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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

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Abstract

Background—To determine whether post-traumatic stress disorders (PTSD) is a risk factor for myocardial infarction (MI) and stroke, beyond the expected effects from recognized cardiovascular risk factors and depression.

Methods and Results—World Trade Center (WTC)-Heart is an observational prospective cohort study of 6481 blue-collar first responders nested within the WTC Health Program in New York City. Baseline measures in 2012 and 2013 included: blood pressure, weight and height, and blood lipids. PTSD, depression, smoking, and dust exposure during the 2001 cleanup were self-reported. During the 4-year follow-up, outcomes were assessed through: 1. Interview-based incident, non-fatal MI and stroke, validated in medical charts (n=118). 2. Hospitalizations for MI and stroke for New York City and State residents (n=180)). Prevalence of PTSD was 19.9% in men and 25.9% in women, that is, at least twice that of the general population. Cumulative incidence of MI or stroke was consistently larger for men or women with PTSD across follow-up. Adjusted hazard ratios (aHR) were: 2.22 (95% CI: 1.30–3.82) for MI, 2.51 (1.39–4.57) for stroke. For pooled MI and stroke, aHRs were 2.35 (1.57–3.52) in all, and 1.88 (1.01–3.49) in men free of depression. Using hospitalization registry data, aHR were 2.17 (1.41–3.32) for MI, 3.01 (1.84–4.93) for stroke, and, for pooled MI and stroke, 2.40 (1.73–3.34) in all, HR=2.44 (1.05–5.55) in women, and aHR=2.27 (1.41–3.67) in men free of depression. WTC dust exposure had no effect.

Conclusions—This cohort study confirms that PTSD is a risk factor for MI and stroke of similar magnitude in men and women, independently of depression.

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CONFLICT OF INTEREST DISCLOSURES:

None.

Keywords

PTSD; Myocardial Infarction; Stroke; Depression; Air pollution; Disaster

This is the first report from World Trade Center (WTC)-Heart, a cohort study specifically designed to assess the association between early response to the WTC September 11 attack and cardiovascular outcomes. WTC-Heart comprises blue-collar workers who were involved in the cleaning of the debris of the WTC complex during the months immediately following the attack The cohort offers the ability to assess whether post-traumatic stress disorder (PTSD) is involved in cardiovascular disease (CVD) incidence because of three cohort characteristics: 1) it underwent the same, extraordinary traumatic event at a discrete point in time and suffers from an excess burden of PTSD symptoms at least twice that of the general population,[1, 2] 2) being nested within a long term program offering free care to first responders, can explore long-term risk 11 to 15 years after the event, and 3) since first responders were on average in their forties in 2001 it is a relatively young cohort in which cardiovascular diseases would be expected to be rare if it were not because of this unique exposure to the psychologically and environmentally traumatic event in 2001.

PTSD, a syndrome characterized by re-experiencing the traumatic exposure, avoidance of reminders, hyperarousal, and negative mood and cognitions, highly prevalent among WTC responders,[3] has been consistently shown to be an independent risk factor for incident MI or stroke in cohort studies.[4–10] Previous reports from a different WTC cohort have related psychological stress to heart diseases among people who worked at or were living in the vicinity of Ground Zero in 2001–2002.[11, 12] However, despite the wealth of evidence, PTSD is still not recognized as a risk factor for CVD because available studies are all affected by one or several limitations such as self-report of established CVD risk factors (e.g., lipids, blood pressure, weight and height), self-reported CVD events, exclusively male or female cohorts, or non adjustment for depression. In contrast, in WTC-Heart, CVD risk factors were measured in a standardized fashion and incident cases of myocardial infarction (MI) and stroke were assessed using an active follow-up including personal interviews and validation of events in medical charts simultaneously in men and women.

METHODS

The data, analytic methods, and study materials will not be automatically made available to other researchers for purposes of reproducing the results or replicating the procedure. The cohort is nested within the WTC Health Program (WTCHP) and authorization will have to be obtained from the WTCHP too.

WTC-Heart is a prospective, fixed cohort of 6481 WTC first responders involved in cleaning up the debris of the WTC complex in New York (NY) City in 2001 and 2002 and recruited within the WTCHP. Active follow-up for incident and recurrent CVD was conducted from January 2012 until June 2016, remotely via email, mail, and phone interviews in English, Spanish, and Polish. The cohort was also linked with the NY State hospitalization database, SPARCS.

