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The association of Gastro-esophageal reflux disease and self-reported myocardial-infarction among World Trade Center disaster exposed persons

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ABSTRACT

Objective: Gastroesophageal reflux disease (GERD) is the third most prevalent condition among responders and survivors who are monitored and treated in World Trade Center Health Program. Given the evidence of a potential association between GERD and myocardial-infarction, this study used observational data from the World Trade Center Health Registry (WTCHR) located in New York City Department of Health and Mental Hygiene, to evaluate the risk of myocardial-infarction from GERD.

Methods: The study used five waves of survey data from 2003 to 2020 of the WTCHR to estimate the hazard ratio for persons who reported both GERD and gastroesophageal symptoms (GERS) compared to those with no-GERD in relation to a self-reported myocardial infarction. GERD cases and no-GERD controls were matched by age and sex; pairs of cases and controls were assigned index GERD diagnosis year for start of observation.

Results: With a final sample of 13,234, proportional hazard analysis yielded an unadjusted HR = 2.0 (95 % CI, 1.67, 2.49) and HR = 1.7 (95 % CI, 1.32, 2.19) after adjusting for putative confounders.

Conclusions: A nearly two-fold risk of myocardial-infarction among those with 9/11 disaster related GERD is concerning given the high prevalence of GERD among persons who were exposed to the 9/11 disaster.

1. Introduction

The terrorist attacks on the World Trade Center (WTC) in New York City on September 11, 2001, exposed an estimated 400,000 people who were present on 9/11 or living or working in the area in following weeks. Over the past 20 years, surveillance and observational research has identified mental health disorders (post-traumatic stress disorder, depression), aerodigestive disorders including asthma and chronic rhinosinusitis, Gastro-esophageal reflux disease (GERD), cardiovascular diseases and autoimmune diseases as associated with exposure on 9/11 (Daniels et al., 2021).

In addition, the occurrence of 9/11 related conditions such as chronic GERD has shown to promote or exacerbate post-traumatic stress disorder (PTSD) and asthma (Li et al., 2016). Although not addressed in 9/11 related research, there is evidence that GERD may also increase the risk for coronary heart disease (Gries et al., 2023). This evidence has included cross-sectional (Jansson et al., 2008), longitudinal (Chen et al., 2016; Lei et al., 2017), and cloud-based data sources (Eisa et al., 2020). Mendelian randomization has also been used to establish a causal relationship between GERD and cardiovascular diseases (Sun et al., 2022) and specifically atherosclerosis (Song et al., 2022). (See Gries et al., 2023, Table 1 for summary of studies on GERD and MI).

The connection between and GERD and coronary heart disease may stem from increased levels of inflammatory agents from chronic GERD (cytokines Interleukin-1, 6, 8) in the blood stream, which promote atherosclerotic lesions and vulnerability to acute coronary trauma through plaque rupture (Gries et al., 2023; Eisa et al., 2020). Also, there is potential autonomic imbalance that is mediated by the vagus nerve as a result of endothelial inflammation of the esophagus (Thayer et al., 2009).

The objective of the current study is to evaluate the association between self-reported GERD and self-reported diagnosed non-fatal myocardial-infarctions. Cohort based longitudinal data from the WTC Health Registry (WTCHR) provided 18 years of follow-up data including first year of diagnosis of GERD since 9/11 and reported year of diagnosis of myocardial-infarction. It is hypothesized that symptomatic GERD independently increases the risk of myocardial-infarction.

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Table 1
Descriptive analysis of exposure variable-gastro-esophageal reflux disease, by self-reported myocardial infarction, and demographic and confounder variables among adult World Trade Center Health Registry enrollees, 2002 to 2021 (N = 13,234).

