Form Approved
OMB No. 0920-0891
Exp. Date 12/31/2021

Petition for the Addition of a New WTC-Related Health Condition for Coverage under the World Trade Center (WTC) Health Program

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

General Instructions

Any interested party may petition the WTC Program Administrator to add a condition to the List of WTC-Related Health Conditions (List) in 42 C.F.R. Part 88 (see http://www.cdc.gov/wtc/faq.html#hlthcond for the complete list).

Please use this form to petition the Administrator to add a health condition (any recognized medical condition requiring treatment or medication) to the List. Please use a separate form for each health condition.

Use of this petition *form* is voluntary, but any petition must include all of the information identified below, as required by 42 C.F.R. Part 88. Petitions that do not provide the required information will not be considered by the WTC Program Administrator. Additional supporting materials may be submitted and are encouraged.

Please note, however, the petition and all supporting materials submitted to the WTC Health Program are part of the public record and may be subject to public disclosure. Personal information will be redacted prior to public disclosure.

Please TYPE or PRINT all information clearly on the form.

If you need more space to provide the required information, please attach additional pages to this form.

Mail or email this form to: World Trade Center Health Program
395 E. Street, S.W., Suite 9200
Washington, D.C. 20201
WTC@cdc.gov

A. Interested Party Information

A1. Do you represent an organization (are you submitting this petition on behalf of an organization)?
☐ Yes (Go to A2) ☒ No (Go to A3)

A2. Organization Information:

Name of organization

A3. Name of Individual Petitioner or Organization Representative:

[redacted]

First name Last name

Position, if representative of organization

A4. Mailing Address:

[redacted]

Street

[redacted]

City State Zip code

A5. Telephone Number: [redacted]

A6. Email Address: [redacted]

B. Proposed WTC-Related Health Condition Information

B1. Health Condition Information:

cardiomyopathy, atrial fibrillation

Name of health condition you wish to petition to add to the List of covered conditions

If the name of the condition is not known, please provide a description of the condition or the name of the diagnosis provided by a physician or other healthcare provider.

C. Basis for Proposing that the Condition Be Added to the List of WTC-Related Health Conditions

C1. Describe the reasons the WTC Program Administrator should consider the addition of this health condition. Explain how the health condition you are proposing relates to the exposures that may have occurred from the September 11, 2001, terrorist attacks. Your explanation must include a medical basis for the relationship/association between the 9/11 exposure and the proposed health condition. The medical basis may be demonstrated by reference to a peer-reviewed, published, epidemiologic study about the health condition among 9/11 exposed populations or to clinical case reports of health conditions in WTC responders or survivors. First-hand accounts or anecdotal evidence may not be sufficient to establish medical basis. If you need more space, please attach additional pages to this form.

Peer reviewed studies have demonstrated that first responders' exposure to air pollution, radioactive materials, environmental toxic metals, and other particulate matter identified in the WTC disaster have been associated with increased risk of cardiac dysfunction, heart disease, and death.

The peer reviewed studies and/or citations are attached.

D. Signature of Petitioner

Sign your name below to indicate that you are petitioning the WTC Program Administrator to consider adding a hea[redacted]ted health conditions identified in 42 C.F.R. Part 88.

[redacted]

Signa[redacted] Date

Privacy Act Statement

In accordance with the Privacy Act of 1974, as amended (5 U.S.C. § 552a), you are hereby notified of the following:

Title I of the James Zadroga 9/11 Health and Compensation Act of 2010 amended the Public Health Service Act (PHS Act) to establish the World Trade Center (WTC) Health Program. Sections 3311, 3312, and 3321 of Title XXXIII of the PHS Act require that the WTC Program Administrator develop regulations to implement portions of the WTC Health Program established within the Department of Health and Human Services (HHS). The WTC Health Program is administered by the Director of the National Institute for Occupational Safety and Health (NIOSH), within the Centers for Disease Control and Prevention (CDC). The information provided with this form and supporting documentation will be used by the WTC Program Administrator to consider the disposition of a petitioned-for health condition. Disclosure of this information is voluntary.

Records containing information in identifiable form become part of an existing NIOSH system of records under the Privacy Act, 09-20-0147, "Occupational Health Epidemiological Studies and EEOICPA Program Records and WTC Health Program Records, HHS/CDC/NIOSH." These records are treated in a confidential manner, unless otherwise compelled by law.

Information submitted to WTC Health Program which may be considered "protected health information" pursuant to the Health Insurance Portability and Accountability Act of 1996 (HIPAA) (Pub. L. 104–191; 42 U.S.C. § 1320d) and the HIPAA Privacy, Security, Breach Notification, and Enforcement Rules (45 C.F.R. pts. 160, 162, and 164) will be maintained in accordance with all applicable laws.

NIOSH may disclose information in identifiable form only insofar as such disclosure is permitted pursuant to the HIPAA Privacy Rule; this may include disclosure to the WTC Health Program Scientific/Technical Advisory Committee (STAC), which may be asked to consider the petition and issue a recommendation to the WTC Program Administrator. Information in identifiable form will be redacted from submitted petition forms and supporting documentation that become a part of the public record (e.g. in conjunction with STAC consideration or a rulemaking).

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Respiratory and Cardiovascular Hospitalizations After the World Trade Center Disaster

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Cardiovascular disease in the World Trade Center Health Program General Responder Cohort

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AUTHOR CONTRIBUTIONS

Nancy L. Sloan and Susan L. Teitelbaum conceived and designed the work; Michael A. Crane, Denise J. Harrison, Benjamin J. Luft, Jacqueline M. Moline, and Iris G. Udasin participated in data acquisition, Nancy L. Sloan conducted the analysis, and Nancy L. Sloan, Moshe Z. Shapiro, Ahmad Sabra, Christopher R. Dasaro, and Susan L. Teitelbaum participated in the interpretation of data; all authors participated in drafting or critically revising the work for important intellectual content in its final approval and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest.

DISCLOSURE BY AJIM EDITOR OF RECORD

Paul A. Landsbergis declares that he has no conflict of interest in the review and publication decision regarding this article.

DATA AVAILABILITY STATEMENT

The relevant data are available within the manuscript. The access to the study's de-identified data and analytic code requires IRB approval, and submission and approval of the WTC Data Center Data Use Agreement, and Data Request Form (including an attestation), which may be requested of the corresponding author.

ETHICS APPROVAL AND INFORMED CONSENT

This study has been conducted in accordance with the principles of the Declaration of Helsinki 1975, as revised in 2013, and complies with the ethical standards of the relevant national and institutional committees on human experimentation. The work was performed and the WTCHP research has been approved by the Institutional Review Boards (IRBs) of the Icahn School of Medicine at Mount Sinai (formerly Mount Sinai School of Medicine), New York, NY and the program's other clinical sites, including New York University Langone Medical Center, New York University School of Medicine, New York, NY; Department of Medicine, Stony Brook University Medical Center, Stony Brook, NY; Department of Occupational Medicine, Epidemiology and Prevention, Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, Hempstead, NY; and Environmental and Occupational Health Sciences Institute, Rutgers University, Piscataway, NJ.

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Abstract

Background: Over 90,000 rescue and recovery responders to the September 2001 World Trade Center (WTC) attacks were exposed to toxic materials that can impair cardiac function and increase cardiovascular disease (CVD) risk. We examined WTC-related exposures association with annual and cumulative CVD incidence and risk over 17 years in the WTC Health Program (HP) General Responder Cohort (GRC).

Methods: Post 9/11 first occurrence of CVD was assessed in 37,725 responders from self-reported physician diagnosis of, or current treatment for, coronary artery disease, myocardial infarction, stroke and/or congestive heart failure from WTCHP GRC monitoring visits. Kaplan–Meier estimates of CVD incidence used the generalized Wilcoxon test statistic to account for censored data. Cox proportional hazards regression analyses estimated the CVD hazard ratio associated with 9/11/2001 arrival in responders with and without dust cloud exposure, compared with arrival on or after 9/12/2001. Additional analyses adjusted for comorbidities.

Results: To date, 6.3% reported new CVD. In covariate-adjusted analyses, men's CVD 9/11/2001 arrival risks were 1.40 (95% confidence interval [CI] = 1.26, 1.56) and 1.43 (95% CI = 1.29, 1.58) and women's were 2.16 (95% CI = 1.49, 3.11) and 1.59 (95% CI = 1.11, 2.27) with and without dust cloud exposure, respectively. Protective service employment on 9/11 had higher CVD risk.

Conclusions: WTCHP GRC members with 9/11/2001 exposures had substantially higher CVD risk than those initiating work afterward, consistent with observations among WTC-exposed New York City firefighters. Women's risk was greater than that of men's. GRC-elevated CVD risk may also be occurring at a younger age than in the general population.

Keywords

cardiovascular disease; environmental exposure; occupation; responder/recovery worker; World Trade Center

1 | INTRODUCTION

Heart disease continues to be the leading cause of death worldwide,[1–3] only recently surpassed by cancer among middle-aged people in the United States and other high-income countries.[4] Major risk factors include older age, cigarette smoking, diet and obesity, high blood pressure, high cholesterol, diabetes, and work psychosocial stressors.[1–3,5–7] In some studies,[8–14] exposure to air pollution, radioactive materials, some environmental toxic metals, and other particulate matter have been associated with cardiac function damage and increased risk of heart disease and death. Others have found null or negative associations of such exposures with cardiovascular risk and disease, and this lack of association is sometimes attributed to a healthy worker effect.[15–18]

In response to the 9/11/2001 terrorist attacks on the World Trade Center (WTC), more than 90,000 people participated in rescue and recovery efforts and in debris cleanup.[19,20] These responders were exposed to a complex mix of toxins, including burning jet fuel from the hijacked airplanes and hazardous particulate matter from the collapse of the WTC towers.[21,22] In addition to physiologic stress, many responders experienced work psychosocial stressors that can initiate or exacerbate unhealthy chronic biologic processes and behavioral responses and influence CVD risk.[7,23] These exposures have been associated with increased short-term and persistent risk of morbidity, including respiratory disease, gastro–esophageal reflux disorder, posttraumatic stress disorder (PTSD), and certain cancers.[20,24–26]

An analysis of WTC Registry responders and non-responders through 2009 who had high levels of exposure were found to have death rates from heart disease that were more than twice the rates observed in those with less exposure to the environmental site contaminants.[27] By 2014, female WTC Registry members who had intense dust cloud exposure had a 1.28 times higher risk of physician-diagnosed heart disease than less exposed women (95% confidence interval [CI] = 1.02, 1.61), whereas men with intense dust cloud exposure had a 1.14 (95% CI = 0.97, 1.34) times higher rate of physician-diagnosed heart disease than men with less exposure.[28] In an early 4-year responder follow-up, higher risks for PTSD-mediated heart attack and stroke were observed in surviving responders involved in debris cleanup.[29] For the earliest arriving firefighter responders, who were exposed to the toxic dust cloud on 9/11/2001, a 1.44 (95% CI = 1.09, 1.90) higher risk of primary cardiovascular disease (CVD) was observed compared with responders who had less toxic exposure arriving on or after 9/12/2001.[30]

Using 17 years of follow-up of the WTC Health Program's (HP) large, diverse General Responder Cohort (GRC) of both men and women, this study's objective was to examine the annual and cumulative incidence of CVD. We also investigated the association of CVD risks with 9/11/2001 exposures, based on whether or not the responders reported being exposed to the dust cloud, compared with those arriving on or after 9/12/2001.

2 | MATERIALS AND METHODS

This study was conducted in adherence to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidance.

2.1 | Study population

The WTCHP GRC is an open (continuing to enroll) cohort. Through March 31, 2019, 32,539 men and 5186 women general responders who reported whether they had or had not been diagnosed with or treated for CVD have been followed.

2.2 | WTCHP GRC eligibility criteria and recruitment

The WTCHP GRC recruited its participants using publicity and broad outreach through various mechanisms (volunteers, community and labor organizations; governmental and legislative agencies and media). Men and women eligible for the WTCHP GRC include those who worked or volunteered at the tower sites in lower Manhattan, at the Staten Island landfill or on certain barge-loading piers for at least 4 h between September 11 and 14, 2001, who worked at least 24 h between September 11 and 30, 2001 or at least 80 h between September 11 and July 31, 2002. The WTCHP GRC comprises rescue and recovery workers and volunteers who were enrolled in the HP on or after July 16, 2002.[31] This includes staff who had contact with WTC-related human remains from the Office of the Chief Medical Examiner between September 11, 2001 and July 31, 2002, and those from the Port Authority Trans-Hudson Corporation who cleaned tunnels for at least 24 h between February 1, 2002 and July 1, 2002.[32] Other responders who may not be included in the WTCHP GRC are employees of the Fire Department of the City of New York (FDNY), individuals participating in the New York City Department of Health Registry (~23% also participate in the WTCHP GRC), Pentagon and Shanksville responders, Nationwide Provider Network members (not WTCHP GRC participants), and others participating and not participating in similar programs.[20,28]

2.3 | Data collection

Responders voluntarily participating in the program were first interviewed (beginning July 2002) to assess their socio-demographic characteristics and WTC-related exposures, after which they received a comprehensive, standardized physical and mental health examination. Program participants were subsequently interviewed and examined every 12–18 months, depending upon their presentation for program visits. The data were collected, using a standardized clinical interview, from each WTCHP GRC participant at each monitoring visit from July 16, 2002. Data available at the time of analysis, through March 31, 2019, were analyzed.

2.4 | Data source

CVD was defined as a responder's self-report of a physician's first diagnosis or responders' reporting that they were under current treatment for first-time coronary artery disease (CAD), myocardial infarction (MI), stroke, or congestive heart failure (CHF). Only monitoring visit reports of diagnosis or treatment on or after 9/11/2001 are classified as having post 9/11 CVD. Responders were classified as not having post 9/11 CVD if they did not report a physician diagnosis or treatment for any of these conditions. Additionally, reported heart murmur and other heart diseases were not included in the definition of CVD. Individuals solely reporting heart murmur or other heart diseases were retained as noncases in the analysis.

Exposure was categorized by self-report as follows: first arrival at the WTC site on 9/11/2001 reporting exposure to the dust cloud (very high exposure); first arrival on 9/11/2001 not reporting exposure to the dust cloud (high exposure); and first arrival on or after 9/12/2001 (low/intermediate exposure).

2.5 | Statistical analyses

We conducted analyses to compare the effects of more intense (very high and high) with less intense (low/intermediate) WTCHP GRC exposures. The conditional cumulative incidence of post 9/11 first-time CVD was assessed by Kaplan–Meier analyses, using the generalized Wilcoxon test statistic to account for censored data. Responders with pre-existing (before 9/11/2001) CVD were excluded from this evaluation to ensure any association with WTC exposure was not attributable to prior CVD history ($n = 302$). Age-to-diagnosis, which simultaneously adjusts for time since 9/11 and age at 9/11, was estimated as the age at the reported earliest post 9/11 date of a physician's diagnosis of CAD, MI, stroke, or CHF and 9/11/2001.[33] As the analysis includes a maximum of 17 years' follow-up, results were censored at the most recent follow-up fewer than 17 years before March 31, 2019. The annual incidence was calculated as each year's difference in cumulative incidence. Cox proportional hazards regression analyses were conducted to estimate the risk of CVD associated with high and very high exposure compared with low/intermediate exposure. As men constitute the large majority of WTCHP GRC and their results were almost identical to the total sample results, adjusted Cox proportional hazards regression analyses were conducted stratified by responders' sex. To determine the influence of potential confounders, covariate-adjusted analyses included race/ethnicity. Additional Cox proportional hazards models included comorbidities and risk factors that could have been influenced by their WTC exposure and are in the causal pathway for CVD. These variables include self-reported lifetime cigarette smoking, cholesterol, hypertension, and diabetes status, and measured initial visit body mass index (BMI). Cigarette smoking was classified as never (the referent value), former, or current smoker. Height and weight were measured at each visit. Initial visit BMI was calculated as $703 \times$ measured weight in pounds/(measured height in inches)^2 and then categorized as <25 (normal), 25 to <30 (overweight), ≥30 (obese). At each monitoring visit, responders reported whether they had ever been diagnosed with high cholesterol, hypertension, and diabetes. Associations with these comorbidities, (not having high cholesterol, hypertension, or diabetes, respectively, are the referent groups), were examined in the comorbidity-adjusted analyses as risk factors in the causal pathway of WTC exposure and CVD. Because the responders' reported dates were frequently missing for the first physician diagnosis of high cholesterol, high blood pressure, and/or diabetes; their categories are reported as "ever or never diagnosed" and do not reflect cases solely diagnosed before the first CVD date of diagnosis.