The cohort was recruited from two WTCHP sites (i.e., the Icahn School of Medicine at Mount Sinai or North Shore Long Island Jewish Hospital) in NY City. The WTCHP, established in 2002, comprises non-firefighter workers and volunteers engaged in rescue, recovery, restoration of services, cleanup, or other support work on or after 9/11. These first responders were eligible in the WTCHP if they had worked for ≥4 hours on September 11 to 14, 2001, ≥24 hours during the month of September 2001, or ≥80 hours total during the period of October through December 2001.[13] Also eligible were employees of the Office of the Chief Medical Examiner who processed human remains, of the Port Authority Trans-Hudson Corporation who participated in the cleanup efforts for ≥24 hours from February to July 2002, and workers who drove, repaired, cleaned, or maintained vehicles that handled WTC debris for at least one day between September 11th, 2001 and July 31st, 2002.[14]

Sample

The cohort is a consecutive sample of 6481 women and men attending one of their up to 10th annual clinical visits at the WTCHP between January 2012 and June 2013 (baseline). Annual visits are not necessarily associated with medical problems requiring care. Informed consent was provided on the day of recruitment, in English, Spanish and Polish. The Queens College IRB and those of the two recruitment sites approved the WTC-Heart protocol.

Exposures

Recognized cardiovascular risk factors comprised: a) standardized 2 measurements of blood pressure to the 3rd digit (e.g., 12.2) after sitting down; b) a comprehensive smoking history; c) body weight and height measured in lightly dressed subjects; d) blood lipids: total cholesterol, low-density lipoprotein, and triglycerides. Clinical personnel were trained specifically for this study and provided identical, new instruments (scales, sphygmomanometers). Baseline mental health symptoms included the PTSD checklist PCL-C (civilian) for DSM-IV,[15] with a specific preamble relating the answers to the participation in the WTC cleanup. The cut off for probable PTSD was >= 44 points as recommended given the prevalence of PTSD in the cohort.[16] Possible depression was defined as a score of 10 or more (in a range of 0 to 27) on the 9 questions of the Patient Health Questionnaire-9 (PHQ-9)[17]

In a detailed questionnaire each participant indicated: 1) which day on or after September 11 they began working on the WTC complex; 2) whether they were “directly in the cloud of dust (or blackout) from the collapse of the WTC buildings” or not; 3) whether their work was “adjacent” or “on” the pile or pit, terms that referred to the former location of the twin towers of the WTC complex; and 4) whether they wore a protective mask.[18]

Active Follow-up

Attempts were made to contact every participant once a year from the 2012 baseline to June 2016. At each annual contact, incident cardiac and incident neurologic events were reported in a standardized, self-administered survey in English, Spanish, and Polish either online, by mail, or by telephone. In case of death reported by a relative or by the WTCHP a questionnaire and request of death certificate was mailed to either relatives or health care provider.

Outcomes

All self-reported CV events on the follow-up forms, including positive report of cardiac symptoms, myocardial infarction (MI), neurological symptoms, and/or stroke, were tentatively confirmed by specific additional personal interviews conducted by phone by trained interviewers in English, Spanish, or Polish. Determination of incident and recurrent cardiovascular outcomes in medical charts was performed by a blinded adjudicating panel comprising the principal investigator (AM) and a senior research worker (CMM) with 24 years of experience adjudicating acute MI and stroke in several large population-based studies. An MI was confirmed in the presence of typical symptoms, EKG signs, Troponin, and/or enzymatic movement. A stroke or a transient ischemic attack was confirmed in the presence of typical symptoms and medical imaging or sonographic signs.

From January 1st, 2012 through December 31st, 2016 (latest year available) all WTC-Heart participants were electronically linked to NY State Department of Health's SPARCS program, which includes diagnoses and dates for all patients discharged from acute care hospitals, excluding psychiatric and federal hospitals. The coding of discharge diagnoses may not be accurate but the degree of inaccuracy is likely to be homogeneous across the cohort. Matched records had identical key identifying information, such as name, date of birth, 4 last digits of the Social Security number, or address. Analyses were restricted to the 5484 NY City or State residents. We used, for MI, ICD-9 codes 410–411, and ICD-10 codes I21–I22, and for stroke, ICD-9 codes 430–432 and ICD-10 codes I60–I63. For the follow-up years 2012 through 2016, 180 WTC-Heart study participants were identified in the SPARCS database as having been hospitalized with a discharge diagnosis of MI or stroke.

Statistical analysis

Kaplan-Meier survival analysis was used to plot the cumulative incidence of MIs and/or stroke. Cox proportional hazards models were used to estimate adjusted hazard ratios (aHR) and 95% confidence intervals (CI) of MI or stroke, adjusted for use of a respirator and for recognized cardiovascular risk factors: age, blood pressure, total cholesterol, body mass index, tobacco use, and, when relevant, gender. Firth's procedure was applied to improve aHR and CI estimates from Cox proportional hazard models with small sample sizes and few measured outcome events.[19]

Since PTSD may have increased weight gain, tobacco use, blood pressure, and possibly cholesterol after the 2001–2002 trauma, resulting in these factors being on the pathway to MI and stroke and biasing the HRs towards the null, we present primary analyses both age-adjusted and, when sample size allows, adjusted for all these factors.

