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Act 46 of 2011
Firefighter Cancer Presumption and Workers' Compensation Coverage

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December 18, 2013

Chairman Barrar, Chairman Sainato, Representative Farry, Chairman Scavello, Chairman Keller, and members of the Committees - thank you for inviting us to provide testimony regarding Act 46. I am Robert Anspach, Director of Insurance Services for the PennPRIME Workers' Compensation Trust (PennPRIME). PennPRIME is a service program of the Pennsylvania Municipal League. The PennPRIME Trust provides workers' compensation and liability insurance to Pennsylvania municipalities. We insure Boroughs, Townships and Cities.

We have faced some significant challenges emanating from Act 46 which has affected insurers, municipalities, and both career and volunteer firefighters. We heard representations before Act 46 was enacted that there would be a limited number of Act 46 claims and a small number of prostate cancer claims. This has not been the case due to the filing of over 96 Act 46 claims including 31 prostate cancer claims. Another serious concern is the filing of many medical subrogation claims that appear to be chiefly targeted on awarding legal recovery fees to attorneys and not wage loss payments to firefighters. We believe that our goal should be to make the adjustments needed to provide a non-State Workers Insurance Fund (SWIF) product to the municipalities and firefighters of Pennsylvania. The challenges to insurers have emanated from the potential claims which have increased the cost of insurance that is borne by municipalities.

From an underwriting standpoint, we noted the following concerns that caused us to withdraw from the market:

1. There is no reliable statistical basis upon which to base workers' compensation loss forecasts for Act 46 claims. This would include cradle to grave costs for cancer claims and the relative frequency and severity of each type of cancer claims.
2. The scope of interpretation by the judiciary is immature.
3. Our Trust has a new reserve for the retroactive liability associated with Act 46 claims which has significantly reduced our required surplus.
4. There is also the real potential for compounding of costs due to the risk of health insurer subrogation. In addition to workers' compensation benefits paid, health insurers that bargained for and received substantially higher premiums to accept the cancer risk in its entirety are permitted to seek recovery from workers' compensation underwriters.

The PennPRIME cancer claims have numbered six and include cancer of the skin (melanoma), colon, prostate, lung, brain/colon, and thyroid. They are evenly divided between career and volunteer claimants. As a result of these claims, our reserves will be in excess of $1 million to pay claims related expenses. I should note that these are costs that were not included in our cost modeling in past years so the money will have to come out of our required surplus and, therefore, will not be returned our municipal members. Another significant concern is subrogation claims for medical liens that are brought by claimant's attorneys. The claims are significant and are expected to be significantly more than the current claims reserve.

As noted previously, we hope that we can move this coverage out of the SWIF. There is much to be done, however, in order to accomplish that goal. We have focused on three areas that we thought should be addressed in Act 46:

1. Subrogation;
2. Identification methodology of those cancers tied to firefighting; and
3. Potential financial support for municipalities and municipal trusts for Act 46 claims and loss control and risk management as part of the volunteer fire coverage.

Subrogation

Subrogation is defined as the ability of a disenfranchised third party (in this case the health insurer) to step into the shoes of someone with standing (the claimant), because the third party cannot make its own claim. The basis of the subrogation claim from the health insurer is to transfer coverage of health costs to workers' compensation carriers in the event of a firefighter with cancer. Health insurers are in the business of insuring risk and have always covered cancer treatment in their insurance contracts. After payment for treatment is made, that insurer will adjust premiums based on the experience and collect higher premiums thereafter. The issue here is that the health insurers had the ability to reserve for the huge expense of cancer treatment and set premiums accordingly. In any individual case, the insurer paid for the treatment and went on with business as usual, since that cancer treatment was something that the insurer knew was a possible liability. Workers' compensation insurers never reserved for payment of cancer treatment, since claims were not anticipated (unlike the health insurer). Now, Act 46 has forced workers' compensation insurers to plan for up to 12 years of possible claims. If the workers' compensation insurer is forced to reimburse health insurers (who planned for cancer expenses and charged premiums accordingly), the health insurer is going to receive a windfall, since it has already collected premiums to support the cost of cancer treatment. That windfall is coming at the expense of the municipalities, which are being asked to fund cancer treatment coverage from trusts like PennPRIME and other insurers are going to be forced to pay for unplanned past medical expenses under Act 46 cases, if this trend continues. The result, and an unintended consequence of Act 46, is sharply increased rates for workers' compensation insurance.

We would suggest an amendment to the bill that would not allow subrogation of the medical costs. This provision will not take one nickel away from firefighters, and will not impact the benefits they can receive. Data developed by our counsel at The Chartwell Law Offices reveals that in a review of 12 high-exposure, pending cases the amount of indemnity benefits at issue

and payable to firefighters is less than $180,000. In these same 12 cases, the exposure for liens under Section 319 of the Workers' Compensation Act to health insurers is over $2.4 million.

In dozens of cases, there is minimal exposure for indemnity benefits to the firefighter, such as in cases in which a cancer was diagnosed after retirement. The only apparent reason the cases are clogging the system and sapping resources is that the claimant's lawyers are salivating over a 20% fee on the medical bills paid, some that were paid more than a decade ago.

Eliminating the health care lien in Act 46 cases will reduce potential exposure by as much as 75% in some cases, and hence, increases in premiums for Act 46 coverage will fall to more modest and, hopefully, more palatable levels.

To address the subrogation issue, we would suggest an amendment to some sort of insurance bill that:

1. Prohibits subrogation in cases filed under Act 46 of 2011.
2. The provisions of Act 46 of 2011 shall not apply retroactively to any alleged entitlement under Section 319 of the Workers' Compensation Act.
3. No compensation, including subrogation, shall be awardable if the wage loss or medical treatment predates July 7, 2011, the effective date of the amendments contained in Act 46 of 2011.

Covered Cancers

We feel that the law needs to look at the cancer equitably. While we certainly see that some cancers could be caused by firefighting duties, there should be some consideration as to what is truly caused by their duties. To that end, we would ask the legislature to consider the following amendments to Act 46:

Clarify "caused by"

In the language of Act 46, the legislature noted that, to invoke the presumption, the firefighter must prove that his cancer was "caused by" exposure to Group I carcinogens encountered on the job. How can the notion of a presumption of causation live in the same provision requiring proof of causation?

The answer has been provided in virtually every other state with a presumption statute. Some states enumerate specific cancers that are covered by the legislation. Other states place a burden on the claimant to prove that the cancer is one that CAN be caused by firefighting exposures. In those states, the claimant must first prove that he has a cancer that CAN be caused by

firefighting, and then it is presumed that his particular case of cancer was in fact caused by the exposure. The language was intended to require the claimant to prove "general" causation.

Claimant's lawyers around the Commonwealth have twisted the language of Act 46, and have successfully argued that a cancer is compensable if the claimant proves exposure to any Group I carcinogen, even if there is no suspected link between the two. For example, claimant's lawyers argue that, if a firefighter was ever outside in the sunlight, and then develops rectal cancer, the firefighter wins. Why? Sunlight is a Group I carcinogen, and the firefighter has cancer. Without clarification that the claimant must prove general causation, the presumption becomes universal and un-rebuttable.

In the following recommendations, we suggest that, among other things, the Act restrict the presumption to cancers about which the generally accepted scientific literature finds a significantly increased association between a given cancer and firefighting by a statistically significant increased Standardized Incident Rate (SIR). The National Institute for Occupational Safety and Health (NIOSH) completed the most comprehensive study to date regarding firefighter cancer, involving almost 30,000 firefighters in Philadelphia, Chicago and San Francisco. The study covered firefighters who were employed from 1950 to 2009 and is at Enclosure 1. The raw data for the study is also provided at Enclosure 2. The study request was supported by the International Association of Fire Fighters and the National League of Cities, as well as others, and was funded by the U.S. Fire Administration. The NIOSH study gives us a much broader and deeper analysis of firefighter cancers. Prior to enactment of Act 46, the limited LeMasters study was used to support a position that the two most likely firefighting related cancers were multiple myeloma and testicular cancer. The NIOSH study completely reversed LeMasters, finding that multiple myeloma and testicular cancer do not occur more frequently in firefighters. The NIOSH study revealed that multiple myeloma and testicular cancers in firefighters actually fell below the national average, having SIRs of 0.72 and 0.75, respectively, well below the 1.0 which is used to identify the national average. A NIOSH study analysis prepared by the National League of Cities Risk Information Sharing Consortium is at Enclosure 3.

Dr. Tee Guidotti, Consultant, Occupational and Environmental Health and Medicine, Washington, D.C., and Vice President for Health/Safety, Environment, and Sustainability at Medical Advisory Services of Rockville, Md., has been recognized for his extraordinary scientific achievements in the field of occupational and environmental medicine (OEM). An internationally recognized expert in OEM, Dr. Guidotti's expertise includes epidemiology, toxicology, and occupational risk management. Dr. Guidotti's testimony regarding causation notes that in order for an epidemiologist to state that there is a causal relationship between a

studied exposure and a medical endpoint, a study should reach a relative risk of 2.0 or greater. It may be "statistically relevant to causation" to have a very tightly calculated 1.8 or 1.9 SIR, but not dispositive of causation. The goal, therefore, is to differentiate cancers that are very rarely, if ever, related to firefighting from those that have a scientifically proven link through a general causation framework. For instance, there is no study that shows a statistically significant positive association between firefighting and thyroid cancer, nor are there any exposures at a fire scene that are reasonably thought to cause thyroid cancer. In fact, most studies show that there is something protective about being a firefighter, since firefighters develop thyroid cancer less than the general population. Specifically, the NIOSH study found that firefighters develop thyroid cancer up to 43% less than the general population. (See Enclosure 2 at Table S4). Under our proposal, a firefighter would be permitted to file a claim for thyroid cancer, but the presumption would not be available, since general causation does not support the proposition that working as a firefighter leads to the development of thyroid cancer, generally. The firefighter would, however, be able to prove that his or her case was an outlier from the general rule by proving that his or her thyroid cancer was caused by exposure to Group 1 carcinogens on the job.

We acknowledge that few cancers have an SIR of greater than 2.0 in the scientific literature. Therefore, we propose that the target for applying a presumption for a given cancer be set at an SIR of 1.5, so long as the result reaches statistical significance. For cancers with an SIR of 1.5 or greater, the rebuttable presumption would apply. An employer would still have the opportunity to rebut the presumption with substantial competent evidence that the occupation of firefighter was not the cause of the cancer.

We would suggest that we follow the lead of other states. Other states use one or more of the following strategies in their laws:

1. Restrict and specify the cancers that are presumptively firefighting related;
2. Specify that the cancer must be a type of cancer that has been proven to be generally caused by firefighting;
3. Restrict the presumption to cancers about which the generally accepted scientific literature finds an increased association between a given cancer and firefighting by an increased SIR of 1.5 (statistically significant) or greater;
4. Restrict the presumption to non-smokers;
5. Restrict the presumption to people under age 65;
6. Require proof of general causation under generally accepted scientific methodologies; and
7. Specify that the presumption can be rebutted by evidence that the type of cancer the firefighter has not been proven to be causally related to firefighting based as a matter of general causation.

ACT 46 Benefits Fund or Program

The fact is that coverage became too expensive based on the uncertainty of claims filed and the potential catastrophic cost of each claim. I would point to the testimony of Richard Duffy from the IAFF (Enclosure 4), who testified before the Legislature on March 30, 2011 as follows:

> "In Pennsylvania there [are] 7,133 active (and retired active) career fire fighters (sic). Using the assumption that Pennsylvania has a rate that does not exceed the average of the above States' cancer related disabilities -- .034% of the active fire fighting (sic) workforce – the expected number of initial annual cancer claims for career fire fighters would be 3 career fire fighters. Pennsylvania has approximately 70,000 volunteer fire fighters. We would expect their longevity and exposures to be very different from career fire fighter, however even if we assumed their cancer experience would be the same, the annual cancer claims, based on the above assumptions, would be 24 volunteer fire fighters."

Mr. Duffy's testimony clearly does not reflect reality in Pennsylvania. There have been over 96 claims to date across Pennsylvania in less than 3 years with claims averaging $50,000 - $100,000.

A significant issue causing the insurers to no longer cover the firefighters is that there was no provision in Act 46 for the funding of the municipal employers statutory obligations created therein, except through the purchase of workers' compensation coverage by the individual municipal entities. Even if credible forecasting of such claims was possible, premium levels charged by SWIF set the floor for what other parties could charge to finance the newly covered cancer claims both prospective and retrospective. The reaction to the cost increases resulting from SWIF placements was swift and angry, yet the uncertainty associated with retaining and paying for these potentially large Act 46 claims revealed financial implications that made the transfer the risk to SWIF necessary. The philosophical decision to make the move to SWIF was not simple but in the end, the need to ensure the financial health of the trust had to be acknowledged.

While the underwriting uncertainties surrounding Act 46 abound, they come down to not being able to quantify the risk in order to establish a sustainable rate. Just some of the observations and questions that are of interest to the insurance community are:

1. The historical exposure basis is nonsensical (population served) rather than number of volunteers and/or extent of activity.
2. What is the total pool of potential claimants? How many active, inactive and or retired volunteer and paid firefighters are there?
3. What are their full time occupations? The law is silent on the cancer risks from their full time occupations be it steel worker, teacher, painter, etc.
4. What is their lifestyle (smoker/nonsmoker)?
5. When does the employment end for members of VFC's? Does this increase the average age of members relative to other employments? Cancer rates increase exponentially with age.

Historically, volunteer fire companies have been extraordinarily costly in the workers' compensation arena. PennPRIME's history indicates that we paid two dollars for every dollar in contribution (premium) collected. We would submit that while the work can be dangerous, a significant part of that cost is due to lack of risk management and loss control. Please understand that, while this is a dollar issue, it is, more importantly, a safety issue for the firefighters. In order for loss control and risk management to work for the benefit of the insured and insurer, there has to be a set of standards and accountability. In the case of PennPRIME, the standards exist for our Members but when we insured volunteer fire companies, there was no accountability because the volunteer fire companies were statutory employees of the municipality but the municipality had no control over the safety operations of firefighting or non-firefighting activities. Therefore, if a firefighter chose not to wear self-contained breathing apparatus, there is nothing that the municipality paying the insurance bill could do about it.

We believe that the volunteer fire companies would benefit from basic loss control and risk management practices. This could be accomplished by the creation of an organization, perhaps a trust that handles only volunteer fire, is financially supported or assisted by the Commonwealth for coverage of cancer presumption claims, and provides services such as claims adjudication, loss control and risk management training. With the Commonwealth's direction and guidance, bringing a program like Labor and Industry's Accident and Injury Prevention Program to the fire companies would be immense.

In order to address this, we would suggest that the Commonwealth provide some form of financial relief for the mandate, including but not limited to the following:

1. The Commonwealth creates a fund or office through which employers can secure payment of or reimbursement for any Act 46 claim obligations incurred; or
2. The Commonwealth assumes all Act 46 claim obligations with the statutory employer retaining all other obligations; or
3. Amend the Workers' Compensation Act so members of volunteer fire companies are employees of the volunteer fire companies, rather than the municipality, and create a new program to afford the specialized coverage and service needs of volunteer fire companies.

This support could be in the form of a risk financing vehicle providing direct coverage or some form of backstop or stop loss protection for municipalities and municipal trusts like the PennPRIME Workers' Compensation Trust.

We believe that our recommendations are just a beginning. The potential of narrowing the cancer presumption scope, limiting subrogation, creating a Commonwealth mechanism to fund the cancer presumption costs, and develop an organization or trust that assists volunteer fire companies in insuring their personnel while providing the insurance services to them are all viable ideas that will need to be discussed in-depth. We are one part of a puzzle that includes the Legislature, the Administration, firefighters, municipalities, the insurance community, and probably others. On behalf of PennPRIME and its Board, we stand ready to work this issue and get to a point where the firefighters are getting the proper, affordable coverage and a safer workplace.

Mortality and cancer incidence in a pooled cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950–2009)

Robert D Daniels, Travis L Kubale, James H Yiin, et al.

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Enclosure 1

Notes

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ORIGINAL ARTICLE

Mortality and cancer incidence in a pooled cohort of US □re□ghters from San Francisco, Chicago and Philadelphia (1950□2009)

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ABSTRACT

Objectives To examine mortality patterns and cancer incidence in a pooled cohort of 29 993 US career □re□ghters employed since 1950 and followed through 2009.

Methods Mortality and cancer incidence were evaluated by life table methods with the US population referent. Standardised mortality (SMR) and incidence (SIR) ratios were determined for 92 causes of death and 41 cancer incidence groupings. Analyses focused on 15 outcomes of a priori interest. Sensitivity analyses were conducted to examine the potential for signi□cant bias.

Results Person-years at risk totalled 858 938 and 403 152 for mortality and incidence analyses, respectively. All-cause mortality was at expectation (SMR=0.99, 95% CI 0.97 to 1.01, n=12 028). There was excess cancer mortality (SMR=1.14, 95% CI 1.10 to 1.18, n=3285) and incidence (SIR=1.09, 95% CI 1.06 to 1.12, n=4461) comprised mainly of digestive (SMR=1.26, 95% CI 1.18 to 1.34, n=928; SIR=1.17, 95% CI 1.10 to 1.25, n=930) and respiratory (SMR=1.10, 95% CI 1.04 to 1.17, n=1096; SIR=1.16, 95% CI 1.08 to 1.24, n=813) cancers. Consistent with previous reports, modest elevations were observed in several solid cancers; however, evidence of excess lymphatic or haematopoietic cancers was lacking. This study is the □rst to report excess malignant mesothelioma (SMR=2.00, 95% CI 1.03 to 3.49, n=12; SIR=2.29, 95% CI 1.60 to 3.19, n=35) among US □re□ghters. Results appeared robust under differing assumptions and analytic techniques.

Conclusions Our results provide evidence of a relation between □re□ghting and cancer. The new □nding of excess malignant mesothelioma is noteworthy, given that asbestos exposure is a known hazard of □re□ghting.

What this paper adds

- From previous studies, there is limited epidemiological evidence of increased risk of cancer from □re□ghting.
- We examined cancer in 30 000 career □re□ghters by pooling information from urban □re departments in three large US cities. The large sample size and long follow-up period improved risk estimates compared with previous studies.
- We report that □re□ghting may be associated with increased risk of solid cancers. Furthermore, we report a new □nding of excess malignant mesothelioma among □re□ghters, suggesting the presence of an occupational disease from asbestos hazards in the workplace.

Linked

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INTRODUCTION

There are approximately 1.1 million volunteer and career □re□ghters in the US.[1] During □re□ghting activities, these workers may be exposed to many known carcinogens (eg, polycyclic aromatic hydrocarbons (PAHs), formaldehyde, benzene, 1,3-butadiene, asbestos and arsenic) in volatilised combustion and pyrolysis products or debris.[2] These exposures have raised concerns of increased cancer among □re□ghters and have prompted a number of exposure assessment and epidemiologic investigations. Some studies have found excess cancers of the brain,[3 8] digestive tract,[4 5 7□10] genitourinary tract[5 7 11 12] and lymphohematopoietic organs.[6 8 13] In a recent meta-analysis of 32 studies, signi□cant excess risk was reported for brain, stomach, colon, rectum, prostate, testes, multiple myeloma and non-Hodgkin lymphoma (NHL).[14] Similarly, the International Agency for Research on Cancer (IARC) reviewed 42 studies and reported signi□cant summary risks for prostatic and testicular cancers and NHL.[2] Given limited evidence, however, IARC concluded that □re□ghter exposures were only possibly carcinogenic to humans (Group 2B).

Most studies have examined mortality, but not cancer incidence, among relatively few □re□ghters recruited from one □re department. The current study examines mortality and cancer incidence in a pooled cohort of □re□ghters employed in three major US cities. Malignancies of the brain, stomach, oesophagus, intestines, rectum, kidney, bladder, prostate, testes, leukaemia, multiple myeloma and NHL were of a priori interest in the current study, based on possible sites identi□ed in previous reviews.[2 14] Lung cancer and chronic obstructive pulmonary disease (COPD) were also of interest because inhalation is a major pathway for □re□ghter exposures, and there is evidence of

chronic and acute in□ammatory respiratory effects in □re□ghters, which may be linked to cancer.[2] Breast cancer was included as a result of interests shared in researcher discussions with □re□ghters.

METHODS

Data collection methods

This research was approved by the Institutional Review Boards of the National Institute for Occupational Safety and Health (NIOSH) and the National Cancer Institute (NCI). Personnel records and previous study data were used to assemble the study roster, which comprised male and female career □re□ghters of all races employed for at least 1 day in □re departments serving San Francisco, Chicago, or Philadelphia, from 1 January 1950, through 31 December 2009. Fire departments were selected based on size, location, work experience, records availability and the willingness of labour and city management to participate. 'Career □re□ghter' status was determined from job titles categorised by researchers and vetted by each □re department. Selected job titles included general classi□cations of □re□ghters, □re□ghter paramedics, and □re department arson investigators. Persons of known race were mostly Caucasian (81%) and those missing race (2.5%) were hired in earlier periods of lower minority hiring (median year at hire= 1955). Therefore, persons missing race were assumed Caucasian and retained in main analyses to maximise study size. Analyses were also conducted excluding persons of unknown race.

Vital status was ascertained from the National Death Index-Plus (NDI-Plus), the Social Security Administration Death Master File (SSA-DMF), personnel and pension board records, and records from the previous studies.[9 10] Fire□ghters not found to be deceased were con□rmed alive by matches to employment records, Internal Revenue Service (IRS) records, and data accessible through LexisNexis (a private vendor of residential information).

Causes of death were obtained from previous studies,[9 10] NDI-Plus, and death certi□cates collected from state vital records and retirement boards. Deaths of Philadelphia □re□ghters through 1986 were previously determined by Baris et al,[9] who retrieved and coded death certi□cates to the ninth revision of the International Classi□cation of Diseases (ICD-9). San Francisco □re□ghter deaths were determined through 1982 by Beaumont et al.[10] In that and the current study, causes of death were coded to the ICD revision in effect at the time of death. The underlying cause of death determined by a trained nosologist was used for all mortality analyses.