Being employed in high-stress occupations has been associated as a CVD risk factor.[34,35] As over half of the WTCHP GRC were engaged in the high-stress occupation of protective services on 9/11, sensitivity analyses were conducted to evaluate whether protective services occupation on 9/11/2001 influenced the observed results. Cross-tabulations were conducted to determine whether the proportion of those actively engaged in protective services varied by WTC 9/11 arrival time with and without self-reported exposure to the dust cloud, and dichotomous variables were added to the Cox regressions to assess their associations with CVD risk and how 9/11 protective services occupation influenced the effects of WTC 9/11 exposures. Further analyses were conducted by including, in the statistical models, diagnosis of cancer since 9/11/2001, and unknown cancer status before CVD as proxies for having potentially received cancer treatment that may have increased the risk of CVD.[36] To assess recall bias, we also examined whether the age of reported CVD diagnosis was influenced by when the WTCHP GRC participants enrolled in the health monitoring program.

All variables calculated using dates imputed missing days as each month's midpoint, missing month as June, and missing year only when information from a consecutive visit could clarify the missing year. Missing covariate values were recoded to the sex-specific mode value in the multivariate analyses, which was the referent value of zero except for high cholesterol, BMI, and occupation on 9/11. For consistency with the other comorbidities (and because nearly 50% of men and women reported ever being diagnosed or treated for high cholesterol), missing data for high cholesterol was set to the referent value of zero for both sexes. Missing BMI was set to obese for men and overweight for women. For the sensitivity analyses for protective services, missing occupation was conservatively coded as the referent value of 'not protective services.'

The Cox proportional hazards assumption was assessed via Schoenfeld residuals. No violations were found for the primary factors of interest except in the comorbidity-adjusted sensitivity analyses for protective services. Sensitivity analysis adding log survival time interactions for the violating covariates and comorbidities was used to address the variables where proportionality was violated. Analyses included all WTCHP GRC who provided written voluntary consent for research data aggregation and who presented for at least one monitoring visit. In addition to the previously mentioned exclusions, responders with unknown arrival date on the WTC site or unknown dust cloud exposure ($n = 1005$); and responders with unknown sex ($n = 1$) or age ($n = 1$) were also excluded from the analysis.

Descriptive statistics were conducted using SPSS 24.0 (IBM Corp.). The Cox proportional hazards analyses and Schoenfeld tests of the proportional hazards models were conducted in Stata (StataCorp. 2019, Stata Statistical Software: Release 16; StataCorp LLC.).

3 | RESULTS

This analysis includes 37,725 WTCHP GRC, 86% male (Table 1). Most were between 30 and 59 years old with a median age of 37 (ranging from 19 to 80) years old on 9/11/2001. Most of the responders were married, white non-Hispanic, never smoked cigarettes, and employed in protective services on 9/11/2001. Half the participants enrolled in the WTCHP GRC before 2008, another 25% enrolled between 2008 and 2013, and 25% enrolled after 2013. Those enrolled before 2008 have participated in 6 ± 4 health monitoring visits (range, 1–14), and those enrolled between 2008 and 2013 have participated in 5 ± 3 (range, 1–11) visits. Those enrolled in 2014 and after have participated in 2 ± 2 health monitoring visits. Twenty-one percent have made only one health monitoring visit, 53% of whom enrolled in 2014 or after, 32% of whom enrolled before 2008, and 14% of whom enrolled between 2008 and 2013. Nineteen percent first arrived at the WTC site on 9/11/2001 and reported being exposed to the toxic dust cloud (men: 19.5%, women: 16.2%); 25.6% arrived on 9/11/2001 and reported that they were not exposed to the dust cloud (men: 26.2%; women: 22.3%); and 55.3% (men: 54.3%; women: 61.5%) arrived on or after 9/12/2001.

To date, 6.3% ($n = 2385$) of WTCHP GRC reported having been diagnosed with or treated for first-time CVD (Table 2). More than half of the reported CVD cases are CAD (3.8%), followed by stroke (1.8%), MI (1.3%), and CHF (0.7%). While male and female responders' prevalence of post 9/11 stroke and CHF are similar, men's prevalence of CAD and MI are nearly three times that of women. The mean age at CVD diagnosis was 52.4 ± 9.0 years, with an average of 7.9 ± 4.7 years since 9/11/2001 when diagnosed. The age and years since 9/11/2001 at diagnosis did not vary by sex; there was also little variation in age at diagnosis across CVD conditions.

Some CVD comorbidity was observed. A considerable percentage diagnosed with CAD also reported heart attacks (15.3%), stroke (5.1%), and CHF (2.8%; Figure 1); few (n = 36, 1.5%) had reported three or more CVD conditions.

Within 5 years of 9/11/2001, the annual CVD incidence increased from <1% in all responders by 0.7% in the general responders who initiated work on or after 9/12, compared with 1.2% among those presenting for work on 9/11/2001 (Table 3, Figures 2 and 3). Eight years after 9/11/2001, the mean time for the cohort's CVD diagnoses, the annual incidence in those arriving on or after 9/12/2001 had quadrupled to 2.0% compared with 2.5% and 3.1%, among those arriving on 9/11/2001 who were not and who were exposed to the dust cloud, respectively. The cumulative incidence across these exposure groups remained similar until the fourth year after 9/11/2001. By eight years after 9/11/2001, the cumulative CVD incidence was 12.5% among those arriving on 9/11/2001 not exposed to the dust cloud and 13.5% among those exposed to the dust cloud, compared with 9.6% in those arriving on or after 9/12. The CVD incidence patterns were virtually identical among men and women (data not shown).

Men's covariate-adjusted risk of CVD was substantially higher in 9/11/2001 arrivals not exposed to the dust cloud (HR_{cov} = 1.43; 95% CI = 1.29, 1.58) and in those exposed to the dust cloud (HR_{cov} = 1.40; 95% CI = 1.26, 1.56) than that of general responders arriving on or after 9/12/2001 (Table 4). Additional adjustment for comorbidities reduced men's overall risk compared with those arriving later to HR_{cov_comorb} = 1.33; 95% CI = 1.20, 1.47 in those without dust cloud exposure and hazard ratio (HR) = 1.29; 95% CI = 1.16, 1.44 in those with dust cloud exposure. There was no substantial violation of the proportional hazards assumptions in the covariate-adjusted model; however, some violation of the assumptions was observed for smoking, cholesterol, and BMI status in the model adjusted for comorbidities. The sensitivity analysis adding log survival time interactions for the violating variables (BMI, smoking, and cholesterol status) produced nearly identical risk estimates as the non-timed dependent model (HR_{cov_comorb} = 1.33; 95% CI = 1.20, 1.47 in those without dust cloud exposure and HR_{cov_comorb} = 1.28; 95% CI = 1.15, 1.43 in those with dust cloud exposure).

Women's covariate-adjusted risks were HR_{cov_comorb} = 1.59; 95% CI = 1.11, 2.27 in responders arriving on 9/11/2001 without dust cloud exposure and HR_{cov_comorb} = 2.16; 95% CI = 1.39, 3.11 in responders arriving on 9/11/2001 with dust cloud exposure, compared with women arriving on or after 9/12. When adjusted for comorbidities, women arriving on 9/11/2001 without dust cloud exposure had HR_{cov_comorb} = 1.49; 95% CI = 1.04, 2.13 and HR_{cov_comorb} = 2.17; 95% CI = 1.34, 3.14 with dust cloud exposure compared with women arriving on or after 9/12/2001. No violation of the proportional hazards assumptions was observed in the women's analyses.

Except for Hispanic men who had lower CVD risk than white non-Hispanic responders in the covariate-adjusted model, the hazards ratios for race/ethnicity all had 95% CIs that included the null value of 1. Current cigarette smokers had higher CVD risk in men and women, but former smokers only had higher CVD risk in women than lifetime nonsmokers. Overweight and obesity at a responder's first visit was associated with CVD risk but only significantly so in men. Responders who had ever been diagnosed with diabetes, high blood pressure, and/or high cholesterol had significantly higher CVD risk than those never diagnosed with those conditions, except in women with high blood pressure. The associations with smoking, cholesterol, and BMI status should not be over-interpreted as violations of the proportional hazards assumptions were observed for these indices. In additional multivariate analyses (data not shown), having been diagnosed with cancer before CVD was highly associated with CVD.

WTCHP GRC protective services occupational status on 9/11/2001 was significantly different among those arriving on or after 9/12/2001 than those responding on 9/11/2001. Over 65% of men and 71% of women 9/11 arrivals (in those exposed and not exposed to the dust cloud) were engaged in protective services employment on 9/11. Only 42% of men and 44% of women arriving on or after 9/12 were engaged in protective services on 9/11. Therefore, sensitivity analyses adjusting for protective services occupation on 9/11 were conducted. Compared with all other responders, active occupation in protective services on 9/11/2001 was associated with increased risk of CVD in men (HR_{cov} = 2.07; 95% CI = 1.89, 2.27) and women (HR_{cov} = 2.81; 95% CI = 2.02, 3.90; Table 5). Higher protective services HRs were observed in the comorbidity-adjusted models. Adjustment for 9/11/2001 protective services occupational status reduced the CVD risks associated with 9/11/2001 arrival, particularly in the comorbidity-adjusted analysis of men reporting dust cloud exposure and in both the covariate and covariate/comorbidity-adjusted analysis of women not reporting dust cloud exposure, to risks with confidence limits crossing the null value of 1. Sensitivity analyses adding log survival time interactions for the violating covariates and comorbidities produced similar results. Analyses of the total sample (both sexes) combining arrivals on 9/11 with and without dust cloud exposure that were adjusted for 9/11 protective services occupation had a 9/11 arrival HR_{cov} = 1.21; 95% CI = 1.12, 1.32 in the covariate-adjusted model and HR_{cov_comorb} = 1.12; 95% CI = 1.03, 1.22 (with no violations for the primary factor of interest).

Because those enrolling later in the health monitoring program had fewer visits, we assessed whether age at diagnosis varied by enrollment period. Men who enrolled in the first 5 years of the monitoring program (50% of men's CVD cases) reported they were 53 ± 9 years old at their first CVD diagnosis, as did the men who enrolled in the last 4 years of the program (26% of men's CVD cases). Men who enrolled between 2008 and 2013 reported being a year and a half younger (±9 years) at their first CVD diagnosis. Women who enrolled in the first 5 years (before 2007, 58% of women's cases) and the following 5 years (2008–2013, 25% of women's cases) reported being 51 ± 10 years old at their first CVD diagnosis, whereas the 17% (n = 33) enrolled in the last 4 years reported they were 56 ± 13 years old at their first diagnosis of CVD.

4 | DISCUSSION

Globally, heart disease remains the leading cause of death.[1–3] In some studies, exposure to air pollution, radioactive materials, environmental toxic metals, and other particulate matter identified in the WTC disaster have been associated with increased risk of cardiac dysfunction, heart disease, and death. With 17 years' follow-up of a large cohort of WTC general responders, we observed substantially higher risks of CVD associated with initiating work on 9/11/2001 compared with responders who initiated work on or after 9/12.

This study found 9/11/2001 arrival was associated with elevated CVD risk in men and women compared with responders arriving on or after 9/12. Despite the somewhat different definitions of CVD used in our and in the FDNY analyses, the analysis of men's risk adjusted for nearly identical covariates and comorbidities demonstrate WTC 9/11 exposures are associated with large, statistically significant increased hazards for CVD, consistent with observations in the all-male sample of firefighter responders.[30] This consistency with the male FDNY results is notable, as their criteria for CVD was based upon the theoretically more accurate (than a responder's recall, particularly regarding event dates) physicians' diagnosis from electronic medical records.[30] Except for women presenting on 9/11/2001 without dust cloud exposure, the WTCHP GRC lower 95% CIs were all well above 1. The association with dust cloud exposure was less clear among men, who comprise the vast majority of the sample, whereas a clear elevated CVD risk was observed with arrival on 9/11 in the total sample.

Distinct from the FDNY responders, the WTCHP GRC includes a substantial female cohort. With its large sample of female general responders, the WTCHP GRC also identified large risk differences associated with 9/11/2001 compared with later exposure among female responders, even though there was a 45% lower post 9/11/2001 CVD prevalence among female than male responders. The risk in women associated with 9/11/2001 exposure was much greater than the elevated men's 9/11/2001 CVD risk compared with those presenting on or after 9/12/2001. This may indicate that the female general responders have less chronic or previous hazardous environmental exposures and, therefore, have a greater reaction to the toxic 9/11/2001 exposures than do men. Alternatively, because the cohort of female general responders is so much smaller than that of men, they may simply represent a less diverse group of responders than men. Women's hazards ratios had wider 95% CIs than that of men's, reflecting the smaller sample size for female responders.

Over half of the WTCHP GRC were employed in protective services (high-stress occupations) which, among other occupations (such as transportation and moving materials) have been identified as CVD risk factors.[34,35] Employment in protective services was associated with CVD, and specifically attenuated the risks of men with dust cloud exposure risks to the extent that their confidence limits crossed the value of 1 (no effect). Conversely, adjustment for 9/11 occupation in protective services obviated the excess CVD risk in women without dust cloud exposure. These observations suggest that the risks associated with protective services employment share common characteristics with 9/11 arrival, although there was no significant interaction between arrival time and 9/11 protective services occupation (data not shown). Still, when all 9/11 arrivals for the total sample (both sexes, whether exposed to the dust cloud or not) were combined, adjustment for being engaged in protective services on 9/11 reduced but did not nullify the excess CVD risk in the WTCHP GRC.

Known risk factors for CVD include age, poor diet, cigarette smoking, high blood pressure, high cholesterol, diabetes, and work psychosocial stressors.[2,3,5–7,23,34,35] Analyses adjusted and stratified for these risk factors were conducted. Consistent with the literature,[1–3] being Hispanic was associated with lower men's CVD risk in the covariate-adjusted analyses, but not in the remaining analyses where the confidence limits for race/ethnicity risk crossed the null value of 1. The associations of measured BMI at the first health monitoring visit and smoking were generally in the expected direction. Although the observed associations with the participants' reports of ever being diagnosed with high blood pressure, high cholesterol, and diabetes were in the expected direction, they are flawed in this evaluation because dates of their initial diagnoses were often missing, offering no assurance that these conditions occurred before the self-reported CVD diagnosis. As violations of the proportional hazards assumptions were observed for smoking, cholesterol, and BMI status, their associations with CVD risk in this study should not be over-interpreted. Adjustment for comorbidities and risk factors, including smoking, overweight and obesity, hypertension, diabetes, and high cholesterol, all of which may be partially caused or exacerbated by the 9/11/2001 exposures, and are in the causal pathway for CVD, may overcontrol and underestimate the 9/11 exposure effects.