Time of event was retrieved from the participant's interview or SPARCS records. For event-free participants censoring for interview-based incident events occurred at the date of completion of the most recent annual survey, or, for SPARCS-derived events, on December 31st, 2016, the last date of available SPARCS data. Analyses of post-baseline, incident outcomes were therefore repeated including and excluding MIs and strokes recurring during follow-up among subjects who already had an MI or stroke before 2012. We report the analysis including all cases in the article for four reasons: 1) because this cohort had a mean

age of 51y in 2012, all cardiovascular events occurred after 2001; 2) PTSD is a chronic disease, most likely secondary to 2001, for which it is difficult to set an exact date of onset; 3) these middle aged subjects who had cardiovascular events before baseline were subjects at highest risk in the cohort and excluding them would also introduce selection bias; 4) removing 35 recurrent cases did not alter the statistical power for the full analysis (as shown in the paper) but left thin data for the gender- and disease-specific analyses. Thus, subjects who had had pre-baseline cardiovascular events were excluded for some of the exploratory full sample analyses but kept in all other analyses, with only the post baseline events going into the numerator of the hazard rates and risks.

MI and stroke were analyzed both separately and pooled as a single outcome (MI/stroke) because separate analyses of self-reported MI and stroke, provided in an appendix, yielded associations of consistent magnitudes. Similarly, men and women were analyzed estimating both separate and pooled, gender-adjusted HRs. The number of female cases (9 MIs, 7 strokes and 22 hospitalizations for MI or stroke) precluded statistical power for most analyses, but, as shown in the appendix, the magnitude of the associations was remarkably consistent across gender allowing for meaningful gender-adjusted HRs. The participants who had both MI and stroke were analyzed as per the first of the two outcomes in the analyses pooling MI and stroke.

Because PTSD and depression are highly correlated the analyses for pooled MI and stroke adjusted for recognized cardiovascular risk factors were repeated among n=4120 non-depressed men (the number of female cases was too small to perform gender adjusted analyses), after exclusion of men having either depression only (n=97), depression and PTSD (n=477) or missing depression data (n=248).

RESULTS

The socio-demographic, cardiovascular, and mental characteristics of the WTC-Heart cohort were similar to those of the whole WTCHP in which it was nested (Table S1). As of July 1st, 2016, 510 participants (7.9%) had no follow-up information. The analyses are therefore based on 4942 men and 1029 women (n=5971).

Baseline (2012–2013) characteristics

Mean age was 51.3y in men and 51.1y in women. The cohort was 82.8% men, 54.1% white, and 25.4% lower than college education. Table 1 shows these characteristics by gender. Prevalence of PTSD was 19.9% in men and 25.9% in women.

Recognized cardiovascular risk factors and dust

Figure 1 and Table S2 show current smoking standing out – besides PTSD not shown in Figure 1 - as the main recognized risk determinant of incident (including recurrent) pooled MI and stroke, after adjustment for other cardiovascular risk factors and PTSD. Specific results for MI or stroke are shown in Tables S3–S4. Moreover, resting heart rate per minute at baseline was, respectively for those without and with PTSD, 68.9 and 70.8 in men, and 69.5 and 69.9 in women (not shown in a Table).

None of the variables differently expressing the timing or intensity of exposure to the WTC dust and dust cloud were independently associated with subsequent MI or stroke in these analyses (Table S5). Donning of a protective mask was deemed the closest approximation of the amount of dust inhaled and was adjusted for in all multivariate analyses (Figure 1).

Cumulative risk of CVD by PTSD

By June 30th, 2016 reported cases were: 70 MIs, of which 20 were recurrent, and 53 strokes, of which 15 were recurrent. Figure 2 shows the cumulative risks of MI and stroke for the whole sample by PTSD status. The curves diverge early, divergence increases over time, with statistically significant differences at p<.0001. Specific curves for MI or stroke by gender are shown in Supplemental Figures S1–S4.

Multivariable analysis

In Figure 3 and Table S6 the aHRs of PTSD were 2.22 (95% CI: 1.30–3.82) for 70 MIs, 2.51 (1.39–4.57) for 53 strokes, 2.35 (1.57–3.52) for 118 incident (including recurrent) pooled MI and stroke, and (not shown in a table or figure) 2.24 (1.39–3.63) for 83 incident (excluding recurrent) pooled MI and stroke. Five subjects had both MI and stroke. Specific results by gender and outcomes are provided in Table S6. In women, the estimated HRs for MI, stroke, and for pooled MI and stroke were consistent with those observed among men.