Incident cases were de□ned as all primary invasive cancers, and in situ bladder cancers among □re□ghters matched to state cancer registries on name, gender, race, date of birth and Social Security number. The last known residence and the state of death were used to narrow inclusion of registries for case ascertainment to 11 states (ie, Arizona, California, Florida, Illinois, Indiana, Michigan, Nevada, New Jersey, Oregon, Pennsylvania and Washington) where nearly 95% of all deaths in known states occurred (see online supplementary table S1). The site and histology of each tumour were used to classify cancers in one of 41 diagnostic groups using the International Classi□cation of Diseases for Oncology, 3rd Edition (ICD-O-3).[13] The conversion from ICD-O-3 to ICD-10 used the Surveillance, Epidemiology and End Results Program (SEER) recodes (dated 27 January 2003) following slight modi□cation to align with mortality groupings and to account for recent classi□cation changes. Diagnosis dates were assigned as of 1 July of the year of diagnosis if only the diagnosis year was known, and on the 15th of the month of diagnosis if only the diagnosis month and year were known. The death date was used when death preceded the estimated date.

Statistical methods

The NIOSH Life Table Analysis System (LTAS.NET) was used to examine mortality and cancer incidence.[16] Main analyses used the US population as referent. In all analyses, person-years at risk (PYAR) were strati□ed by gender, race (Caucasian, other races), age (age 15–85+ years in 5-year categories), and calendar year (in 5-year categories). Con□dence limits for risk measures were estimated based on a Poisson distribution for the observed outcome, with exact limits for outcomes with 10 or fewer occurrences.

For mortality analyses, PYAR began on the latest of 1 January 1950 or the date of cohort inclusion, and ended the earliest of the date of death (DOD), the date last observed (DLO), or 31 December 2009. US mortality rates (1950–2009) were used to estimate the expected numbers of deaths for all causes, all cancers and 92 categories of underlying cause of death.[17] Additional mortality rates were developed to separately report on cancers of the small intestine, large intestine and testes to coincide with incidence rates; however, these rates were limited to time periods after 1959. In both cases, the subsites of interest (ie, colon and testes) account for the largest proportion of the deaths in the respective aggregate site (ie, intestine or male genital organs excluding prostate); therefore, the aggregate site reasonably approximates the subsite. The standardised mortality ratio (SMR) was calculated as the ratio of the observed to the total number of expected deaths.

Two approaches were used to examine cancer incidence. The main analyses included □rst and later primary cancers (ie, multiple-cancer approach) occurring within the risk period. PYAR accrued from the date of statewide ascertainment by the respective □re department's state cancer registry (eg, 1 January 1988 for San Francisco □re□ghters (see online supplementary table S1)) or cohort inclusion, whichever was latest, and ended at the earliest of the DOD, DLO, or 31 December 2009. Secondary analyses were restricted to the □rst occurrence of invasive cancer (ie, □rst-cancer approach). In these analyses, PYAR for cases ended on the date of □rst diagnosis. In both approaches, the standardised incidence ratio (SIR) was calculated as the ratio of observed malignancies to the expected number of cases estimated using US incidence rates (1985–2009) calculated from SEER data.[18] Additional steps required for □rst-cancer analyses were: selecting the most common cancer when diagnoses included multiple primary tumours on the same day (n= 21), excluding □re□ghters known to have a cancer diagnosis prior to the start of the risk date (n= 55), and adjusting US rates for cancer prevalence using methods described by Merrill et al.[19]

Heterogeneity in □re department-speci□c SMRs and SIRs was examined using Poisson regression modelling. To control for gender, age, calendar year and race, an offset term was set to the expected number of deaths or cases in each stratum of the classi□cation table. To address differences between □re departments, a mixed model was used that speci□ed a random intercept term. Thus, the model intercept is the log of the pooled SMR, adjusted for heterogeneity among the □re departments. The signi□cance of heterogeneity was assessed by likelihood ratio test (signi□cance level of 0.05).

Several sensitivity analyses were conducted. First, we examined the effects of including prevalent hires (workers employed before 1950) and short□term workers (those employed < 1 year)

in mortality analyses. Prevalent hires must be employed long enough to be recruited into the study; thus, these workers may have a survival advantage compared with persons hired during the follow-up period (ie, incident hires).[20] Short-term workers include temporary hires and probationary □re□ghters whose health and lifestyle patterns may differ from those employed one or more years. Short-term workers may also have had substantial occupational histories other than as □re□ghters, possibly in jobs with hazardous exposures. Second, we examined age effects on risk estimates in two age-at-risk categories (17–64, 65+ years). Testing of an effect across all 5-year age groups was accomplished using mixed models adjusted for age-at-risk groups. Third, we conducted SMR analyses restricting observation to age 84 years or less. Including PYAR for ages 85+ years could bias results from: rates used in analyses that are open-ended, more uncertainty in underlying cause of death at later ages, and subjects who are incorrectly traced as alive having a disproportionate effect in the open-ended age group.[21] Fourth, we calculated SMRs using California, Illinois and Pennsylvania State populations as referent for □re□ghters from San Francisco, Chicago and Philadelphia, respectively. Last, SMRs and standardised rate ratios (SRRs) were calculated for categories of employment duration (<10, 10–<20, 20–<30, 30+ years). Trend slopes with Wald-based two-sided p values (signi□cance level of 0.05) were calculated for the change in SRRs with increasing duration.

RESULTS

There were 29 993 □re□ghters available for study, contributing 858 938 PYAR (table 1). The cohort was largely male (97%), with mean age at □rst employment and total years employed of 29 and 21 years, respectively. Fewer than 5% of □re□ghters were short-term workers and approximately 30% were □rst employed prior to 1950. A higher percentage of women (9.4%) were short-term workers compared with men (4.3%) (see online supplementary table S2). Prevalent hires, on average, tended to be employed longer (+7.9 years, t test $p<0.001$) and had a greater attained age (+17.0 years, t test $p<0.001$) than incident hires. Persons eligible for incidence analyses using the multiple-cancer approach (n=24 453) contributed 403 152 PYAR. The □rst-cancer approach included 24 398 persons contributing 383 577 PYAR. There were 4461 malignant tumours distributed among 3903 □re□ghters with cancer. Among these, 488 reported cancers at multiple primary sites. Mortality and cancer incidence results are summarised in table 2 and in online supplementary tables S3–S5. To aid in comparisons with previous studies, table 2 also shows summary risk estimates (SREs) reported by LeMasters et al[14], whose meta-analysis included studies published through 2003.

Mortality

With the US population referent, all-cause mortality was at expectation (SMR=0.99, 95% CI 0.97 to 1.01, n=12 028). Ischaemic heart disease was the leading cause of death (SMR=1.01, 95% CI 0.98 to 1.04, n=3619). There was signi□cantly decreased mortality in other outcomes that may be related to healthy worker selection and survivor effects (HWE), such as non-malignant respiratory diseases (SMR=0.80, 95% CI 0.74 to 0.86, n=796), cerebrovascular disease (SMR=0.91, 95% CI 0.84 to 0.98, n=636), diabetes mellitus (SMR=0.72, 95% CI 0.62 to 0.83, n=175), nervous system disorders (SMR=0.80, 95% CI 0.69 to 0.93, n=187), and alcoholism (SMR=0.61, 95% CI, 0.41 to 0.86, n=31). In particular, there was a strong decrease in COPD mortality (SMR=0.72, 95% CI

Table 1 Demographic characteristics of the cohort by fire department and combined (1950–2009)

Description	All fire departments	San Francisco	Chicago	Philadelphia
Study cohort:				
Eligible for mortality analysis	29 993	5313	15 185	9495
PYAR	858 938	154 317	419 414	285 207
Years of follow-up; avg. (SD)	29 (16)	29 (16)	28 (16)	30 (16)
Race (%):				
White	24 244 (80.8)	4254 (80.1)	11 736 (77.3)	8254 (86.9)
Other	5008 (16.7)	986 (18.6)	2808 (18.5)	1214 (12.8)
Unknown	741 (2.5)	73 (1.4)	641 (4.2)	27 (<1.0)
Gender (%):				
Male	29 002 (96.7)	5009 (94.3)	14 694 (96.8)	9299 (97.9)
Female	991 (3.3)	304 (5.7)	491 (3.2)	196 (2.1)
Vital status:				
Alive (%)	17 965 (59.9)	3239 (61.0)	9241 (60.9)	5485 (57.8)
Deceased (%)	12 028 (40.1)	2074 (39.0)	5944 (39.1)	4010 (42.2)
Unknown cause of death	144	9	91	44
Attained age*; avg. (SD)	60 (16)	62 (16)	59 (16)	61 (16)
LTFU	175	1	32	142
PYAR potentially LTFU (%)	8809 (1.0)	59 (<1.0)	1483 (<1.0)	7267 (2.5)
Employment:				
Avg. hire year	1968	1967	1970	1965
Age at hire; avg. (SD)	29 (5)	29 (5)	29 (5)	27 (5)
Employment years; avg. (SD)	21 (11)	22 (11)	21 (11)	21 (11)
Hired before 1950 (%)	8085 (27)	1682 (32)	3294 (22)	3109 (33)
Employed <1 year (%)	1328 (4.4)	194 (3.7)	891 (5.9)	243 (2.6)

*Age attained at earliest of the date of death, date LTFU or 31 December 2009.
Avg., average; LTFU, lost to follow-up; PYAR, person-years at risk.

Table 2 Standardised mortality and incidence ratios in firefighters for select outcomes compared to results from a recent meta-analysis

	Current study results (US population referent)						Meta-analys
	Mortality (1950–2009)*		Cancer incidence (1985–2009)				
			All cancers		First cancer		
Underlying cause (ICD-10 codes)	Obs	SMR (95% CI)	Obs	SIR (95% CI)	Obs	SIR (95% CI)	Studies
All cancers (C00-C97)	3285	1.14 (1.10 to 1.18)	4461	1.09 (1.06 to 1.12)	3890	1.09 (1.06 to 1.12)	25
MN oesophagus (C15)	113	1.39 (1.14 to 1.67)	90	1.62 (1.31 to 2.00)	80	1.71 (1.36 to 2.13)	8
MN stomach (C16)	110	1.10 (0.91 to 1.33)	93	1.15 (0.93 to 1.40)	72	1.02 (0.80 to 1.28)	13
MN intestine (C17-C18)	326	1.30 (1.16 to 1.44)	398	1.21 (1.09 to 1.33)	351	1.29 (1.16 to 1.43)	NA
MN large intestine (C18)	264	1.31 (1.16 to 1.48)	381	1.21 (1.09 to 1.34)	335	1.28 (1.15 to 1.43)	25
MN small intestine (C17)	8	1.66 (0.72 to 3.27)	17	1.15 (0.67 to 1.85)	16	1.43 (0.82 to 2.33)	NA
MN rectum (C19-C21)	89	1.45 (1.16 to 1.78)	166	1.11 (0.95 to 1.30)	140	1.09 (0.91 to 1.28)	13
MN lung (C33-C34)	1046	1.10 (1.04 to 1.17)	716	1.12 (1.04 to 1.21)	602	1.13 (1.04 to 1.22)	19
MN breast (C50)	8	1.39 (0.60 to 2.73)	26	1.26 (0.82 to 1.85)	24	1.32 (0.84 to 1.96)	NA
MN prostate (C61)	282	1.09 (0.96 to 1.22)	1261	1.03 (0.98 to 1.09)	1176	1.03 (0.97 to 1.09)	13
MN other male genital (C60, C62-C63)	<5	0.47 (0.13 to 1.20)	17	0.62 (0.36 to 0.99)	17	0.67 (0.39 to 1.07)	NA
MN testes (C62)	<5	0.73 (0.15 to 2.14)	15	0.75 (0.42 to 1.24)	15	0.79 (0.44 to 1.30)	4
MN kidney (C64-C66)	94	1.29 (1.05 to 1.58)	166	1.27 (1.09 to 1.48)	129	1.24 (1.04 to 1.48)	12
MN bladder (C67-C68)‡	84	0.99 (0.79 to 1.22)	316	1.12 (1.00 to 1.25)	272	1.18 (1.05 to 1.33)	11
MN brain (C47, C70-C72)	73	1.01 (0.79 to 1.27)	51	1.02 (0.76 to 1.34)	48	1.06 (0.78 to 1.41)	19
NHL (C46.3, C82-C85, C88.0, C88.3, C91.4, C96)§	123	1.17 (0.97 to 1.40)	170	0.99 (0.85 to 1.15)	145	0.99 (0.83 to 1.16)	8
Leukaemia (C91.0-C91.3, C91.5-C91.9, C92-C95)	122	1.10 (0.91 to 1.31)	100	0.94 (0.77 to 1.15)	85	0.93 (0.74 to 1.15)	8
Multiple myeloma (C88.7, C88.9, C90)	42	0.89 (0.64 to 1.20)	36	0.72 (0.50 to 0.99)	33	0.75 (0.52 to 1.06)	10
Other cancers¶							
Mesothelioma (C45)	12	2.00 (1.03 to 3.49)	35	2.29 (1.60 to 3.19)	26	2.00 (1.31 to 2.93)	NA
MN buccal and pharynx (C00-C14)	94	1.40 (1.13 to 1.72)	174	1.39 (1.19 to 1.62)	148	1.41 (1.20 to 1.66)	9

*Results from Table 5 of LeMasters et al[14]; likelihood of cancer risk by meta-analysis criteria: 1=probable, 2=possible, 3=unlikely.
†SMRs restricted to 1960–2009 for MN large intestine, MN small intestine, and MN testes and 2000–2009 for mesothelioma.
‡Urinary bladder incidence included in situ (D09.0) and invasive cases as per SEER protocol.
§NHL incidence data exclude Kaposi sarcoma (C46.3).
¶Sites not listed among cancers of a priori interest but reporting statistically significant excess mortality and cancer incidence.
ICD-10, International Classification of Diseases, 10th Revision, MN, malignancy, NA, not applicable, NHL, non-Hodgkin lymphoma, Obs, observed, SEER, Surveillance, Epidemiology and End Results, SIR, standardis
mortality ratio; SRE, summary risk estimate.

0.65 to 0.80, n= 367). Few non-malignant outcomes were elevated, although statistically signi□cant excess mortality was observed for cirrhosis and other chronic liver disease (SMR= 1.26, 95% CI 1.12 to 1.41, n= 299) and acute glomerulonephritis with renal failure (SMR= 1.56, 95% CI 1.07 to 2.20, n= 32). Deaths from falls (SMR= 1.31, 95% CI 1.08 to 1.58, n= 113) and other accidents (SMR= 1.17, 95% CI 1.01 to 1.34, n= 197) were also elevated.

By contrast with non-malignant outcomes, we observed excess overall cancer mortality (SMR= 1.14, 95% CI 1.10 to 1.18, n= 3285) table 2). The elevation was largely attributable to excess cancers of the lung (SMR= 1.10, 95% CI 1.04 to 1.17, n= 1046), oesophagus (SMR= 1.39, 95% CI 1.14 to 1.67, n= 113), intestine (SMR= 1.30, 95% CI 1.16 to 1.44, n= 326) rectum (SMR= 1.45, 95% CI 1.16 to 1.78, n= 89) and kidney (SMR= 1.29, 95% CI 1.05 to 1.58, n= 94). There was little evidence of excess mortality from the remaining cancers of a priori interest; however, statistically signi□cant SMRs were apparent for buccal and pharynx cancers (SMR= 1.40, 95% CI 1.13 to 1.72, n= 94), malignancies of the liver, gall bladder and biliary tract (SMR= 1.30, 95% CI 1.06 to 1.57, n= 107), and malignant mesothelioma (SMR= 2.00, 95% CI 1.03 to 3.49, n= 12).

Women and non-Caucasians

All-cause mortality among women was near expectation (SMR= 0.91, 95% CI 0.59 to 1.33, n= 26). Accidental death was the leading cause (SMR= 2.79, 95% CI 1.21 to 5.50, n= 8) resulting in 31% of the total deaths among women. While there was little evidence of excess overall cancer mortality among women (SMR= 0.74, 95% CI 0.27 to 1.61, n= 6), most cancer deaths were from breast cancer (SMR= 1.46, 95% CI 0.30 to 4.26, n< 5). Bladder cancer mortality was statistically signi□cant (SMR= 33.51, 95% CI 4.06 to 121.05, n< 5) based on few cases. Non-Caucasian males were characterised by decreased all-cause mortality (SMR= 0.68, 95% CI 0.62 to 0.74, n= 453) and all-cancers (SMR= 0.80, 95% CI 0.65 to 0.97, n= 104). They had few observed deaths in any a priori outcome, and lung cancer mortality was below expectation (SMR= 0.67, 95% CI 0.44 to 0.97, n= 27). Only prostate cancer mortality showed an excess approaching statistical signi□cance (SMR= 1.64, 95% CI 0.95 to 2.63, n= 17) among non-Caucasian males (table 3).

Cancer incidence

There was little difference in SIRs when comparing analysis approaches; therefore, reporting focused on results from the multiple-cancer approach (table 2). All-cancer incidence was slightly above expectation (SIR= 1.09, 95% CI 1.06 to 1.12, n= 4461). Observed elevations in cancers of a priori interest were generally consistent with mortality data as evidenced by signi□cant excess cancers of the oesophagus (SIR= 1.62, 95% CI 1.31 to 2.00, n= 90); large intestine (SIR= 1.21, 95% CI 1.09 to 1.34, n= 381); kidney (SIR= 1.27, 95% CI 1.09 to 1.48, n= 166) and lung (SIR= 1.12, 95% CI 1.04 to 1.21, n= 716). As in mortality analyses, there were excess buccal and pharynx cancers (SIR= 1.39, 95% CI 1.19 to 1.62, n= 174) and malignant mesothelioma (SIR= 2.29, 95% CI 1.60 to 3.19, n= 35). Of those diagnosed with mesothelioma, 31 (88.6%) were pleural. Excess laryngeal cancer incidence was also observed (SIR= 1.50, 95% CI 1.19 to 1.85, n= 84). The incidence of most remaining cancer sites was near expectation; however, multiple myeloma was signi□cantly decreased (SIR= 0.72, 95% CI 0.50 to 0.99, n= 36).

Women and non-Caucasians

Overall cancer incidence among women was elevated, but not signi□cantly (SIR= 1.24, 95% CI 0.89 to 1.69, n= 40). Consistent with mortality, female bladder cancer incidence was statistically signi□cant but based on few cases (SIR= 12.53, 95% CI 3.41 to 32.08, n< 5). Nearly half of all cases were breast cancer (SIR= 1.45, 95% CI 0.86 to 2.29, n= 18). Nearly all breast cancers were diagnosed prior to the attained age of 55 years, with the highest SIR between the ages of 50 and 54 years (SIR= 2.66, 95% CI 0.86 to 6.21, n= 5). Left-sided disease appeared more frequent (61%, n= 11). Overall cancer incidence among non-Caucasian male □re□ghters was near expectation (SIR= 0.92, 95% CI 0.81 to 1.05, n= 240). There was excess prostate cancer (SIR= 1.26, 95% CI 1.02 to 1.54, n= 94) but decreased lung cancer (SIR= 0.67, 95% CI 0.43 to 1.00, n= 24) (tables 3 and 4).

Sensitivity analyses

Except for COPD and cancers of the lung, prostate and brain, there was little evidence of heterogeneity in SMRs (see online supplementary table S6) or SIRs (see online supplementary table S7) across □re departments for outcomes of a priori interest. For mortality, the between-department variance was largely attributable to outlying decreased lung cancer (SMR= 0.76, 95% CI 0.64 to 0.89, n= 142) and COPD (SMR= 0.53, 95% CI 0.40 to 0.69, n= 57) in San Francisco □re□ghters, and excess cancers of the prostate (SMR= 1.28, 95% CI 1.08 to 1.50, n= 152) and lung (SMR= 1.23, 95% CI 1.13 to 1.34, n= 566) in Chicago □re□ghters. The between-department variance in mortality persisted when using state populations as referent (see online supplementary table S8). Similarly, heterogeneous lung cancer incidence stemmed from decreased cases among San Francisco □re□ghters (SIR= 0.70, 95% CI 0.56 to 0.87, n= 81); however, there was outlying excess prostate cancer incidence among San Francisco □re□ghters (SIR= 1.22, 95% CI 1.08 to 1.37, n= 276). Brain cancer SIRs varied widely across □re departments; excess cancer was observed in San Francisco □re□ghters (SIR= 1.95, 95% CI 1.14 to 3.12, n= 17), while decreased cancer was reported for Chicago (SIR= 0.53, 95% CI 0.28 to 0.91, n= 13).