The WTCHP GRC CVD risk may also be occurring much earlier than observed in the general population. The WTCHP GRC's average age at heart attack was 52 years old compared with the US general population with an average age at heart attack of 64 in men and 70 in women.[2,3,5,6,37] In the WTCHP GRC, only 4% of CVD occurred at or after age 65. Among the WTCHP GRC, 16% of men's heart attacks occurred before age 45 compared with 4%–10% of men's heart attacks in the general population.[38]

This investigation used robust statistical methods to assess the association of WTCHP GRC environmental exposure and CVD risk among a large cohort of men and women. Categorizing those with a reported heart murmur and other heart diseases as noncases may have attenuated the observed results. Similarly, if responders, particularly those engaged in physically demanding work, are relatively healthy and thus at lower risk for adverse health outcomes, this healthy worker effect may have biased our observed effects of 9/11 exposure toward the null. The results also depend upon the accuracy of participant reports of diagnosis and/or treatment for CVD, in this study. The CVD conditions assessed in this study are major life events. Regardless, the validity of self-reported CVD is imperfect. Correct case identification of MI has been found to be better (ranging from 50% to 98%) than stoke (ranging from 38% to 81%) in various populations.[39–41] While overreporting is more common than underreporting, bidirectional misreporting has been observed. The lack of lifetime primary care medical records, against which to validate the health monitoring program's self-reported CVD diagnoses, is a study limitation. The consistency of men's elevated CVD risk with that of the FDNY suggests that the men's self-reported physician's diagnosis of CVD was not unduly influenced by recall bias. Relatively few women reporting CVD enrolled in the last 4 years of the monitoring program, and the influence of any women's recall bias may be limited. The results are consistent with the extant literature in both the direction and magnitude of 9/11/2001 exposure effects and its higher risk of heart disease.[27–30] While the FDNY data were limited to male responders, Jordan et al. also found an increased risk of heart disease in female responders with intense dust cloud exposure compared with less exposed women.[28,30,42]

As a voluntary program, the extent to which WTCHP GRC participants represent all WTC first responders is unknown. Compared with the FDNY program, the WTCHP GRC provides services to and obtains information on a more diverse group of general responders. While continuing to enroll responders, the WTCHP GRC has experienced diminishing numbers of responders presenting for monitoring visits over time. The considerable decline in and the possible self-selection of responders presenting for HP monitoring visits among those who are symptomatic or ill potentially explains the increasingly high cumulative incidence over time.

In summary, this evaluation observed a substantially higher risk of CVD associated with WTCHP GRC members initiating work on 9/11/2001 compared with those who initiated work on or after 9/12, with women's risk greater than that of men's. Elevated CVD risk may also be occurring at a younger age in responders than in the general population. Active engagement in protective services on 9/11/2001 is associated with increased CVD risk. The study findings reinforce the need for broadening public awareness about the associations of WTC exposure and CVD and for continued monitoring of the WTCHP GRC to identify and treat heart disease and promote CVD prevention (e.g., through diet, exercise, weight control, smoking cessation, work stressor reduction, etc.) as a means to minimize its life-altering and life-threatening consequences.

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REFERENCES

1. Benjamin EJ, Muntner P, Alonso A, et al. Heart Disease and Stroke Statistics-2019 update: a report from the American Heart Association. Circulation. 2019;139(10):e56–e528. [PubMed: 30700139]
2. Heron M. Leading Causes of Death for 2017 Washington, DC: National Center for Health Statistics; 2019. https://www.cdc.gov/nchs/data/nvsr/nvsr68/nvsr68_06-508.pdf. Accessed September 5, 2019.
3. Nowbar AN, Gitto M, Howard JP, Francis DP, Al-Lamee R. Mortality from ischemic heart disease. Circ Cardiovasc Qual Outcomes. 2019; 12(6):e005375. [PubMed: 31163980]
4. Dagenais GR, Leong DP, Rangarajan S, et al. Variations in common diseases, hospital admissions, and deaths in middle-aged adults in 21 countries from five continents (PURE): a prospective cohort study. Lancet. 2019;395:785–794. [PubMed: 31492501]
5. Dawber TR, Meadors GF, Moore FE Jr. Epidemiological approaches to heart disease: the Framingham Study. Am J Public Health Nations Health. 1951;41(3):279–281. [PubMed: 14819398]

6. Mahmood SS, Levy D, Vasan RS, Wang TJ. The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective. Lancet. 2014;383(9921):999–1008. [PubMed: 24084292]

7. Backe EM, Seidler A, Latza U, Rossnagel K, Schumann B. The role of psychosocial stress at work for the development of cardiovascular diseases: a systematic review. Int Arch Occup Environ Health. 2012; 85(1):67–79. [PubMed: 21584721]

8. Brook RD, Rajagopalan S, Pope CA 3rd, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation. 2010; 121(21):2331–2378. [PubMed: 20458016]

9. Chowdhury R, Ramond A, O'Keeffe LM, et al. Environmental toxic metal contaminants and risk of cardiovascular disease: systematic review and meta-analysis. BMJ. 2018;362:k3310. [PubMed: 30158148]

10. Miller KA, Siscovick DS, Sheppard L, et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med. 2007;356(5):447–458. [PubMed: 17267905]

11. Pope CA 3rd, Dockery DW. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc. 2006;56(6): 709–742. [PubMed: 16805397]

12. World Health Organization. Air Quality Guidelines Global Update 2005: Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. Germany: WHO Regional Office for Europe; 2006. http://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf

13. Yang BY, Guo Y, Morawska L, et al. Ambient PM1 air pollution and cardiovascular disease prevalence: insights from the 33 Communities Chinese Health Study. Environ Int. 2019;123:310–317. [PubMed: 30557810]

14. Zhang Z, Guo C, Lau AKH, et al. Long-term exposure to fine particulate matter, blood pressure, and incident hypertension in Taiwanese adults. Environ Health Perspect. 2018;126(1):017008. [PubMed: 29351544]

15. Gellissen J, Pattloch D, Mohner M. Effects of occupational exposure to respirable quartz dust on acute myocardial infarction. Occup Environ Med. 2019;76(6):370–375. [PubMed: 31010894]

16. Ljungman PLS, Li W, Rice MB, et al. Long- and short-term air pollution exposure and measures of arterial stiffness in the Framingham Heart Study. Environ Int. 2018;121(Pt 1):139–147. [PubMed: 30205320]

17. Sjogren B. Occupational exposure to dust: inflammation and ischaemic heart disease. Occup Environ Med. 1997;54(7): 466–469. [PubMed: 9282121]

18. Boffetta P, Zeig-Owens R, Wallenstein S, et al. Cancer in World Trade Center responders: findings from multiple cohorts and options for future study. Am J Ind Med. 2016;59(2):96–105. [PubMed: 26725936]

19. New York City 9/11 Health. Rescue and Recovery Workers: Search City of New York; 2019. https://www1.nyc.gov/site/911health/enrollees/rescue-recovery-workers.page. Accessed December 16, 2019.

20. Savitz DA, Oxman RT, Metzger KB, et al. Epidemiologic research on man-made disasters: strategies and implications of cohort definition for World Trade Center worker and volunteer surveillance program. Mt Sinai J Med. 2008;75(2):77–87. [PubMed: 18500709]

21. Lioy PJ, Weisel CP, Millette JR, et al. Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC 11 September 2001. Environ Health Perspect. 2002;110(7):703–714. [PubMed: 12117648]

22. Howard J. World Trade Center Health Program Development of the Inventory of 9/11 Agents ResearchGateway: World Trade Center Health Program; 2018. https://wwwn.cdc.gov/ResearchGateway/Content/pdfs/Development_of_the_Inventory_of_9-11_Agents_20180717.pdf. Accessed September 5, 2019.

23. Schnall PL, Dobson M, Landsbergis P. Globalization, work, and cardiovascular disease. Int J Health Serv. 2016;46(4):656–692. [PubMed: 27604540]

24. Herbert R, Moline J, Skloot G, et al. The World Trade Center disaster and the health of workers: five-year assessment of a unique medical screening program. Environ Health Perspect. 2006;114(12):1853–1858. [PubMed: 17185275]

25. Landrigan PJ, Lioy PJ, Thurston G, et al. Health and environmental consequences of the World Trade Center disaster. Environ Health Perspect. 2004;112(6):731–739. [PubMed: 15121517]
26. Wisnivesky JP, Teitelbaum SL, Todd AC, et al. Persistence of multiple illnesses in World Trade Center rescue and recovery workers: a cohort study. Lancet. 2011;378(9794):888–97. [PubMed: 21890053]
27. Jordan HT, Brackbill RM, Cone JE, et al. Mortality among survivors of the Sept 11, 2001, World Trade Center disaster: results from the World Trade Center Health Registry cohort. Lancet. 2011;378(9794):879–887. [PubMed: 21890052]
28. Jordan HT, Stein CR, Li J, et al. Mortality among rescue and recovery workers and community members exposed to the September 11, 2001 World Trade Center terrorist attacks, 2003–2014. Environ Res. 2018;163:270–279. [PubMed: 29477875]
29. Remch M, Laskaris Z, Flory J, Mora-McLaughlin C, Morabia A. Post-traumatic stress disorder and cardiovascular diseases: a cohort study of men and women involved in cleaning the debris of the World Trade Center complex. Circ Cardiovasc Qual Outcomes. 2018; 11(7):e004572. [PubMed: 29991645]
30. Cohen HW, Zeig-Owens R, Joe C, et al. Long-term cardiovascular disease risk among firefighters after the World Trade Center disaster. JAMA Netw Open. 2019;2(9):e199775. [PubMed: 31490535]
31. Dasaro CR, Holden WL, Berman KD, et al. Cohort profile: World Trade Center Health Program General Responder Cohort. Int J Epidemiol. 2017;46(2):e9. [PubMed: 26094072]
32. Moline JM, Herbert R, Levin S, et al. WTC medical monitoring and treatment program: comprehensive health care response in after-math of disaster. Mt Sinai J Med. 2008;75(2):67–75. [PubMed: 18500708]
33. Caldwell DM, Welton NJ, Dias S, Ades AE. Selecting the best scale for measuring treatment effect in a network meta-analysis: a case study in childhood nocturnal enuresis. Res Synth Methods. 2012;3(2):126–141. [PubMed: 26062086]
34. Moline JM, McLaughlin MA, Sawit ST, et al. The prevalence of metabolic syndrome among law enforcement officers who responded to the 9/11 World Trade Center attacks. Am J Ind Med. 2016;59(9):752–760. [PubMed: 27582477]
35. Shockey TM,SA, Odom EC. Cardiovascular Health Status by Occupational Group—21 States, 2013. Centers for Disease Control and Prevention. 2016;65:793–798.
36. Chang HM, Okwuosa TM, Scarabelli T, Moudgil R, Yeh ETH. Cardiovascular complications of cancer therapy: best practices in diagnosis, prevention, and management: part 2. J Am Coll Cardiol. 2017;70(20):2552–2565. [PubMed: 29145955]
37. Memorial Hermann Heart & Vascular Institute. Heart Disease and Age: Memorial Hermann; 2019. http://heart.memorialhermann.org/education-prevention/heart-disease-and-age/. Accessed October 7, 2019.
38. Harvard Medical School. Premature Heart Disease, Cambridge. MA: Harvard Health Publishing; 2009. https://www.health.harvard.edu/heart-health/premature-heart-disease
39. Smith B, Chu LK, Smith TC, et al. Challenges of self-reported medical conditions and electronic medical records among members of a large military cohort. BMC Med Res Methodol. 2008;8:37. [PubMed: 18644098]
40. Machón M, Arriola L, Larrañaga N, et al. Validity of self-reported prevalent cases of stroke and acute myocardial infarction in the Spanish cohort of the EPIC study. J Epidemiol Community Health. 2013;67(1):71–75. [PubMed: 22577182]
41. Barr EL, Tonkin AM, Welborn TA, Shaw JE. Validity of self-reported cardiovascular disease events in comparison to medical record adjudication and a statewide hospital morbidity database: the AusDiab study. Intern Med J. 2009;39(1):49–53. [PubMed: 19290982]
42. Jordan HT, Miller-Archie SA, Cone JE, Morabia A, Stellman SD. Heart disease among adults exposed to the September 11, 2001 World Trade Center disaster: results from the World Trade Center Health Registry. Prev Med. 2011;53(6):370–376. [PubMed: 22040652]

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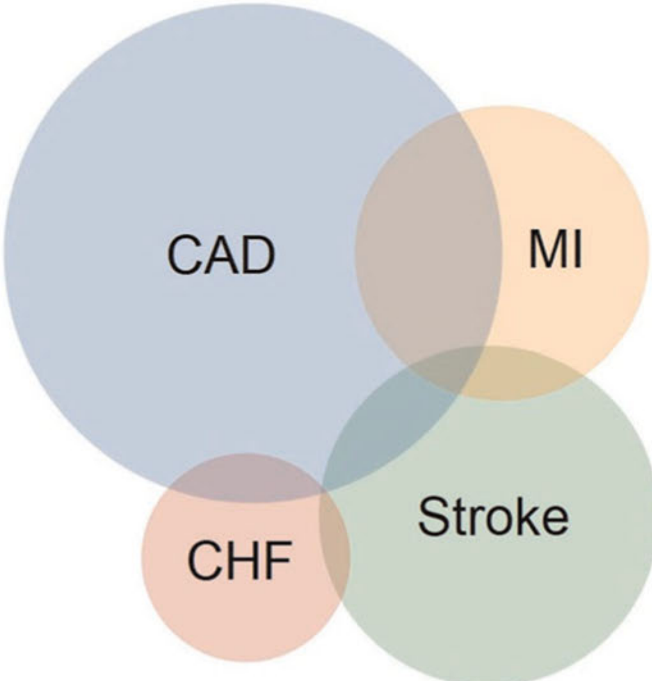

	Total
CAD	1441
MI	502
Stroke	670
CHF	253
Any CVD	**2385**

	CAD	MI	Stroke	CHF
CAD	1073	220	73	40
MI		192	47	12
Stroke			509	16
CHF				167

CAD x MI x Stroke	18
CAD x MI x CHF	11
CAD x Stroke x CHF	5
MI x Stroke x CHF	1
CAD x MI x Stroke x CHF	1

FIGURE 1.
Distribution of post 9/11 CVD ($n = 2385$) in World Trade Center Health Program General Responder Cohort. CAD, coronary artery disease; CHF, congestive heart failure; CVD, cardiovascular disease; MI, myocardial infarction