	Gastroesophageal Reflux Disease				
	Yes		No		
Variable	n	%	n	%	*P* value**
Self-reported myocardial infarction					<0.0001
Yes	418	6.3	208	3.1	
No	6199	93.7	6409	96.9	
Sex***					1.0
Male	4362	65.9	4362	65.9	
Female	2255	34.1	2255	34.1	
Age at Wave 1***					0.96
18–29	337	5.1	324	4.9	
30–39	1526	23.1	1551	23.4	
40–49	2682	40.5	2683	40.5	
50–59	1672	25.3	1664	25.1	
60–69	374	5.7	374	5.7	
70–79	26	0.4	21	0.3	
Race/ethnic group					<0.0001
White	4940	74.7	4937	74.6	
Black	455	6.9	688	10.4	
Hispanic	803	12.1	502	7.6	
Asian	218	3.3	351	5.3	
Other	201	3.0	139	2.1	
Level of exposure to WTC disaster					<0.0001
Low/none	2596	39.2	3632	54.9	
Moderate	3144	47.5	2552	38.6	
High	977	14.8	433	6.5	
History of obesity*					<0.0001
Yes	3385	51.3	4313	65.6	
No	3214	48.7	2257	34.4	
History of probable PTSD*					<0.0001
Yes	3887	58.8	5730	86.7	
No	2729	41.2	883	13.3	
History of current smoking*					<0.0001
Yes	5412	81.8	5630	85.1	
No	1202	18.2	984	14.9	
History of binge drinking*					<0.0001
Yes	2810	42.4	2365	35.7	
No	3802	57.5	4246	64.3	
Self-reported diagnosed Angina*					>0.0001
Yes	6233	94.2	6383	96.5	
No	384	5.8	234	3.5	

* Missing: 65 for History of obesity; 5 for History of probable PTSD; 6 for History of current smoking; 11 for History of binge drinking;208 for Self-reported diagnosed angina.
** Chi square.
*** Sex and age are not included in analysis models.

2. Methods

2.1. Data source and study population

The WTCHR was established in 2002 to monitor the physical and mental health consequences of the September 11, 2001 terrorist attacks in New York City. In 2003–2004, 71,424 enrollees completed the baseline survey (Wave one). Four follow-up surveys between 2006 and 2020 have been done. (Brackbill et al., 2009; Sisti et al., 2025).

The WTCHR sample included enrollees who completed Waves one (2003–04, n = 71,424) and two (2006–07, n = 46,600) and three (2011–12, n = 43,132) or four (2015–16, n = 36,862) and Wave five (2020–21, n = 27,408). Interview data for enrollees under 18 at Wave one, deceased, or unknown whether Wave five participants were excluded (n = 362). Those missing GERD diagnosis information were also excluded leaving an available sample of 25,471 before defining symptomatic GERD and no-GERD exposure and self-reported myocardial-infarction outcome (see Fig. 1).

2.2. Study variables

2.2.1. Exposure: symptomatic GERD

Symptomatic GERD was defined by both self-reported physician diagnosis and self-reported gastroesophageal reflux symptoms (GERS).

Starting at Wave 3, the survey asked about physician diagnosed GERD with the lead question: "Have you ever been told by a doctor or other health professional that you had any of these conditions?" Enrollees who reported GERD with a diagnosis year before 2002 or after 2016 were excluded from analysis. This exclusion assured temporality for myocardial-infarction first reported in Wave five (year of diagnosis for myocardial-infarction, 2017 to 2021).

For symptoms, information on frequency, medication, and doctor visits was obtained with the following questions: For Wave two: "Have you experienced any of these symptoms in the last 30 days? – heartburn, indigestion, or reflux" and for "How many days?"; For Waves three and four: "In the last 30 days have you experienced heart burn or acid reflux?" and "How many days?", "In the last 12 months how often have you experienced heart burn or acid reflux – never, <1 once a month, about 1 once a month, about once a week, at least twice a week", "Seen a doctor in last 12 months? – heart burn or reflux", and "Are you taking medication for heart burn?" Enrollees diagnosed with GERD had to report a symptom frequency more than once a month, seen a doctor for GERS or took medication for GERS; otherwise those with GERD diagnosis who did not report symptoms or treatment were excluded from analysis (See Fig. 1, excluded n = 2943).

The no-GERD group was defined by those who did not report a diagnosis of GERD and did not report GERS, seeing doctor for heartburn or taking medication for GERS on any of the WTCHR surveys.

The GERD group was 6770 after excluding 2943 of those missing year of diagnosis or year of diagnosis prior to 2002 or after 2016 and 274 who reported no GERS. The no-GERD group was 8110 after excluding those with GERS or having any medication or doctor visits related to GERS in Waves two to four. The total sample before defining the myocardial-infarction outcome was 14,880 (Fig. 1).