Restricting analyses to □re□ghters with one or more years of employment had negligible effects (see online supplementary table S9). Slight increases in SMRs were observed for most a priori outcomes when restricting the cohort to incident hires, although these differences were not statistically signi□cant. Age-at-risk differences in mortality also lacked statistical signi□cance, but SMRs generally appeared greater at older ages. SMRs for cancers of the breast (SMR= 1.42, 95% CI 0.46 to 3.32, n= 5), oesophagus (SMR= 1.41, 95% CI 1.05 to 1.86, n= 51), and kidney (SMR= 1.47, 95% CI 1.09 to 1.95, n= 48) were highest among workers less than 65 years of age (see online supplementary table S10). Signi□cant age-at-risk differences in SIRs were evident for prostate (p< 0.001) and bladder (p= 0.002) cancers (see online supplementary table S11). The heterogeneity was largely attributable to signi□cant increases in prostate (SIR= 1.21, 95% CI 1.10 to 1.33, n= 426) and bladder (SIR= 1.33, 95% CI 1.08 to 1.62, n= 97) cancer risks among □re□ghter aged 64 years or less. Excess prostate cancer was limited to ages 45□59 years (SIR= 1.45, 95% CI 1.28 to 1.64, n= 249), while the age pattern of excess bladder cancer incidence was unclear. The effects of restricting PYAR to age-at-risk < 85 were inconsequential (see online supplementary table S12). Excluding □re□ghters without race information also had little

Table 3 Standardised mortality and incidence ratios among men compared with the US population for causes of a priori interest

	Mortality (1950–2009)				Cancer incidence (1985–2009)*	
	Caucasian		Other		Caucasian	
Underlying cause (ICD-10 codes)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SIR (95% CI)
All causes	11 549	1.01 (0.99 to 1.03)	453	0.68 (0.62 to 0.74)	NA	NA
All cancers (C00-C97)	3175	1.16 (1.12 to 1.20)	104	0.80 (0.65 to 0.97)	4181	1.10 (1.07 to 1.13)
MN oesophagus (C15)	110	1.46 (1.20 to 1.75)	<5	0.51 (0.11 to 1.49)	87	1.70 (1.36 to 2.09)
MN stomach (C16)	105	1.12 (0.92 to 1.36)	5	0.81 (0.26 to 1.89)	87	1.19 (0.96 to 1.47)
MN intestine (C17-C18)	319	1.32 (1.18 to 1.48)	7	0.68 (0.27 to 1.40)	379	1.23 (1.11 to 1.36)
MN rectum (C19-C21)	86	1.46 (1.17 to 1.81)	<5	1.21 (0.25 to 3.53)	159	1.16 (0.99 to 1.36)
MN lung (C33-C34)	1019	1.12 (1.05 to 1.19)	27	0.67 (0.44 to 0.97)	689	1.15 (1.07 to 1.24)
MN breast (C50)	5	1.43 (0.46 to 3.34)	0	NC	6	0.79 (0.29 to 1.72)
MN prostate (C61)	265	1.06 (0.94 to 1.20)	17	1.64 (0.95 to 2.63)	1167	1.02 (0.96 to 1.08)
MN other male genital (C60, C62-C63)	<5	0.49 (0.13 to 1.26)	0	NC	16	0.64 (0.37 to 1.04)
MN kidney (C64-C66)	91	1.31 (1.05 to 1.60)	<5	1.05 (0.22 to 3.07)	151	1.26 (1.06 to 1.47)
MN bladder (C67-C68)†	80	0.96 (0.76 to 1.19)	<5	1.19 (0.14 to 4.30)	305	1.11 (0.99 to 1.24)
MN brain (C47, C70-C72)	72	1.03 (0.81 to 1.30)	<5	0.44 (0.01 to 2.47)	49	1.05 (0.78 to 1.39)
NHL (C46.3, C82-C85, C88.0, C88.3, C91.4, C96)‡	119	1.18 (0.98 to 1.41)	<5	1.01 (0.28 to 2.60)	161	1.02 (0.87 to 1.19)
Leukaemia (C91.0-C91.3, C91.5-C91.9, C92-C95)	117	1.10 (0.91 to 1.32)	5	1.28 (0.41 to 2.98)	88	0.88 (0.71 to 1.09)
Multiple myeloma (C88.7, C88.9, C90)	41	0.92 (0.66 to 1.25)	<5	0.35 (0.01 to 1.97)	35	0.76 (0.53 to 1.06)
COPD (J40-J44)	362	0.73 (0.65 to 0.81)	5	0.50 (0.16 to 1.16)	NA	NA

*Incidence results based on analysis of all invasive primary cancers (ie, multiple-cancer approach).
†Urinary bladder incidence included in situ (D09.0) and invasive cases as per SEER protocol.
‡NHL incidence data exclude Kaposi sarcoma (C46.3).
COPD, chronic obstructive pulmonary disease; ICD-10, International Classification of Diseases, 10th Revision; MN, malignancy; NA, not applicable; NC, not calculated; NHL, non-Hodgkin lymphoma; Obs, observed; Surveillance, Epidemiology, and End Results; SMR, standardised mortality ratio.

Table 4 Standardised mortality ratios (US population referent) and rate ratios for select outcomes* by employment duration (lagged 10 years)

	Employment duration (years)							
	0–<10		10–<20		20–<30		30+	
Underlying cause (ICD-10 codes)	Obs	SMR (95% CI) SRR (95% CI)	Obs	SMR (95% CI) SRR (95% CI)	Obs	SMR (95% CI) SRR (95% CI)	Obs	SMR (95% SRR (95% C
MN oesophagus (C15)	13	1.17 (0.62 to 2.00) (Reference)	28	1.72 (1.14 to 2.48) 2.43 (1.07 to 5.50)	53	1.40 (1.05 to 1.83) 1.17 (0.56 to 2.41)	19	1.18 (0.71 to 0.60 (0.27 to
MN stomach (C16)	12	0.80 (0.41 to 1.40) (Reference)	18	0.92 (0.54 to 1.45) 0.33 (0.08 to 1.43)	47	1.07 (0.79 to 1.43) 0.39 (0.10 to 1.55)	33	1.53 (1.06 to 0.40 (0.10 to
MN intestine (C17-C18)	27	0.86 (0.57 to 1.26) (Reference)	52	1.27 (0.95 to 1.67) 1.16 (0.38 to 3.54)	171	1.42 (1.22 to 1.66) 0.62 (0.27 to 1.44)	76	1.28 (1.01 to 0.40 (0.17 to
MN rectum (C19-C21)	13	1.48 (0.79 to 2.54) (Reference)	19	1.58 (0.95 to 2.46) 0.99 (0.33 to 2.97)	37	1.35 (0.95 to 1.86) 0.61 (0.24 to 1.52)	20	1.52 (0.93 to 0.43 (0.16 to
MN lung (C33-C34)	123	1.02 (0.85 to 1.22) (Reference)	184	1.03 (0.88 to 1.19) 1.32 (0.97 to 1.80)	523	1.14 (1.05 to 1.24) 1.24 (0.91 to 1.68)	216	1.12 (0.98 to 0.80 (0.59 to
MN prostate (C61)	24	1.39 (0.89 to 2.07) (Reference)	23	1.08 (0.68 to 1.62) 0.66 (0.31 to 1.41)	148	1.10 (0.93 to 1.29) 0.84 (0.47 to 1.50)	87	1.01 (0.81 to 0.69 (0.39 to
MN kidney (C64-C66)	12	1.10 (0.57 to 1.92) (Reference)	18	1.24 (0.73 to 1.95) 0.61 (0.26 to 1.48)	47	1.43 (1.05 to 1.90) 1.25 (0.58 to 2.69)	17	1.19 (0.69 to 0.70 (0.29 to
MN bladder and other urinary (C67-C68)	8	1.05 (0.45 to 2.08) (Reference)	7	0.65 (0.26 to 1.34) 0.25 (0.08 to 0.79)	46	1.08 (0.79 to 1.45) 1.15 (0.49 to 2.70)	23	0.94 (0.60 to 1.03 (0.38 to
MN brain and other nervous (C47, C70-C72)	12	0.65 (0.34 to 1.13) (Reference)	15	0.88 (0.49 to 1.46) 0.80 (0.30 to 2.19)	32	1.17 (0.80 to 1.65) 1.48 (0.60 to 3.68)	14	1.47 (0.80 to 1.52 (0.53 to
NHL (C46.3, C82-C85, C88.0, C88.3, C91.4, C96)	18	0.98 (0.58 to 1.55) (Reference)	9	0.51 (0.23 to 0.96) 1.18 (0.41 to 3.45)	63	1.35 (1.04 to 1.73) 1.15 (0.60 to 2.22)	33	1.47 (1.01 to 1.04 (0.51 to
Leukaemia (C91.0-C91.3, C91.5-C91.9, C92-C95)	18	0.91 (0.54 to 1.44) (Reference)	23	1.36 (0.86 to 2.05) 2.24 (0.92 to 5.50)	54	1.11 (0.83 to 1.45) 1.36 (0.65 to 2.87)	27	1.06 (0.70 to 1.13 (0.48 to
Multiple myeloma (C88.7, C88.9, C90)	5	0.84 (0.27 to 1.96) (Reference)	<5	0.52 (0.14 to 1.34) 0.56 (0.11 to 2.82)	22	0.97 (0.61 to 1.47) 1.59 (0.47 to 5.41)	11	0.99 (0.49 to 1.25 (0.33 to
COPD (J40-J44)	33	0.78 (0.54 to 1.10) (Reference)	38	0.69 (0.49 to 0.94) 1.07 (0.60 to 1.91)	185	0.70 (0.60 to 0.81) 1.03 (0.67 to 1.60)	111	0.75 (0.62 to 0.83 (0.53 to

*Excluding a priori causes with total observations <20.
†Cause-specific deaths per year of employment-person-year.
COPD, chronic obstructive pulmonary disease; ICD-10, International Classification of Diseases, 10th Revision; MN, malignancy; NHL, non-Hodgkin lymphoma; Obs, observed; SMR, standardised mortality ratio; SRR

effect on a priori outcomes (results not shown). Finally, there was no apparent trend in increasing risk with employment duration; however, negative trends in COPD and colorectal cancer SRRs were evident (table 4). Subsequent sensitivity analyses revealed that SRRs were largely dependent on selection of cut-points and lag periods (results not shown).

DISCUSSION

This study is among the largest examining cancer risk in career □re□ghters. The pooled approach and long follow-up period improved risk estimates relative to previous studies. With few exceptions, there was little evidence of signi□cant cancer risk heterogeneity across □re departments or age groups. Furthermore, sensitivity analyses did not suggest the potential for signi□cant bias from including short-term workers, prevalent hires, or person-time in the open-ended age-group (85+ years). Despite notable differences in the analytical approaches, we observed remarkable similarities between mortality and incidence analyses. Additionally, the results of incidence analyses were not signi□cantly affected by the choice of including multiple primaries or only the □rst cancer diagnosis. The lack of signi□cant differences in results between □re departments, end-points, and analytic techniques suggest that the pooled study □ndings are robust and generalisable to similar □re□ghter populations.

We observed decreases in many non-malignant diseases that suggest improved health in these □re□ghters compared with the general population. This □nding is not surprising given health requirements for entering and remaining in the □re service. Nevertheless, there was a modest excess in overall cancer mortality and incidence brought about by excess solid cancers at several sites of a priori interest. With few exceptions, our results are consistent with those previously reported and similar to SREs presented in the meta-analysis by LeMasters et al.[14] Nevertheless, we found little evidence of excess cancers of the testes, brain and lymphohematopoietic systems, which is contrary to the synthesis by LeMasters et al[14] and subsequently published studies.[8 11]

We observed about a twofold increase in malignant mesothelioma mortality and incidence compared with the US population. Malignant mesothelioma is largely attributable to asbestos exposure, with sparse evidence of other causes.[22] Excess malignant mesothelioma in US □re□ghters was not previously described; however, excess incidence was recently observed in Nordic □re□ghters aged 70+ years,[23] and increased risk of asbestos-induced pulmonary and pleural □brosis was reported in a study of New York City □re□ghters.[24] Although □re□ghter exposures to asbestos are known, the absence of previous reports of malignant mesothelioma is not surprising given the rarity and extremely long latency (20–40 years) of the disease. The average time between the date □rst employed and the date of diagnosis in the current study was 45 years; therefore, □re□ghting exposure-induced disease may be discernible only after lengthy follow-up. Also, previous studies have been hindered by the lack of speci□c codes for mesothelioma deaths before ICD-10.

We observed excess digestive cancers, mainly of oesophageal and colorectal sites. Information on occupational causes is sparse, although there is limited evidence suggesting asbestos and diesel exhaust exposures may be weakly associated with gastrointestinal cancers.[25 26] Still, the relation between these hazardous exposures and digestive cancers appears small compared to the effects of other factors such as diet, obesity, physical activity, tobacco use and alcohol consumption.[22 27] We also found increased risk of oral, pharyngeal and laryngeal cancers, compared with the US population. Similar to digestive cancers, important risk factors for these sites are tobacco and alcohol consumption, with lesser evidence that exposures to wood dusts, smoke, asbestos, PAHs and acid mists may also increase risk.[22 28 29]

Some insight into the degree of a potential bias from the lack of controlling for lifestyle factors can be gained from previous surveillance of □re□ghter behaviours. For example, the prevalence of smoking among current □re□ghters appears less than the general population, and is decreasing,[30–33] a trend that is consistent with observed decreases in non-malignant smoking-related diseases (eg, COPD, stroke) but contradictory to excess digestive, oral and respiratory cancers. As another example, previous studies suggest there is increased obesity among □re□ghters compared with the general population.[34–36] Obesity, or a dietary intake that is high in meat, fat, or overall caloric intake could contribute increased gastric or colorectal cancer risk, although concomitant elevations in health outcomes that are more strongly related to these factors (eg, ischaemic heart disease, diabetes mellitus, hypertension and stroke) were not found. Last, information on alcohol consumption within the □re service is sparse and inconsistent.[37–40] Some studies suggest that □re□ghter behaviours may differ from the general population, although it is not clear that any perceived behavioural difference is su□cient to explain disparities in alcohol-related health outcomes. In the current study, the information on non-malignant and potentially alcohol-related mortality was at con□ict; there was excess mortality from cirrhosis and other chronic liver disease, but fewer than expected alcoholism deaths. Alternate explanations for increased cirrhosis mortality may be exposures to chemical toxins or infectious disease,[41–43] which may also account for excess acute renal dysfunction, a disease that is more common among those with chronic liver disease.

Fewer than 4% of □re□ghters in our study were women. There was evidence of excess female bladder and breast cancers; however, only bladder cancer mortality and incidence reached statistical signi□cance. Modest excess bladder cancer has been observed in some occupations involving known or suspected bladder carcinogens (eg, PAHs, and diesel exhaust), yet contrary to our □ndings, risk patterns by occupation tend not to differ by gender.[22] There is little evidence linking female breast cancer to workplace exposures; however, prolonged shift work may be a risk factor (and to a lesser extent a risk factor for prostate, colon and endometrial cancers).[2] Moreover, similar □ndings had not been reported previously, although increased risk of Hodgkin lymphoma and cancers of the cervix and thyroid among women □re□ghters (n=2017) was recently described.[11] Given the small sample and the lack of con□rmatory results, our □ndings on female outcomes merit cautious interpretation.

Excess bladder and prostate cancer incidence was found among □re□ghters less than 65 years of age. Interestingly, the prostate cancer excess was limited to ages between 45years and 59 years, which was consistent with recent observations in Nordic □re□ghters.[23] Similar mortality patterns were not observed. These cancers have relatively high survival; therefore, the underlying cause of death may be an inferior risk measure compared to cancer diagnoses. The early onset of these cancers suggests an association with □re□ghting. Prostate and bladder cancer diagnoses can occur following routine screening.[44 45] As an alternative explanation, differences in medical screening (eg, prostate-speci□c antigen tests) among □re□ghters compared to the general population could have contributed to the observed excess. Data on cancer screening practices are lacking; however,

it is plausible that screening may be more frequent among □re□ghters with improved healthcare availability and heightened cancer awareness.

There was little evidence of increasing cancer risk with increasing employment; however, there were notable analytical shortcomings that merit discussion. First, rather than specifying cut-points and an exposure lag period speci□c to each outcome, we applied cut-points (10, 20 and 30 years) used in earlier studies[5 9 46] and a common exposure lag period (10 years) to all outcomes; these choices were found to be in□uential in subsequent sensitivity analyses. Second, our methods have limited capability to account for HWE or other sources of bias that may have masked a dose response. Last, employment duration may poorly represent exposure potential given that some jobs are prone to lower exposures compared with others. For these reasons, a detailed exposure assessment is underway to support multivariable regression modelling for improved dose-response analyses.

Death certi□cates and registry data used in the current study are imperfect measures of cancer risk. In the absence of a national cancer registry, coverage is limited geographically; therefore, cases occurring outside catchment areas would be missed. Cases occurring before the registries attained comprehensive coverage have also been missed. Mortality analyses have the advantage of broader temporal and spatial coverage, but may poorly characterise cancers with relatively high survival (eg, cancers of the breast, bladder, testes and larynx). Finally, there may have been errors in tracing which can also bias study results. Although errors in ascertainment cannot be ruled out, our use of multiple information sources and end points, and the low numbers of participants lost to follow-up or moving out of catchment areas, act to minimise these errors.

CONCLUSION

In this □rst phase of examining health effects in career □re□ghters, we report on mortality and cancer incidence among nearly 30 000 career □re□ghters followed from 1950 through 2009. Compared with the US population, we found small to moderate increases in risk for several cancer sites and for all cancers combined, stemming mostly from excess malignancies of the respiratory, digestive and urinary systems in otherwise healthy individuals. Our □ndings are consistent with previous studies and strengthen evidence of a relation between □re□ghters' occupational exposure and cancer. We found a previously unreported twofold excess of malignant mesothelioma among □re□ghters. Given that asbestos is the only known causal agent for malignant mesothelioma, and □re□ghter exposures are probable, the excess is likely to be a causal association.

This report provides the foundation for subsequent analyses of □re□ghter risks, some of which are ongoing. In upcoming research, detailed employment histories (eg, number and types of □re runs) and institutional knowledge (eg, use of respiratory protection and source capture ventilation of diesel exhaust) will be used to derive exposure metrics to more accurately examine dose response. Future regression modelling will also enable examination of temporal effects that are poorly suited to life-table analyses, such as time since □rst exposure. Expansion and continued follow-up of this cohort would enhance future analyses, particularly among women and non-Caucasian □re□ghters.

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Contributors RDD participated in design, data collection, analysis and manuscript development. TLK conceived the study and participated in design and data collection. JHY participated in design, data collection and analysis. MMD, TRH, DB, SHZ, JLB and KMW participated in design and data collection. LEP participated in design and critical appraisal. All authors participated in the interpretation and presentation of results and have read and approved the □nal manuscript.

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Competing interests None.

Ethics approval This research was approved by the Institutional Review Boards of the National Institute for Occupational Safety and Health (NIOSH) and the National Cancer Institute (NCI). Approvals for cancer registry access were granted by 11 states (ie, Arizona, California, Florida, Illinois, Indiana, Michigan, Nevada, New Jersey, Oregon, Pennsylvania and Washington). Approvals were also granted by vital records centres for death certi□cates maintained in 25 states (Alaska, Arizona, Arkansas, California, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Massachusetts, Michigan, Minnesota, Mississippi, New Jersey, New York, Ohio, Oklahoma, Oregon, Pennsylvania, Texas, Virginia, Washington and Wisconsin). The state public health entities provided vital status information in accordance with state policies, and disclaim responsibility for any analyses, interpretations, or conclusions herein.

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Supplementary (online only) data for:

Mortality and cancer incidence in a pooled cohort of U.S. firefighters from San Francisco, Chicago, and Philadelphia (1950-2009)

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Enclosure 2

Table S1. Cancer registry information

Cancer Registry	State	NPCR participant	SEER participant	Statewide Data Collection Began	No. of cases (%
Arizona Cancer Registry	Arizona	Yes	No	1981	105 (2
California cancer Registry	California	No	Yes	1988	881 (1
Florida Cancer Data System	Florida	Yes	No	1981	301 (6
Illinois State Cancer Registry	Illinois	Yes	No	1986	1783 (4(
Indiana Sate Cancer Registry	Indiana	Yes	No	1987	24 ((
Michigan Cancer Surveillance Program	Michigan	Yes	Yes	1985	31 ((
Nevada Central Cancer Registry	Nevada	Yes	No	1995	35 ((
New Jersey State Cancer Registry	New Jersey	No	Yes	1979	216 (4
Oregon State Cancer Registry	Oregon	Yes	No	1996	9 ((
Pennsylvania Cancer Registry	Pennsylvania	Yes	No	1985	1071 (24
Washington State Cancer Registry	Washington	Yes	Yes	1994	5 ((
NA	Others	NA	NA	NA	1

*Includes persons with multiple primary tumors and excludes duplicate reporting (n=4461).

†Number and percentage of deaths among cohort members with known death state (n=9290).

NA, not applicable; NPCR, National Program of Cancer Registries; SEER, Surveillance, Epidemiology, and End Results Pro

Table S2. Firefighter employment and vital status by gender (1950-2009).