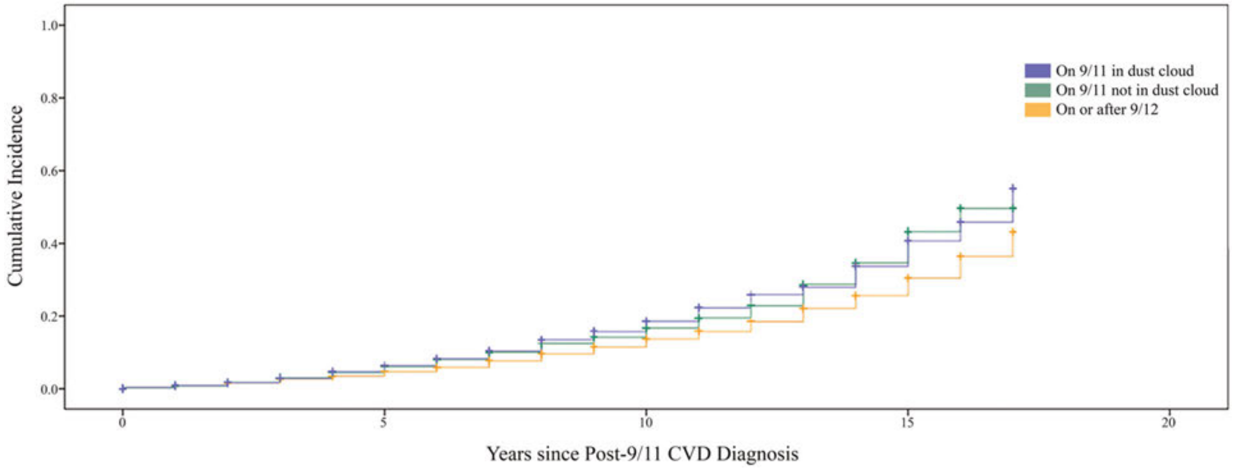

FIGURE 2.
Kaplan–Meier post 9/11 cardiovascular disease (CVD) incidence by years since 9/11/2001

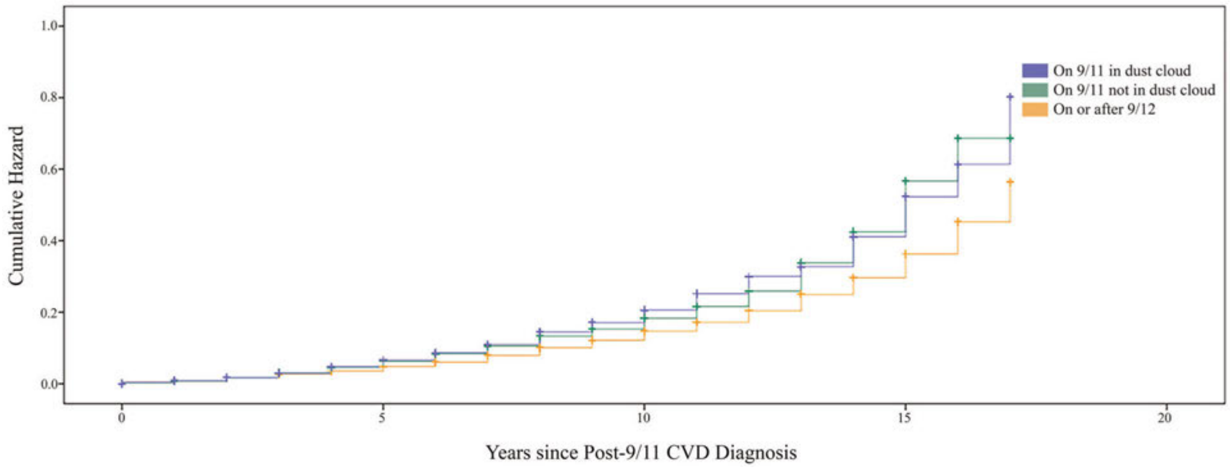

FIGURE 3.

Kaplan–Meier post 9/11 cardiovascular disease (CVD) cumulative hazard by years since 9/11/2001

TABLE 1

WTCHP GRC participant characteristics (*n* = 37,725)

	N	%
Age (years) on 9/11/2001		
20–29	1153	3.1
30–39	8871	23.5
40–49	15,021	39.8
50–59	9188	24.4
60+	3492	9.3
Sex		
Male	32,539	86.3
Female	5186	13.7
Race/ethnicity		
White	20,241	53.7
Black	3422	9.1
Asian	464	1.2
Hispanic	6992	18.5
Other, not reported	6606	17.5
Cigarette smoker		
Never	21,754	57.7
Former	9713	25.7
Current	2651	7.0
Missing	3607	9.6
Occupation on 9/11/2001		
Protective services/military	19,968	52.9
Construction	6601	17.5
Electrical, telecom, and other installation and repair	2463	6.5
Transportation and material moving occupations	2008	5.3
Other jobs	4744	12.6
Unemployed/retired	516	1.4
Not reported	1425	3.8
Marital status on 9/11/2001 (*n* = 37,434)		
Single	4184	11.1
Married or partnered	27,084	71.8
Separated or divorced	5539	14.7
Widowed	627	1.7
Missing	291	0.8
Initial body mass index (703 × pounds/inches^2)		
<25 (normal)	5263	14.3
25 to <30 (overweight)	15,373	41.8
≥30	16,146	42.8
Missing	943	2.5

	N	%
Ever diagnosed with diabetes	5193	13.8
Missing	125	3.3
Ever diagnosed with high cholesterol	19,672	52.1
Missing	4099	10.9
Ever diagnosed with high blood pressure	16,167	42.9
Missing	155	0.4
WTCHP GRC exposure based on arrival at site		
On or after 9/12	20,866	55.3
On 9/11, not dust cloud	9673	25.6
On 9/11, in dust cloud	7186	19.0
Year of WTCHP GRC enrollment		
≤2007	18,874	50.0
2008–2013	9231	24.5
≥2014	9620	25.5

Abbreviations: GRC, General Responder Cohort; WTCHP, World Trade Center Health Program.

TABLE 2

CVD (CAD, MI, stroke, CHF) prevalence in WTCHP GRC[a]

	Diagnosed on/after 9/11								
	Men (*n* = 32,539)			Women (*n* = 5186)			Total (*n* = 37,725)		
	N	%	Age at diagnosis; mean (*SD*)	*N*	%	Age at diagnosis; mean (*SD*)	*N*	%	Age at diagnosis; mean (*SD*)
Coronary artery disease	1360	4.2	53.2 (8.6)	81	1.6	53.1 (9.9)	1441	3.8	53.2 (8.7)
Heart attack (MI)	476	1.1	52.3 (7.6)	26	0.5	54.4 (10.2)	502	1.3	52.4 (7.8)
Stroke	586	1.8	52.8 (9.6)	84	1.6	51.2 (10.6)	670	1.8	52.6 (9.7)
Congestive heart failure	226	0.7	51.3 (10.1)	27	0.5	49.5 (10.6)	253	0.7	51.2 (10.2)
Total reporting ≥1 CVD	2192	6.7	52.5 (8.8)	193	3.7	51.8 (10.4)	2385	6.3	52.4 (9.0)

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; CVD, cardiovascular disease; GRC, General Responder Cohort; MI, myocardial infarction; WTCHP, World Trade Center Health Program.

[a]Conditions not mutually exclusive.

TABLE 3

Kaplan–Meier cumulative and annual cardiovascular disease incidence by years since 9/11/2001 ($n = 37{,}725$)

Years since 9/11/2001																		
Cumulative incidence (%)																		
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
≥9/12	0.4	0.9	1.6	2.7	3.5	4.7	5.9	7.6	9.6	11.5	13.7	15.8	18.5	22.1	25.6	30.4	36.4	43.1
9/11, not in dust	0.3	0.7	1.7	3.0	4.5	6.1	8.0	10.0	12.5	14.2	16.7	19.4	22.8	28.7	34.6	43.3	49.7	NA
9/11, in dust cloud	0.4	0.9	1.7	2.9	4.7	6.4	8.3	10.4	13.5	15.8	18.6	22.3	25.9	27.9	33.7	40.7	45.8	55.2
Annual incidence (%)																		
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
≥9/12		0.5	0.7	1.1	0.8	1.2	1.2	1.7	2.0	1.9	2.2	2.1	2.7	3.6	3.5	4.8	6.0	6.7
9/11, not in dust		0.4	1.0	1.3	1.5	1.6	1.9	2.0	2.5	1.7	2.5	2.7	3.4	5.9	5.9	8.7	6.4	NA
9/11, in dust cloud		0.5	0.8	1.2	1.8	1.7	1.9	2.1	3.1	2.3	2.8	3.7	3.6	2.0	5.8	7.0	5.1	9.4
Number at risk																		
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
≥9/12	20,865	15,032	8676	7300	6392	5954	5463	4924	4360	3825	3287	2846	2381	1823	1347	994	640	56
9/11, not in dust	9672	6725	3622	2974	2584	2373	2127	1904	1666	1441	1257	1071	883	645	480	316	159	15
9/11, in dust cloud	7185	5124	2860	2309	2004	1834	1642	1457	1289	1100	955	793	661	477	362	263	161	28

TABLE 4

Cox proportional hazards regression of age to CVD diagnosis

	Men (n = 32,539)				Women (n = 5186)			
		95% CI				95% CI		
	HR	Lower	Upper	*p*	HR	Lower	Upper	*p*
Covariate adjusted								
9/11, not in dust cloud	1.43	1.29	1.58	≤0.001	1.59	1.11	2.27	0.01
9/11, in dust cloud	1.40	1.26	1.56	≤0.001	2.16	1.49	3.11	≤0.001
White, non-Hispanic								
Black, non-Hispanic	0.79	0.68	0.92	0.003	1.84	1.28	2.64	≤0.001
Asian, non-Hispanic	1.23	0.84	1.81	0.28	0.44	0.06	3.18	0.41
Hispanic	0.83	0.73	0.95	0.006	1.28	0.89	1.82	0.18
Other or not reported	1.03	0.91	1.16	0.66	0.90	0.51	1.58	0.72
Covariate and comorbidity adjusted								
9/11, not in dust cloud	1.33	1.20	1.47	≤0.001	1.49	1.04	2.13	0.03
9/11, in dust cloud	1.29	1.16	1.44	≤0.001	2.17	1.50	3.14	≤0.001
White, non-Hispanic								
Black, non-Hispanic	0.87	0.74	1.01	0.07	1.52	1.04	2.24	0.03
Asian, non-Hispanic	1.13	0.77	1.67	0.53	0.62	0.08	4.54	0.64
Hispanic	0.95	0.83	1.08	0.41	1.31	0.91	1.90	0.15
Other or not reported	1.03	0.91	1.15	0.67	0.95	0.54	1.66	0.85
Nonsmoker								
Former smoker	0.98	0.89	1.07	0.59	1.42	1.03	1.96	0.03
Current smoker	1.55	1.33	1.80	≤0.001	1.89	1.09	3.28	0.02
Normal (BMI < 25)								
Overweight (BMI, 25–29)	1.25	1.07	1.47	0.005	1.04	0.71	1.51	0.86
Obese (BMI ≥ 30)	1.56	1.34	1.83	≤0.001	1.31	0.89	1.92	0.17
Ever had diabetes	1.21	1.10	1.33	≤0.001	1.52	1.07	2.16	0.02
Ever had high blood pressure	1.46	1.31	1.63	≤0.001	1.28	0.92	1.78	0.14
Ever had high cholesterol	5.61	4.92	6.39	≤0.001	2.76	1.93	3.94	≤0.001

Abbreviations: BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio.

TABLE 5

Cox proportional hazards regression of age to CVD diagnosis including protective services occupation on 9/11/2001

	Men (n = 32,539)				Women (n = 5186)			
		95% CI				95% CI		
	HR	Lower	Upper	p	HR	Lower	Upper	p
Covariate adjusted								
9/11, not in dust cloud	1.24	1.12	1.38	≤0.001	1.25	0.87	1.80	0.23
9/11, in dust cloud	1.14	1.02	1.28	0.02	1.71	1.17	2.48	0.005
White, non-Hispanic								
Black, non-Hispanic	0.83	0.71	0.97	0.02	1.49	1.03	2.16	0.04
Asian, non-Hispanic	1.31	0.89	1.91	0.17	0.34	0.05	2.47	0.29
Hispanic	0.86	0.75	0.98	0.02	1.25	0.88	1.78	0.21
Other or not reported	1.04	0.92	1.17	0.53	0.81	0.46	1.42	0.47
Protective services	2.07	1.89	2.27	≤0.001	2.81	2.02	3.90	≤0.001
Covariate and comorbidity adjusted								
9/11, not in dust cloud	1.16	1.05	1.28	0.005	1.18	0.82	1.70	0.38
9/11, in dust cloud	1.04	0.93	1.17	0.46	1.61	1.10	2.36	0.02
White, non-Hispanic								
Black, non-Hispanic	0.91	0.78	1.06	0.22	1.20	0.81	1.78	0.37
Asian, non-Hispanic	1.16	0.79	1.71	0.44	0.42	0.06	3.14	0.40
Hispanic	0.97	0.85	1.11	0.66	1.28	0.89	1.85	0.19
Other or not reported	1.00	0.89	1.13	0.97	0.83	0.47	1.47	0.53
Nonsmoker								
Former smoker	1.02	0.93	1.12	0.62	1.33	0.97	1.84	0.08
Current smoker	1.77	1.52	2.06	≤0.001	1.67	0.96	2.91	0.07
Normal (BMI < 25)								
Overweight (BMI, 25–29)	1.20	1.02	1.41	0.03	0.96	0.66	1.41	0.84
Obese (BMI ≥ 30)	1.46	1.25	1.71	≤0.001	1.19	0.81	1.75	0.38
Ever had diabetes	1.25	1.14	1.38	≤0.001	1.44	1.01	2.06	0.04
Ever had high blood pressure	1.48	1.33	1.65	≤0.001	1.33	0.96	1.85	0.09
Ever had high cholesterol	5.50	4.83	6.27	≤0.001	2.90	2.03	4.15	≤0.001

	Men (n = 32,539)				Women (n = 5186)			
		95% CI				95% CI		
	HR	**Lower**	**Upper**	***p***	**HR**	**Lower**	**Upper**	***p***
Protective services	2.16	1.97	2.37	≤0.001	2.94	2.10	4.11	≤0.001

Abbreviations: BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio.

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Respiratory and Cardiovascular Hospitalizations After the World Trade Center Disaster

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ABSTRACT. The objective of this study was to determine whether there were increases in respiratory and cardiovascular hospital admissions among residents of lower Manhattan after the destruction of the World Trade Center. The authors used hospital admission records from 1991 to 2001 with a diagnosis of respiratory, cardiovascular, or cerebrovascular illness and a residential address in lower Manhattan or Queens. The authors assessed the change in admissions by comparing lower Manhattan to Queens (the control area) and before and after 9/11 admissions in lower Manhattan. They found the following significant increases in hospital admissions: for respiratory illnesses during the weeks of 9/11/01 and 10/16/01; asthma during the week of 9/11/01; cardiovascular during the weeks of 9/18/01 and 10/9/01; cerebrovascular during the weeks of 9/11/01, 9/18/01, 10/2/01, and 10/9/01. There was an immediate increase in respiratory admissions after the disaster and a delayed increase in cardiovascular and cerebrovascular admissions.

KEYWORDS: asthma, cardiovascular diseases, environmental disasters, respiratory diseases, World Trade Center

The collapse of the World Trade Center (WTC) on September 11, 2001 ("9/11") resulted in a large and intense environmental disaster in New York City (NYC). The collapse of the buildings released pulverized cement, glass, building materials, and building contents; air contaminants were also released by the combustion of jet fuel, on-going fires, and clean-up activities (eg, demolition of damaged buildings). The huge cloud of dust covered streets, residences, and workplaces of lower Manhattan. Landrigan et al reviewed the published research related to the WTC disaster and stated that the sources of exposure included settled dust, airborne particulate matter, trace elements, and asbestos.[1] The settled dust consisted mostly of coarse particles and contained pulverized building materials, lead, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), polychlorinated furans, and dioxins.[1]

To date, researchers have published health studies that focus on firefighters, other rescue workers, and clean-up workers.[1–3] In general, these studies showed an association between the high exposures to airborne contaminants and the presence of respiratory symptoms among these workers and volunteers. There have been relatively few studies to assess the effects on the respiratory and cardiovascular health of the residents living near the WTC on 9/11. The pollutants that were released by the WTC disaster may have caused exacerbations of symptoms or resulted in new disease among these residents.

