fire (■); postfire testing (▲). Statistically significant declines were observed from mean preseason FEV_1 to mean preshift FEV_1 on first day of fire ($P < 0.05$), from mean preseason FEV_1 to mean postfire FEV_1 ($P < 0.05$), and in mean cross-shift FEV_1 ($P < 0.001$). No statistically significant difference was observed preseason to postseason.

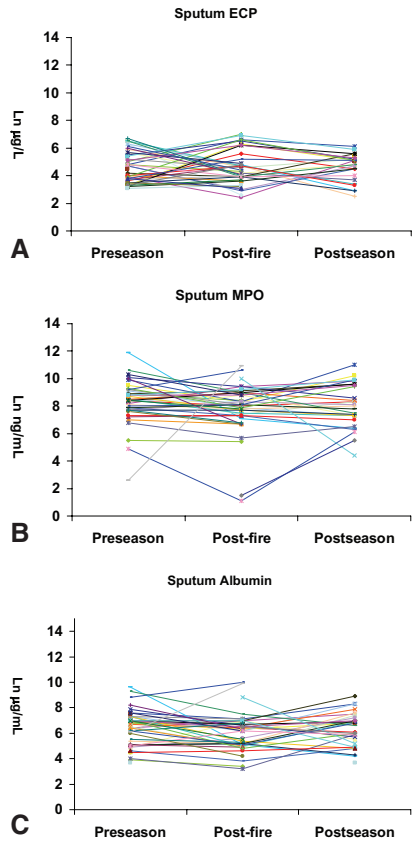

Fig. 2. ECP (A), MPO (B), and albumin (C) concentrations in sputum (n = 56 participants). Measurements reflect both intracellular and extracellular ECP and MPO (see Methods). ECP, eosinophil cationic protein; MPO, myeloperoxidase.

plus extracellular content) of albumin, ECP, and MPO were not significantly different at preseason, postfire, or postseason (Table 3 and Figs. 2A–C). There was marked variability, both within individuals and between surveys, in all of these measures (Figs. 2A–C). Mean sputum volume was significantly increased postfire compared to preseason ($P < 0.05$).

In multifactor analyses of inflammatory markers in sputum, higher MPO concentrations were associated with higher scores for both upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.01$); higher ECP concentrations were associated with higher scores for both upper respiratory symptoms ($P < 0.05$) and lower respiratory symptoms ($P < 0.05$); and albumin concentrations were not significantly associated with any of the examined factors. Each model was adjusted for significant association between preseason and subsequent values of the respective outcome variable. Comparable associations were observed in models examining total recovered amount of each of these three inflammatory markers.

Within individuals, postfire respiratory symptom scores were significantly associated with preseason to postfire differences in total recovered amount of albumin, ECP, or MPO in sputum (Table 4). The greater the increase in sputum ECP from preseason to postfire, the higher the postfire scores for upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.001$). Greater increases in sputum MPO from preseason to postfire were also associated with higher scores for postfire upper respiratory symptoms ($P < 0.001$) and lower respiratory symptoms ($P < 0.001$). In contrast to other parameters measured in sputum, differences in sputum albumin were inversely related to respiratory symptoms: the greater the increase in albumin from preseason to postfire, the lower the postfire scores for upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.001$). Similar associations were observed when we examined concentration values of these three inflammatory markers.

Nasal Lavage. Compared with sputum values, there was marked variability in nasal lavage concentrations (reflecting intracellular plus extracellular content) of ECP and MPO (Table 3 and Figs. 3A–C). Still, mean ECP concentration in nasal lavage fluid increased significantly from preseason to postfire ($P < 0.01$) and from preseason to postseason ($P < 0.05$). In contrast, mean albumin concentration in nasal lavage fluid decreased significantly from preseason to postfire ($P < 0.05$) and from preseason to postseason ($P < 0.05$). Mean MPO concentration did not significantly change over the three time points.