Misclassification of outcomes

Access to medical charts in order to validate the interview-based diagnosis was similar in cohort participants with (60%) and without PTSD (62%). The confirmation fraction was also similar across groups for stroke. However, for MIs, it was smaller for PTSD (47%) than for non-PTSD cases (74%). Such source of differential misclassification could spuriously strengthen the observed associations. We therefore repeated the analyses using discharge diagnoses for hospitalization in NY State for MI or stroke as outcomes, a measure independent of participant reports.

Hospitalization for MI or stroke

These analyses were restricted to NY City or State residents (n=5484). All incident MI and strokes identified in interviews, whether confirmed or not in medical charts, were present in the SPARCS database. In contrast, 49 events (19 among subjects with PTSD and 30 among subjects without PTSD) were recorded in SPARCS but had been missed by the active follow-up. As shown in Figure 4 and Table S7 for hospitalizations for MI or stroke in NY State, aHRs were 2.17 (1.41–3.32) for 112 MIs, 3.01 (1.84–4.93) for 77 strokes, and 2.40 (1.73–3.34) for pooled 180. Nine subjects had been hospitalized for both MI and stroke. The association of PTSD and hospitalization for pooled MI and stroke adjusted for age only was statistically significant among women too (HR=2.44, 1.05–5.55).

Of note, of the 415 lost to active follow-up, 228 (54.9%) had a record in SPARCS between January 2012 and December 2016. Of these 228, 4 had had an MI or a stroke, that is, 1.8% (95% CI: 0.0–3.5). This cumulative incidence of hospitalizations for MI or stroke in the lost to follow-up is of the same order of magnitude as that of 2.8% observed in the full cohort of (123/5971) during the same period.

Control of depression

In the full cohort there were 6 cases of MI or stroke in men with depression but no PTSD, and 29 cases of MI or stroke in men with both depression and PTSD. Among men free of depression the crude (not shown in the Figures), age-adjusted, and multivariate (shown in Figures 3 and 4) HRs of MI or stroke were, respectively, 1.94 (1.07–3.50), 2.19 (1.21–3.95), and 1.88 (1.01–3.49) for incidence, and 1.93 (1.19–3.02), 2.41 (1.49–3.75), and 2.27 (1.41–3.67) for hospitalization.

Mortality

There were 43 deaths identified in the cohort, of which 3 were from cardiovascular diseases, 18 from other causes and 22 from unknown causes.

PCL-Score

When using the continuous PCL-C score instead of the dichotomized PTSD variable, the aHR of pooled MI or stroke for each SD increase in PCL score was identical for both incident events and for hospitalizations: 1.58 (1.35–1.84) including recurrent cases, and 1.36 (1.17–1.95) excluding recurrent cases.

DISCUSSION

After 4 years of active follow-up of WTC-Heart, PTSD comes out as a strong, independent determinant of MI and stroke, in both men and women, before and after controlling for recognized use of a respirator during clean-up of debris, cardiovascular risk factors, and depression. Consistent hazard ratios were observed when restricting the analyses to discharge diagnoses of MI and stroke derived from linkage to a NY hospital registry and therefore independent of patient reports.

Because of its methodological strength (i.e., active follow-up, medical chart validation, and adjustment for depression) this study validates previous reports of exclusively male or female cohorts[4, 5] and those of the WTC-Registry reports.[11, 12] The independence of the PTSD and MI or stroke association from depression is consistent with most[4] but not all reports.[20, 21]

PSTD and CVD in women

The associations with PTSD were in the same order of magnitude for men and women, and, specifically, the association of PTSD and hospitalization for pooled MI and stroke was statistically significant in both genders. Of the three previous cohort studies which examined women, two had only women, [7, 10] and one lacked measured conventional cardiovascular risk factors.[11, 12] The average age of women at baseline in these 3 cohorts were 44.4 years,[7] 34y [10] and 32y,[12] whereas women in WTC-Heart were 51.1y at baseline. Thus, the present results add qualitatively to the evidence indicating that PTSD before menopause increases women's long-term risk of MI and stroke. [22]

Biological plausibility

PTSD is a polythetic disorder consisting of clusters of symptoms potentially impacting cardiometabolic risk.[23, 24] For example, PTSD may develop and persist because of fear manifested by nightmares, intense emotional and physiological re-experiencing of the trauma, active avoidance of cues or reminders of the trauma, and exaggerated startle response to threat.[25] Another cluster groups symptoms evoking major depression such as anhedonia, detachment, restricted affect, sleep difficulty and failing concentration.[26–28] In humans the stress associated with these PTSD dimensions may upregulate amygdalar activity, activating both the sympathetic nervous system, releasing inflammatory cells from the bone marrow, and leading to atherosclerotic inflammation, MI, and stroke. [29–36] The similar effect sizes in this study suggest similar mechanisms relating PTSD and incident MI or stroke in men and women.[37]

Recognized cardiovascular risk factors

Current smoking was the only recognized cardiovascular risk determinant in this cohort. Blood lipids, blood pressure, and body mass were not associated with MI or stroke. These recognized risk factors may play a role for individual cohort members, but they do not discriminate cardiovascular risk in this homogeneous cohort of blue-collar workers.