	Men		Women	
Employment	Alive	Deceased	Alive	Deceased
Employed 1+ years	15969	11798	875	23
Employed <1 year	1031	204	90	3
Total	17000	12002	965	26

Table S3. Cause-specific standardized mortality ratios compared to the US population by fire department (1950-2009, *n*=29993,

Minor ID*	Cause	All		San Francisco		Chicago	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
1-92	All causes	12028	0.99 (0.97 to 1.01)	2074	0.84 (0.80 to 0.87)	5944	1.05 (1.02 to 1.08)
1-2	Tuberculosis	12	0.35 (0.18 to 0.61)	<5	0.30 (0.04 to 1.09)	<5	0.25 (0.07 to 0.65)
1	Respiratory tuberculosis	11	0.35 (0.18 to 0.63)	<5	0.33 (0.04 to 1.19)	<5	0.21 (0.04 to 0.61)
2	Other tuberculosis	<5	0.33 (0.01 to 1.85)	0	NC	<5	0.69 (0.02 to 3.83)
3-38	All Cancers	3285	1.14 (1.10 to 1.18)	578	1.00 (0.92 to 1.09)	1670	1.22 (1.16 to 1.28)
3-6	MN buccal cavity & pharynx	94	1.40 (1.13 to 1.72)	22	1.69 (1.06 to 2.56)	41	1.28 (0.92 to 1.73)
3	MN lip	<5	0.80 (0.02 to 4.44)	<5	3.70 (0.09 to 20.61)	0	NC
4	MN tongue	25	1.61 (1.04 to 2.38)	6	1.99 (0.73 to 4.33)	11	1.49 (0.74 to 2.66)
5	MN other buccal	25	1.43 (0.93 to 2.12)	5	1.45 (0.47 to 3.38)	8	0.97 (0.42 to 1.91)
6	MN pharynx	43	1.31 (0.95 to 1.77)	10	1.59 (0.76 to 2.92)	22	1.39 (0.87 to 2.10)
7-13	MN digestive & peritoneum	928	1.26 (1.18 to 1.34)	179	1.21 (1.04 to 1.40)	463	1.33 (1.21 to 1.46)
7	MN esophagus	113	1.39 (1.14 to 1.67)	23	1.45 (0.92 to 2.18)	58	1.47 (1.12 to 1.90)
8	MN stomach	110	1.10 (0.91 to 1.33)	25	1.23 (0.80 to 1.82)	53	1.14 (0.86 to 1.50)
9	MN intestine	326	1.30 (1.16 to 1.44)	56	1.09 (0.82 to 1.41)	157	1.33 (1.13 to 1.55)
10	MN rectum	89	1.45 (1.16 to 1.78)	20	1.59 (0.97 to 2.46)	47	1.65 (1.21 to 2.20)
11	MN biliary, liver, gall bladder	107	1.30 (1.06 to 1.57)	21	1.28 (0.79 to 1.96)	60	1.51 (1.15 to 1.95)
12	MN pancreas	168	1.13 (0.97 to 1.32)	33	1.11 (0.76 to 1.56)	81	1.15 (0.91 to 1.43)
13	MN peritoneum, other & unspecified	<29	1.42 (0.80 to 2.35)	<5	0.47 (0.01 to 2.60)	7	1.42 (0.57 to 2.93)
14-17	MN respiratory system	1096	1.10 (1.04 to 1.17)	147	0.75 (0.63 to 0.88)	594	1.24 (1.14 to 1.34)
14	MN larynx	<46	1.26 (0.91 to 1.69)	<5	0.44 (0.09 to 1.28)	26	1.55 (1.01 to 2.27)
15	MN trachea, bronchus, lung	1046	1.10 (1.04 to 1.17)	142	0.76 (0.64 to 0.89)	566	1.23 (1.13 to 1.34)
16	MN pleura	<5	0.81 (0.10 to 2.93)	0	NC	<5	1.71 (0.21 to 6.19)
17	MN other respiratory sites	<5	0.66 (0.18 to 1.69)	<5	1.71 (0.21 to 6.18)	0	NC
18	MN breast	8	1.39 (0.60 to 2.73)	<5	2.43 (0.50 to 7.11)	<5	1.29 (0.35 to 3.29)
19-22	MN female genital organs	0	NC	0	NC	0	NC
19	MN cervix	0	NC	0	NC	0	NC
20	MN other parts of uterus	0	NC	0	NC	0	NC
21	MN ovary	0	NC	0	NC	0	NC
22	MN other female genital	0	NC	0	NC	0	NC
23-24	MN male genital organs	286	1.07 (0.95 to 1.20)	52	0.90 (0.67 to 1.18)	152	1.23 (1.05 to 1.45)
23	MN prostate	282	1.09 (0.96 to 1.22)	51	0.91 (0.68 to 1.19)	152	1.28 (1.08 to 1.50)
24	MN other male genital	<5	0.47 (0.13 to 1.20)	<5	0.63 (0.02 to 3.50)	0	NC
25-26	MN urinary	178	1.13 (0.97 to 1.31)	29	0.89 (0.60 to 1.28)	96	1.30 (1.06 to 1.59)

Minor ID*	Cause	All		San Francisco		Chicago	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
25	MN kidney	94	1.29 (1.05 to 1.58)	13	0.90 (0.48 to 1.54)	56	1.62 (1.23 to 2.11)
26	MN bladder & other urinary	84	0.99 (0.79 to 1.22)	16	0.88 (0.50 to 1.43)	40	1.02 (0.73 to 1.39)
27-34	MN other & unspecified	397	1.09 (0.98 to 1.20)	89	1.23 (0.99 to 1.51)	181	1.04 (0.89 to 1.20)
27	MN skin	56	0.94 (0.71 to 1.22)	14	1.19 (0.65 to 1.99)	22	0.78 (0.49 to 1.19)
28	Mesothelioma	12	2.00 (1.03 to 3.49)	<5	2.41 (0.50 to 7.05)	8	2.73 (1.18 to 5.38)
29	MN eye	<5	2.28 (0.62 to 5.84)	0	NC	<5	4.95 (1.35 to 12.67)
30	MN brain & other nervous system	73	1.01 (0.79 to 1.27)	16	1.16 (0.66 to 1.89)	34	0.98 (0.68 to 1.37)
31	MN thyroid gland	<5	0.56 (0.11 to 1.62)	<5	0.93 (0.02 to 5.17)	<5	0.39 (0.01 to 2.18)
32	MN bone	9	1.16 (0.53 to 2.19)	<5	0.65 (0.02 to 3.63)	6	1.66 (0.61 to 3.61)
33	MN connective tissue	10	0.68 (0.32 to 1.24)	<5	0.70 (0.08 to 2.52)	<5	0.56 (0.15 to 1.44)
34	MN other & unspecified sites	230	1.16 (1.02 to 1.32)	52	1.31 (0.98 to 1.72)	102	1.08 (0.88 to 1.32)
35-38	MN lymphatic & hematopoietic	298	1.07 (0.95 to 1.20)	57	1.01 (0.77 to 1.31)	139	1.05 (0.88 to 1.24)
35	Non-Hodgkin lymphoma	123	1.17 (0.97 to 1.40)	25	1.19 (0.77 to 1.75)	53	1.06 (0.80 to 1.39)
36	Hodgkin lymphoma	11	0.68 (0.34 to 1.22)	<5	0.66 (0.08 to 2.37)	6	0.79 (0.29 to 1.72)
37	Leukemia & aleukemia	122	1.10 (0.91 to 1.31)	23	1.02 (0.65 to 1.53)	61	1.17 (0.90 to 1.50)
38	Multiple myeloma	42	0.89 (0.64 to 1.20)	7	0.74 (0.30 to 1.52)	19	0.84 (0.50 to 1.30)
39-41	Benign & unspecified neoplasms	40	1.07 (0.77 to 1.46)	8	1.06 (0.46 to 2.10)	17	0.97 (0.56 to 1.55)
39	Benign eye, brain, other nervous system	<5	0.92 (0.25 to 2.35)	<5	1.16 (0.03 to 6.45)	<5	0.97 (0.12 to 3.52)
40	Unspecified eye, brain, other nervous system	<15	0.96 (0.52 to 1.61)	5	1.75 (0.57 to 4.09)	5	0.72 (0.23 to 1.68)
41	Other benign & unspecified	<25	1.20 (0.75 to 1.82)	<5	0.53 (0.06 to 1.90)	10	1.17 (0.56 to 2.15)
42	Diabetes mellitus	175	0.72 (0.62 to 0.83)	17	0.35 (0.20 to 0.55)	89	0.77 (0.62 to 0.94)
43-46	Diseases of blood & blood-forming organs	50	1.11 (0.82 to 1.46)	11	1.16 (0.58 to 2.07)	24	1.13 (0.73 to 1.69)
43	Pernicious Anemia	0	NC	0	NC	0	NC
44	Other & unspecified anemia	16	1.01 (0.58 to 1.64)	<5	0.60 (0.07 to 2.15)	10	1.34 (0.64 to 2.47)
45	Coagulation & hemorrhagic conditions	7	0.77 (0.31 to 1.60)	<5	0.55 (0.01 to 3.07)	<5	0.92 (0.25 to 2.36)
46	Other diseases of blood-forming organs	27	1.37 (0.91 to 2.00)	8	1.91 (0.83 to 3.77)	10	1.09 (0.52 to 2.01)
47-48	Mental, psychoneurotic, and personality disorders	120	0.81 (0.68 to 0.97)	20	0.65 (0.39 to 1.00)	60	0.87 (0.66 to 1.12)
47	Alcoholism	31	0.61 (0.41 to 0.86)	<5	0.44 (0.12 to 1.13)	19	0.74 (0.45 to 1.16)
48	Other mental disorders	89	0.92 (0.74 to 1.14)	16	0.73 (0.42 to 1.19)	41	0.94 (0.67 to 1.28)

Minor ID*	Cause	All		San Francisco		Chicago	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
49-50	Nervous system disorders	187	0.80 (0.69 to 0.93)	43	0.86 (0.62 to 1.15)	82	0.76 (0.61 to 0.95)
49	Multiple sclerosis	<10	0.57 (0.21 to 1.23)	0	NC	5	0.99 (0.32 to 2.30)
50	Other nervous system diseases	181	0.82 (0.70 to 0.94)	43	0.89 (0.64 to 1.20)	77	0.75 (0.59 to 0.94)
51-55	Diseases of the heart	4289	0.99 (0.96 to 1.02)	720	0.80 (0.74 to 0.86)	2124	1.06 (1.02 to 1.11)
51	Rheumatic heart disease	51	0.89 (0.66 to 1.17)	6	0.54 (0.20 to 1.17)	24	0.90 (0.58 to 1.35)
52	Ischemic heart disease	3619	1.01 (0.98 to 1.04)	598	0.80 (0.74 to 0.87)	1812	1.10 (1.05 to 1.15)
53	Chronic Diseases of endocardium	56	0.99 (0.75 to 1.29)	15	1.22 (0.68 to 2.02)	25	0.97 (0.63 to 1.44)
54	Hypertension w/heart disease	111	0.84 (0.69 to 1.02)	24	0.89 (0.57 to 1.32)	54	0.89 (0.67 to 1.16)
55	Other heart disease	452	0.88 (0.81 to 0.97)	77	0.72 (0.57 to 0.90)	209	0.88 (0.76 to 1.00)
56-58	Other diseases circulatory system	967	0.91 (0.85 to 0.97)	201	0.88 (0.76 to 1.01)	439	0.91 (0.83 to 1.00)
56	Hypertension w/o heart disease	46	0.85 (0.62 to 1.13)	10	0.88 (0.42 to 1.61)	25	0.98 (0.64 to 1.45)
57	Cerebrovascular disease	636	0.91 (0.84 to 0.98)	131	0.86 (0.72 to 1.02)	298	0.95 (0.84 to 1.06)
58	Diseases of the arteries, Veins, and pulmonary circulation	285	0.93 (0.82 to 1.04)	60	0.91 (0.70 to 1.18)	116	0.82 (0.68 to 0.99)
59-64	Diseases of the respiratory system	796	0.80 (0.74 to 0.86)	137	0.65 (0.54 to 0.76)	411	0.89 (0.81 to 0.98)
59	Acute respiratory infections except influenza and pneumonia	0	NC	0	NC	0	NC
60	Influenza	<5	0.22 (0.03 to 0.81)	<5	0.51 (0.01 to 2.83)	<5	0.26 (0.01 to 1.43)
61	Pneumonia	269	0.90 (0.79 to 1.01)	55	0.84 (0.64 to 1.10)	129	0.95 (0.79 to 1.12)
62	Chronic obstructive pulmonary diseases	367	0.72 (0.65 to 0.80)	57	0.53 (0.40 to 0.69)	206	0.87 (0.75 to 0.99)
63	Asthma	<10	0.36 (0.14 to 0.74)	<5	0.51 (0.06 to 1.84)	5	0.55 (0.18 to 1.27)
64	Pneumoconiosis & other respiratory diseases	151	0.97 (0.82 to 1.14)	22	0.67 (0.42 to 1.01)	70	0.97 (0.76 to 1.23)
65-68	Diseases of the digestive system	572	1.13 (1.04 to 1.23)	110	1.11 (0.91 to 1.34)	277	1.16 (1.02 to 1.30)
65	Diseases of the stomach & duodenum	64	1.16 (0.89 to 1.48)	12	1.06 (0.54 to 1.84)	29	1.15 (0.77 to 1.66)
66	Hernia & intestinal obstruction	<23	0.69 (0.41 to 1.07)	<5	0.17 (0.00 to 0.93)	8	0.64 (0.28 to 1.27)
67	Cirrhosis & other chronic liver disease	299	1.26 (1.12 to 1.41)	65	1.47 (1.13 to 1.87)	144	1.25 (1.05 to 1.47)

Minor ID*	Cause	All		San Francisco		Chicago	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
68	Other diseases digestive system	190	1.04 (0.89 to 1.19)	32	0.85 (0.58 to 1.21)	96	1.11 (0.90 to 1.36)
69-77	Diseases of the genitourinary system	201	1.00 (0.87 to 1.15)	36	0.84 (0.59 to 1.16)	91	0.98 (0.79 to 1.21)
69	Acute glomerulonephritis nephrotic syndrome and acute renal failure	32	1.56 (1.07 to 2.20)	7	1.61 (0.65 to 3.31)	14	1.46 (0.80 to 2.46)
70	Chronic and unspecified nephritis and renal failure & other renal sclerosis	91	0.86 (0.69 to 1.05)	9	0.40 (0.18 to 0.77)	48	0.96 (0.71 to 1.28)
71	Kidney infection	9	0.75 (0.34 to 1.42)	<5	1.14 (0.24 to 3.34)	<5	0.77 (0.21 to 1.96)
72	Urinary system calculi	<5	1.29 (0.35 to 3.30)	<5	1.51 (0.04 to 8.43)	<5	0.73 (0.02 to 4.06)
73	Prostate hyperplasia	11	1.27 (0.63 to 2.28)	<5	1.84 (0.50 to 4.71)	5	1.50 (0.49 to 3.51)
74	Other diseases of male genital organs	<5	0.34 (0.01 to 1.87)	0	NC	0	NC
75	Diseases of breast	0	NC	0	NC	0	NC
76	diseases female genital organs	0	NC	0	NC	0	NC
77	Other genitourinary diseases	53	1.12 (0.84 to 1.46)	12	1.18 (0.61 to 2.05)	19	0.87 (0.52 to 1.36)
78-79	diseases of skin & subcutaneous tissue	13	1.18 (0.63 to 2.02)	<5	1.77 (0.48 to 4.53)	<5	0.77 (0.21 to 1.97)
78	Skin & subcutaneous infections	5	1.21 (0.39 to 2.83)	<5	2.35 (0.28 to 8.48)	<5	1.03 (0.12 to 3.71)
79	Other diseases skin & subcutaneous tissue	8	1.17 (0.50 to 2.30)	<5	1.42 (0.17 to 5.13)	<5	0.62 (0.07 to 2.23)
80-82	diseases musculoskeletal & connective tissue	15	0.53 (0.30 to 0.88)	<5	0.35 (0.04 to 1.25)	7	0.53 (0.21 to 1.09)
80	Arthritis & spondylitis	<5	0.18 (0.02 to 0.65)	0	NC	0	NC
81	Osteomyelitis & periostitis	<5	0.91 (0.19 to 2.67)	0	NC	<5	1.27 (0.15 to 4.60)
82	Other diseases musculoskeletal	10	0.73 (0.35 to 1.35)	<5	0.72 (0.09 to 2.61)	5	0.76 (0.25 to 1.78)
83	Symptoms & ill-defined conditions	161	1.31 (1.11 to 1.53)	<5	0.08 (0.01 to 0.30)	54	0.92 (0.69 to 1.19)
84-88	Accidents	524	0.86 (0.79 to 0.94)	87	0.78 (0.62 to 0.96)	289	0.99 (0.88 to 1.11)
84	Transportation accidents	160	0.56 (0.48 to 0.65)	29	0.57 (0.38 to 0.82)	89	0.65 (0.52 to 0.80)
85	Accidental poisoning	45	0.88 (0.64 to 1.18)	6	0.70 (0.26 to 1.51)	26	0.98 (0.64 to 1.43)
86	Accidental falls	113	1.31 (1.08 to 1.58)	27	1.49 (0.98 to 2.17)	62	1.57 (1.20 to 2.01)
87	Other accidents	197	1.17 (1.01 to 1.34)	24	0.77 (0.50 to 1.15)	109	1.34 (1.10 to 1.61)
88	Medical causes	9	0.63 (0.29 to 1.20)	<5	0.35 (0.01 to 1.95)	<5	0.44 (0.09 to 1.29)

Minor ID*	Cause	All		San Francisco		Chicago	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
89-91	Violence	251	0.78 (0.69 to 0.89)	57	1.00 (0.76 to 1.30)	120	0.75 (0.63 to 0.90)
89	Suicide	193	0.87 (0.75 to 1.00)	50	1.24 (0.92 to 1.63)	86	0.81 (0.65 to 1.00)
90	Homicide	58	0.59 (0.45 to 0.76)	7	0.43 (0.17 to 0.88)	34	0.65 (0.45 to 0.91)
91	Terrorism	0	NC	0	NC	0	NC
92	Other & unspecified causes	370	1.15 (1.04 to 1.27)	39	0.63 (0.45 to 0.86)	182	1.14 (0.98 to 1.32)

*Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas
MN, malignancy; NC, not calculated; Obs, observed; SMR, standardized mortality ratio.

Table S4. Cancer-specific standardized incidence ratios compared to the US population by fire department (1985-2009, *n*=24453

Minor ID[†]	Cause	All		San Francisco		Chicago	
		Obs	SIR (95% CI)	Obs	SIR (95% CI)	Obs	SIR (95% CI)
1-41	All Cancers	4461	1.09 (1.06 to 1.12)	855	1.14 (1.06 to 1.22)	2186	1.09 (1.04 to 1.13)
1-4	MN buccal cavity & pharynx	174	1.39 (1.19 to 1.62)	43	1.96 (1.42 to 2.64)	81	1.32 (1.05 to 1.64)
1	MN lip	<20	1.11 (0.65 to 1.78)	10	3.71 (1.78 to 6.82)	5	0.68 (0.22 to 1.58)
2	MN tongue	52	1.74 (1.30 to 2.28)	9	1.70 (0.78 to 3.23)	23	1.58 (1.00 to 2.38)
3	MN other buccal	46	1.24 (0.91 to 1.65)	11	1.67 (0.83 to 2.99)	25	1.37 (0.89 to 2.03)
4	MN pharynx	59	1.39 (1.06 to 1.79)	13	1.77 (0.94 to 3.02)	28	1.33 (0.88 to 1.92)
5-6	MN colon & rectum	537	1.18 (1.08 to 1.28)	100	1.20 (0.98 to 1.46)	267	1.20 (1.06 to 1.35)
5	MN large intestine	381	1.21 (1.09 to 1.34)	72	1.24 (0.97 to 1.56)	186	1.21 (1.04 to 1.40)
6	MN rectum	156	1.11 (0.94 to 1.30)	28	1.12 (0.74 to 1.61)	81	1.17 (0.93 to 1.46)
7-12	MN digestive other & peritoneum	393	1.16 (1.05 to 1.28)	69	1.11 (0.87 to 1.41)	200	1.20 (1.04 to 1.37)
7	MN esophagus	90	1.62 (1.31 to 2.00)	15	1.49 (0.83 to 2.45)	49	1.79 (1.33 to 2.37)
8	MN stomach	93	1.15 (0.93 to 1.40)	12	0.82 (0.42 to 1.43)	50	1.24 (0.92 to 1.64)
9	MN small intestine	<18	1.15 (0.67 to 1.85)	<5	1.49 (0.41 to 3.82)	5	0.69 (0.22 to 1.61)
10	MN biliary, liver, gall bladder	85	1.10 (0.88 to 1.36)	16	1.12 (0.64 to 1.82)	47	1.22 (0.89 to 1.62)
11	MN pancreas	90	0.96 (0.77 to 1.18)	19	1.10 (0.66 to 1.72)	43	0.94 (0.68 to 1.27)
12	MN peritoneum, other & unspecified	<20	1.10 (0.65 to 1.74)	<5	1.01 (0.21 to 2.96)	6	0.74 (0.27 to 1.62)
13-16	MN respiratory & Intrathoracic	813	1.16 (1.08 to 1.24)	91	0.72 (0.58 to 0.88)	463	1.33 (1.21 to 1.46)
13	MN larynx	84	1.50 (1.19 to 1.85)	10	1.02 (0.49 to 1.88)	42	1.51 (1.08 to 2.03)
14	MN trachea, bronchus, lung	716	1.12 (1.04 to 1.21)	81	0.70 (0.56 to 0.87)	409	1.30 (1.17 to 1.43)
15	MN pleura	0	NC	0	NC	0	NC
16	MN other respiratory & intrathoracic	<17	1.37 (0.73 to 2.34)	0	NC	12	2.55 (1.32 to 4.45)
17	MN breast	26	1.26 (0.82 to 1.85)	6	1.23 (0.45 to 2.67)	14	1.19 (0.65 to 1.99)
18-21	MN female genital organs	<5	0.62 (0.13 to 1.81)	<5	0.77 (0.02 to 4.28)	<5	0.66 (0.08 to 2.40)
18	MN cervix uteri	<5	1.20 (0.15 to 4.33)	<5	2.06 (0.05 to 11.49)	<5	1.02 (0.03 to 5.67)
19	MN other & unspecified parts of uterus	<5	0.56 (0.01 to 3.11)	0	NC	<5	0.86 (0.02 to 4.81)
20	MN ovary, fallopian tube, & broad ligament	0	NC	0	NC	0	NC
21	MN other female genital organs	0	NC	0	NC	0	NC
22-24	MN male genital organs	1278	1.02 (0.97 to 1.08)	278	1.21 (1.07 to 1.36)	602	0.98 (0.91 to 1.07)
22	MN prostate	1261	1.03 (0.98 to 1.09)	276	1.22 (1.08 to 1.37)	592	0.99 (0.91 to 1.07)
23	MN testes	<18	0.75 (0.42 to 1.24)	<5	0.74 (0.09 to 2.67)	8	0.76 (0.33 to 1.50)
24	MN other & unspecified male genital organs	<5	0.26 (0.03 to 0.93)	0	NC	<5	0.53 (0.06 to 1.92)
25-26	MN urinary organs	482	1.17 (1.06 to 1.27)	89	1.15 (0.93 to 1.42)	234	1.17 (1.02 to 1.32)
25	MN kidney	166	1.27 (1.09 to 1.48)	26	1.10 (0.72 to 1.61)	83	1.30 (1.04 to 1.61)

Minor ID†	Cause	All		San Francisco		Chicago	
		Obs	SIR (95% CI)	Obs	SIR (95% CI)	Obs	SIR (95% CI)
26	MN bladder & other urinary organs	316	1.12 (1.00 to 1.25)	63	1.18 (0.91 to 1.51)	151	1.10 (0.93 to 1.29)
27-28	MN thyroid & other endocrine glands	28	0.91 (0.60 to 1.31)	<5	0.72 (0.20 to 1.84)	15	0.98 (0.55 to 1.61)
27	MN thyroid gland	25	0.87 (0.56 to 1.28)	<5	0.57 (0.12 to 1.68)	13	0.90 (0.48 to 1.55)
28	MN other endocrine glands	<5	1.50 (0.31 to 4.39)	<5	2.90 (0.07 to 16.18)	<5	2.03 (0.25 to 7.32)
29-35	MN other solid cancers	275	0.97 (0.86 to 1.09)	85	1.69 (1.35 to 2.09)	104	0.76 (0.62 to 0.92)
29	MN bone	<13	2.62 (1.35 to 4.57)	0	NC	8	3.49 (1.51 to 6.87)
30	MN melanoma (skin)	141	0.87 (0.73 to 1.03)	56	1.89 (1.43 to 2.46)	44	0.56 (0.41 to 0.76)
31	Kaposi sarcoma	<5	0.17 (0.05 to 0.43)	<5	0.30 (0.01 to 1.69)	<5	0.08 (0.00 to 0.47)
32	MN mesothelioma	35	2.29 (1.60 to 3.19)	6	2.05 (0.75 to 4.47)	20	2.71 (1.65 to 4.18)
33	MN connective tissue	22	1.07 (0.67 to 1.62)	<5	1.06 (0.29 to 2.72)	10	0.99 (0.47 to 1.82)
34	MN brain & other nervous system	51	1.02 (0.76 to 1.34)	17	1.95 (1.14 to 3.12)	13	0.53 (0.28 to 0.91)
35	MN eye	<18	1.45 (0.69 to 2.66)	<5	0.81 (0.02 to 4.53)	8	2.44 (1.05 to 4.80)
36-40	MN lymphohematopoietic tissue	345	0.94 (0.84 to 1.04)	68	1.00 (0.78 to 1.27)	154	0.85 (0.72 to 1.00)
36	Hodgkin lymphoma	<17	0.96 (0.54 to 1.59)	6	2.36 (0.87 to 5.15)	6	0.76 (0.28 to 1.65)
37	Non-Hodgkin lymphoma	169	0.99 (0.84 to 1.15)	28	0.90 (0.60 to 1.30)	79	0.94 (0.75 to 1.18)
38	Multiple myeloma	36	0.72 (0.50 to 0.99)	9	0.97 (0.44 to 1.84)	16	0.65 (0.37 to 1.05)
39	Leukemia & aleukemia	100	0.94 (0.77 to 1.15)	22	1.12 (0.70 to 1.69)	43	0.83 (0.60 to 1.12)
40	Other lymphohematopoietic neoplasms	<27	0.97 (0.63 to 1.43)	<5	0.56 (0.12 to 1.64)	10	0.79 (0.38 to 1.45)
41	MN Ill-specified & residual	107	1.04 (0.86 to 1.26)	21	1.10 (0.68 to 1.68)	50	1.00 (0.75 to 1.32)

*Analysis of all occurrences of invasive cancer (i.e., multiple-cancer approach).