2.2.2. Outcome: self-reported myocardial-infarction

Myocardial-infarction was defined by the response to the question "Have you ever been told by a doctor or other health care professional that you had heart attack or myocardial-infarction?" and reporting a diagnosis year in Waves two to five. Those with missing data on myocardial-infarction or the year of diagnosis prior to 2002 were excluded (N = 254), resulting in pre-matching dataset of 14,626.

2.2.3. Confounders

A Directed Acyclic Graph (DAG) was used to identify confounders for the analytic model (Fig. 2).

The relationships provided by the DAG can be summarized as follows: Sex, age, race/ethnicity are assumed to have associations with both GERD and myocardial-infarctions. WTC disaster exposure is a confounder because it is associated with GERD (Li et al., 2016), diabetes (Miller-Archie et al., 2014), and PTSD (Brackbill et al., 2009) and myocardial-infarction (Jordan et al., 2011). Obesity and PTSD are both associated with both GERD/GERS (Kent, 2024; Li et al., 2016)) and myocardial-infarction (Edmondson et al., 2013; Powell-Wiley et al., 2021) and thus are also confounders. Smoking and alcohol consumption have been shown to exacerbate GERD and are also risk factors for heart

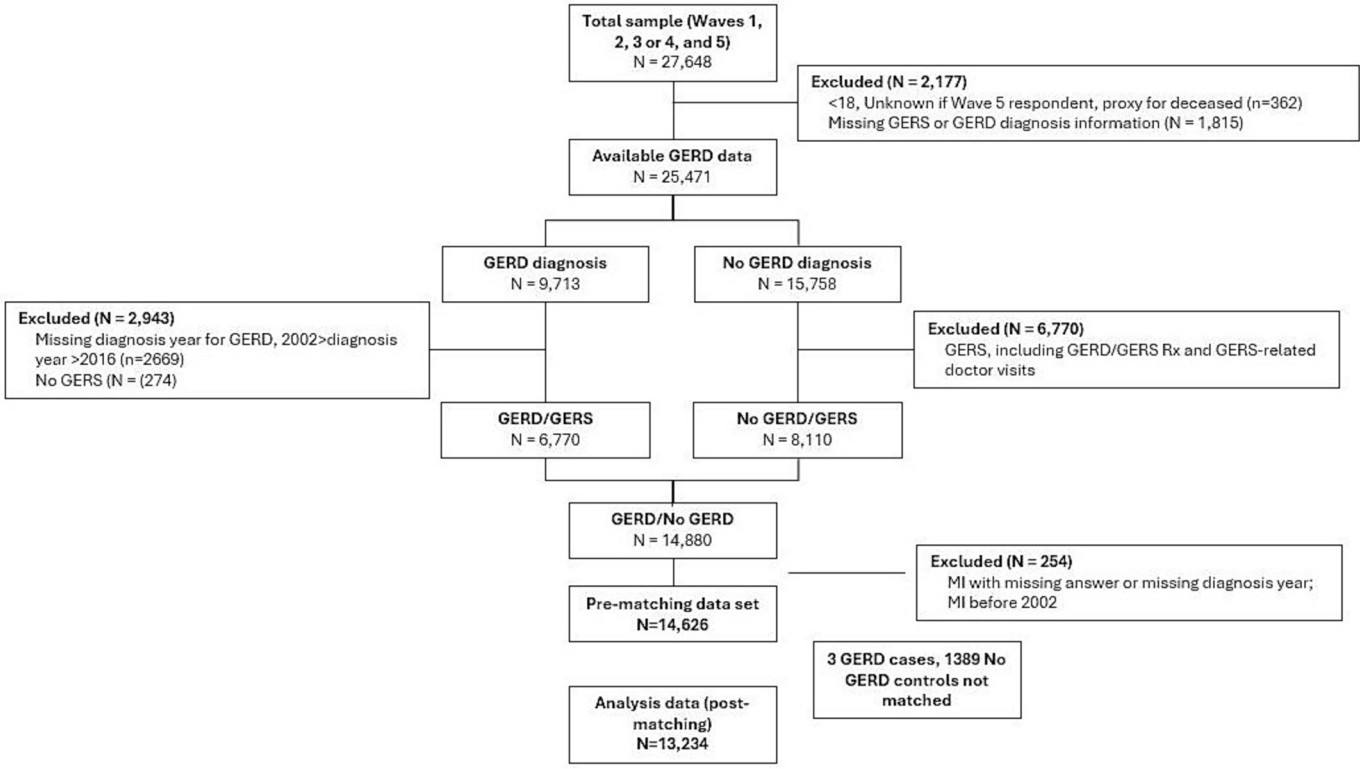

Fig. 1. Flow chart showing sample creation for gastroesophageal reflux disease and self-reported myocardial infarction analysis, World Trade Center Health Registry, 2002–2021.

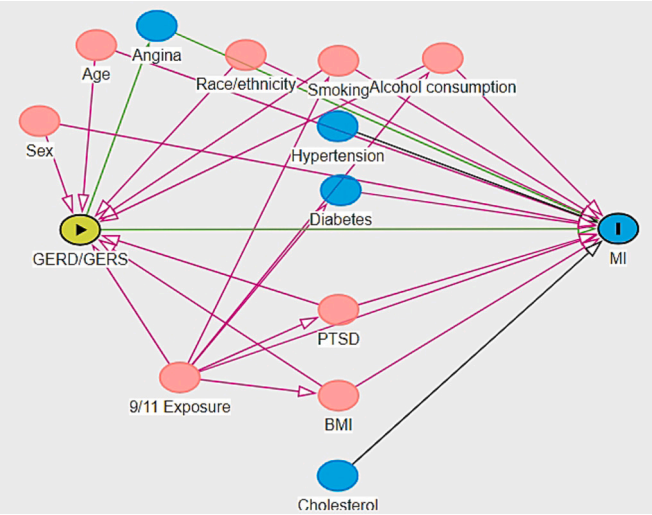

Fig. 2. Direct Acyclic Graph (DAG) used to identify confounders for analytical model for gastroesophageal reflux disease and self-reported myocardial-infarction, World Trade Center Health Registry, 2002 to 2021.

disease and thus were also included as confounders in the analytic models. Angina acts as a mediator between GERD and myocardial-infarctions [GERD and angina (Kato et al., 2009); and angina and myocardial-infarction (Dunder et al., 2002)] and is thus not included in model.