There are several reports on the respiratory and cardiovascular health effects among residents in the vicinity of the WTC. Szema et al found that children with asthma who lived within 5 miles of the WTC and were seen at a clinic in lower Manhattan's Chinatown had more asthma-related clinic

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visits and received more prescriptions for asthma medications.[4] Reibman and Lin and colleagues conducted a series of surveys to examine upper and lower respiratory symptoms of residents living near Ground Zero as compared to residents of a control area in upper Manhattan.[5,6] Lin et al reported that residents living near Ground Zero had higher rates of new upper respiratory symptoms after 9/11 and that most of these symptoms persisted 1 year after 9/11.[6] In addition, these residents also had higher rates of respiratory-related unplanned medical visits and new medication use. Riebman et al reported that residents near Ground Zero who had no respiratory illnesses before 9/11 reported more new lower respiratory symptoms after 9/11.[5] Wagner et al surveyed Medicaid enrollees residing in NYC who had asthma before 9/11 and found that living in lower Manhattan and western Brooklyn was associated with worsened asthma.[7] Finally, Allegra et al studied cardiac events in New Jersey and found a significant increase in myocardial infarction admissions to emergency departments within 50 miles of the WTC after 9/11.[8]

There are no published studies that assess the potential effect of the WTC disaster on hospital admissions, a measure of more severe health outcomes. The purpose of our study was to examine whether there was an increase in hospital admissions due to respiratory and cardiovascular illnesses after the WTC disaster among residents of lower Manhattan. We also assessed whether there were any changes on the distributions of the cases in terms of demographic factors and specific disease categories.

METHODS

Study design and study areas

This study used 2 types of controls: a control area similar to the area that was affected by the disaster, and admissions before 9/11. The affected-control area component compared the rates of hospital admissions among the residents living near Ground Zero to residents in a control area of Queens during the same time period to minimize the effect by seasonal and temporal trends. The pre- and post-9/11 component compared hospital admissions in the affected area before and after 9/11 to minimize bias due to socioeconomic differences between the affected and control areas by using the affected area prior to 9/11 as its own control. Furthermore, by using the average of the rates for the past 10 years in the affected area before and after 9/11 as the reference, we controlled for seasonal and socioeconomic influences.

The affected area was defined as the part of lower Manhattan below Canal Street that encompasses the “hot zone” and “near zone” identified by the US Environmental Protection Agency (EPA). According to the 2000 US Census, this area covered 33 block groups and had a population of 56,013 persons. The control area was composed of 68 block groups in the Borough of Queens, NYC, with a population of 88,017. The control area was selected using 3 criteria: (1) it was at least 5 miles from the affected area, which minimized the possibility of selecting a control area that was affected by air pollutants produced by the collapse of the towers; (2) it was less likely to have been affected by the plume of smoke and dust released by the collapse (namely, the Boroughs of Brooklyn and Staten Island); (3) it was similar to the affected area with respect to population density, race, ethnicity, median household income, and percentage of households below the poverty level. By “similar” we mean that the percentage of the population in each demographic category in the affected area and the control area were within 5 percentage points. We did not use statistical tests (such as the chi-square test) to compare the areas because, due to the large population numbers, the test would detect negligible differences.

Hospital admission data and diagnoses

Data from the New York State Department of Health, Statewide Planning and Research Cooperative System (SPARCS), were used to identify all hospital admission cases. SPARCS is a legislatively mandated database that contains hospital discharge data for at least 95% of all acute care hospital admissions in New York State.[9] The database does not include admissions to psychiatric and federal hospitals. With the approval of the SPARCS Data Protection Review Board, we were provided with the data for all hospital admissions between 1/1/91 and 12/31/01, including principal diagnoses, hospital admission date, medical record number, source of payment, date of birth, gender, race, ethnicity, and street address. We selected the records with a primary diagnosis of respiratory diseases and cardiovascular diseases for all patients with residential addresses in the affected and control areas.

This study examined 2 major health outcomes: respiratory diseases (including asthma) and cardiovascular diseases (including cerebrovascular disease). The measures included the number and rate of hospital admissions for each health outcome. Respiratory diseases included the following principal diagnoses based on the International Classification of Disease (ICD-9, US DHHS, 1991)[10]: chronic bronchitis (ICD code 491); emphysema (492); asthma (493); chronic obstructive pulmonary disease (COPD) (496); and, for children aged 0 to 4 years, acute bronchiolitis (466) and acute bronchitis (490). Acute bronchitis and bronchiolitis were included because they are common respiratory illnesses among very young children and are difficult to distinguish from asthma in young children. Cardiovascular disease was defined as: chronic rheumatic heart disease (ICD codes 393–396), hypertension (401–405), acute coronary artery disease (CAD) (410, 411), chronic CAD (412–414), cardiac dysrhythmia (427), congestive heart failure (428), and cerebrovascular disease (430–438). These conditions have been found to be related to various air pollutants, including particulate matter.[11] Because asthma and cerebrovascular diseases are major categories among respiratory and cardiovascular diseases, we examined them separately.

To define the study population more precisely, the residential address of each patient was geocoded and assigned a latitude and longitude using MapMarker software.[12] Most addresses were geocoded automatically; the remaining addresses were geocoded interactively. Finally, we selected the admission records for patients with a residential latitude and longitude within the affected area or the control area using MapInfo software.[13] Population estimates for the affected and control areas (to use as the denominators) were determined using 1990 and 2000 US Census block group data.

Statistical analyses

The weekly hospital admission rates in the affected area were compared to the weekly rates in the control area (affected-control area comparison). Because hospital admissions in this study included new cases and readmitted cases, we chose to use a conservative prevalence estimate. We computed the average weekly rate of the 4 weeks before 9/11 and the average weekly rate for the 4 weeks immediately after 9/11. (We used the average of the 4 weeks before 9/11 for a more stable estimate that would be less prone to weekly and seasonal fluctuations.) We also computed the weekly rates for each of the 8 weeks after 9/11 because the chemical and dust levels, which dropped off quickly, were relatively localized and would be more likely to cause acute health effects.[1] We also used 8 weeks for all of the comparisons to observe temporal patterns of the admissions. We computed the prevalence ratio (PR) by dividing the rate in the affected area by the rate in the control area. For each PR we computed the 95% confidence interval (95% CI). In addition, for cardiovascular disease admissions, we computed the relative change in each week after 9/11 compared to the baseline rate within each area.

The pre- versus post-9/11 (before-after) comparisons were conducted within the affected area only. First, we compared the weekly hospital admission rate for each week from 9/11/01 to 11/5/01 (8 weeks) to the average weekly admission rate for the 4 weeks before 9/11 (the reference period). We also compared the 4-week average rate immediately after 9/11 to the 4-week average before 9/11. The PR was calculated by dividing the hospital admission rate for each week after 9/11 by the average rate during the reference period.

Second, we compared the weekly hospital admission rates before and after 9/11 to the average rate during the same week in the preceding 10 years. Using the population of the affected area from the 1990 and 2000 US Censuses, we interpolated the population for the intervening years and 2001. For each year we computed the weekly hospital admission rate (number of admissions/population) for 2 weeks before 9/11 and 8 weeks after 9/11. For 1991–2000, we averaged the weekly rates across the 10-year period and computed the 95% CI. We considered an elevated weekly rate in 2001 statistically significant if it was above the upper 95% confidence limit of the 1991–2000 average weekly rate.

Third, we used a temporal scan statistic to identify significant temporal clusters of hospital admissions after 9/11. For this analysis, the cases were the hospital admissions between 9/11/01 and 11/5/01 (8 weeks) and the controls were the admissions for the same days in the preceding 10 years (1991 to 2000). We used daily admission counts, assumed a Bernoulli model, and allowed for flexible time windows (ie, we did not specify the length of the cluster).[14] We present the most likely time cluster, the ratio of the observed to expected cases (ie, prevalence ratio), and the p value from Monte Carlo replications. A p value less than .05 was considered a statistically significant temporal cluster.

RESULTS

Demographic and economic characteristics

The affected and control areas were generally comparable with respect to the distribution by age, race, Hispanic ethnicity, and income. However, the population of the control area tended to be younger ($\leq$19 years, 22.3% versus 17.4% in the affected area) and older ($\geq$65 years, 16.7% versus 12.1%). The distributions of race and ethnicity in these 2 areas were similar: black, 6.5% in the affected area versus 6.7% in the control area; Hispanic, 9.4% versus 12.7%, respectively). The percent of the population living below the poverty level was 18.5% in the affected area and 13.5% in the control area, but the median household income was similar ($47,127 versus $50,074, respectively).

Affected versus control area comparison

The affected-control area analysis is presented in Table 1. The baseline (pre-9/11) hospital admission rates were similar in the affected and control areas for respiratory diseases, asthma, and cerebrovascular diseases. However, the baseline rate for cardiovascular diseases was significantly lower in the affected area before 9/11 (PR = 0.51, 95% CI 0.26–1.00). During the week of 9/11, the respiratory hospital admission rate was significantly higher in the affected area (1.79 per 10,000) compared to the control area (0.34 per 10,000) (PR = 5.24, 95% CI 1.44–19.0). During the same week, there were 6 admissions for asthma in the affected area (2 to 3 times more than in the subsequent weeks) and none in the control area, although the PR could not be computed. In data not shown, the 4-week average rate after 9/11 was not significantly higher for respiratory diseases (PR = 0.66, 95% CI 0.21–2.13) or for asthma (PR = 1.08, 95% CI 0.23–5.02). There were no other significant differences between the 2 areas for respiratory diseases and asthma in the weeks that followed.

For cardiovascular and cerebrovascular diseases, the affected-control area comparison did not show any statistically significant increases. Because the baseline (pre-9/11) rate of cardiovascular disease admissions was much higher in the control area (3.86 per 10,000) than that in the affected area (1.96 per 10,000), we also examined the relative change

Table 1.—Comparison of the Affected and Control Areas, Weekly Respiratory and Cardiovascular Hospital Admissions 8 Weeks After September 11, 2001 ("9/11"), New York City

	Affected area		Control area			
Illness, weeks before and after 9/11	Admission number	Admission rate[a]	Admission number	Admission rate[a]	PR	95% CI[b]
Respiratory diseases						
08/14–09/10[c]	2	0.36	4.25	0.48	0.74	0.14–3.97
09/11–09/17	10	1.79	3	0.34	**5.24**	**1.44–19.00**
09/18–09/24	3	0.54	11	1.25	0.43	0.12–1.54
09/25–10/01	3	0.54	10	1.14	0.47	0.13–1.71
10/02–10/08	0	0	14	1.59	—	—
10/09–10/15	3	0.54	5	0.57	0.94	0.23–3.95
10/16–10/22	5	0.89	2	0.23	3.93	0.76–20.25
10/23–10/29	2	0.36	7	0.80	0.45	0.09–2.16
10/30–11/05	2	0.36	10	1.14	0.31	0.07–1.43
Asthma						
08/14–09/10[c]	1.25	0.22	1.5	0.17	1.31	0.12–14.06
09/11–09/17	6	1.07	0	0	—	—
09/18–09/24	3	0.54	5	0.57	0.94	0.23–3.95
09/25–10/01	2	0.36	5	0.57	0.63	0.12–3.24
10/02–10/08	0	0	6	0.68	—	—
10/09–10/15	3	0.54	1	0.11	4.71	0.49–45.32
10/16–10/22	1	0.18	1	0.11	1.57	0.10–25.12
10/23–10/29	1	0.18	1	0.11	1.57	0.10–25.12
10/30–11/05	1	0.18	3	0.34	0.52	0.05–5.04
Cardiovascular diseases						
08/14–09/10[c]	11	1.96	34	3.86	0.51	0.26–1.00
09/11–09/17	11	1.96	31	3.52	0.56	0.28–1.11
09/18–09/24	19	3.39	39	4.43	0.77	0.44–1.32
09/25–10/01	10	1.79	32	3.64	0.49	0.24–1.00
10/02–10/08	15	2.68	24	2.73	0.98	0.52–1.87
10/09–10/15	18	3.21	26	2.95	1.09	0.60–1.98
10/16–10/22	12	2.14	38	4.32	**0.50**	**0.26–0.95**
10/23–10/29	8	1.43	28	3.18	**0.45**	**0.20–0.98**
10/30–11/05	10	1.79	33	3.75	**0.48**	**0.23–0.97**
Cerebrovascular diseases						
08/14–09/10[c]	3	0.54	4.75	0.54	0.94	0.23–3.95
09/11–09/17	3	0.54	8	0.91	0.59	0.16–2.22
09/18–09/24	6	1.07	5	0.57	1.89	0.58–6.18
09/25–10/01	3	0.54	6	0.68	0.79	0.20–3.14
10/02–10/08	7	1.25	5	0.57	2.20	0.70–6.93
10/09–10/15	4	0.71	8	0.91	0.79	0.24–2.61
10/16–10/22	1	0.18	8	0.91	0.20	0.12–1.57
10/23–10/29	1	0.18	4	0.45	0.39	0.04–3.51
10/30–11/05	1	0.18	6	0.68	0.26	0.03–2.18

[a]Per 10,000 persons in the population.
[b]PR = prevalence ratio; CI = confidence interval; bold face indicates a 95% CI that does not include 1.0.
[c]For 8/14/01–9/10/01, the average of the weekly number of admissions and average of the weekly admission rate are presented.

in the number of admissions from the baseline number in each area (data not shown). For the week of 9/18, the relative change in the affected area was 72.7% compared to 14.7% in the control area. There were relative increases in the affected area but decreases in the control area during the weeks of 10/2 (36.4% and −29.4%, respectively) and 10/9 (63.6% and −23.5%, respectively).