† Minor cause-of-death category in Table S4 of this appendix.

MN, malignancy; NC, not calculated; Obs, observed; SIR, standardized incidence ratio.

Table S5. Recode from ICD-O-3 codes reported by cancer incidence registries to diagnostic minor codes used in NIOSH LTAS*.

Major Category	Minor ID	Minor Category	ICD-10 Codes	ICD-O-3 Site Codes	ICD-O-3 Histology Cod
MN of buccal cavity and pharynx	1	MN of lip	C00	C000-C009	All excluding 9140, 905(
	2	MN of tongue	C01, C02	C019-C029	
	3	MN of other buccal cavity	C03-C08	C039-C069, C079-C089	
	4	MN of pharynx	C09-C14	C090- C119, C129-C148	
MN of colon and rectum	5	MN of colon	C18	C180-C189	
	6	MN of rectum	C19, C20	C199, C209	
MN of other digestive organs and peritoneum	7	MN of esophagus	C15	C150- C159	
	8	MN of stomach	C16	C160-C169	
	9	MN of small intestine	C17	C170-C179	
	10	MN of biliary, liver, gall bladder	C22-C24	C220, C221, C239-C249	
	11	MN of pancreas	C25	C250-C259	
	12	MN of anus, peritoneum, other, and unspecified digestive	C21, C26, C48	C210-C212, C218, C260, C268, C269, C422, C480-C482, C488	
MN of respiratory and Intrathoracic organs	13	MN of larynx	C32	C320-C329	
	14	MN of trachea, bronchus, and lung	C33, C34	C339-C349	
	15	MN of pleura	C38.4	C384	
	16	MN of other respiratory and intrathoracic organs	C30, C31, C37, C38.0-C38.3, C38.8, C39	C300,C301, C310-C319, C379, C380-C383, C388, C390, C398, C399	
MN of breast	17	MN of breast	C50	C500-C509	
MN of female genital organs	18	MN of cervix uteri	C53	C530-C539	
	19	MN of other and unspecified parts of uterus	C54, C55, C58	C540-C549, C559, C589	
	20	MN of ovary, fallopian tube, and broad ligament	C56, 57.0-C57.4, C57.8	C569-C574, C578	

Major Category	Minor ID	Minor Category	ICD-10 Codes	ICD-O-3 Site Codes	ICD-O-3 Histology Cod
	21	MN of other and unspecified female genital organs	C51, C52, C57.7, C57.9	C510-C519, C529, C577, C579	
MN of male genital organs	22	MN of prostate	C61	C619	
	23	MN of testes	C62	C620-C629	
	24	MN of other and unspecified male genital organs	C60, C63	C600-C609, C630-C639	
MN of urinary organs	25	MN of kidney	C64-C66	C649, C659, C669	
	26	MN of bladder and other urinary organs	C67, C68, D09.0†	C670-C689	
MN of thyroid and other endocrine glands	27	MN of thyroid gland	C73	C739	
	28	MN of other endocrine glands	C74, C75	C740-C749, C750-C759	
MN of other solid cancers	29	MN of bone	C40, C41	C400-C419	
	30	Malignant melanoma of skin	C43	C440-C449	8720-8790
	31	Kaposi sarcoma	C46	Not used	9140
	32	Mesothelioma	C45	Not used	9050-9055
	33	MN of connective tissue	C49	C490-C499	All excluding 9140, 905(
	34	MN brain and other parts of nervous system	C47, C70-C72	C470-C479, C700-C729	
	35	MN eye	C69	C690-C699	
Malignant neoplasms of lymphatic and hematopoietic tissue	36	Hodgkin lymphoma	C81	Not used	9650- 9667
	37	Non-Hodgkin lymphoma	C82-C85, C88.0, C88.3, C91.4, C96.0-C96.3, C96.7	Not used	9590, 9591, 9596, 9670, 9680, 9684, 9687, 9688, 9702, 9705, 9708, 9709, 9729, 9735, 9737, 9738, 9761, 9764, 9940
	38	Multiple myeloma	C90	Not used	9731-9734
	39	Leukemia and aleukemia	C91.0-C91.3, C91.5, C91.7, C91.9, C92-C95	Not used	9742, 9800, 9801, 9805, 9831-9837, 9840, 9860, 9870-9876, 9891, 9895-9 9931, 9945, 9946, 9948,

Major Category	Minor ID	Minor Category	ICD-10 Codes	ICD-O-3 Site Codes	ICD-O-3 Histology Cod
	40	Other lymphatic and hematopoietic neoplasms	C88.2, C88.7, C88.9, C96.9, D45, D46.1-D46.4, D46.7, D46.9, D47.1, D47.3, D47.7	Not used	9751, 9760, 9762, 9950, 9980, 9982-9987, 9989
Ill-specified and residual	41	MN of Ill-specified and residual sites	C44, C76, C77, C80, C97	C440-C449	All excluding 8720-8790 9590-9989
				C760-C768, C809, C420-C424, C770-C779	All excluding 9140, 905(

*Results in Table 2 of the main manuscript differ slightly from Table S3 due to classification adjustment made to better align can with mortality results in Table 2. Specifically, Table 2 differs from this recode by including ICD-10 codes C21 with MN rectum, C88.7 and C88.9 with multiple myeloma.

†Urinary bladder incidence cases originally coded *in situ* (Behavior=2) were recoded to invasive (Behavior=3) per SEER protoco

ICD-O-3, International Classification of Diseases for Oncology, 3rd Edition; ICD-10, International Classification of Diseases, 10 malignancy; SEER, Surveillance, Epidemiology, and End Results Program

Table S6. Heterogeneity in standardized mortality ratios across fire departments with U.S. population referent (1950-2009).

Minor ID*	Underlying Cause	All fire departments†		San Francisco		Chicago		Philadelp
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMI
7	MN esophagus	113	1.39 (1.15 to 1.67)	23	1.45 (0.92 to 2.18)	58	1.47 (1.12 to 1.90)	32	1.22 (0.
8	MN stomach	110	1.10 (0.91 to 1.33)	25	1.23 (0.80 to 1.82)	53	1.14 (0.86 to 1.50)	32	0.97 (0.
9	MN intestine	326	1.30 (1.16 to 1.44)	56	1.09 (0.82 to 1.41)	157	1.33 (1.13 to 1.55)	113	1.38 (1.
10	MN rectum	89	1.45 (1.16 to 1.80)	20	1.59 (0.97 to 2.46)	47	1.65 (1.21 to 2.20)	22	1.08 (0.
15	MN lung	1046	1.02 (0.81 to 1.29)	142	0.76 (0.64 to 0.89)	566	1.23 (1.13 to 1.34)	338	1.11 (0.
23	MN prostate	282	1.05 (0.87 to 1.27)	51	0.91 (0.68 to 1.19)	152	1.28 (1.08 to 1.50)	79	0.94 (0.
25	MN kidney	94	1.23 (0.90 to 1.67)	13	0.90 (0.48 to 1.54)	56	1.62 (1.23 to 2.11)	25	1.05 (0.
26	MN bladder	84	0.99 (0.80 to 1.22)	16	0.88 (0.50 to 1.43)	40	1.02 (0.73 to 1.39)	28	1.00 (0.
30	MN brain	73	1.01 (0.80 to 1.27)	16	1.16 (0.66 to 1.89)	34	0.98 (0.68 to 1.37)	23	0.96 (0.
35	NHL	123	1.17 (0.98 to 1.40)	25	1.19 (0.77 to 1.75)	53	1.06 (0.80 to 1.39)	45	1.32 (0.
37	Leukemia	122	1.10 (0.92 to 1.32)	23	1.02 (0.65 to 1.53)	61	1.17 (0.90 to 1.50)	38	1.05 (0.
38	Multiple myeloma	42	0.89 (0.66 to 1.20)	7	0.74 (0.30 to 1.52)	19	0.84 (0.50 to 1.30)	16	1.06 (0.
62	COPD	367	0.68 (0.53 to 0.85)	57	0.53 (0.40 to 0.69)	206	0.87 (0.75 to 0.99)	104	0.63 (0.

*Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas restricted to 20 or more total cases.

†Results from Poisson mixed model that adjusts for fire department-specific effects as a random variable.

‡Results of testing against the null model (i.e., no between-department variance).

COPD, chronic obstructive pulmonary disease; ICD-10, International Classification of Diseases, 10th Revision; LRT, likelihood malignancy; NC. Not calculated; NHL, non-Hodgkin lymphoma; Obs, observed; SMR, standardized mortality ratio.

Table S7. Heterogeneity in standardized incidence ratios by department compared to the US population for cancers of *a priori* int

Minor ID*	Cause	All departments† Obs	All departments† SIR (95% CI)	San Francisco Obs	San Francisco SIR (95% CI)	Chicago Obs	Chicago SIR (95% CI)	Philadelp Obs	Philadelp SIF
5	MN large intestine	381	1.21 (1.09 to 1.34)	72	1.24 (0.97 to 1.56)	186	1.21 (1.04 to 1.40)	123	1.19 (0.
6	MN rectum	156	1.11 (0.95 to 1.30)	28	1.12 (0.74 to 1.61)	81	1.17 (0.93 to 1.46)	47	1.02 (0.
7	MN esophagus	90	1.62 (1.32 to 1.99)	15	1.49 (0.83 to 2.45)	49	1.79 (1.33 to 2.37)	26	1.44 (0.
8	MN stomach	93	1.15 (0.94 to 1.40)	12	0.82 (0.42 to 1.43)	50	1.24 (0.92 to 1.64)	31	1.18 (0.
14	MN lung	716	1.01 (0.76 to 1.35)	81	0.70 (0.56 to 0.87)	409	1.30 (1.17 to 1.43)	226	1.10 (0.
17	MN breast	26	1.26 (0.86 to 1.85)	6	1.23 (0.45 to 2.67)	14	1.19 (0.65 to 1.99)	6	1.53 (0.
22	MN prostate	1261	1.05 (0.95 to 1.17)	276	1.22 (1.08 to 1.37)	592	0.99 (0.91 to 1.07)	393	0.99 (0.
25	MN kidney	166	1.27 (1.09 to 1.48)	26	1.10 (0.72 to 1.61)	83	1.30 (1.04 to 1.61)	57	1.33 (1.
26	MN bladder	316	1.11 (1.00 to 1.25)	63	1.18 (0.91 to 1.51)	151	1.10 (0.93 to 1.29)	102	1.10 (0.
34	MN brain	51	1.07 (0.59 to 1.95)	17	1.95 (1.14 to 3.12)	13	0.53 (0.28 to 0.91)	21	1.25 (0.
37	NHL	169	0.99 (0.85 to 1.15)	28	0.90 (0.60 to 1.30)	79	0.94 (0.75 to 1.18)	62	1.10 (0.
38	Multiple myeloma	36	0.72 (0.52 to 0.99)	9	0.97 (0.44 to 1.84)	16	0.65 (0.37 to 1.05)	11	0.68 (0.
39	Leukemia	100	0.94 (0.78 to 1.15)	22	1.12 (0.70 to 1.69)	43	0.83 (0.60 to 1.12)	35	1.01 (0.

* Minor cause shown in Table S4 of this appendix. Reporting restricted to 20 or more total cases.

†Results from Poisson mixed model that adjusts for department-specific effects as a random variable.

‡Results of testing against the null model (i.e., no between-department variance).

COPD, chronic obstructive pulmonary disease: LRT, likelihood ratio test; MN, malignancy; NHL, non-Hodgkin lymphoma; Obs standardized mortality ratio.

Table S8. Standardized mortality ratios using State mortality rates for causes of death of *a priori* interest (1950-2009).

Minor ID*	Underlying Cause	All departments†		San Francisco (California rates)		Chicago (Illinois rates)		Philadel (Pennsylvan	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMF
7	MN esophagus	113	1.28 (1.06 to 1.54)	23	1.57 (1.00 to 2.36)	58	1.32 (1.00 to 1.71)	32	1.07 (0.
8	MN stomach	110	1.05 (0.87 to 1.26)	25	1.20 (0.78 to 1.77)	53	1.10 (0.82 to 1.43)	32	0.90 (0.
9	MN intestine	326	1.19 (1.07 to 1.33)	56	1.22 (0.92 to 1.59)	157	1.17 (1.00 to 1.37)	113	1.19 (0.
10	MN rectum	89	1.30 (1.01 to 1.68)	20	1.67 (1.02 to 2.59)	47	1.45 (1.07 to 1.93)	22	0.92 (0.
15	MN lung	1046	1.06 (0.92 to 1.23)	142	0.87 (0.73 to 1.02)	566	1.20 (1.10 to 1.30)	338	1.10 (0.
23	MN prostate	282	1.04 (0.86 to 1.26)	51	0.90 (0.67 to 1.19)	152	1.26 (1.07 to 1.48)	79	0.92 (0.
25	MN kidney	94	1.24 (0.96 to 1.61)	13	0.97 (0.51 to 1.66)	56	1.51 (1.14 to 1.96)	25	1.06 (0.
26	MN bladder	84	0.94 (0.76 to 1.17)	16	0.91 (0.52 to 1.47)	40	0.98 (0.70 to 1.33)	28	0.92 (0.
30	MN brain	73	1.05 (0.83 to 1.32)	16	1.13 (0.64 to 1.83)	34	1.01 (0.70 to 1.42)	23	1.05 (0.
35	NHL	123	1.11 (0.93 to 1.33)	25	1.17 (0.76 to 1.73)	53	0.99 (0.74 to 1.30)	45	1.25 (0.
37	Leukemia	122	1.07 (0.90 to 1.28)	23	1.07 (0.68 to 1.60)	61	1.10 (0.84 to 1.42)	38	1.03 (0.
38	Multiple myeloma	42	0.91 (0.67 to 1.23)	7	0.75 (0.30 to 1.55)	19	0.86 (0.52 to 1.34)	16	1.07 (0.
62	COPD	367	0.71 (0.55 to 0.93)	57	0.54 (0.41 to 0.69)	206	0.93 (0.81 to 1.07)	104	0.69 (0.

* Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas restricted to 20 or more total cases.

†Results from Poisson mixed model that adjusts for department-specific effects as a random variable.

‡Results of testing against the null model (i.e., no between-department variance).

COPD, chronic obstructive pulmonary disease; LRT, likelihood ratio test; MN, malignancy; NC, not calculated; NHL, non-Hodg observed; SMR, standardized mortality ratio.

Table S9. Standardized mortality ratios by hire type, with U.S. population referent (1950-2009).

Minor ID*		All hires employed 1+ years		Hired on or after 1950 (incid All		
	Underlying Cause	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Ot
1-92	All causes	11821	0.99 (0.97 to 1.01)	4441	0.91 (0.89 to 0.94)	423
3-38	All cancers	3231	1.14 (1.10 to 1.18)	1454	1.12 (1.07 to 1.18)	140
7	MN esophagus	111	1.39 (1.14 to 1.67)	67	1.51 (1.17 to 1.92)	6
8	MN stomach	109	1.11 (0.91 to 1.34)	45	1.29 (0.94 to 1.73)	4
9	MN intestine	322	1.30 (1.16 to 1.45)	120	1.18 (0.98 to 1.41)	11
10	MN rectum	88	1.45 (1.17 to 1.79)	36	1.56 (1.09 to 2.15)	3
15	MN lung	1028	1.10 (1.03 to 1.17)	476	1.05 (0.96 to 1.15)	45
18	MN breast	7	1.25 (0.50 to 2.57)	6	1.68 (0.62 to 3.67)	
23	MN prostate	278	1.09 (0.96 to 1.22)	95	1.18 (0.96 to 1.45)	9
24	MN other male genital organs	<5	0.48 (0.13 to 1.23)	<5	0.42 (0.05 to 1.52)	<
25	MN kidney	91	1.28 (1.03 to 1.57)	53	1.46 (1.10 to 1.92)	5
26	MN bladder& other urinary	84	1.00 (0.80 to 1.24)	27	0.89 (0.59 to 1.30)	2
30	MN brain & other nervous	73	1.03 (0.81 to 1.29)	36	0.90 (0.63 to 1.25)	3
35	NHL	120	1.16 (0.96 to 1.39)	56	1.06 (0.80 to 1.38)	5
37	Leukemia	121	1.11 (0.92 to 1.33)	55	1.10 (0.83 to 1.43)	5
38	Multiple myeloma	42	0.90 (0.65 to 1.22)	17	0.76 (0.44 to 1.22)	1
62	COPD	361	0.72 (0.65 to 0.80)	145	0.75 (0.63 to 0.88)	13

*Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas
COPD, chronic obstructive pulmonary disease; ICD-10, International Classification of Diseases, 10th Revision; MN, malignancy lymphoma; Obs, observed; SMR, standardized mortality ratio.

Table S10. Heterogeneity in standardized mortality ratios by age compared to the US population for causes of death of *a priori* ir

Minor ID*	Underlying Cause	All Ages† Obs	All Ages† SMR (95% CI)	Age 65-85+ Obs	Age 65-85+ SMR (95% CI)	Age 17-64 Obs	Age 17-64 SMR (95% C
7	MN esophagus	113	1.39 (1.15 to 1.67)	62	1.36 (1.05 to 1.75)	51	1.41 (1.05 to 1
8	MN stomach	110	1.02 (0.76 to 1.37)	74	1.33 (1.05 to 1.68)	36	0.81 (0.57 to 1
9	MN intestine	326	1.23 (1.03 to 1.48)	231	1.42 (1.24 to 1.61)	95	1.07 (0.87 to 1
10	MN rectum	89	1.45 (1.18 to 1.78)	51	1.49 (1.11 to 1.96)	38	1.40 (0.99 to 1
15	MN lung	1046	1.09 (1.01 to 1.18)	662	1.17 (1.09 to 1.27)	384	0.99 (0.90 to 1
23	MN prostate	282	1.09 (0.97 to 1.22)	245	1.08 (0.95 to 1.23)	37	1.10 (0.77 to 1
25	MN kidney	94	1.29 (1.06 to 1.58)	46	1.15 (0.84 to 1.53)	48	1.47 (1.09 to 1
26	MN bladder	84	0.97 (0.74 to 1.26)	65	1.01 (0.78 to 1.29)	19	0.90 (0.54 to 1
30	MN brain	73	1.01 (0.80 to 1.27)	34	1.20 (0.83 to 1.67)	39	0.89 (0.63 to 1
35	NHL	123	1.15 (0.90 to 1.46)	86	1.37 (1.09 to 1.69)	37	0.88 (0.62 to 1
37	Leukemia	122	1.10 (0.92 to 1.31)	81	1.17 (0.93 to 1.46)	41	0.98 (0.70 to 1
38	Multiple myeloma	42	0.89 (0.66 to 1.20)	29	0.93 (0.63 to 1.34)	13	0.80 (0.42 to 1
62	COPD	367	0.70 (0.59 to 0.82)	313	0.77 (0.69 to 0.86)	54	0.53 (0.40 to 0

* Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas restricted to 20 or more total cases.

†Results from Poisson mixed model that adjusts for age-specific effects as a random variable.

‡Results of testing against the null model (i.e., no between-age group variance). Age defined as all ages in 5-year periods.

COPD, chronic obstructive pulmonary disease; LRT, likelihood ratio test; NC, not calculated; MN, malignancy; NHL, non-Hodg observed; SMR, standardized mortality ratio.