The definition of confounders used available data from Waves one to four. Race and/ethnicity were grouped into five categories (White Non-Hispanic; Black Non-Hispanic; Hispanic; Asian; and Non-Hispanic, race unknown).

WTC disaster exposure, based on Waves one and two, was defined using a 12-item summary measurement that was used in previous WTCHR reports (Brackbill et al., 2013). The categories of exposure were none/low (0–2), medium (3–5), and high (6 or more). Each item in the scale has shown an association with physical or mental health outcomes in previous Registry studies (Brackbill et al., 2013).

History of obesity used self-reported weight and height available from Waves three to five with reporting of BMI = >30 at any wave.

History of probable PTSD was assessed at Waves one to four using the PTSD Checklist-Specific (PCL-S), a 17-item self-reported symptom scale specifically related to the events of September 11. The 17 items correspond to the Diagnostic and Statistical Manual of Mental Disorders: DSM IV, 1994 PTSD symptoms. Enrollees were asked to rate the degree to which they were bothered by symptoms in the past four weeks from one (not at all) to five (extremely). Responses for the 17 items were summed, with a possible total score of 17–85. A PCL-S score ≥44 at any one of Waves one to four indicated a history of probable PTSD (Ruggiero et al., 2003). Missing was defined for all variables as missing a response at all survey waves.

Smoking history was ever reporting current smoking in contrast to reporting only former or never smoking at any or all survey waves. Binge drinking was based on reporting an episode of five or more drinks for men and four or more drinks for women on a single occasion. If an enrollee reported having a binge drinking episode at least once in the last 30 days (preceding the survey date) anytime in Waves two to four, they were defined as having a history of binge drinking.