Because PTSD is more prevalent in this cohort (20.9%) than current smoking (7.1%) for aHRs of a similar magnitude of 2, about 3 times more cardiovascular cases are attributable to PTSD than to current smoking.

Dust exposure

None of the variables expressing exposure to the WTC dust, such as the timing and intensity of exposure to the dust cloud, or the donning of a protective mask, were independently associated with subsequent MI or stroke in these analyses.

Limitations

The exposure severity and cardiovascular profile of the sub-cohort of survivors enrolled in the WTC-Heart subjects may not be representative of the estimated 90,000 individuals that originally responded to the 9/11 disaster (Figure 5). However, given the homogeneity of the cohort of blue-collar worker in terms of cardiovascular risk, the selection process is unlikely to have biased the risk comparison among the enrollees between those who developed PTSD and those who did not. Moreover, the small losses to follow-up (7.9%) offers some protection against selection bias, in particular since we could establish that: 1) The proportion of lost to follow-up was identical in men (7.9%) and women (7.7%); 2) The subjects lost to follow-up had a similar cardiovascular risk profile as those who remained in the cohort (Table S8); 3) the cumulative risk of MI and stroke for more than half of those who were never contacted after baseline but had a record in the New York Sate SPARCS program was of the same order of magnitude as the risk of the rest of the cohort.

We were not able to obtain the causes of death for 22 participants and do not know which proportion of the lost to follow-up died of cardiovascular diseases. Incorporating cardiovascular deaths could have attenuated or exacerbated the present findings. However,

the small number of cardiovascular deaths expected given the age distribution of the cohort is unlikely to have qualitatively modified the cumulative risks of events, which progressively diverged over time between participants with and without PTSD (Figure 2).

Also, since participants were sampled consecutively among subjects attending the WTCHP either for their annual visit or for health problems, we may have included people visiting the clinic more frequently because of a poorer health problems than those only visiting the clinic once per year. However, as shown in Table S1 the demographics of WTC-Heart were virtually identical to those of the whole WTCHP in which it was nested. Prevalences of PSTD in WTC-Heart were almost identical to those published for the whole WTCHP.[38]

Additional limitations include a self-report of PTSD based on the PCL-C questionnaire; residual confounding from self-report of depression based on the PHQ-9 questionnaire and recall of respirator use and exposure to the dust cloud; and the lack of assessment of potential biological mediators and unmeasured confounding (e.g., prior trauma) of the association between PTSD and MI/Stroke. However, similar baseline heart rate for those with and without PTSD spoke against specific protective factor against PTSD in a subgroup of the cohort.

Strengths

Because WTC-Heart is nested within the health program (i.e., WTCHP) begun one year after the attack and currently providing lifetime free medical care for WTC-related medical conditions to first responders, the cohort study had a unique opportunity to recruit exposed workers 11 to 15 years after 9/11 as they entered ages at which incidence of CVD increases rapidly, and follow them up for four years with small losses (7.9%).

In agreement with previous WTC reports,[3] prevalence of PTSD in this cohort comprising a majority of non-trained responders was more than twice that in the general population. This excess prevalence of PTSD provided statistical power to study the association of PTSD and cardiovascular outcomes in both genders. The young age of the cohort participants, associated with low rates of MI or stroke, negatively affected statistical power but allowed us to reasonably assume that few cases were missed during the 2001–2012 period. The young age may also have facilitated singling out the effect of PTSD for lack of competing causes.

In terms of validity and confounding controls: analyses were performed before and after adjustment for measured recognized cardiovascular risk factors; in men, the association was observed in the subgroup free of depression; interview-based outcomes were tentatively confirmed in medical charts; findings for interview-based diagnoses of incident MIs and stroke were reproduced after linkage to hospitalization records.

Conclusions

Because of its design, this cohort study offers unique and strong evidence that WTC attack–related PTSD is a risk factor for MI and stroke, in men and women, and independently of recognized cardiovascular risk factors and depression.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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WHAT IS KNOWN

Post-traumatic stress disorder (PTSD) has been consistently shown to be an independent risk factor for incident myocardial infarct or stroke in cohort studies

However, despite the wealth of evidence, PTSD is still not recognized as a risk factor for CVD because available studies are all affected by one or several limitations.

WHAT THE STUDY ADDS

WTC-Heart offers unique and strong evidence that WTC attack–related PTSD is a risk factor for MI and stroke, in men and women, and independently of recognized cardiovascular risk factors and depression.

Because of its methodological strength (i.e., active follow-up, medical chart validation, and adjustment for depression) this study validates previous reports of exclusively male or female cohorts.