Table S11. Heterogeneity in standardized incidence ratios by age compared to the US population for cancers of *a priori* interest (

Minor ID*	Underlying Cause	All Ages†		Age 65-85+		Age 17-64	
		Obs	SIR (95% CI)	Obs	SIR (95% CI)	Obs	SIR (95% CI
5	MN large intestine	381	1.21 (1.09 to 1.34)	269	1.20 (1.06 to 1.35)	112	1.23 (1.01 to 1
6	MN rectum	156	1.11 (0.95 to 1.30)	97	1.12 (0.91 to 1.37)	59	1.10 (0.83 to 1
7	MN esophagus	90	1.60 (1.27 to 2.03)	55	1.59 (1.20 to 2.07)	35	1.68 (1.17 to 2
8	MN stomach	93	1.15 (0.94 to 1.40)	62	1.15 (0.88 to 1.48)	31	1.14 (0.77 to 1
14	MN lung	716	1.12 (1.05 to 1.21)	494	1.13 (1.03 to 1.23)	222	1.12 (0.98 to 1
17	MN breast	26	1.26 (0.86 to 1.85)	<5	0.53 (0.11 to 1.56)	23	1.53 (0.97 to 2
22	MN prostate	1261	1.10 (0.95 to 1.28)	835	0.96 (0.90 to 1.03)	426	1.21 (1.10 to 1
25	MN kidney	166	1.27 (1.09 to 1.48)	87	1.17 (0.94 to 1.44)	79	1.41 (1.12 to 1
26	MN bladder	316	1.11 (0.90 to 1.36)	219	1.04 (0.91 to 1.19)	97	1.33 (1.08 to 1
34	MN brain	51	1.02 (0.77 to 1.34)	25	1.04 (0.67 to 1.54)	26	1.00 (0.65 to 1
37	NHL	169	0.98 (0.82 to 1.17)	107	1.10 (0.90 to 1.33)	62	0.84 (0.64 to 1
38	Multiple myeloma	36	0.72 (0.52 to 0.99)	26	0.77 (0.50 to 1.13)	10	0.60 (0.29 to 1
39	Leukemia	100	0.94 (0.78 to 1.15)	61	0.86 (0.66 to 1.10)	39	1.12 (0.80 to 1

* Minor cause shown in Table S4 of this appendix. Reporting restricted to 20 or more total cases.
† Results from Poisson mixed model that adjusts for age-specific effects as a random variable.
‡ Results of testing against the null model (i.e., no between-age group variance). Age defined as all ages in 5-year periods.
LRT, likelihood ratio test; MN, malignancy; NHL, non-Hodgkin lymphoma; Obs, observed; SIR, standardized incidence ratio.

Table S12. Standardized mortality ratios with censoring at age ≥85 for causes of death of *a priori* interest (1950-2009), US population referent.

Minor ID*	Underlying Cause	All ages		Age <85	
		Obs	SMR (95% CI)	Obs	SMR (95% CI)
7	MN esophagus	113	1.39 (1.14 to 1.67)	108	1.38 (1.13 to 1.66)
8	MN stomach	110	1.10 (0.91 to 1.33)	102	1.07 (0.87 to 1.30)
9	MN intestine	326	1.30 (1.16 to 1.44)	301	1.29 (1.15 to 1.44)
10	MN rectum)	89	1.45 (1.16 to 1.78)	82	1.40 (1.12 to 1.74)
15	MN lung	1046	1.10 (1.04 to 1.17)	1001	1.09 (1.03 to 1.16)
18	MN breast	8	1.39 (0.60 to 2.73)	7	1.27 (0.51 to 2.62)
23	MN prostate	282	1.09 (0.96 to 1.22)	235	1.08 (0.95 to 1.23)
24	MN other male genital	<5	0.47 (0.13 to 1.20)	<5	0.48 (0.13 to 1.24)
25	MN kidney	94	1.29 (1.05 to 1.58)	91	1.32 (1.06 to 1.62)
26	MN bladder	84	0.99 (0.79 to 1.22)	75	1.00 (0.78 to 1.25)
30	MN brain	73	1.01 (0.79 to 1.27)	71	1.00 (0.78 to 1.26)
35	NHL	123	1.17 (0.97 to 1.40)	113	1.15 (0.95 to 1.39)
37	Leukemia	122	1.10 (0.91 to 1.31)	116	1.13 (0.93 to 1.36)
38	Multiple myeloma	42	0.89 (0.64 to 1.20)	39	0.88 (0.63 to 1.20)
62	COPD	367	0.72 (0.65 to 0.80)	321	0.71 (0.64 to 0.79)

* Minor cause-of-death category in NIOSH Life Table Analysis System (LTAS) 92-cause rate file: http://www.cdc.gov/niosh/ltas/rates.html.

COPD, chronic obstructive pulmonary disease; LRT, likelihood ratio test; MN, malignancy; NHL, non-Hodgkin lymphoma; Obs, observed; SMR, standardized mortality ratio.

NIOSH Publishes Study of Cancer Among Firefighters

Claire Reiss
National League of Cities Risk Information Sharing Consortium
November 14, 2013

The National Institute for Occupational Safety and Health has released the attached new study[1], Mortality and Cancer Incidence in a Pooled Cohort of US Firefighters from San Francisco, Chicago and Philadelphia (1950-2009). The research has been underway for several years, and we have previously brought it to your attention. This paper summarizes and identifies questions about the study's important conclusions, and discusses how it relates to the 2009 study published by the National League of Cities, Assessing State Firefighter Cancer Presumption Laws and Current Firefighter Cancer Research.[2]

The NIOSH study may not be representative of the typical exposures faced in the fire departments insured by NLC-RISC member pools. The NIOSH report focuses on 59 years of data about 30,000 career firefighters at three big-city fire departments: Philadelphia, Chicago and San Francisco. These are all old-line cities where the firefighters would be expected to fight more fires and encounter asbestos and chemicals more often and in greater concentrations than in the smaller cities and towns typical of our membership. The career service is important, certainly, as it protects 66% of the U.S. population, but almost 70% of 1,129,250 firefighters in the U.S. are volunteers, and 85% of U.S. fire departments are all or mostly volunteer.[3] A large part of the U.S. is protected by those volunteers in low population or rural settings. The type and extent of their exposure may well differ, and they are not part of the NIOSH study cohort.

NIOSH says that this study strengthens the evidence of a relationship between firefighting and certain cancers. The report and commentary suggest that firefighters have a slightly elevated risk compared to the general population, but when you look at the body of the report the significance of the excess experience for some cancers is less certain. Discerning the real significance requires careful reading of the discussion section, not just review of the statistical results. There are important issues NLC-RISC member pools may want to consider for use in their advocacy efforts on presumption legislation.

First, the NIOSH study does not identify the strength of association criteria it uses to evaluate the causal relationship between an activity and an illness. However, a presentation about the NIOSH study by one of its primary researchers at the Redmond Symposium on the Occupational Health and Hazards of the Fire Service does identify

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[1] Mortality and Cancer Incidence in a Pooled Cohort of US Firefighters from San Francisco, Chicago and Philadelphia (1950-2009), National Institute of Occupational Safety and Health, 2013. http://www.cdc.gov/niosh/firefighters/cancer.html
[2] Assessing State Firefighter Cancer Presumption Laws and Current Firefighter Cancer Research, National League of Cities, April 2009.
[3] NFPA Fire Department Profile for 2012, www.nfpa.org

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strength of association criteria.[4] According to that presentation, ratios equal to 1 indicate cancer mortality or incidence similar to the overall population. Any ratio in excess of 1, however small, indicates an excess experience. Neither the study nor the presentation indicate when an excess experience becomes sufficiently strong to show an association.

The 2009 National League of Cities research adopted specific strength of association criteria that were identified during the literature review. Those criteria require a range of 1.2 to 1.5 excess experience to show even a weak strength of association. 1.5 to 3 was considered to be a moderate strength of association. Association was not considered to be strong until the ratio reached 3 and above.[5] The NIOSH strength of association criteria thus appear to be significantly more liberal than those used in the NLC study, because they do not establish even a minimal buffer for sampling variability.

Second, despite its use of weaker standards for determining strength of association, the NIOSH study still finds only "small to moderate increases in risk for several cancer sites and for all cancers combined, stemming mostly from excess malignancies of the respiratory, digestive, and urinary systems."[6]

The ratios for the list of cancers NIOSH finds to show small to moderate increase in mortality and/or incidence are:

Cancer	SMR (Mortality ratio)	SIR (Incidence ratio)
All cancer	1.14	1.09
Bladder	.99	1.12
Buccal and pharynx	1.40	1.39
Esophagus	1.39	1.62
Intestine	1.30	1.21
Kidney	1.29	1.27
Laryngeal	N/A	1.50
Liver, gall bladder, biliary	1.30	1.06
Lung	1.10	1.12
Malignant mesothelioma	2.0	2.29
Rectum	1.45	1.11

When these ratios are evaluated using the strength of association criteria from the NLC study, malignant mesothelioma is the lone cancer to reflect both a moderate level of excess mortality/incidence (2.0/2.29 in a range of 1.5 – 3.0) and an association with a risk

[4] NIOSH Firefighter Cancer Study Workshop, August 24, 2013, Robert D. Daniels, PhD and Thomas Hales, MD, MPH, IAFF John P. Redmond Symposium on the Occupational Health and Hazards of the Fire Service, Slide 7. Available online http://www.iaff.org/Comm/PDFs/NIOSH_Cancer_Study.pdf (cut and paste link into a browser to access)

[5] Supra, p. 38.

[6] Mortality and Cancer Incidence in a Pooled Cohort of US Firefighters, supra, p. 9

factor, asbestos exposure, which is recognized to occur in firefighting. The mortality and incidence for "all cancers" and for "lung cancer" are so low as to be in the "no association" category (less than 1.2). Of the remaining cancers NIOSH identifies as "excess", all show only a "weak association" under the NLC study strength of association criteria except esophageal cancer, laryngeal cancer and malignant mesothelioma. Those cancers show a moderate association. However, as the NIOSH authors acknowledge, the primary known risk factors for esophageal and laryngeal cancer are not related to firefighting, so the elevation may be due to some other characteristic that firefighters have in common.

Third, the NIOSH study shows that numerous cancers already targeted by state presumption statutes do not have a significant excess incidence or mortality in firefighters as compared to the rest of the population. Even where there is an apparent excess of cases, the authors sometimes conclude that other risk factors were more likely causes. The discussion section of the NIOSH report made the following important observations:

- There is "little evidence of excess cancers of the testes, brain and lymphohematopoietic systems."[7]
- In women, there is statistical evidence of excess female bladder and breast cancers, but only bladder cancer mortality and incidence reach significance. The report notes: "There is little evidence linking female breast cancer to workplace exposures, however prolonged shift work may be a risk factor." The report urges cautious interpretation of the findings on female firefighters due to the "small sample size and lack of confirmatory results".[8]
- There are excess digestive cancers, primarily of esophageal and colorectal sites, but the report notes that information on occupational causes is "sparse", with only "limited evidence suggesting asbestos and diesel exhaust exposure may be weakly associated with gastrointestinal cancers." The report observes "the relation between these hazardous exposures and digestive cancers appears small compared to the effects of other factors such as diet, obesity, physical activity, tobacco use and alcohol consumption."[9]
- The important risk factors for the increased oral, pharyngeal and laryngeal cancers are tobacco and alcohol consumption, with "lesser evidence that exposures to wood dusts, smoke, asbestos, PAHs and acid mists may also increase risk."[10]
- The excess bladder and prostate cancer incidence (there is no excess mortality) is limited to firefighters between 45 and 59 year old. The report notes that "differences in medical screening (e.g., prostate-specific antigen tests) among

[7] Supra, p. 8.
[8] Supra, p. 8.
[9] Supra, p. 8.
[10] Supra, p. 8.

firefighters compared to the general population", as well as firefighting itself, could have contributed to the observed excess.[11]

Malignant mesothelioma is an exception. The study finds a "previously unreported twofold excess of malignant mesothelioma among firefighters." It notes that asbestos is the "only known causal agent" for this cancer and that firefighter exposures to asbestos "are probable", so it is likely that this excess represents a true causal connection. [12] This result should not be entirely unexpected, given the age and construction of the old-line cities studied, which would be expected to pose a greater risk of exposure to asbestos.

Fourth, much work remains to be done. The study describes itself as the "first phase of examining health effects in career firefighters", and as the "foundation for subsequent analysis", not as the last word. The presentation by the NIOSH study authors at the Redmond Symposium acknowledges several limitations:

- Low statistical power – it is difficult to observe the effect due to the long latency of the disease and the small effect size[13].
- Few women and minority firefighters are included.
- Estimates could be influenced by other factors, including other risk factors, such as tobacco use, alcohol consumption, diet and obesity, information on which was noted to be lacking.

The need to further evaluate "other risk factors" is particularly important, and the Redmond Symposium presentation notes that they will be the subject of follow up research to evaluate exposure and response. That research will estimate exposure for each firefighter by looking at all jobs held and their duration, defining the exposure potentials for those jobs, and modifying the exposure potentials during fire service based on information about fire runs, diesel exhaust controls in the station, PPE use and other factors that may affect exposure.[14]

Finally, compared to the incidence in the population as a whole, the "excess" cancers identified by NIOSH are relatively few. For example, the mortality of 1.10 for lung cancer is based on 1,046 "observations": firefighters in the cohort identified as dying from lung cancer. With a ratio of 1.10, only 10% of those deaths are actually in excess of what would be expected in the population as a whole. Thus, the actual number of excess lung cancer deaths identified over the entire 59-year period studied is 95, as compared to 951 firefighters whose disease is consistent with the incidence in the population as a

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[11] Supra, p. 8. Also note that recently a number of organizations are cautioning against the routine use of this screening for several reasons, including the incidence of false positives. http://www.cancer.gov/cancertopics/factsheet/detection/PSA

[12] Supra, p. 9.

[13] "A measure describing the magnitude of the difference between two groups." Texas Education Agency, http://www.tea.state.tx.us/Best_Practice_Standards/How_To_Interpret_Effect_Sizes.aspx

[14] NIOSH Firefighter Cancer Study Workshop, Supra, Slide 18.

whole, and the 160,340 Americans that were expected to die from lung cancer in 2012 alone.[15] The same analysis can be applied to any of the other cancers studied, and it illustrates how small an excess experience has been identified thus far, and the need for additional research to determine whether there is a true causal relation with firefighting.

Conclusion

The relationship between firefighting and cancer is an issue that is likely to remain on the front burner. More study is needed, especially about the effect of non-employment related risk factors, before any conclusions can be drawn that are sufficiently robust to support a change in public policy. Nor is the issue exclusive to firefighters, as many people in other lines of work are exposed to carcinogenic substances without any worker's compensation presumption to benefit them. The ongoing nature of this issue and the difficulty of establishing any strong relationship in many cases raises the question of whether our efforts should be directed to reducing the risk to firefighters rather than to establishing programs that will provide compensation on a presumptive basis to a large group of people who statistically would have developed the illness whatever their occupation.

We are available for questions and further exploration of this issue.

[15] Lung Cancer Fact Sheet, American Lung Association, www.lung.org

Testimony of
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International Association of Fire Fighters

On behalf of the
Pennsylvania Professional Fire Fighters Association

March 30, 2011

Good afternoon, I am Richard Duffy, the Assistant to the General President for Occupational Health, Safety and Medicine for the International Association of Fire Fighters. This morning I will discuss the important topic of chemical-induced cancers that our Public Safety and Emergency Response personnel, fire fighters in particular, may be subjected to while performing their duties. On behalf of fire fighters throughout the State of Pennsylvania, we are here today to discuss evidence that links higher rates of certain cancers with tasks that involve fire fighting emergency response activities, particularly when that response occurs in a dangerous environment containing unknown hazards. The known and potential risks to which these individuals are exposed, on our collective behalf, certainly warrants the passage of legislation that addresses the job-related health consequences suffered by our emergency responders.

Before going ahead, I believe it is important for you to understand what our organization is and whom we represent at these hearings. The IAFF is an international union affiliated with the AFL-CIO and the Canadian Labour Congress. At the present time, we represent over 298,000 paid professional fire service employees in the United States and Canada, including 7,133 IAFF members in Pennsylvania. The membership of the IAFF is employed by various parties that include the federal government, states, counties, municipalities, fire districts, airports, and industrial manufacturers.

The profession of fire fighting is and has always been a hazardous occupation. Fire fighter line-of-duty fatalities and injuries have ranked fire fighting above other publicized hazardous occupations in the private sector, such as mining and construction.

Enclosure 4

Fire Fighters and Occupational Cancer

Practically every emergency situation encountered by a fire fighter has the potential for exposure to carcinogenic agents. The list of potential carcinogenic agents to which fire fighters can be exposed is almost as long as the list of all known or suspected carcinogens, or over 700 agents. Despite the ominous risk of exposures, fire fighters knowingly enter potentially toxic atmospheres without adequate protection or knowledge of the environment. Fire fighters in Pennsylvania are exposed to toxic and carcinogenic substances at fire scenes as well as other emergencies such as chemical spills.

The long term health effects of exposures from routine fires combined with unique chemical spills may not be apparent to fire fighters until long after the memory of that incident is gone.

Fire fighters, unlike most workers in this country, have little information about the many materials to which they are potentially exposed or the hazards of such exposures. Nevertheless, fire fighters continue to respond to the scene and work immediately to save lives and reduce property damage without regard to the potential hazards that may exist. A fire emergency is an uncontrolled environment that is managed by fire fighters using heavy, bulky, and often times, inadequate personal protective equipment. The experience is not only physically demanding, but also involves exposures that are known to cause cancer.

Fire fighters are routinely exposed to complex and dynamic mixtures of chemical substances that are contained in fire smoke and building debris. Despite the large numbers of people employed in this occupation, the nature of these exposures is not well defined. Nevertheless, I will outline numerous studies to date that demonstrate that fire fighters are routinely exposed to carcinogens including the following:

Benzene

Benzene is firmly established as a human carcinogen. Numerous studies have shown that benzene is a common airborne contaminant in fire smoke and occurs in concentrations that are considered deleterious in the context of chronic exposures.

In the Harvard study, Treitman, Burgess, and Gold examined ambient environmental levels of a number of air contaminants, including benzene, at more than 200 structural fires. Benzene was detected in 181 of 197 (92%) samples taken at fire scenes by air sampling units placed on the chests of fire fighters. Half of the samples showed benzene over 1 part per million (ppm), the current Occupational Safety and Health Administration (OSHA) permissible exposure level. Approximately 5% of the samples were above 10 ppm benzene.

In Dallas, Lowry and colleagues studied fire fighters' exposure to benzene at nearly 100 structural fires. They found benzene at the majority of the fires and also detected the presence of at least 70 organic chemical species regardless of whether synthetic materials were a major part of the materials burned.

In Buffalo, Brandt-Rauf and colleagues used personal portable sampling devices to measure exposures of 51 fire fighters at 14 fires. The tubes of the sampling devices were attached to the fire fighters' turnout gear, thereby representing ambient air outside the mask. Benzene was second only to carbon monoxide as the most common chemical substance detected at the fires. It was detected in 18 of 26 samples from 12 of 14 fires. When detectable, the concentration of benzene ranged from 8.3 to 250 ppm. In only one sample where benzene was detected was its concentration below 10 ppm. Even when the smoke's intensity was rated as low, benzene was usually present in concentrations ranging from 22 to 54 ppm. The authors noted that respiratory protection was only partially used or not used at all at the fires judged to be of low smoke intensity.

Jankovic and colleagues at the National Institute for Occupational Safety and Health (NIOSH - the research institute for OSHA) studied benzene and other exposures at 22 fires, including 6 training fires, 15 residential fires, and 1 automobile fire. Samples were collected via probes placed inside and outside the masks of working fire fighters. In addition, industrial hygienists used a variety of sampling devices at the fire scene. Samples were taken separately during the two phases of a fire: knockdown and overhaul. Half of the samples taken during the knockdown phase of the fire showed benzene in concentrations of 1-22 ppm. Of the 29 organic substances analyzed, benzene was the most common compound detected and was the only substance present in all eight samples. To measure the efficacy of respiratory protection, samples for benzene were taken inside and outside the mask. Surprisingly, the levels of benzene inside the mask were as high as those taken outside the mask and ranged from nondetectable to 21 ppm. The authors attributed this equivalence in benzene concentrations inside and outside the mask to partial nonuse of the mask at the fire, especially after the initial phase of fire knockdown. They further suggested that the presence of benzene may begin only during the latter part of knockdown. During the overhaul phase of the fire, when respiratory protection is frequently removed, benzene was also found.

Asbestos

Asbestos, which has been used widely in buildings for its insulation properties, is universally recognized as a human carcinogen and is responsible for an excess risk of a variety of cancers in numerous occupations. Since the building destruction caused by fires and the building demolition actively performed by fire fighters during overhaul are likely to dislodge respirable asbestos fibers, the likelihood that fire fighters have exposure to asbestos is high.

In New York City, Markowitz and colleagues studied 212 fire fighters who had begun employment in the New York City Fire Department at least 25 years previously. Twenty of the 152 (13%) fire fighters, without any documented exposure to asbestos, had pleural thickening and/or parenchymal opacities on chest x-ray that were consistent with prior asbestos exposure.

The finding of excess risk of lung and pleural fibrosis due to asbestos among fire fighters indicated that significant asbestos exposure has occurred in this group. Since

significant asbestos exposure confers excess risk for selected cancers, it is reasonable to expect that fire fighters have an increased risk of various cancers as a result of their exposure to asbestos.

Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) are a class of organic substances that have been implicated as the carcinogens in coal tar pitches, coal tar, and selected mineral oils. They have been associated with excess risk of a variety of cancers, including cancer of the skin, lung, kidney, bladder, colon, pancreas, stomach, pharynx, brain, and leukemia.

Given the combustion of diverse materials at fires, it is likely that fire fighters would be exposed to significant levels of PAHs. A study by Jankovic et al. evaluated the presence of PAHs at the scene of fires. All 14 PAHs measured were present during the knockdown phase of the fire.

Formaldehyde

Formaldehyde is considered a probable carcinogen. It has been measured at the fire scene in a variety of studies. The current OSHA permissible exposure limit is 0.75 ppm for an 8-hour-time-weighted average and 2 ppm for a 15-minute short-term exposure. Lowry et al. reported combined formaldehyde and acetaldehyde levels, with a mean of 5 ppm and a range of 1 to 15 ppm. Brandt-Rauf and colleagues found aldehydes, including formaldehyde, at 4 of 14 fires at concentrations of 0.1 to 8.3 ppm. Jankovic et al. detected formaldehyde at levels up to 8 ppm during knockdown and 0.4 ppm during overhaul.

Diesel Exhaust

Considerable experimental and epidemiologic evidence gathered over the past 15 years suggests that constituents of diesel exhaust emissions are carcinogenic. Fire fighters have significant potential for exposure to diesel exhaust because fire trucks with diesel engines are routinely started inside of and backed into firehouses. Fire fighters spend much of the work shift inside the firehouse and obviously do not wear respiratory protection at the firehouse. Froines and colleagues studied the concentration of diesel exhaust particulates in the air inside firehouses in New York, Boston, and Los Angeles and detected airborne particulates from diesel exhaust at levels which were associated with a significant carcinogenic risk.