In multifactor analyses of inflammatory markers in nasal lavage fluid, higher ECP concentrations were significantly associated with higher lower respiratory symptom scores ($P < 0.05$); MPO and albumin concentrations were not significantly associated with any of the examined factors. The ECP and MPO models were adjusted for significant associations between preseason and subsequent values of ECP and MPO,

TABLE 4
Within-Individual Associations Between Changes in Inflammatory Markers and Postfire Respiratory Symptom Scores

Preseason to Postfire Change in Total Recovered Amount*	*P*	
	Postfire Upper Respiratory Symptom Score	Postfire Lower Respiratory Symptom Score
Sputum		
ECP (increase)	$P < 0.01$	$P < 0.001$
MPO (increase)	$P < 0.001$	$P < 0.001$
Albumin (decrease)	$P < 0.01$	$P < 0.001$
Nasal Lavage Fluid		
ECP (increase)	$P < 0.01$	$P < 0.001$
MPO (increase)	NS	$P < 0.05$
Albumin (decrease)	NS	NS

*Parenthetical "increase"/"decrease" indicates direction of association.

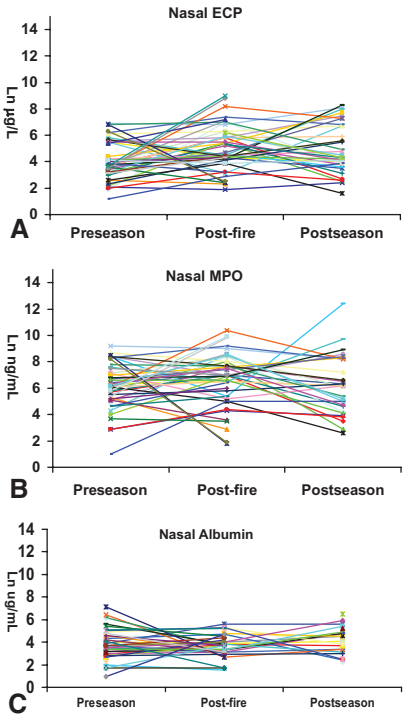

Fig. 3. ECP (A), MPO (B), and albumin (C) concentrations in nasal lavage fluids (n = 56 participants). Measurements reflect both intracellular and extracellular ECP and MPO (see Methods). ECP, eosinophil cationic protein; MPO, myeloperoxidase. Mean nasal ECP concentration was significantly lower preseason compared to postfire ($P < 0.01$) and postseason ($P < 0.05$). Mean nasal albumin was significantly lower postfire compared to preseason ($P < 0.05$) and postseason ($P < 0.05$).

respectively. The MPO model was furthermore adjusted for a significant association between MPO concentration and older age. Cumulative time spent fighting fires (throughout career), days spent fighting fires (that season), having an upper respiratory infection in the week preceding testing, allergies, asthma, and smoking status were not significantly associated with sputum or nasal fluid inflammatory markers or volume.

Within individuals, postfire respiratory symptom scores were significantly associated with preseason to postfire differences in nasal lavage fluid ECP and MPO (Table 4). The significance of these associations was essentially the same regardless of whether ECP and MPO values were expressed as concentrations or total recovered amounts. The larger the increase in ECP from preseason to postfire, the higher the postfire score for upper respiratory symptoms ($P < 0.01$). Also, the larger the increase in ECP ($P < 0.001$) or MPO ($P < 0.05$) from preseason to postfire, the higher the postfire score for lower respiratory symptoms. Changes in albumin from preseason to postfire were not significantly associated with postfire respiratory symptom scores.

Exhaled Breath Carbon Monoxide

The mean preshift exhaled breath CO level was 2.7 parts per million (ppm) and the mean postshift level was 3.7 ppm ($P < 0.001$). No individual CO levels exceeded 16 ppm.

Discussion

We observed significantly increased respiratory symptom scores postfire compared to preseason. This finding is consistent with observations made by Rothman et al,[15] who observed a significant increase in eye irritation, nose irritation, cough, phlegm, and wheezing from preseason to late-season among 52 wildland firefighters, with strong associations noted for recent firefighting activity. In our study, the increased scores for lower respiratory symptoms observed postfire returned to near preseason levels during the postseason. Upper respiratory symptom scores remained significantly elevated at postseason compared to preseason, although scores were significantly lower at postseason compared to postfire. These observations suggest substantial recovery from respiratory tract effects of firefighting by the time of our postseason assessment. Nevertheless, the finding in multifactor analyses that cumulative time spent fighting fires over a career was significantly associated with increased upper (but not lower) respiratory symptoms suggests that wildfire-associated exposure may produce a more sustained rhinitis/sinusitis. Betchley et al[11] observed no significant increase in symptoms cross-season in their study of 53 wildland firefighters, but their postseason testing was done well over a month later in the season than ours and may have allowed for more complete recovery. However, previous NIOSH investigators who made postseason assessments earlier in the calendar year than ours also found no cross-season increase in symptoms.[13]

Corresponding to our symptoms findings, we observed a statistically significant reduction in mean FEV_1 postfire compared to preseason. Likewise, we observed recovery of FEV_1 from postfire to postseason.