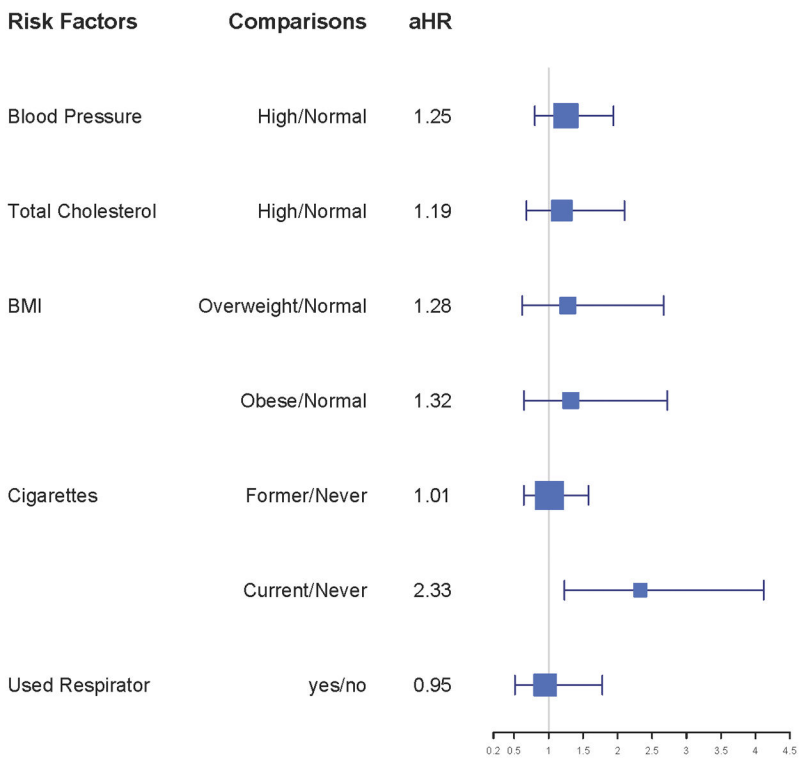

Figure 1.

Adjusted* hazard ratios (aHR) of pooled incident (including recurrent) myocardial infarctions and strokes and recognized risk factors in the WTC-Heart cohort (N=5971). New York, 2012–2016. *Adjusted for use of a respirator and for recognized cardiovascular risk factors: age, blood pressure, total cholesterol, body mass index, tobacco use, and, when relevant, gender.