Other Agents

Acrolein is present in most fires as a combustion product of wood, cotton, carpeting and upholstery. Although its carcinogenicity is not well studied, one of its metabolites is a known carcinogen. Acrylonitrile is used in textiles and rubber for clothing, building materials, and household products. It is converted in the body to cyanide and causes cancer in animals and probably humans, especially cancers of the lung, prostate, stomach, colon, brain, blood, and lymphatic system. Vinyl chloride is used in the

manufacture of plastics and present in building materials and consumer goods. It is known to cause cancer in humans, especially cancer of the liver, brain, lung, blood, lymphatic system, gastrointestinal system, and malignant melanoma.

Carbon monoxide and soots are found in all fires. Carbon monoxide is a natural product of combustion and, when inhaled, it blocks the body from being able to carry and use oxygen. It is believed to cause cancer in animals and possibly humans, especially liver and kidney cancer. Soots contain a variety of chemicals including PAHs and fire fighters often have direct skin contact with soot that penetrates their clothing. Soots are known to cause cancer in humans, especially cancer of the skin, scrotum, lung, liver, esophagus, and leukemia.

Since the beginning of World War II, the production of synthetic chemicals has increased 350-fold in the United States. With the addition of thousands of synthetic chemicals annually, it becomes impossible to study the carcinogenic properties of each chemical. Furthermore, the latency period (the time from exposure to disease manifestation) for many cancers may be many years, and, therefore, it is difficult to identify the exposures responsible for adverse health effects (including cancer).

Fire fighters have a potential for exposure to multiple carcinogenic agents; many are known and many have likely not been identified. Despite protective gear, fire fighters are exposed to a variety of cancer causing agents.

Epidemiologic Studies:

When reviewing occupational studies of fire fighters, it is important to keep several points in mind. The first concept to remember is the healthy worker effect. Workers in general and fire fighters in particular, tend to be healthier compared to the general population, which includes those who cannot work due to illness or disability. This idea is supported by the low all-cause mortality rates of fire fighters. In fact, a Paris study found the mortality rate of fire fighters to be half of the general population and a study in Seattle found a 25% lower mortality rate for fire fighters. A report by Samet pooled estimates from available studies in the literature and found an overall 10% lower mortality rate for fire fighters. These findings support the proposition that fire fighters are healthier than the general population. Therefore, when a study finds a mild to moderate increase in cancer or a lack of increase in cancer in fire fighters compared to the general population it is very likely an underestimate. When a study finds fire fighters to have *any* increase in cancer rates relative to the general population it is unsettling. When significantly higher than expected rates of cancer mortality are found in fire fighters compared to the general population it is very concerning. Comparisons with another group of "healthy" workers, such as police officers, rather than with the general population are therefore more likely to provide accurate estimates of occupational risks.

Second, the shortcomings of epidemiological studies are more likely to dilute or mask associations between occupational exposures of fire fighting and cancer than to create falsely positive associations. Fire fighters who are diagnosed with cancer after retirement from the fire service may not be included in these studies. In addition, death

certificate information is often incomplete and may not reflect all cases of cancer, especially if cancer was not the primary cause of death. These oversights would further contribute to the underestimation of cancer rates and cancer deaths in fire fighters. For any given study, the lack of an association between fire fighting and a type of cancer is simply uninformative. It does not mean the relationship doesn't exist.

Third, when results are found to be "statistically significant," it means we can be confident that the differences between 2 groups (for example, fire fighters and the general population) is real and did not occur by chance. But, in order for scientific studies to report "statistically significant" conclusions, typically the number of people studied must be large, especially when studying relatively rare diseases like certain cancers. Even if fire fighters from several regions are studied together, there may not be enough cancer cases to report "statistical significance" even though a relationship between exposure and disease may be present.

Some studies investigate dose-response relationships to examine if the risk of disease increases as the dose of exposure increases. If a dose-response relationship is present, it is strong evidence for a causal relationship. However, the absence of a dose-response relationship does not rule out a causal relationship. In some cases in which a threshold may exist, no disease may develop up to a certain level of exposure, but above this level, disease may develop.

Finally, length of follow-up is important when studying cancer since many cancers develop decades after the exposure. Some studies that do not find an association simply may not have been long enough or did not include fire fighters who develop cancer after retirement.

Nevertheless, a number of studies have identified and established increased risk of cancer in fire fighters and identified associations with carcinogenic occupational exposures. The majority of studies that examined these cancers found *markedly* elevated risks for fire fighters, and there were usually no alternative viable hypotheses that could readily explain their increased prevalence.

<u>Meta-analysis of 32 Fire Fighter Cancer Studies:</u>

I would like to highlight a recent cancer study, titled "Cancer Risk Among Firefighters: A Review and Meta-analysis of 32 Studies" conducted by the University of Cincinnati and published in November 2006 in the Journal of Occupational and Environmental Medicine that found that on-the-job exposure to soot and toxins creates an increased risk for various cancers among fire fighters. The study and their analysis used data from 32 health studies conducted among fire fighters over the past 50 years, and then quantitatively and qualitatively assessed the risk of 21 cancers among fire fighters. The authors categorized the final risk as "probable," "possible," or "unlikely" patterned after the International Agency for Research on Cancer (IARC) risk assessment of human carcinogenicity.

The IARC uses the designation of "probable" when there is sufficient evidence (a causal relationship has been established) of carcinogenicity from animal studies, the same

mechanism of action is believed to occur in the human body, and a limited number of studies in humans show a carcinogenic effect. The designation of "possible" is used when there is less than sufficient evidence in animals and limited evidence in humans or sufficient evidence in animals and inadequate data in humans. The designation of "unlikely" is not used by the IARC.

The IARC's next category is "not classifiable," which is used when there is not enough convincing evidence from human or animal studies.

In order to understand the findings of this meta-analysis, it is important to understand the methodology that was used. The authors determined the risk classification of "probable", "possible," and "unlikely" risk for each cancer in a unique way that was based on three criteria.

The first criterion was the "pattern of meta-relative risk association." In order to be placed in either the "probable" or "possible" category, the risk of a certain cancer in a fire fighter had to be statistically significantly elevated *when averaged out* over all studies which examined that cancer. I have explained the many reasons it can be difficult to achieve a statistically significant result in occupational cancer studies. Even if several studies did show a statistically significant result, this can be diluted by averaging with other studies (which may not be as well designed) and the summary estimate would be lower and possibly not statistically significant.

The second criterion was "study type" and this step served to downgrade the risk classification of a cancer from the first step (for example, from "probable" to "possible") if the study type didn't meet certain criteria.

Finally, the third criterion, which was "heterogeneity," further downgraded the risk classification of a given cancer if a certain level of consistency among all studies was not achieved. It should be noted that it is very unlikely that an investigation of heterogeneity will produce useful findings unless there is a substantial number of studies, typically at least 10 in a meta-analysis. There were very few cancers that had more than 10 study results in this meta-analysis, making it more difficult to achieve statistical consistency.

Overall, this meta-analysis had extremely stringent criteria for classifying a cancer as "probable" or "possible" increased risk for fire fighters. The summary estimates that are listed on page 1199 of the article are very likely substantial underestimates. Given the limitations of this meta-analysis, *the finding that all cancers studied were increased in fire fighters is convincing evidence that supports the position that fire fighters suffer from cancer due to their fire fighting exposures.*

Another important point is while some meta-analysts assign "quality weights" to the component studies; this meta-analysis gave all studies the same weight by averaging the results. However, in some cases, incidence studies, dose-response studies, or studies comparing fire fighters to police were available and would have been more relevant in assessing the true relationship of fire fighting and those particular cancers.

For example, the authors gave bladder cancer a final designation of "unlikely" because the increased summary risk estimate was not statistically significant. However, it's important to know that bladder cancer has a 5 year survival rate of over 70%. If an individual with bladder cancer dies from another cause, bladder cancer may not be listed on the death certificate. For this reason incidence studies are a much better measure for risk of bladder cancer than mortality studies. There is an important incidence study of bladder cancer which compared fire fighters to the general population *and* police, and found statistically significant increased rates for fire fighters in both analyses. However, this study was simply averaged with the other studies, which were almost all mortality studies. In addition, two studies found statistically significant dose-response relationships between bladder cancer and fire fighting, but this was not taken into consideration.

According to expert epidemiologists, "the information achieved by the meta-analytical approach cannot transcend the quality of the individual studies." I will now go through individual studies that are pertinent to the understanding of the relationship between fire fighting and the following specific cancers.

Fire Fighter Cancers

Brain Cancer

Chemical exposures that are suspected causes of brain tumors include vinyl chloride, benzene, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), N-nitroso compounds, triazenes and hydrazines.

Epidemiologic studies consistently have found that brain cancer is strongly associated with fire fighting. Several studies have found a 2-3 times excess risk of death for fire fighters compared to the general population. Statistically significant elevated risk of brain cancer death in fire fighters ranges from double the risk in a study of almost 6,000 fire fighters from Toronto to 3.8 times the risk in 205 fire fighters from Hawaii.

Notably, a study by Demers and co-workers compared 4,546 fire fighters with police officers. The all-cause mortality for both fire fighters and police was lower than expected to a statistically significant degree, indicating a healthy worker effect for both groups. Brain cancer rates, however, showed statistically significant increases among fire fighters compared to US males with 2.07 times the risk. An elevated rate also appeared when fire fighters were compared to police with 1.63 times the risk. The increase among fire fighters compared to police is particularly important because police also had a higher rate of brain cancer than expected compared to US white men.

A study by Tornling and colleagues found dose response relationships between brain cancer incidence and increasing age, duration of employment, and years since hire, and between brain cancer mortality and increasing age, duration of employment, and estimated number of fires fought.

Skin Cancer

The most common risk factor for cancers of the skin is prolonged and intense exposure to sunlight. Occupational exposure to soot and tars, coke oven emissions, arsenic, and cutting oils have also been associated with increased risk. Substances containing carcinogenic agents such as PAHs and PCBs may be absorbed by the skin of exposed body areas, including the hands, arms, face and neck, and other sites when protective clothing is permeated. Contact with these substances can occur during fire knockdown and overhaul and during the cleaning of clothing or equipment.

Most epidemiologic studies have found an increased risk of skin cancer among fire fighters. Feuer and Rosenman found a statistically significant 2.7 (or almost three-fold) increase in skin cancer mortality for New Jersey fire fighters compared to the U.S. population. Risk among fire fighters clearly increased with duration of employment. Sama and colleagues found that fire fighters had a statistically significant 2.92 (or almost three times) increase in the risk of melanoma, compared to the state population, when incident cases reported to the Massachusetts Cancer Registry were examined. Baris and colleagues found a statistically significant 3.1 (or greater than three times) increased risk of skin cancer in a subgroup of fire fighters from Philadelphia.

Cancers of Blood and Lymphatic Systems

Leukemia and lymphoma are associated with environmental and occupational exposure to asbestos, benzene and 1,3 butadiene. The prevalence of benzene as a solvent, as a component of gasoline, and as a combustion product that forms during the burning of plastics and synthetics, and of 1,3 butadiene, a monomer found in tires and synthetic rubber products, guarantees that fire fighters will be exposed to gases released by these materials as they burn. Chemical exposures that have been associated with multiple myeloma include benzene and petroleum products.

Leukemia

The majority of epidemiologic studies have found that fire fighters are at increased risk of leukemia. For example, Feuer and Rosenman reported a statistically significant increased risk of 2.76 times for fire fighters compared to police officers in New Jersey and an almost two fold increase in mortality compared to the general population in New Jersey and in the United States (1.77 and 1.86). Similarly, Sama and colleagues found that fire fighters had 2.67 times (or almost three times) the risk of police officers when incident cases reported to the Massachusetts Cancer Registry were examined. A large 1994 study from NIOSH combining mortality data from 27 states reported an excess risk of 1.71 for fire fighters younger than 65. Some studies found that the highest risk occurred among those with the longest employment, suggesting a dose-response relationship.

Lymphoma

Several studies of fire fighters have evaluated this group of malignant diseases. Without exception, marked increases in risk were found. The study from the

Massachusetts Cancer Registry by Sama found a statistically significant risk of 3.27 times for fire fighters relative to police officers. Studies by Giles from Melbourne, Australia, and Aronson from Toronto, Canada, reported that fire fighters had twice the risk of non-Hodgkin's lymphoma compared to males in the general population.

Multiple Myeloma

Several studies have shown an increased risk of multiple myeloma among fire fighters. The analysis of a cohort of Seattle fire fighters by Heyer and colleagues reported a 2.25 (or greater than two fold) increase in risk of death from multiple myeloma for fire fighters. This risk increased to a statistically significant 9.89 for men with 30 years or more of fire combat duty. Howe and Burch combined the results of all cancer mortality studies of fire fighters available as of 1989 (including four unpublished reports) and concluded that there was a consistent evidence of a causal association between multiple myeloma and fire fighting. The meta-analysis by LeMasters and colleagues combined results from available studies through 2003 and found a statistically significant increase of 1.69 (or almost 70%) for death from multiple myeloma among fire fighters. An incidence study by Demers and colleagues reported a 1.90 (or almost two fold) increase in risk for fire fighters.

Some studies have analyzed lymphatic and hematopoietic cancers together as a group. A review by the Industrial Disease Standards Panel (IDSP) of Ontario found that a strong, statistically significant association between fire fighting and blood and lymph cancers was identified in six studies with increase in risk ranging from 2.05 to 9.89. Four analyses also identified a dose-response trend.

Cancers of the Digestive System

Several established occupational exposures increase the risk of cancer of the digestive system including asbestos, cutting and lubricating oils, dyes, solvents, and metallic compounds. In addition, fire fighters are exposed to soots and vinyl chloride, which are known human carcinogens that can cause cancer in the gastrointestinal system. Once cleared from the airways, inhaled particles and the carcinogens that adhere to them are transferred to the gastrointestinal tract by swallowing and exert their effect on the digestive epithelium. Some of the cancers that can result include:

Colon and Rectal Cancer

Of particular relevance to fire fighters are the higher than expected rates of colon and rectal cancer observed in workers with exposure to asbestos. Excess colon and rectal cancer has been found consistently in many studies of fire fighters.

Ma and colleagues found more than double the risk of colon cancer for African American fire fighters, which was statistically significant. Vena and Fiedler, who studied 1867 fire fighters from Buffalo, found a statistically significant increase of colon cancer risk for fire fighters that was 1.83 (or almost double) that of the general population. Further, they found that the risk increased to a statistically significant 4.71 (or almost 5 times) higher for fire fighters with the longest employment, suggesting a dose-response

trend. Demers also found that colon cancer risk increased with length of employment, supporting a dose-response relationship. In addition, Demers found that when compared to police, fire fighters had a 58% (or 1.58) excess risk of colon cancer. In a study of 7789 Philadelphia fire fighters, Baris and others found a significant increase in the risk of colon cancer which increased with over 20 years of employment.
Many studies have shown an increased risk of rectal cancer, with at least three studies showing a greater than two-fold risk. Orris and colleagues reported a statistically significant increase in rectal cancer among more than 3000 Chicago fire fighters. An analysis by Burnett and colleagues of mortality data for fire fighters from 27 states found a statistically significant excess risk of rectal cancer in fire fighters, which was almost double (1.86) for those under age 65.

Pancreatic Cancer

Several studies have found an increased risk of pancreatic cancer among fire fighters, ranging from slightly elevated to two times the risk. When studies reporting pancreatic cancer were combined in a report by Samet, the pooled estimate revealed a statistically significant increase in risk of pancreatic cancer for fire fighters. A Massachusetts study by Sama and colleagues found that the incidence of pancreatic cancer among fire fighters was more than three times the incidence in police officers.

Liver Cancer

Primary liver cancer is rare in the general population of the United States. Angiosarcoma of the liver has been associated with occupational and environmental exposures, including arsenic and vinyl chloride monomer from PVC. PVC can be assumed to be present at every structural fire site in recent years involving furniture, electrical wire, and cable insulation and water pipes, and at automobile fires. Furthermore, Hepatitis B and C, which are now beginning identified as fire fighter occupational illness, also leads to chronic liver diseases, including liver cancer.

The largest study of liver cancer and fire fighting, by Beaumont, found a two-fold excess of liver cancer mortality relative to the United States population among fire fighters in San Francisco.

Stomach Cancer

Most of the epidemiologic studies that addressed stomach cancer found a positive association with fire fighting. The results ranged from a small increase in risk to a two fold increase in risk. Tomling found that both stomach cancer incidence and mortality increased with duration of employment and number of fires fought. Stomach cancer incidence was statistically significantly elevated by almost three times (2.89) for those with more than 30 years employments and by over two and a half times (2.64) for those who fought more than 1,000 fires.

Esophageal Cancer

Some studies have found an increased risk of esophageal cancer among fire fighters. A study by Beaumont and colleagues of over 3,000 San Francisco fire fighters found that mortality from esophageal cancer occurred at twice the expected rate among fire fighters and this result was statistically significant.

Oral and Pharyngeal Cancer

Few studies have reported on oral and pharyngeal cancer, but generally rates have been increased in fire fighters. The meta-analysis of 32 studies by LeMasters and colleagues reported a summary risk estimate of 1.23 (or a 23% increase) based on available studies of buccal (oral) cavity and pharyngeal cancer, which was almost statistically significant.

Cancers of the Genitourinary System

Bladder Cancer

Occupational chemical exposures known to cause bladder cancer include several aromatic amines, solvents, benzidine, PAHs, coal tars and pitches, soot and oils. These substances are commonly encountered by fire fighters, particularly at fires in commercial establishments.

The majority of epidemiologic studies found that fire fighting was associated with increased risk of bladder cancer deaths. Sama and colleagues found a statistically significant 2.11 (or more than double) increase in risk for fire fighters compared with police. Demers also found an almost two fold (1.7) increase in bladder cancer risk for fire fighters relative to police. When compared to the general population, in a study of over 1800 fire fighters from Buffalo, Vena and Fiedler reported an almost three fold (2.86) increase in risk, which was statistically significant. Guidotti's study of more than 3300 Canadian fire fighters found a greater than threefold (3.16) increase in risk of bladder cancer compared to the general population. Further, both of these studies (Vena & Fiedler and Guidotti) found the highest rates in fire fighters with the longest duration of employment or greatest exposure index. These dose-response findings were statistically significant.

Kidney Cancer

Occupational exposures that have been implicated as risk factors for renal cell carcinoma include asbestos, PAHs, lead phosphate, dimethyl nitrosamine, coke oven emissions, and gasoline. This list clearly includes agents encountered in fire fighting.

Several studies have found an increased risk of kidney cancer in fire fighters. Guidotti's study of more than 3,300 Canadian fire fighters reported a greater than fourfold increase (4.14) in risk, which was statistically significant. This study also reported statistically significant highest risk of kidney cancer among those with the longest employment and those with the greatest exposure index. The study by Tornling also

found a dose-response trend, supporting this finding. The large study by Baris of more than 7,700 Philadelphia fire fighters found a statistically significant elevated kidney cancer risk that was 2.2 times (or more than twice) the rate of the general population for fire fighters employed over 20 years.

Prostate Cancer

High rates of prostate cancer have been reported among workers in a variety of occupations including chemists, farmers, loggers, textile workers, painters, and rubber industry workers. Fire fighters, specifically, are exposed to acrylonitrile and formaldehyde, both of which are considered probable causes of prostate cancer in humans.

Studies on prostate cancer have consistently found an increased risk in fire fighters. While the majority of studies found a 30-50% increase in risk, at least two studies have found a greater than double risk for fire fighters. Giles and colleagues found 2.09 times the rate of prostate cancer in Australian fire fighters compared to the general population. Grimes and colleagues found a statistically significant increase that was 2.61 times higher in fire fighters in Honolulu as compared to the general population.

Testicular Cancer

Fire fighters report that their groin area frequently becomes covered with "black soot." Soot is a human carcinogen that is known to cause cancer of the scrotum.

Only a few studies have specifically addressed testicular cancer in fire fighters. Aronson and colleagues found higher than expected mortality for men employed by the Toronto Fire Department during a 40 year period, with an overall 2.52 times increased risk for fire fighters. An incidence study by Stang and colleagues found fire fighters were four times more likely to get testicular cancer. The meta-analysis by LeMasters determined a statistically significant summary risk estimate of two times (2.02) increased risk for fire fighters based on available studies.

<u>Breast and Gynecologic Cancers</u>

There is little literature on the health effects of fire fighting in female fire fighters, even though increased risks with selected cancers among female workers have been reported in a number of professions (Ma). However, epidemiological data suggest that the potency of certain carcinogens may vary by gender and that women may be at greater risk. Also, the dose of carcinogen exposures per body weight is greater in women than men.

In a study conducted by the University of Maryland for the International Association of Fire Fighters, distinct associations of fire fighter exposures with breast cancer, gynecologic malignancies, and lymphomas in women fire fighters were found.

A recent study of female fire fighters in Florida by Ma and colleagues showed a statistically significant increase in cervical cancer that was more than 5 times (5.24)

higher than the general population. In fact, the "all site" cancer risk was statistically significantly increased in female fire fighters by 63% (1.63).

Lung Cancer

Fire fighters may be routinely exposed to many known or suspected lung carcinogens, including asbestos, arsenic, PAHs, vinyl chloride, and formaldehyde. Inhalation can occur during active fire combat as well as during the overhaul phase when protective breathing equipment is usually removed.

A few studies have found slightly increased rates of lung cancer in fire fighters and two studies found moderately increased rates, though the results were not statistically significant. Guidotti from Canada found a 1.42 (or 40%) increase in risk and Hansen from Denmark found a 1.63 (or 60%) increase in risk. When studies were "averaged" in the LeMasters meta-analysis, the summary risk estimate for lung cancer in fire fighters was 1.03, or just slightly higher than the general population. However, if the healthy worker effect and other study limitations could be adjusted for, this estimate could be significantly higher.

In summary, there is ample data to support the notion that fire fighters are exposed to carcinogens in their work environment.

The respiratory protection and other personal protective equipment used by fire fighters are of uncertain efficacy. Additionally, the protective equipment is often not used in overhaul and it carries carcinogens back to the fire station.

Apart from known carcinogens, fire fighters are potentially exposed to thousands of new synthetic chemicals being introduced into houses and commercial structures annually. The addition of these new chemicals adds to the uncertainty of risk that fire fighters face.