Similarly, a previous study of type I Hotshot crews found no significant mean cross-season FEV_1 decline.[13] These findings contrast with those from somewhat larger studies by Liu et al[12] and Betchley et al,[11] who did observe significant mean cross-season FEV_1 declines among wildland firefighters.

We observed an overall mean cross-shift decline in FEV_1 during firefighting that was not statistically significant. However, the magnitude and statistical significance varied by individual fire, and the mean cross-shift decline at the Alaska fire was highly statistically significant. Although reasons for cross-shift differences by fire are uncertain, the fire intensity and the extended shifts worked during the Alaska fire may have contributed to the greater cross-shift decline observed for that fire. Betchley et al,[11] Slaughter et al,[16] and previous NIOSH investigators[13] also observed statistically significant mean cross-shift FEV_1 declines among wildland firefighters during fires.

Only one participant in our study experienced a cross-season decline in FEV_1 as much as 12%, a criterion recommended by an ATS/European Respiratory Society Task Force for defining statistically significant FEV_1 change for an individual.[20] A total of four of the 33 (12%) participants with cross-season measurements experienced a cross-season decline in FEV_1 of at least 8%, an alternative criterion for defining significant FEV_1 change for an individual over a 6- to 12-month period.[21]

A different four participants experienced a single cross-shift decline in FEV_1 of 10% or greater, a criterion for defining a statistically significant cross-shift drop in FEV_1.[22] Notably, all four of the significant cross-shift changes occurred at the Alaska fire. Although our study cannot indicate whether individual firefighters with greater cross-shift and cross-season declines are at greater risk for progressive declines in lung function over subsequent years, evidence from worker populations chronically exposed to other occupational agents with acute respiratory effects would suggest that they are at greater risk.[28,29]

We predicted that respiratory symptoms or changes in lung function associated with wildland firefighting would be associated with airways inflammation. Inflammation of the lower airways is found in both asthma and COPD and plays an important role in the pathogenesis of these disorders.[30] COPD has been associated with neutrophilic inflammation and the neutrophil-related enzyme MPO in sputum.[31] Allergic asthma is commonly associated with increases in eosinophils and ECP in sputum, though some work-related asthma has been found to be predominantly mediated by neutrophils.[32,33] Wildland firefighting is stressful; psychological stress has been associated with exacerbation of eosinophilic inflammatory changes in sputum of asthmatics.[34] Wildland firefighting is also physically demanding; intense physical exertion has been associated with neutrophilic inflammatory changes in sputum.[35] Eosinophilic and neutrophilic inflammation in upper airway conditions, including allergic and nonallergic rhinitis, have been associated with increased inflammatory markers in nasal lavage fluid.[36–38]

We found that assessments of population mean concentrations of ECP and MPO were somewhat informative for nasal lavage fluid but were not informative for sputum. Nevertheless, within-person analyses of airways inflammation in wildland firefighters indicated that greater preseason to postfire increases in sputum ECP and MPO were significantly associated with higher postfire respiratory symptom scores. Similarly, greater preseason to postfire increases in ECP and MPO in nasal lavage fluid were significantly associated with higher postfire respiratory symptom scores. These results suggest that symptoms reflect induction of airways inflammation by wildfire-related exposures.

In contrast to results of the within-person analyses of sputum ECP and MPO, within-person analyses of sputum albumin indicated that greater preseason to postfire increases in sputum albumin were significantly associated with lower postfire respiratory symptom scores (Table 4). Also, in contrast to mean nasal lavage fluid ECP, mean nasal lavage albumin was reduced postfire. These findings seem paradoxical, as increased airway albumin is commonly employed as a marker of transudation. During inflammation, transudation can be mediated both pharmacologically by mediators such as histamine and pathologically by loss of integrity of the endothelial and/or epithelial barriers. It is possible that compensatory mechanisms such as development of tolerance to repeated insult and/or increased albumin clearance (eg, through digestion by inflammatory proteases in the airways) could have contributed to the decreased postfire albumin levels we observed in nasal lavage fluid.