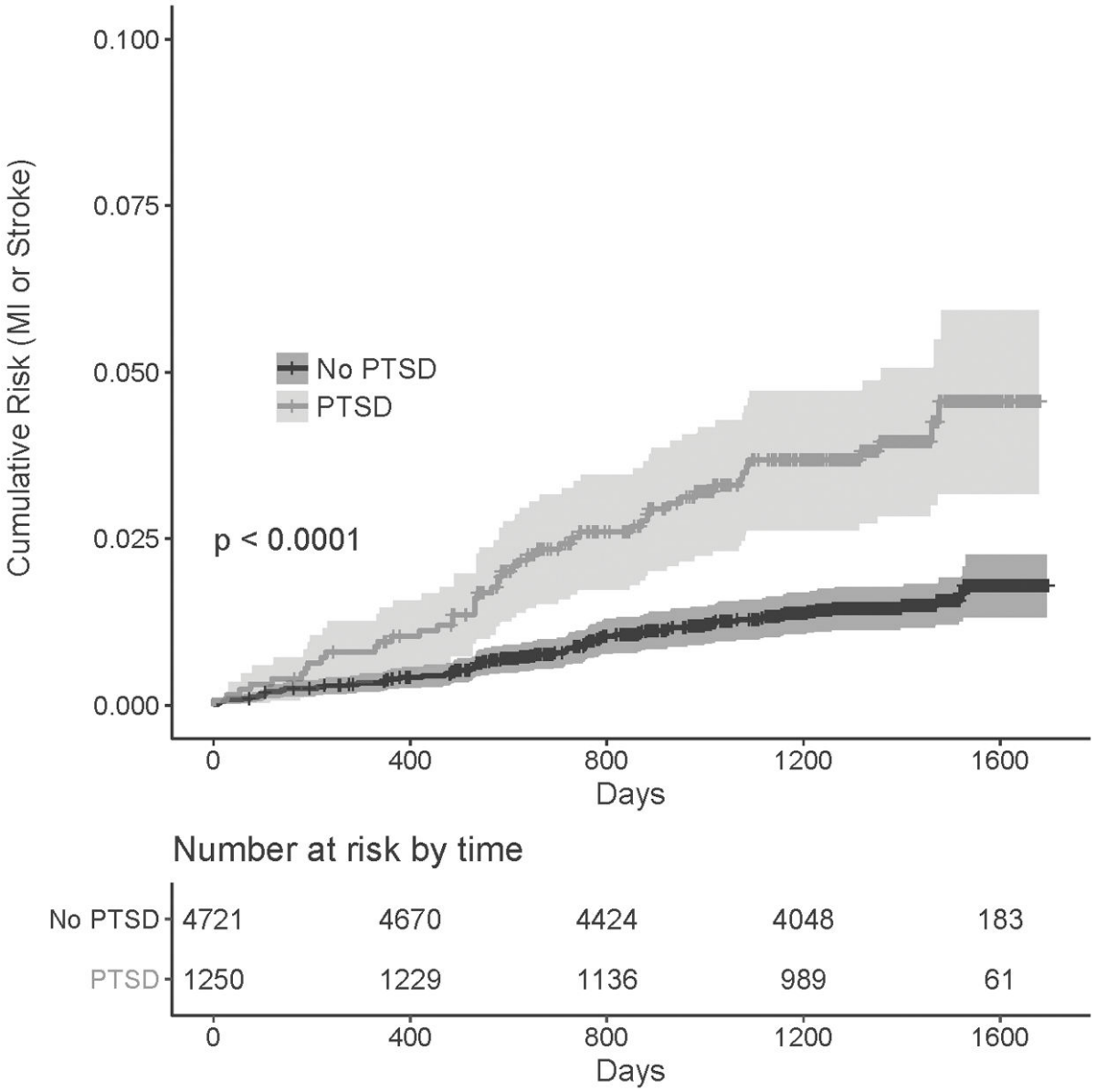

Figure 2.

Cumulative risk of pooled incident (including recurrent) myocardial infarctions and strokes by PTSD status in the WTC-Heart cohort (N=5971). New York, 2012–2016.

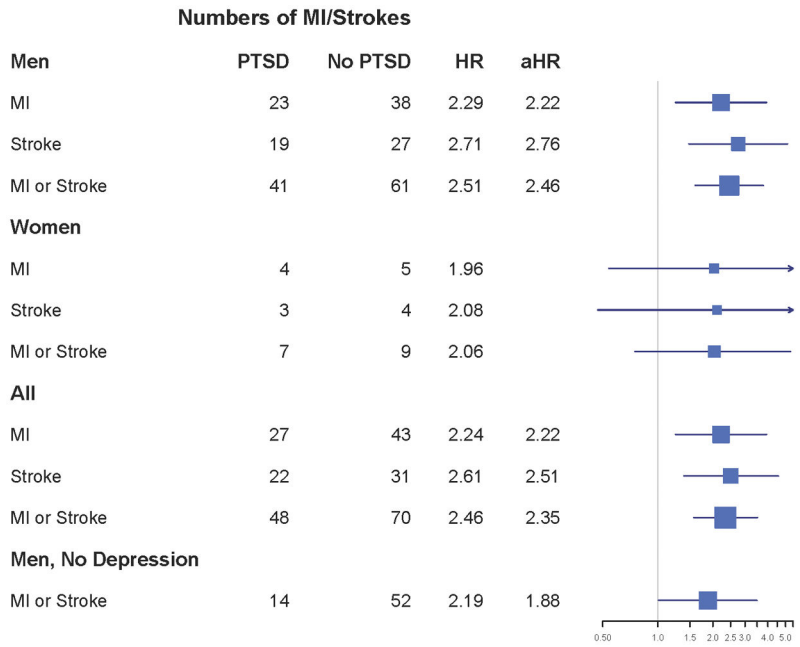

Figure 3.

Age-adjusted (HR) and adjusted* hazard ratios (aHR) for PTSD and pooled incident (including recurrent) myocardial infarctions and strokes in the WTC-Heart cohort (N=5971). New York, 2012–2016. *Adjusted for use of a respirator and for recognized cardiovascular risk factors: age, blood pressure, total cholesterol, body mass index, tobacco use, and, when relevant, gender.