Pre- versus post-9/11 comparison

We compared the hospital admission rates before and after 9/11 in the affected area (Table 2). The respiratory admission rate was found to be significantly higher during the first week after 9/11 (1.79 per 10,000) compared to the average rate for the 4 weeks before 9/11 (0.36 per 10,000) (PR = 5.00, 95% CI 1.10–22.83). This increase was not significant

Table 2.—Comparison of Pre- and Post-September 11, 2001 ("9/11") Weekly Respiratory and Cardiovascular Hospital Admissions, Affected Area of New York City

Illness, weeks before and after 9/11	Admission rate[a]	PR	95% CI[b]
Respiratory diseases			
08/14–09/10[c]	0.36	1.00	reference
09/11–09/17	1.79	**5.00**	**1.10–22.83**
09/18–09/24	0.54	1.50	0.25–8.98
09/25–10/01	0.54	1.50	0.25–8.98
10/02–10/08	0	—	—
10/09–10/15	0.54	1.50	0.25–8.98
10/16–10/22	0.89	2.50	0.49–12.89
10/23–10/29	0.36	1.00	0.14–7.10
10/30–11/05	0.36	1.00	0.14–7.10
Asthma			
08/14–09/10[c]	0.22	1.00	reference
09/11–09/17	1.07	4.80	0.70–32.98
09/18–09/24	0.54	2.40	0.30–19.34
09/25–10/01	0.36	1.60	0.17–14.95
10/02–10/08	0	—	—
10/09–10/15	0.54	2.40	0.30–19.34
10/16–10/22	0.18	0.80	0.06–11.09
10/23–10/29	0.18	0.80	0.06–11.09
10/30–11/05	0.18	0.80	0.06–11.09
Cardiovascular diseases			
08/14–09/10[c]	1.96	1.00	reference
09/11–09/17	1.96	1.00	0.43–2.31
09/18–09/24	3.39	1.73	0.82–3.63
09/25–10/01	1.79	0.91	0.39–2.14
10/02–10/08	2.68	1.36	0.63–2.97
10/09–10/15	3.21	1.64	0.77–3.47
10/16–10/22	2.14	1.09	0.48–2.47
10/23–10/29	1.43	0.73	0.29–1.81
10/30–11/05	1.79	0.91	0.39–2.14
Cerebrovascular diseases			
08/14–09/10[c]	0.54	1.00	reference
09/11–09/17	0.54	1.00	0.20–4.95
09/18–09/24	1.07	2.00	0.50–8.00
09/25–10/01	0.54	1.00	0.20–4.95
10/02–10/08	1.25	2.33	0.60–9.02
10/09–10/15	0.71	1.33	0.30–5.96
10/16–10/22	0.18	0.33	0.03–3.20
10/23–10/29	0.18	0.33	0.03–3.20
10/30–11/05	0.18	0.33	0.03–3.20

[a]Per 10,000 persons in the population.
[b]PR = prevalence ratio, PR; CI = confidence interval; bold face indicates a 95% CI that does not include 1.0.
[c]For 8/14/01–9/10/01, the average of the weekly number of admissions and average of the weekly admission rate are presented.

when we compared the 4-week average rate after 9/11 to the 4-week average before 9/11 (PR = 2.00, 95% CI 0.37–10.9). The asthma admission rate was also elevated during the first week after 9/11 (PR = 4.80, 95% CI 0.70–32.98) and during the 4 weeks after 9/11 (PR = 2.20, 95% CI 0.27–18.2), but did not reach statistical significance, probably because of the small number of asthma admissions. Although not statistically significant, there were increases in cardiovascular admission rates beginning with the week of 9/18. The average rate of the 4 weeks after 9/11 was also not significantly elevated (PR = 1.25, 95% CI 0.57–2.76). There were no significant increases in cerebrovascular admission rates.

Figure 1 presents the weekly hospital admission rates in the affected area before and after 9/11/01 compared to the same weeks during the preceding 10 years. For respiratory diseases (Figure 1a), the rates during the weeks of 9/11 and 10/16 (6 weeks after 9/11) were significantly higher (above the upper 95% CI) than the mean of the preceding 10 years. For asthma admissions (Figure 1b), the rate was significantly higher during the week of 9/11. The rate of cardiovascular admissions (Figure 1c) was significantly higher during the weeks of 9/4 (before 9/11), 9/18 (1 to 2 weeks after 9/11), and 10/9 (4 to 5 weeks after 9/11). The rate of cerebrovascular admissions was significantly higher during the weeks of 8/28 (before 9/11), 9/11, 9/18, 10/2, and 10/9.

Using the temporal scan statistic, we looked for clusters of hospital admissions in the affected area during the 8 weeks after 9/11 (data not shown). The only statistically significant cluster was for respiratory disease hospital admissions: the most likely cluster was 9/11–9/13 (PR = 3.6, $p = .03$). The most likely clusters for asthma, cardiovascular, and cerebrovascular admissions were not statistically significant. For asthma admissions, the most likely cluster was 9/11–9/17 (PR = 2.4, $p = .53$). The most likely cluster of cardiovascular admissions was on 9/20 (PR = 2.3, $p = .51$) and for cerebrovascular admissions was 10/1–10/2 (PR = 2.2, $p = .98$).

To better understand the increases in hospital admissions for respiratory diseases and asthma during the week of 9/11, we looked at the source of payment (specifically, Worker's Compensation) and demographic characteristics of the hospitalized residents of the affected area (data not shown). Twenty percent of the respiratory admissions during the week of 9/11 were reimbursed by Worker's Compensation compared to 0% during the 4 weeks before 9/11. Patients reimbursed by Worker's Compensation may have had residential exposures as well as possible occupational exposures. Higher rates of admission among certain demographic groups may indicate a differential health effect on susceptible populations and identify a group with increased risk. In the affected area after 9/11, 70% of hospitalized patients for respiratory diseases were women compared to 38% during the 4 weeks before 9/11, and 60% of the admitted patients were at least 65 years old as compared to 25% before 9/11. Similar trends were observed for cardiovascular hospital admissions. These findings indicate that the distributions of payment source (Worker's Compensation), gender, and age among hospitalized cases in the affected area changed after 9/11.

We also compared the hospital admissions by principal diagnosis among residents of the affected area admitted in the 5 weeks after 9/11 to the same 5 weeks in the preceding 10 years (data not shown). Of the 19 residents admitted for respiratory diseases after 9/11, 74% had a

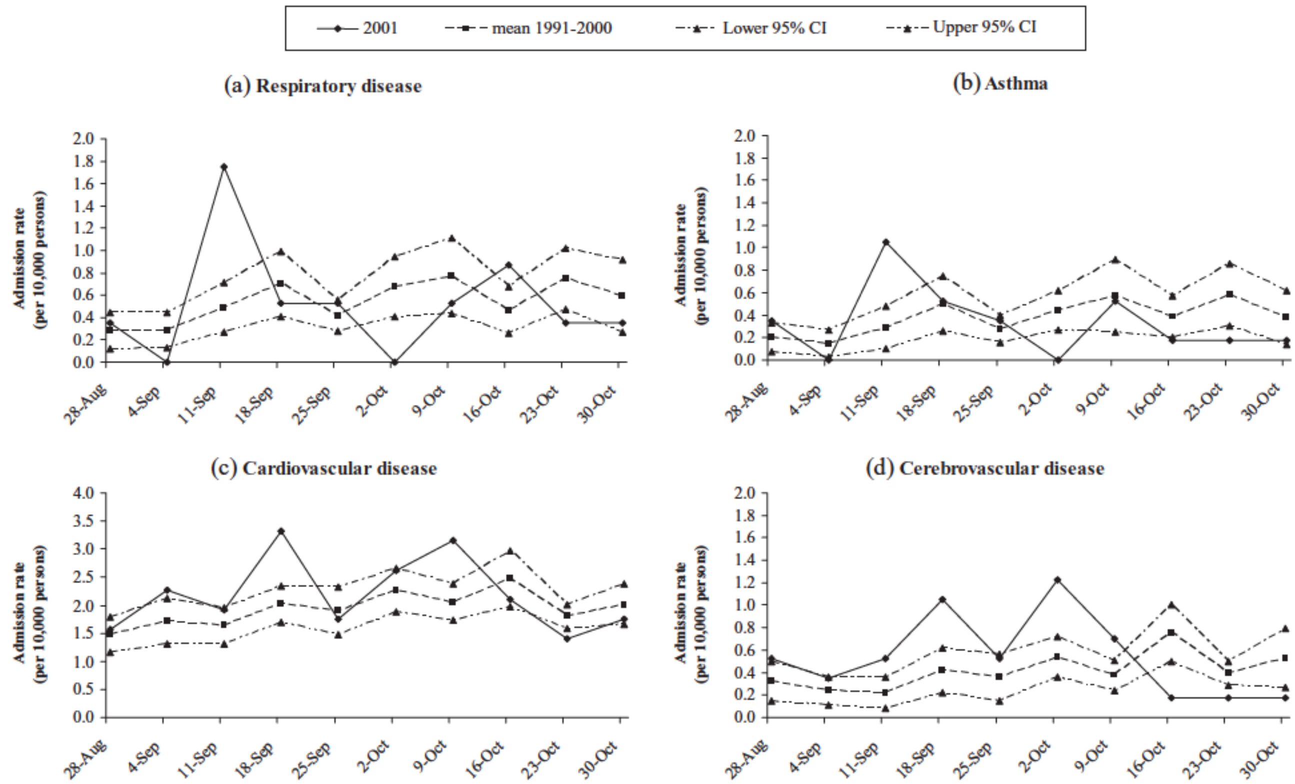

Fig. 1. Respiratory, asthma, cardiovascular, and cerebrovascular hospital admission rates (per 10,000 people) in the weeks before and after September 11, 1991–2000, compared to 2001, affected area of New York City.

principal diagnosis of asthma, followed by 16% with chronic bronchitis and 10% with chronic obstructive pulmonary disease (COPD). The distribution was similar for the preceding 10 years (68% asthma, 15% chronic bronchitis, 11% COPD, 4% emphysema, and 3% acute bronchiolitis). However, the principal diagnoses of the 73 residents admitted for cardiovascular diseases after 9/11 differed from the preceding 10 years. After 9/11, there was a higher percentage of admissions for chronic CAD (25% versus 19% in the preceding 10 years) and cerebrovascular diseases (34% versus 20%) and a lower percentage of admissions for hypertension (4% versus 8%), cardiac dysrhythmias (11% versus 16%), and congestive heart failure (11% versus 18%).

COMMENT

We found consistent increases in hospital admissions for respiratory diseases during the first week after 9/11 using different study designs and statistical methods. The affected-control area comparison showed a significantly elevated PR during this week compared to the control area in Queens. The before-after comparison of the affected area also showed a significantly elevated PR for the week of 9/11 compared to the month before 9/11. We found a significant increase in rate of respiratory disease admissions during the weeks of 9/11 and 10/16 compared to the mean rate during the preceding 10 years. The temporal cluster analysis confirmed the increase during the first few days after 9/11. For asthma we found a significant increase in the rate of admissions during the week of 9/11 as compared to the preceding 10 years, but this increase was not confirmed by other comparisons.

We observed that the residents of the affected area who were hospitalized were disproportionately older and female, and one fifth of the admissions may have been related to an occupational exposure at that time. In addition, this study found that the increase in respiratory admissions probably reflects the increase in asthma admissions and not a change in the diagnosis pattern. Our findings indicate that the disaster may have exacerbated asthma or other respiratory symptoms, especially among older susceptible residents and women.

Since the WTC disaster, many published reports have addressed the health effects, both physical and psychological, on the residents of lower Manhattan. Among published reports, a telephone survey of Manhattan residents was conducted 5 to 9 weeks after 9/11 that found that among adult respondents with asthma, 27% had more severe symptoms after 9/11 compared to the 4 weeks before 9/11. Twenty-eight percent of asthmatic residents with worsened symptoms reported unscheduled medical visits for asthma compared 5% who did not have worsened symptoms.[15]

Szema et al reported that children with asthma living in lower Manhattan have significant increases in the number of asthma-related visits ($p = .002$) and asthma prescriptions ($p = .018$) in the year after 9/11 compared to the year before 9/11.[4] Wagner et al surveyed Medicaid enrollees with persistent asthma before 9/11 residing in NYC and found that living in lower Manhattan and western Brooklyn were associated with worsened asthma (adjusted odds ratios = 2.28 and 4.3, respectively).[7] A survey of residents living near the WTC site found that, among residents without respiratory disease prior to 9/11, those living near the WTC site were significantly more likely to report lower respiratory symptoms (eg, wheeze, shortness of breath, night-time cough) that began after 9/11 than residents of a control area in upper Manhattan (incidence ratios ranged from 2.6 to 4.3).[5] In addition, the incidence of unplanned medical visits and new use of respiratory medication was significantly higher among the residents living near the WTC site than the control residents (incidence ratios = 1.7 and 2.9, respectively).[6] Although these studies differ in their designs and measures of health effects they show significant increases in respiratory symptom incidence and exacerbation. Studies involving workers at the WTC site conclusively demonstrate the health effects of the disaster and document a dose-response relationship between exposure and health outcomes.[3,16] There are no published studies of hospital admissions for respiratory diseases and asthma to directly compare with our findings.

We found a significant increase in the rate of cardiovascular disease admissions for the weeks of 9/18/01 and 10/9/01 compared to the mean rate during preceding 10 years. The rate of cerebrovascular disease admissions significantly increased during the weeks of 9/11/01, 9/18/01, 10/2/01, and 10/9/01 compared to the preceding 10 years. Although not statistically significant, both the affected-control area and before-after comparisons demonstrated similar delayed increases for cardiovascular admissions. The most likely cluster for cardiovascular admissions coincided with the week of 9/18/01 and for cerebrovascular admissions with the week of 10/2/01, a delayed effect. These findings indicate a possible cardiovascular and/or cerebrovascular health effect with a longer latency than the respiratory health effects. We also found increased proportions of CAD and stroke after 9/11 compared to previous years.

To date, only 3 reports have been published that examine the effect of the WTC disaster on cardiovascular outcomes, but no studies of cerebrovascular events have been reported.[8,17,18] Steinberg et al examined the incidence of ventricular arrhythmias recorded by implantable cardioverter-defibrillators (ICD) of 200 patients living an average of 23 miles from the WTC site.[18] They found a significant 2.3-fold increase in the risk of life-threatening arrhythmias after 9/11 and the mean onset of tachyarrhythmia occurred 12.4 days after 9/11. The investigators found a delay in the onset of these events that they believe are more consistent with the health effects of subacute stress, which is consistent with our findings. Shedd et al conducted a similar study among 132 patients with ICDs living in Florida and found a 2.8-fold increase in the risk after 9/11.[17] Allegra et al conducted a retrospective study of patients with a diagnosis of myocardial infarction or tachyarrhythmia seen in 16 emergency departments within 50 miles of the WTC.[8] They found a significant increase in the number of patients with myocardial infarctions in the 2 months after 9/11 compare to 2 months before 9/11, but not for tachyarrhythmias. These studies agree with our finding that increases in cardiovascular events occurred.

In terms of ambient and indoor air quality, Landrigan et al summarized published studies and documented the composition of the dust and debris dispersed by the collapse of the towers.[1] Although the airborne particulate level after 9/11 was reportedly below US EPA limits, short-term peaks occurred.[19] In addition, analysis of indoor dust found that settled dust collected indoors in lower Manhattan was chemically similar to WTC dust collected outdoors.[20] By using a deterministic microenvironmental model, Ng et al found that estimated personal exposure levels were generally higher than outdoor $PM_{2.5}$ concentrations and that one fifth of the population was exposed to $PM_{2.5}$ concentrations over 60 $\mu g/m^3$ after 9/11 (the National Ambient Air Quality Standard for $PM_{2.5}$ is 65 $\mu g/m^3$).[21]

Some residents may have experienced high-level exposures at the time of the collapse of the WTC Towers and lower-level on-going exposures from the settled dusts and continuing fires. In general, we found what appears to be a short-term response to an acute exposure for respiratory and asthma hospital admissions, with a possible delayed response for respiratory disease admissions. We also found both short-term and delayed effect for cardiovascular and cerebrovascular admissions. The possible mechanisms of particulate air pollution on health include oxidative lung damage and inflammation, decreased lung function, and may also produce direct and indirect effects via the autonomic nervous system for cardiovascular diseases.[22,23]

Another important exposure that may contribute to cardiovascular and respiratory diseases and would have certainly played a role after the WTC attack is psychological stress and anxiety. Both Shedd et al and Allegra et al hypothesize that anxiety may have played a role in the increases in arrhythmias observed in their studies and that the effect may extend to persons geographically distant from the WTC.[8,17] Steinberg et al cite a survey that found increased stress levels throughout the United States after the WTC attack and another study that describes the scientific evidence linking acute stress and cardiovascular events.[18,24,25]

Strengths and limitations

This is one of the few studies assessing the potential health impact of the WTC disaster on residents of lower Manhattan. By using hospital admissions, an objective health indicator, reporting bias was not a limitation of this study. In addition to respiratory diseases, which have been studied extensively by other investigators, we also investigated the effect of the WTC disaster on cardiovascular events, another biologically plausible disease outcome.