The data strongly suggest that fire fighters are at increased risk of developing and dying from cancer. Epidemiological studies demonstrate increased risk of several cancers that can be linked with carcinogenic exposures encountered by fire fighters in their work. But how do we know these exposures are directly linked to the increased rate of cancers in fire fighters? In epidemiology there are five key criteria to determine causation between an exposure or activity and development of disease.

- The first is temporality, meaning that there is a logical time frame with the cancer developing after exposure (typically, after years of fire fighting).

- Second is the strength of the association. The fact that fire fighters are 2-4 times more likely to get certain cancers (not just a slightly higher rate) is a strong argument that fire fighting causes these cancers. The strength of the association should be examined in the context of individual studies, not an average (meta-analysis) of studies since a "summary estimate" will significantly underestimate the strength of the association.

- Another criterion is consistency. This does not mean that every study should find elevated rates of cancer in fire fighters since this is not a reasonable expectation. It means results should be reproducible; and all cancers listed in my testimony have been found at elevated rates in multiple studies showing consistency.

- The strongest indication of causality is when a dose-response relationship is found. Many studies show that as length of employment as a fire fighter increases or the number of fires fought increases, the rates of cancer also increase even further. This is strong evidence that the act of (and exposure due to) fire fighting is the cause of the increased rates of cancer that many studies have found.

- Finally, there should be biological plausibility, meaning that there is a biological explanation for the relationship. The repeated exposure to known carcinogens provides the biological pathway for cancers to develop in fire fighters.

When these criteria are met it is generally accepted that the exposure causes the disease. In the case of fire fighting and cancer, all five criteria for causality are met and it would be generally accepted that fire fighting is responsible for the excess cases of cancers found in fire fighters.

The following is a recap of just five of the many examples that demonstrate this connection.

- Brain cancer can be caused by chemical exposures to vinyl chloride, benzene, polycyclic aromatic hydrocarbons (PAHs), and other compounds that fire fighters are exposed to. Fire fighters have 2-3 times higher risk of brain cancer and a dose-response relationship has been shown.

- Skin cancer can result from exposure to soot containing polycyclic aromatic hydrocarbons. Exposure measurements show that fire fighters are exposed to soot and polycyclic aromatic hydrocarbons. Some studies have found fire fighters to have a 3 times increased risk of skin cancer. There is also evidence for a dose-response relationship.

- Leukemia, lymphoma, and multiple myeloma (cancers of the blood and lymphatic system) can result from exposure to benzene, vinyl chloride, and other chemicals. Exposure measurements show that fire fighters are exposed to high concentrations of benzene in almost all fires. Fire fighters have been found to have a 2 times increased risk of blood and lymphatic cancers and there is evidence for a dose-response relationship.

- Digestive system cancers can result from exposure to polychlorinated biphenyl compounds (PCBs), asbestos, soots, and vinyl chloride. Exposure measurements show that fire fighters are exposed to these chemicals. Studies have found up to 2 times higher risk for fire fighters for a variety of gastrointestinal cancers. There is also evidence for a dose-response relationship.

- Genitourinary cancers can result from exposure to gasoline and polycyclic aromatic hydrocarbons in diesel exhaust. Fire fighters are known to be exposed to diesel exhaust. A 2-4 times increased risk of GU cancers in fire fighters has been documented with evidence for a dose-response relationship.

Costs of Fire Fighter Occupational Cancer Legislation

As part of our testimony today, I am pleased to provide you with specific information regarding the claims experience of States that currently have enacted similar presumptive cancer legislation.

As I previously summarized, I have been with the IAFF for over 33 years. Additionally, I have been personally involved in every state and provincial effort to obtain cancer compensation benefits for our members, whether through direct testimony or developing data and information to support these legislative actions. During these efforts over the past three decades, it has become quite obvious to me that the fiscal impact and other financial information provided by opponents to fire fighter cancer legislation might be incomplete, if not just rhetorical. While this was never surprising, it was clear that these individual never understood the true costs of these awards, especially since fire fighters throughout the United States are not universally covered by State Worker's Compensation Programs. Many states, by statute, allow fire departments to cover their employees for worker compensation benefits through the individual retirement systems. Hence, any claims made and or paid would not be recorded by the State Worker Compensation Bureau, but would be recorded by the individual retirement system. This would be the case in a number of other states that currently have cancer presumptive legislation. This data is more easily obtainable from those States that have statewide fire fighter pension systems, since the system collects and records the data. The only exception would be when the employer challenges the presumptive nature of the claim. In this case the State Worker Compensation program would record the claim.

Of course, we believe that it is reasonable to suggest that there would be some claims experience related to this type of coverage. Therefore, I have obtained and wish to share with you some numbers on fire fighter disabilities and cost experience from around the country.

In the State of California, which has the largest career sector of fire fighters in the country (30,000) and one of the largest volunteer sectors (33,000) the addition of cancer presumptive benefits has had "no impact" on the actuarial assumptions or funding of the state's fire fighter retirement system. An actuary for the California Public Employee Retirement System (CALPERS), the largest retirement system in the United States, has declared that the addition of presumptive cancer benefits for fire fighters has had "minimal effect" on the actuarial costs to the retirement system. In fact, the financial implications were so minimal, that CALPERS never had to perform an actuarial impact study after the implementation of the benefit by the California legislature. During the first three years of the California program, an average of 45 annuitants claims have been paid for cancer related disabilities. This is .07% of the active fire fighting workforce. The average claim for total cancer benefits was $14,075.00.

In 1984, the State of Illinois added cancer presumption language to its worker compensation statute. The City of Chicago employs over 50% of the 10,700 career fire fighters in the State of Illinois. During the 6 year period following the implementation of the statute the average number of beneficiaries receiving occupational disability benefits was 8.3% lower than the average number of beneficiaries in the six years prior to passage. Thus the inclusion of cancer benefits in 1984 has obviously had no impact on the funding requirements for the occupational disability benefits portion of the Chicago Firemen's Annuity and Benefit Fund.

In the first six years that they have had fire fighter cancer legislation in Oklahoma, they have had 22 claims paid statewide or 6% of the 378 disability claims paid. This averages to 4 claims per year for a rate of cancer claims of .03% at an average cost to the pension system of $10,409.00 per total cancer claim. There are 3,420 career fire fighters and 9,000 volunteer fire fighters.

In Nevada, there have been 3 cancer claims paid in the first four years after the legislation was enacted. None of these cases include lung cancer, which is covered under separate legislation. There are 1,790 career fire fighters and 2,200 volunteer fire fighters in Nevada. This averages to less than 1 claim per year for a rate of cancer claims of .02%.

In Rhode Island, which passed the legislation in 1986, there have been 6 claims paid in the first 8 years. This averages to less than 1 claim per year for a rate of cancer claims of .02%. There are 2,200 career fire fighters and 2,800 volunteer fire fighters in Rhode Island.

In the first four years that they have had cancer legislation in Massachusetts, there have been 34 cancer claims paid (15 disability and 19 death benefits). This averages to less than 9 claims per year at a rate of .03% of the active fire fighting workforce. There are 14,500 career fire fighters and 11,400 volunteer fire fighters in Massachusetts.

In Pennsylvania there 7,133 active (and retired active) career fire fighters. Using the assumption that Pennsylvania has a rate that does not exceed the average of the above States' cancer related disabilities -- .034% of the active fire fighting workforce -- the expected number of initial annual cancer claims for career fire fighters would be 3 career fire fighters. Pennsylvania has approximately 70,000 volunteer fire fighters. We would expect their longevity and exposures to be very different from career fire fighter, however even if we assumed their cancer experience would be the same, the annual cancer claims, based on the above assumptions, would be 24 volunteer fire fighters.

Based on the above information on actual experience, the cost per cancer claim for those states having presumptive occupational disease statutes is substantially less than the unsubstantiated figures asserted by other parties. The reason for this, unlike benefits for other occupations, is the higher mortality rate and significantly shorter life expectancy associated with fire fighting. Career fire fighters are dying too quickly from cancer and other occupational diseases, unfortunately producing a significant pension annuity saving for states and municipalities.

Conclusions on Fire Fighter Occupational Cancers

We believe that there is sufficient scientific and medical evidence to show that fire fighters suffer from cancer due to their exposures in performing tasks associated with fire fighting.

The compelling body of evidence of an epidemiological correlation between firefighting and cancer has been used by 32 states and 7 Canadian provinces to enact responsible occupational cancer presumptive laws. Again, these laws recognize that fire fighters work in a uniquely dangerous environment that exposes them to carcinogens that cannot be completely controlled by personal protective equipment and safety procedures, placing fire fighters at a substantially increased risk of developing certain cancers.

The attack on that evidence by the National League of Cities and their affiliates, including the Pennsylvania League of Cities and Municipalities, is not surprising since we have collectively fought them on every single piece of presumptive legislation that we have worked to pass on behalf of fire fighters.

The lobbying rhetoric is not credible and is reminiscent of the corrupt strategy of the tobacco industry, which denied for years that smoking causes lung disease and that nicotine is addictive. These claims are just as intellectually dishonest today as those cigarette company claims were decades ago.

Because of sound medical research, this is what we know – cigarette smoke significantly increases a person's chances at contracting lung disease, and the toxic smoke firefighters breathe as an inevitable result of their work places them at an increased risk for leukemia, multiple myeloma, non-Hodgkin's lymphoma, bladder cancer, and brain cancer when compared to other workers. Additional research indicates that firefighters are at increased risk for prostate, large intestine, and skin cancers, as well.

When Vermont Governor Jim Douglas (R) signed that state's fire fighter occupational disease legislation on May 22, 2007, he stated, "This new law will provide peace of mind to all of those who, in order to ensure our safety, willingly expose themselves to potentially carcinogenic agents in the line of duty." As Governor Douglas indicates, firefighters almost never know what they are exposed to when they respond to an emergency. Nevertheless, firefighters continue to save lives and reduce property damage without regard to the health hazards that they may face. We concur. Fire fighters across our great Nation are able to courageously enter buildings and fight

In fact, and contrary to the opposition's statements of those that oppose this legislation, fire fighters are exposed to carcinogens on a frequent basis during their daily work activities. This bill provides for a for a reputable presumption--that is the employer can demonstrate that the exposure did not occur in the line of duty--to compensate a fire fighter if an exposure leads to a disease. Just as a fire fighter would be compensated for injuries that occurred after falling through the roof of a burning structure, a fire fighter that has cancer from a job exposure would be compensated.

Fire fighters face the possibility of death or injury every time they respond to an alarm where they provide emergency assistance to the citizens of Pennsylvania. While risk may be part of the profession, fire fighter occupational cancers and infectious diseases should not be accepted as part of the job. We believe it is time for you to enact legislation to clearly indicate that occupational cancer and infectious diseases are occupationally related to fire fighting.

Thank you

State Presumptive Disability Laws

The following states/provinces have presumptive disability laws which recognize that fire fighters are at increased risk for certain illnesses. The laws create a presumption that the specified diseases are job related. Because the laws vary greatly from state to state and province to province and new legislation continually enacted, please refer to the IAFF's Presumptive Legislation website at http://www.iaff.org/hs/phi/ to review the specific state/provincial laws.

Code Part: WC = Workman's Comp, RS = Retirement / Pension System,
GP = General Provisions / other section

US States:

State	Heart Disease	Lung Disease	Cancer	Infectious Diseases	Code Part
Alabama	✓	✓	✓	✓	GP
Alaska	✓	✓	✓		WC
Arizona			✓	✓	WC
Arkansas			*Pending*		
California	✓		✓	✓	WC & RS
Colorado			✓	✓	WC
Connecticut	✓		✓	✓	GP
District of Columbia					
Delaware					
Florida	✓			✓	GP
Georgia	✓	✓			RS
Hawaii	✓	✓			RS
Idaho	✓	✓		✓	WC
Illinois	✓	✓	✓	✓	RS
Indiana	✓	✓	✓		GP
Iowa	✓	✓	✓	✓	RS
Kansas	✓	✓	✓		RS
Kentucky					
Louisiana	✓	✓	✓	✓	GP
Maine	✓	✓	✓	✓	WC
Maryland	✓	✓	✓		WC
Massachusetts	✓	✓	✓		RS
Michigan	✓	✓			WC
Minnesota	✓		✓	✓	WC
Mississippi					
Missouri	✓	✓	✓		RS
Montana	*Pending*	*Pending*	*Pending*	*Pending*	
Nebraska	✓	✓	✓	✓	GP
Nevada	✓	✓	✓	✓	GP
New Hampshire	✓	✓	✓		WC
New Jersey					
New Mexico	✓		✓	✓	WC
New York	✓	✓	✓	✓	RS
North Carolina					

State	Heart Disease	Lung Disease	Cancer	Infectious Disease	Code Part
North Dakota	✓	✓	✓	✓	GP
Ohio	✓	✓	*Pending*	*Pending*	WC
Oklahoma	✓	✓	✓	✓	RS
Oregon	✓	✓	✓		WC
Pennsylvania			*Pending*	✓	WC
Rhode Island		✓	✓	✓	GP
South Carolina	✓	✓			WC
South Dakota	✓	✓	✓		RS
Tennessee	✓	✓	✓		GP
Texas	✓	✓	✓	✓	GP
Utah	✓	✓		✓	WC
Vermont	✓		✓		WC
Virginia	✓	✓	✓	✓	WC
Washington	✓	✓	✓	✓	WC
West Virginia	✓	✓			WC
Wisconsin	✓	✓	✓	✓	GP
Wyoming					
Totals	**37**	**32**	**32**	**25**	**WC = 20 RS = 10 GP = 12**

Canadian Provinces:

Province	Heart Disease	Lung Disease	Cancer	Infectious Diseases	*Code Part
Alberta	✓		✓		WC
British Columbia		✓	✓	✓	WC
Manitoba	✓		✓		WC
New Brunswick	✓		✓		WC
Newfoundland					
Northwest Territory					
Nova Scotia			✓		WC
Ontario	✓		✓		GP
Prince Edward Island					
Quebec					
Saskatchewan	✓		✓		WC
Yukon					
Totals	**5**	**1**	**7**	**1**	**WC = 6 GP = 1**

US States' Notes

Alabama	Heart disease; hypertension; respiratory disease; disabling cancer which is reasonably linked to a known carcinogen; AIDS and Hepatitis.
Alaska	Cardiovascular events within 72 hours; respiratory disease; brain, malignant melanoma, leukemia, non-Hodgkin's lymphoma, bladder, ureter, kidney
Arizona	Brain, bladder, rectal, colon, lymphoma, leukemia, adenocarcinoma or mesothelioma; occupational disease
California	Heart trouble; exposed to a known carcinogen as defined by the IARC; blood-borne infectious disease, MRSA
Colorado	Brain, skin, digestive system, hematological system, or genitourinary system; Hepatitis C
Connecticut	Hypertension or heart disease
Florida	Heart disease or hypertension; hepatitis, meningococcal meningitis, or tuberculosis
Georgia	Heart disease; respiratory disease

Hawaii	Heart, lungs or respiratory system
Illinois	Heart disease, stroke or any disease of the lungs or respiratory tract; cancer which may be caused by exposure to heat, radiation or a known carcinogen as defined by the IARC; Tuberculosis
Indiana	Disease or impairment of the cardiovascular or respiratory system; cancer that is caused by a known carcinogen to which an individual is at risk for occupational exposure
Iowa	Heart disease or any disease of the lungs or respiratory tract; prostate cancer, primary brain cancer, breast cancer, ovarian cancer, cervical cancer, uterine cancer, malignant melanoma, leukemia, non-Hodgkin's lymphoma, bladder cancer, colorectal cancer, multiple myeloma, testicular cancer, and kidney cancer; HIV or AIDS, hepatitis, meningococcal meningitis, and mycobacterium tuberculosis
Kansas	Heart disease or disease of the lung or respiratory tract; type of cancer which may, in general, result from exposure to heat, radiation or a known carcinogen
Louisiana	Disease or infirmity of the heart or lungs; bladder, brain, colon, liver, pancreas, skin, kidney, gastrointestinal tract, leukemia, lymphoma, multiple myeloma; Hepatitis B or Hepatitis C; hearing loss
Maine	Cardiovascular injury, cardiovascular disease or pulmonary disease; hepatitis, meningococcal meningitis or tuberculosis; cancers of the kidney, prostate, breast, non-Hodgkin's lymphoma, testicular, colon, brain, bladder, leukemia or multiply myeloma
Maryland	Heart disease, hypertension, or lung disease; leukemia or pancreatic, prostate, rectal, or throat cancer that is caused by contact with a toxic substance
Massachusetts	Hypertension or heart disease; disease of the lungs or respiratory tract; cancer affecting the skin or the central nervous, lymphatic, digestive, hematological, urinary, skeletal, oral or prostate systems, lung or respiratory tract
Michigan	Respiratory and heart diseases or illnesses
Minnesota	Myocarditis, coronary sclerosis, pneumonia; cancer of a type caused by exposure to heat, radiation, or a known or suspected carcinogen, as defined by the IARC; infectious or communicable disease
Missouri	Lungs or respiratory tract, hypertension, or disease of the heart; cancer affecting the skin or the central nervous, lymphatic, digestive, hematological, urinary, skeletal, oral, breast, testicular, genitourinary, liver or prostate systems, as well as any condition of cancer which may result from exposure to heat or radiation or to a known or suspected carcinogen as determined by the IARC
Nebraska	Hypertension or heart or respiratory defect or disease; Cancer affecting the skin or the central nervous, lymphatic, digestive, hematological, urinary, skeletal, oral, or prostate systems; blood-borne infectious disease, tuberculosis, meningococcal meningitis, or MRSA
Nevada	Diseases of the heart; diseases of the lungs; exposed to a known carcinogen as defined by the IARC; contagious disease
New Hampshire	Heart or lung disease; cancer involved must be a type which may be caused by exposure to heat, radiation, or a known or suspected carcinogen as defined by the IARC (legislation never funded)
New Mexico	Heart injury or stroke suffered within 24 hours; brain, bladder, kidney, colorectal, non-Hodgkin's lymphoma, leukemia, ureter, testicular, breast, Hodgkin's lymphoma, leukemia, ureter, testicular, breast, esophageal, multiple myeloma; hepatitis, tuberculosis, diphtheria, meningococcal disease and MRSA
New York	Heart and lung (NYC only); cancer affecting the lymphatic, digestive, hematological, urinary, neurological, breast, reproductive, or prostate systems; HIV, tuberculosis or hepatitis
North Dakota	Hypertension, heart disease; lung or respiratory disease; cancer is one which arises due to exposure to smoke, fumes, or carcinogenic, poisonous, toxic, or chemical substances; bloodborne pathogen
Ohio	Cardiovascular, pulmonary, or respiratory diseases
Oklahoma	Heart disease, injury to the respiratory system; existence of any cancer which was not revealed by the physical examination passed by the member upon entry into the department; hepatitis, human immunodeficiency virus, meningitis and tuberculosis
Oregon	Disease of the lungs or respiratory tract, hypertension or cardiovascular renal disease; brain cancer, colon cancer, stomach cancer, testicular cancer, prostate cancer, multiple myeloma, non-Hodgkin's lymphoma, cancer of the throat or mouth, rectal cancer, breast cancer or leukemia
Pennsylvania	Hepatitis C
Rhode Island	Lungs or respiratory tract; disabling occupational cancer which develops as a result of the inhalation of noxious fumes or poisonous gases; infectious disease
South Carolina	Heart disease or respiratory disease
South Dakota	Hypertension, heart disease, or respiratory disease; impairment of health caused by cancer
Tennessee	Disease of the lungs, hypertension or heart disease; cancer resulting in hospitalization, medical treatment or any disability.
Texas	Myocardial infarction or stroke; disease or illness of the lungs or respiratory tract; cancer that may be caused by exposure to heat, smoke, radiation, or a known or suspected carcinogen as determined by the IARC; tuberculosis
Utah	Heart disease, lung disease, or respiratory tract condition; infectious disease as a result of exposure in the performance of duties
Vermont	Heart injury or heart disease; leukemia, lymphoma, or multiple myeloma, and cancers originating in the bladder, brain, colon, gastrointestinal tract, kidney, liver, pancreas, skin, or testicles
Virginia	Hypertension or heart disease; Respiratory diseases, Leukemia or pancreatic, prostate, rectal, throat, ovarian or breast; Hepatitis, meningococcal meningitis, tuberculosis or HIV
Washington	Heart problems, experienced within 72 hours; Respiratory disease; brain cancer, malignant melanoma, leukemia, non-Hodgkin's lymphoma, bladder cancer, ureter cancer, and kidney cancer; HIV/AIDS, all strains of hepatitis, meningococcal meningitis, or mycobacterium tuberculosis
West Virginia	Cardiovascular or pulmonary disease or sustained a cardiovascular injury
Wisconsin	Heart or respiratory impairment or disease; skin, breast, central nervous system or lymphatic, digestive, hematological, urinary, skeletal, oral or reproductive systems; infectious diseases includes the HIV, AIDS, tuberculosis, hepatitis A, hepatitis B, hepatitis C, hepatitis D, diphtheria, meningococcal meningitis, MRSA, and SARS.

Canadian Provinces' Notes:

Alberta	Myocardial infarction within 24 hours; Leukemia, brain, bladder, lung, ureter, kidney, colorectal, non-Hodgkin's Lymphoma

British Columbia	Asthma, Extrinsic allergic alveolitis, Acute upper respiratory inflammation, acute pharyngitis, acute laryngitis, acute tracheitis, acute bronchitis, acute pneumonitis, or acute pulmonary edema; Leukemia, bladder, lung, skin, liver; Staphylococcus aureus, Salmonella organisms, Hepatitis B, Tubercle bacillus
Manitoba	Injury to the heart within 24 hours; Leukemia, brain, bladder, lung, ureter, kidney, colorectal, non-Hodgkin's Lymphoma, testicular, esophageal
Nova Scotia	cancer or other disease that is prescribed by the Governor in Council by regulation
New Brunswick	IAFF is working to obtain specific language
Ontario	Heart injury while, or within 24 hours; Leukemia, brain, bladder, ureter, kidney, colorectal, non-Hodgkin's Lymphoma, esophageal
Saskatchewan	Injury to the heart that manifests within 24 hours; Leukemia, brain, bladder, lung, ureter, kidney, colorectal, non-Hodgkin's Lymphoma, testicular