Typically, sputum is processed for cellular analysis by separating cells and cell-free supernatant.[39] However, given the need to treat all sputum and nasal lavage fluid samples uniformly in difficult wildfire settings, we did not obtain cell counts. Rather, we lysed cells in the liquefied samples and expressed ECP and MPO as concentration and as total amount in recovered sputum and nasal lavage fluid, representing the combined content of the intra- and extra-cellular compartments.[25] Although not the usual approach to evaluation of neutrophilic and eosinophilic inflammation, this method was feasible to perform in this particular study and has been suggested as an option for sputum analysis.[40]

An important limitation of our study is the relatively small number of participants, which may have limited our ability to detect some associations. Although, we did observe some temporal associations of symptoms and pulmonary function results

with firefighting activities, the generally qualitative and self-reported nature of exposure characterization in our study may have limited our ability to have identified statistically significant symptom and pulmonary effects related to firefighting exposures (eg, time spent fighting fires, fire assignment, self-reported smoke exposure rating, and postshift exhaled breath CO). Exhaled breath CO was the one objectively measured and quantitative, though indirect, measurement of exposure that we analyzed. Exhaled breath CO has been shown to be elevated in people with COPD.[41] Exhaled breath CO showed a significant cross-shift increase, offering objective evidence for exposure to the products of combustion during the firefighting shifts. Nevertheless, exhaled breath CO was not found to be a significant determinant of cross-shift FEV_1 decline, perhaps because exhaled breath CO is not a reliable surrogate for the irritant smoke particulate and gas exposures that likely cause airways effects in firefighters.

Our study lacked cross-shift data during periods in which the crews were not fighting fires, which would have enabled comparison of the magnitude of fire-associated cross-shift FEV_1 declines with the magnitude of cross-shift declines, if any, that these same participants may experience when not subjected to firefighting exposures. Having cross-shift data during nonfire periods may have strengthened the inference that the cross-shift declines we observed were caused by fire-related exposures. However, ruling out physical exhaustion as a cause of our cross-shift findings would have required that the participants be subjected to the same physical exertion and exhaustion during the unexposed periods of study as during the firefighting periods.

We collected data from the Alpine IHC at three fires, each lasting 6 to 7 days in duration, and data from the Arrowhead IHC at only one fire lasting 3 days. Thus, both crews were not equally represented in the analyses, and we studied only one of the crews while fighting a largely uncontained wildfire—the Alaska fire—at which we observed the largest cross-shift FEV_1 declines.

Our findings are limited by crew attrition over the course of the wildfire season. It is possible that more susceptible participants might have been more likely to have left the crew midseason and been lost to follow-up. If so, we may have underestimated the apparent effect of firefighting on cross-season change in FEV_1. Some evidence suggests that this may have occurred. Participants who quit the crew during the season ($n = 5$) had a higher mean score for lower respiratory symptoms at preseason than all other participants ($n = 64$) (3.8 vs 2.6; $P = 0.05$). The five who quit also had a slightly lower mean preseason percent predicted FEV_1 (98%) compared to participants who remained (101%), although this difference was not statistically significant.

In summary, we observed cross-shift reductions in lung function among type 1 IHC members while firefighting. We also observed statistically significant differences in mean lung function and respiratory symptom scores at the postfire assessment compared to the preseason assessment. At the individual level, increased ECP and MPO in sputum and nasal lavage fluid were associated with higher postfire respiratory symptom scores. Although we observed evidence of recovery from most of the short-term effects by the end of the firefighting season, they raise the possibility that wildland firefighters may be at increased risk for development of chronic lung and upper airways disease. This possibility is additionally supported by our finding that upper respiratory symptom scores were related to cumulative time spent fighting wildfires over a career. More studies are warranted to investigate potential long-term adverse respiratory effects of firefighting among wildland firefighters. In the meantime, the Federal Interagency Wildland Firefighter Medical Qualification Standards Program was created in 2001 to monitor the health of wildland firefighters employed by the federal government and engaged in arduous duties. Firefighters initially undergo a comprehensive medical examination including spirometry, followed by periodic examinations (depending on employment status). Program information can be found at http://www.nifc.gov/medical_standards/index.htm.

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