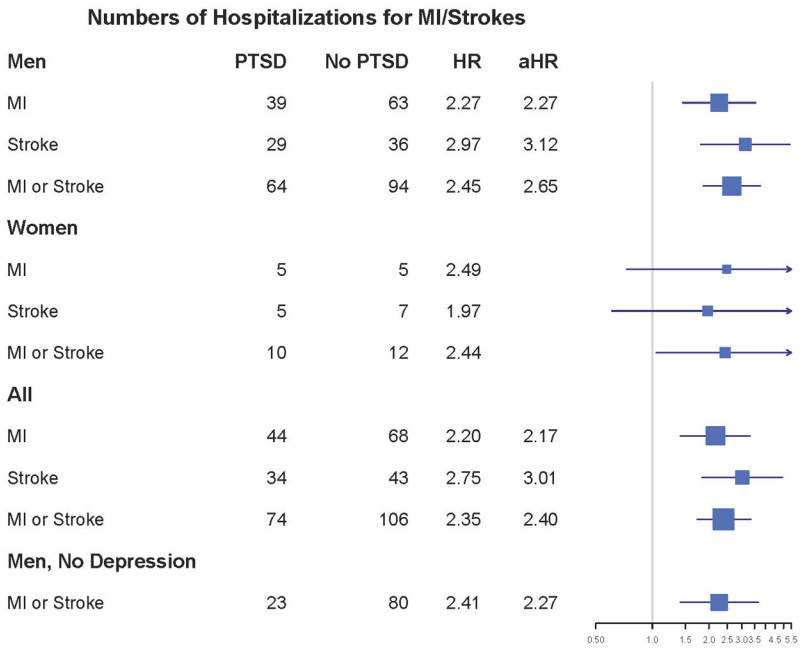

Figure 4.

Age-adjusted (HR) and adjusted* hazard ratios (aHR) for PTSD and hospitalizations for pooled (including recurrent) myocardial infarctions and strokes in the WTC-Heart cohort (N=5484). New York, 2012–2016. *Adjusted for use of a respirator and for recognized cardiovascular risk factors: age, blood pressure, total cholesterol, body mass index, tobacco use, and, when relevant, gender.

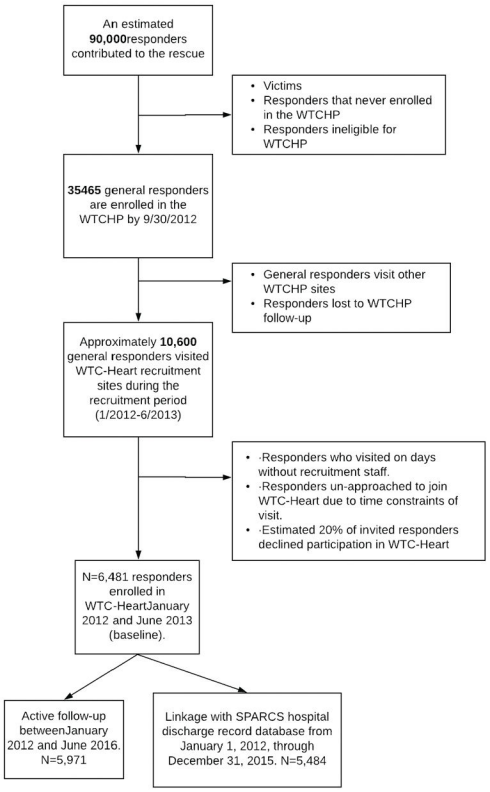

Figure 5.
Flowchart and timeline of the WTC Health Program and of WTC-Heart, 2001–20016.

Table 1

Baseline characteristics of men and women comprising the WTC-Heart cohort (n=5971). New York, 2012–2016.

		Men		Women	
Characteristics	**Categories**	**No.**	**%[a]**	**No.**	**%***
Total		4942	100	1029	100
Race	White	2857	57.8	372	36.2
	Black	669	13.5	240	23.3
	Missing	1141	23.1	348	33.8
Ethnicity	Hispanic	1338	27.1	432	42.0
	Missing	96	1.9	19	1.9
Responder	Trained	2077	42.0	424	41.2
	Non-Traditional	2359	47.7	517	50.2
	Missing	506	10.2	88	8.6
Smoking	Never	2688	54.4	611	59.4
	Current	351	7.1	64	6.2
	Former	1383	28.0	240	23.2
	Missing	520	10.5	114	11.1
Protective mask on 9/11	Donned	4334	87.7	825	80.1
	Missing	99	2.0	23	2.2
Blood pressure	High (Systolic > 140 or diastolic>90 mmHg)	1098	22.2	124	12.1
	Missing	143	2.9	47	4.6
Total cholesterol	High (>6.2 mmol/L)	517	10.5	146	14.2
	Missing	n/a	n/a	n/a	n/a
HDL	Low (men: <1, women:<1.3 mmol/L)	1069	21.6	527	51.2
	Missing	608	12.3	115	11.2
BMI	Normal (<25kg/m^3)	458	9.3	258	25.1
	Overweight (25–29.9 kg/m^2)	2012	40.7	382	37.1
	Obese (30+ kg/m^2)	2472	50.0	389	37.8
	Missing	n/a	n/a	n/a	n/a
PTSD	Yes	984	19.9	266	25.9
	Missing	n/a	n/a	n/a	n/a

		Men		Women	
Characteristics	**Categories**	**No.**	**%[a]**	**No.**	**%***
Depression	Yes	574	11.6	185	18.0
	Missing	248	5.0	59	5.7
Menopause	Yes	--	--	420	40.8
	Missing	--	--	2	0.2
Education	College graduates	1319	26.7	379	36.8
	Missing	570	11.5	112	10.9