There are a few potential concerns when studying respiratory and cardiovascular events. Seasonality and socioeconomic factors are important confounders for the hypotheses being tested. In this study, we used 2 methods to minimize the influence of temporal trend and seasonality: the affected-control area comparison (ie, use of an unaffected area with similar sociodemographics as a control area during the same period) and the comparison of the weekly number of admissions around 9/11 to the average number during the same week in the preceding 10 years, which also provides a more reliable reference estimate. The before-after comparison also helped control for confounding effects of socioeconomics.

Even with careful selection, the population in the control area still tended to be slightly younger ($\leq$19 years) and older ($\geq$65 years), which are known risk factors for asthma and cardiovascular diseases, respectively. If any bias was due to this difference, it would be toward the null; that is, the effect we observed would underestimate the true effect.

The comparability of the baseline cardiovascular event rates in the affected and control areas is another concern. In the affected-control area comparison, the baseline rate of cardiovascular events was much higher than in the control area, which makes comparing the rates in the 2 areas after 9/11 difficult. However, the before-after analyses only included the population in the affected area and used the rates before 9/11 as an internal control for socioeconomic differences. In this case, the comparability of the baseline rates between the affected and control areas is not a concern.

Another potential problem is that we might have underestimated the health impact on respiratory illnesses from the 9/11 disaster. For example, based on the 2000 US Census, residents in the ZIP codes that included the area most affected by the collapse of the WTC Towers (the "hot zone") had a higher median annual income (about $65,000) than the residents living somewhat further away from the WTC site (the "near zone," about $36,000). It is possible that the residents in the "hot zone" may carry more private health insurance and, therefore, may have managed their asthma better and would be less likely to be hospitalized during an exacerbation of symptoms. This would result in an underestimate of the actual respiratory events among the residents with higher exposure by using hospitalization as a surrogate for respiratory disease events. However, by expanding the study areas to include more ZIP codes in the current study, we believe this produced a more representative sample of the population. Another concern related to the use of hospital admissions is that only the most severe respiratory cases are captured and the findings can only be generalized to this special population and not to less severe cases.

One other potential weakness of this study is misclassification bias. Some residents in the affected area temporarily moved out of their residences after 9/11 and may have stayed outside the area. SPARCS requires reporting of the patient's "principal residence at the time of admission." If patients who resided in the affected area on 9/11 reported temporary addresses outside the affected area, and possibly in the control area, then the number of cases in the affected area would have been underestimated, resulting in an underestimate of the association (or bias towards the null).

We were unable to quantify 2 important exposures: air pollution levels in the affected area and psychological stress experienced by the residents of the affected area. One air monitoring site maintained by the New York State Department of Environmental Conservation in one of the Twin Towers was destroyed during the disaster and new monitoring sites in the vicinity were only added in late October 2001. Therefore, no objective data on ambient air pollution measurements near the WTC site were available to study if the changes in health endpoints were associated with changes in air pollution levels. Finally, because this was a data linkage study, we did not have information on the psychological status or stress of the hospitalized patients. Consequently, we cannot verify if the possible health effects found from this study are due to air pollution levels or psychological stress from the WTC disaster, or a combination of the two.

Conclusion

This study demonstrated consistent evidence that hospital admissions due to respiratory diseases (mainly asthma) increased significantly during the first week after 9/11. In addition, hospital admissions for cardiovascular diseases and cerebrovascular diseases also increased significantly after 9/11, but with delayed effects, about 2 to 3 weeks after the disaster. The elderly and women tended to be more susceptible. We estimate that about 20% of these hospital admissions could have been due to occupational exposures, some possibly related to 9/11 rescue and recovery efforts. Based on this and other studies showing acute and persistent health effects after the WTC disaster, residents of lower Manhattan with post-9/11 respiratory or cardiovascular health problems should be followed-up and monitored for potential long-term health effects. This study will also provide information and reference data for on-going disaster and public health preparedness efforts.

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The Institutional Review Board of the New York State Department of Health (NYSDOH) approved the conduct of this study (IRB Study No. 01-062).

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References

1. Landrigan PJ, Lioy PJ, Thurston G, et al. Health and environmental consequences of the world trade center disaster. *Environ Health Perspect*. 2004;112:731–739.
2. Banauch GI, Alleyne D, Sanchez R, et al. Persistent hyperreactivity and reactive airway dysfunction in firefighters at the World Trade Center. *Am J Respir Crit Care Med*. 2003;168:54–62.
3. Prezant DJ, Weiden M, Banauch GI, et al. Cough and bronchial responsiveness in firefighters at the World Trade Center site. *N Engl J Med*. 2002;347:806–815.
4. Szema AM, Khedkar M, Maloney PF, et al. Clinical deterioration in pediatric asthmatic patients after September 11, 2001. *J Allergy Clin Immunol*. 2004;113:420–426.
5. Reibman J, Lin S, Hwang SA, et al. The World Trade Center residents' respiratory health study: new-onset respiratory symptoms and pulmonary function. *Environ Health Perspect*. 2005;113:406–411.
6. Lin S, Reibman J, Bowers JA, et al. Upper respiratory symptoms and other health effects among residents living near the World Trade Center site after September 11, 2001. *Am J Epidemiol*. 2005;162:499–507.
7. Wagner VL, Radigan MS, Roohan PJ, Anarella JP, Gesten FC. Asthma in Medicaid managed care enrollees residing in New York City: results from a post-World Trade Center disaster survey. *J Urban Health*. 2005;82:76–89.
8. Allegra JR, Mostashari F, Rothman J, Milano P, Cochrane DG. Cardiac events in New Jersey after the September 11, 2001, terrorist attack. *J Urban Health*. 2005;82:358–363.
9. New York State Department of Health. *2002 Annual Report: The SPARCS Data System*. Albany, NY: New York State Department of Health; 2002.
10. US Department of Health and Human Services. *The International Classification of Diseases, 9th Revision*. Washington DC: US Department of Health and Human Services; 1991. DHHS Publication No. (PHS) 91–1260 Volume I.
11. Burnett RT, Cakmak S, Brook JR, Krewski D. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ Health Perspect*. 1997;105:614–620.
12. Mapinfo Corp. MapMarker Plus version 11.2. Troy, NY: Mapinfo; 2005.
13. MapInfo Corp. MapInfo version 8.0. Troy, NY: Mapinfo; 2005.
14. Kulldorff M. A spatial scan statistic. *Commun Stat Theory Methods*. 1997;26:1481–1496.
15. Centers for Disease Control and Prevention. Self-reported increase in asthma severity after the September 11 attacks on the World Trade Center—Manhattaan, New York, 2001. *MMWR Morb Mortal Wkly Rep*. 2002;51:781–784.
16. Banauch GI, Alleyne D, Sanchez R, et al. Persistent hyperreactivity and reactive airway dysfunction in fire fighters at the World Trade Center. *Am J Respir Crit Care Med*. 2003;168:54–62.
17. Shedd OL, Sears SF Jr, Harvill JL, et al. The World Trade Center attack: increased frequency of defibrillator shocks for ventricular arrhythmias in patients living remotely from New York City. *J Am Coll Cardiol*. 2004;44:1265–1267.
18. Steinberg JS, Arshad A, Kowalski M, et al. Increased incidence of life-threatening ventricular arrhythmias in implantable defibrillator patients after the World Trade Center attack. *J Am Coll Cardiol*. 2004;44:1261–1264.
19. Lioy PJ, Weisel CP, Millette JR, et al. Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC 11 September 2001. *Environ Health Perspect*. 2002;110:703–714.
20. Offenberg JH, Eisenreich SJ, Gigliotti CL, et al. Persistent organic pollutants in dusts that settled indoors in lower Manhattan after September 11, 2001. *J Expo Anal Environ Epidemiol*. 2004;14:164–172.
21. Ng SP, Dimitroulopoulou C, Grossinho A, Chen LC, Kendall M. PM 2.5 exposure assessment of the population in Lower Manhattan area of New York City after the World Trade Center disaster. *Atmos Environ*. 2005;39:1979–1992.
22. Brook RD, Franklin B, Cascio W, et al. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004;109:2655–2671.
23. Pope CA III. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? *Environ Health Perspect*. 2000;108(Suppl 4):713–723.
24. Schuster MA, Stein BD, Jaycox L, et al. A national survey of stress reactions after the September 11, 2001, terrorist attacks. *N Engl J Med*. 2001;345:1507–1512.
25. Rozanski A, Blumenthal JA, Kaplan J. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*. 1999;99:2192–2217.

FULL TEXT LINKS

Wolters Kluwer

J Occup Environ Med. 2023 Nov 1. doi: 10.1097/JOM.0000000000003007. Online ahead of print.

Self-reported cardiovascular disease in career firefighters with and without World Trade Center exposure

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Affiliations
PMID: 37907410 DOI: 10.1097/JOM.0000000000003007

Abstract

Objective: To assess the effect of World Trade Center (WTC) exposure on cardiovascular disease (CVD) in career firefighters.

Methods: Firefighters from four US cities completed health questionnaires that provide information about demographics, CVD diagnoses, and CVD risk factors. Firefighters were also compared to respondents of the 2019 National Health Interview Survey (NHIS).

Results: Greater WTC exposure was positively associated with combined coronary artery disease, myocardial infarction, and angina (termed "CAD") when comparing WTC-exposed to non-WTC-exposed firefighters. Compared with the NHIS population, firefighters had lower odds of CAD and stroke.

Conclusion: An occupationally appropriate comparison is important to mitigate potential bias from the healthy worker effect. While the risk of CVD in WTC-exposed and non-WTC-exposed firefighters was significantly lower than a general US population, we observed an exposure gradient where greater WTC exposure was associated with greater odds of CVD.

Related information

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NCI CPTAC Assay Portal

Cardiovascular Disease Risk Among WTC Firefighters

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Fire Department of the City of New York, Albert Einstein College of Medicine and Montefiore Medical Center

FDNY Data Center Contracts and U01 OH011309/U01 OH011934

Background – Quick Review

- WTC disaster exposed individuals to fine particulate matter
- Fine particulate matter is a risk factor for cardiovascular disease (CVD)

Background

FDNY has published two cardiovascular disease studies:

1. Cohen HW, Zeig-Owens R, Joe C, Hall CB, Webber MP, Weiden MD, Cleven KL, Jaber N, Skerker M, Yip J, Schwartz T, Prezant DJ. Long-term Cardiovascular Disease Risk Among Firefighters After the World Trade Center Disaster. JAMA Netw Open. 2019 Sep 4;2(9):e199775.

2. Mueller AK, Cohen H, Singh A, Webber MP, Hall CB, Prezant DJ, Zeig-Owens R. Self-reported Cardiovascular Disease in Career Firefighters With and Without World Trade Center Exposure. J Occup Environ Med. 2024 Feb 1;66(2):135-140.

Background – Cohen *et al*

JAMA Network | Open

Original Investigation | Occupational Health

Long-term Cardiovascular Disease Risk Among Firefighters After the World Trade Center Disaster

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Abstract

IMPORTANCE Published studies examining the association between World Trade Center (WTC) exposure on and after September 11, 2001, and longer-term cardiovascular disease (CVD) outcomes have reported mixed findings.

OBJECTIVE To assess whether WTC exposure was associated with elevated CVD risk in Fire Department of the City of New York (FDNY) firefighters.

DESIGN, SETTINGS, AND PARTICIPANTS In this cohort study, the association between WTC

Key Points

Question Is World Trade Center exposure on and after September 11, 2001, associated with long-term cardiovascular disease risk in Fire Department of the City of New York firefighters?

Findings In this cohort study of 9796 firefighters, age-adjusted incident rates

JAMA Network Open. 2019;2(9):e199775. doi:10.1001/jamanetworkopen.2019.9775

September 6, 2019

Background – Cohen *et al*

Aim: assessed whether acute (arrival time) and post-acute (duration) exposures to the WTC site were associated with elevated long-term CVD risk

Methods: Longitudinal cohort using internal analyses

Population: FDNY male firefighters (n= 9,796) who reported first arrival at the WTC site within the two weeks after 9/11, were actively employed on 9/11, no history of CVD on 9/11 and consented to research

Time period: 9/12/2001 to 12/31/2017

Background – Cohen *et al*

Arrival time defined as the time a participant first arrived at the WTC site

Three levels:

- Arrived on the morning of 9/11
- Arrived on the afternoon of 9/11
- Arrived between 9/12 and 9/24 (reference group)

Duration defined as the total number of months (1 -10) a participant reported working at the site ≥ 1 day

Two levels:

- ≥ 6 months (top 25% of cohort)
- < 6 months (reference group)

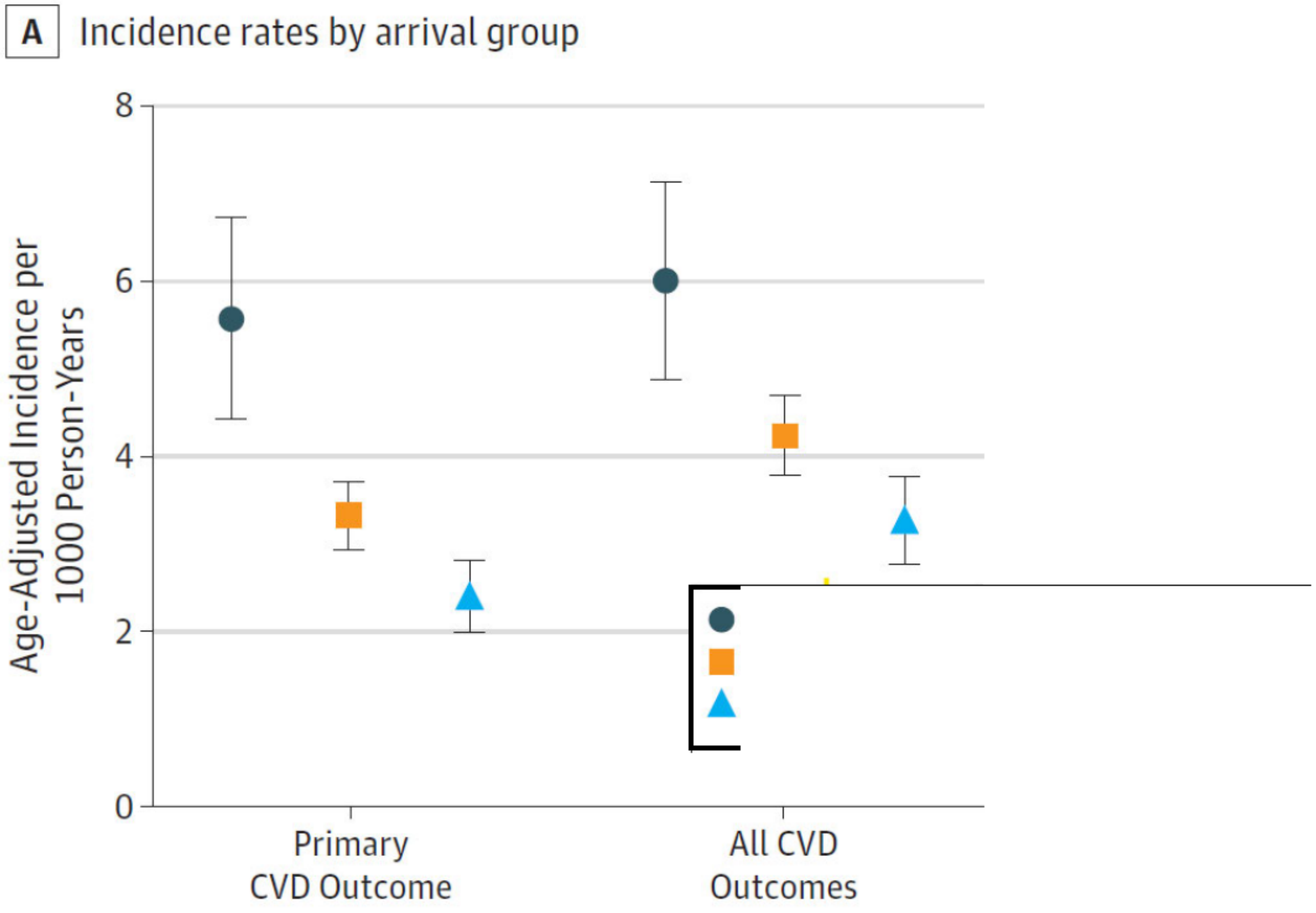
Background – Cohen *et al*

FDNY clinicians confirmed CVD cases via FDNY medical record review

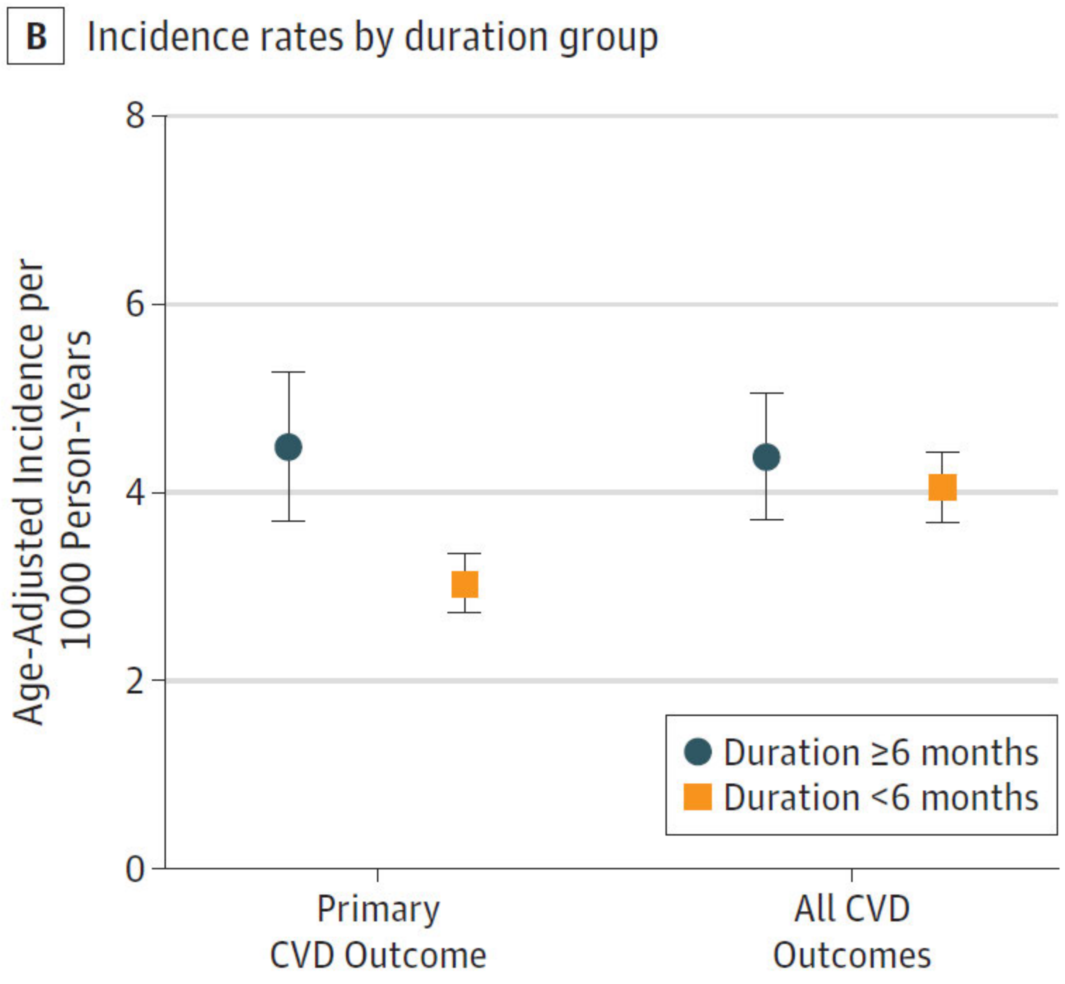
Two case definitions:

Primary Outcome:	All CVD Outcomes:
▪ Myocardial infarction (MI) ▪ Stroke/Cerebrovascular accident ▪ Coronary artery surgery ▪ Unstable angina or angioplasty ▪ Coronary heart failure ▪ CVD death	▪ Myocardial infarction (MI) ▪ Stroke/Cerebrovascular accident ▪ Coronary artery surgery ▪ Unstable angina or angioplasty ▪ Coronary heart failure ▪ CVD death Or ▪ Transient ischemic attack (TIA) ▪ Stable angina ▪ Cardiomyopathy ▪ Other (aortic aneurysm, peripheral arterial vascular intervention, or carotid artery surgery)

Key Findings – Cohen *et al*



Key Findings – Cohen *et al*



Key Findings – Cohen *et al*

Adjusted Hazard Ratio was significant for those who arrived earliest compared with those arriving 9/12 or later:

- Arrived Morning of 9/11: **HR=1.44 (1.09-1.90)**
- Arrived Afternoon of 9/11: HR=1.24 (1.00-1.54)

Adjusted Hazard Ratio was significant for those who worked the most months at the site:

- 6+ months vs <6 months: **HR=1.33 (1.05-1.60)**

Background – Mueller *et al*

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ORIGINAL ARTICLE

Self-reported Cardiovascular Disease in Career Firefighters With and Without World Trade Center Exposure

Alexandra K. Mueller, MPH, Hillel Cohen, DrPH, Ankura Singh, MPH, Mayris P. Webber, DrPH, Charles B. Hall, PhD, David J. Prezant, MD, and Rachel Zeig-Owens, DrPH

Objective: To assess the effect of World Trade Center (WTC) exposure on cardiovascular disease (CVD) in career firefighters. **Methods:** Firefighters from four US cities completed health questionnaires that provide information about demographics, CVD diagnoses, and CVD risk factors. Firefighters were also compared with respondents of the 2019 National Health Interview Survey. **Results:** Greater WTC exposure was positively associated with combined coronary artery disease, myocardial infarction, and angina (termed "CAD") when comparing WTC-exposed with non-WTC–exposed firefighters. Compared with the National Health Interview Survey population, firefighters had lower odds of CAD and stroke. **Conclusions:** An occupationally appropriate comparison is important to mitigate potential bias from the healthy worker effect. While the risk of CVD in WTC-exposed and non-WTC–exposed firefighters was significantly lower than a general US population, we observed an exposure gradient where greater WTC exposure was associated with greater odds of CVD.

Keywords: cardiovascular disease, World Trade Center, firefighting, occupational exposure

LEARNING OUTCOMES

- Describe the association between World Trade Center exposure and cardiovascular disease when accounting for the firefighting occupation
- Recognize how the healthy worker effect can bias association between World Trade Center exposure and cardiovascular disease

Background – Mueller *et al*

Aim: assessed the effect of WTC exposure on CVD in career firefighters

Methods: Cross-sectional prevalence analysis with external, non-WTC-exposed comparison population

Population: Male WTC-exposed (FDNY) and non-WTC–exposed (non-FDNY) firefighters from the Career Firefighter Health Study cohort who were actively employed on 9/11 by their fire depts

Time period: completed a health questionnaire between 2/2019 and 5/2021 – questionnaires evaluated current and past diagnoses

Background – Mueller *et al*

Binary WTC Exposure

Two levels:

- WTC-exposed FDNY firefighters
- Non-WTC-exposed firefighters from Chicago, Philadelphia and San Francisco (reference group)

Categorical WTC Exposure and trend analysis

Four levels:

- Arrived on the morning of 9/11
- Arrived on the afternoon of 9/11
- Arrived between 9/12 and 9/24
- Non-WTC-exposed firefighters (reference group)

Background – Mueller *et al*

Non-Firefighter Comparison

- Additionally compared both WTC-exposed and non-WTC-exposed firefighters with US males who responded to the National Health Interview Survey 2019

Background – Mueller *et al*

- Three Primary Outcomes: **self-reported** cardiovascular diagnoses
  - *Coronary artery disease (CAD)* – includes myocardial infarction, angina, and coronary artery disease
  - *Stroke* – includes stroke/cerebrovascular accident and transient ischemic attack
  - *Stroke/CAD* – includes reporting either or both of the above conditions

Key Findings – Mueller *et al*

TABLE 2. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Status

	Stroke/CAD[a]		Stroke[b]			
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1	Model 2	Model 1	Model 2
WTC-exposed	1.23 (1.06–1.43)	1.03 (0.88–1.20)	1.03 (0.79–1.35)	0.93 (0.71–1.22)	1.25 (1.06–1.47)	1.03 (0.87–1.21)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref

TABLE 3. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Level

	Stroke/CAD[a]		Stroke[b]			
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
High exposure	1.45 (1.18–1.79)	1.19 (0.96–1.48)	1.18 (0.80–1.75)	1.04 (0.70–1.55)	1.48 (1.18–1.86)	1.20 (0.95–1.51)
Moderate exposure	1.31 (1.11–1.54)	1.10 (0.93–1.30)	1.07 (0.79–1.44)	0.97 (0.72–1.30)	1.32 (1.10–1.57)	1.09 (0.91–1.31)
Low exposure	1.00 (0.83–1.21)	0.83 (0.69–1.01)	0.90 (0.64–1.27)	0.81 (0.57–1.15)	1.03 (0.84–1.26)	0.85 (0.69–1.05)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref
P for trend	<0.0001	0.01	0.33	0.74	<0.0001	0.03

[a]Includes any report of stroke or CAD.
[b]Includes diagnoses of stroke/CVA or TIA.
[d]Age, race, and BMI were also included in the model (complete case analysis, $n = 12,516$).
[e]Age, race, BMI, high cholesterol, diabetes, hypertension, and smoking were also included in the model (complete case analysis, $n = 12,435$).

Key Findings – Mueller *et al*

TABLE 2. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Status

	Stroke/CAD[a] OR (95% CI)		Stroke[b] OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1	Model 2	Model 1	Model 2
WTC-exposed	1.23 (1.06–1.43)	1.03 (0.88–1.20)	1.03 (0.79–1.35)	0.93 (0.71–1.22)	1.25 (1.06–1.47)	1.03 (0.87–1.21)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref

TABLE 3. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Level

	Stroke/CAD[a] OR (95% CI)		Stroke[b] OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
High exposure	1.45 (1.18–1.79)	1.19 (0.96–1.48)	1.18 (0.80–1.75)	1.04 (0.70–1.55)	1.48 (1.18–1.86)	1.20 (0.95–1.51)
Moderate exposure	1.31 (1.11–1.54)	1.10 (0.93–1.30)	1.07 (0.79–1.44)	0.97 (0.72–1.30)	1.32 (1.10–1.57)	1.09 (0.91–1.31)
Low exposure	1.00 (0.83–1.21)	0.83 (0.69–1.01)	0.90 (0.64–1.27)	0.81 (0.57–1.15)	1.03 (0.84–1.26)	0.85 (0.69–1.05)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref
P for trend	<0.0001	0.01	0.33	0.74	<0.0001	0.03

[a]Includes any report of stroke or CAD.
[b]Includes diagnoses of stroke/CVA or TIA.
[d]Age, race, and BMI were also included in the model (complete case analysis, n = 12,516).
[e]Age, race, BMI, high cholesterol, diabetes, hypertension, and smoking were also included in the model (complete case analysis, n = 12,435).

Key Findings – Mueller *et al*

TABLE 2. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Status

	Stroke/CAD[a]		Stroke[b]			
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1	Model 2	Model 1	Model 2
WTC-exposed	1.23 (1.06–1.43)	1.03 (0.88–1.20)	1.03 (0.79–1.35)	0.93 (0.71–1.22)	1.25 (1.06–1.47)	1.03 (0.87–1.21)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref

TABLE 3. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Level

	Stroke/CAD[a]		Stroke[b]			
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
High exposure	1.45 (1.18–1.79)	1.19 (0.96–1.48)	1.18 (0.80–1.75)	1.04 (0.70–1.55)	1.48 (1.18–1.86)	1.20 (0.95–1.51)
Moderate exposure	1.31 (1.11–1.54)	1.10 (0.93–1.30)	1.07 (0.79–1.44)	0.97 (0.72–1.30)	1.32 (1.10–1.57)	1.09 (0.91–1.31)
Low exposure	1.00 (0.83–1.21)	0.83 (0.69–1.01)	0.90 (0.64–1.27)	0.81 (0.57–1.15)	1.03 (0.84–1.26)	0.85 (0.69–1.05)
Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref
P for trend	<0.0001	0.01	0.33	0.74	<0.0001	0.03

[a]Includes any report of stroke or CAD.
[b]Includes diagnoses of stroke/CVA or TIA.
[d]Age, race, and BMI were also included in the model (complete case analysis, *n* = 12,516).
[e]Age, race, BMI, high cholesterol, diabetes, hypertension, and smoking were also included in the model (complete case analysis, *n* = 12,435).

Key Findings – Mueller *et al*

TABLE 2. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Status

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	OR (95% CI)		OR (95% CI)		OR (95% CI)	
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Nonexposed	Ref	Ref	Ref	Ref	Ref	Ref

TABLE 3. Estimated Odds Ratios for Self-reported CVD Diagnoses by Exposure Level

	Stroke/CAD[a]		Stroke[b]			
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
High exposure	1.45 (1.18–1.79)	1.19 (0.96–1.48)	1.18 (0.80–1.75)	1.04 (0.70–1.55)	1.48 (1.18–1.86)	1.20 (0.95–1.51)
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[a]Includes any report of stroke or CAD.
[b]Includes diagnoses of stroke/CVA or TIA.
[d]Age, race, and BMI were also included in the model (complete case analysis, $n = 12,516$).
[e]Age, race, BMI, high cholesterol, diabetes, hypertension, and smoking were also included in the model (complete case analysis, $n = 12,435$).

Key Findings – Mueller et al

TABLE 4. Estimated Odds Ratios for Self-reported CVD Diagnoses in Firefighters From All Four Cities Compared With the NHIS Population by Exposure Status

	Stroke/CAD[a]		Stroke[b]		Coronary artery disease (CAD)	
	OR (95% CI)		OR (95% CI)		OR (95% CI)	
	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]	Model 1[d]	Model 2[e]
WTC-exposed	0.81 (0.73–0.90)	0.62 (0.55–0.70)	0.62 (0.51–0.74)	0.54 (0.44–0.66)	0.86 (0.77–0.97)	0.64 (0.57–0.73)
Non-exposed	0.71 (0.62–0.82)	0.59 (0.51–0.68)	0.66 (0.51–0.84)	0.60 (0.47–0.76)	0.74 (0.64–0.87)	0.60 (0.51–0.71)
NHIS Population	Ref	Ref	Ref	Ref	Ref	Ref

Firefighters have lower odds of self-reported CVD diagnoses than US males

Future Research Recommendations

Self-report data has the potential for differential misclassification

- A sensitivity analysis in Mueller et al found among FDNY members the exposure gradient was attenuated when using medical record-confirmed cases.

Medical record data using WTC health records has the potential to miss cases because CVD is not a WTC-covered condition

Future research should use medical record data that does not require a participant to actively inform us of their diagnosis.

- Cause of death data from NDI gets at some of this but not all events are fatal.