Hypertension and high cholesterol, which are both risk factors for myocardial infarction were not included in the model because they are not known be associated with GERD.

2.3. Statistical methods

2.3.1. Matching to create pairs of GERD and no-GERD

GERD and no-GERD were matched at 1:1 ratio on age at the year of GERD diagnosis (plus/minus one year of age difference) and sex using a

matching algorithm typically used for matched case-control studies (Grandits and Neuhaus, 2010), with cases and controls for each matched pair having the same year of diagnosis or index year, similar to matching of cases and controls by Lei et al. (2017). The purpose of the match was to assign the year of GERD diagnosis to the matched no-GERD individual as a start year for the follow-up period so that the start year (index year) of the observation was the same for GERD and no-GERD individuals in the present analysis. The final analytic sample consisted of 13,234 (individuals (6617 matched pairs), with the exclusion of 3 persons with GERD and 1389 with no-GERD due to the unavailability of a matching person with GERD. Only 32 self-reported myocardial-infarctions were removed after matching.

2.3.2. Statistical analysis

Descriptive analysis consisted of computing percentages among those who reported GERD by outcome of myocardial infarction, demographic and confounding variables. In addition, Chi-squares were computed for GERD and each variable; *p*-values for Chi-squares were reported.

Unadjusted and adjusted Cox regression models were run. Adjusted models included confounders regardless of bivariate statistical significance. The start of survival time was index year based on GERD diagnosis year. End time for myocardial-infarction was defined as its year of first diagnosis and was treated as uncensored. The Wave five interview year (2020/2021) was used as the end time for enrollees who did not experience the outcome and were treated as censored. The Cox regression included an option that stratified the analysis by the matched pairs. The assumption for proportional hazard over time was evaluated by the Schoenfeld's global test for proportional hazards (Abeysekera and Soorriyarachich, 2009). A non-significant time exposure-(GERD) interaction indicated that proportionality assumption was not violated.

Several additional sensitivity analyses were performed. First, a logistic odds ratio was computed for when: 1) GERD was defined including those with no symptoms; 2) no-GERD group included those with no diagnosis but had symptoms; and 3) the sample before matching compared to sample after matching.

Second, angina cases were excluded in order to assess whether the presence or absence of diagnosed angina altered the association between GERD and myocardial-infarction. All analyses were performed using SAS version 9.4 (SAS Institute, Cary, North Carolina).

2.4. Ethics approval

The WTCHR was approved by the institutional review boards of the US Centers for Disease Control and Prevention and the New York City Department of Health and Mental Hygiene (Protocol # 02-058) with participants providing verbal consent at enrollment in the WTCHR and implicit consent at each follow-up survey.

3. Results

3.1. Descriptive and proportional hazard analysis

The sex and age distributions for GERD and no-GERD were nearly identical, which is expected as a result of matching. On the other hand, race/ethnic group and analysis covariates were significantly associated with the exposure variable (*p*-value <0.0001) (Table 1).

In an unadjusted model, there was a hazard ratio (HR) of 2.03 (95 % CI, 1.67, 2.49) for GERD as a precursor for myocardial-infarction (Table 2). With confounders included, the HR was attenuated with an adjusted HR (AHR) = 1.7 (95 % CI, 1.32, 2.19).

3.2. Sensitivity analysis

There was no difference detected in the estimate of the association between GERD and myocardial infarction whether GERD was defined considering GERS or not considering GERS. In addition, there was no difference in the estimate of association between GERD and myocardial infarction before or after matching.

When all angina cases that have a diagnosis year were removed, the unadjusted HR was 1.9 (95 % CI, 1.49, 2.38, $N = 12{,}602$).

Table 2
Adjusted and unadjusted hazard ratios for myocardial infarction by Gastro-esophageal reflux disease among adult World Trade Center Health Registry Enrollees, 2002 to 2021 (N = 13,234).

Variable	HR	(95 % CI)	HR	(95 % CI)
Gastro-esophageal reflux disease	Unadjusted		Adjusted*	
Yes	2.0	1.67, 2.49	1.7	1.32, 2.19
No	1.0		1.0	

* Adjusted for race/ethnic group, 9/11 exposure, history of obesity, probable post-traumatic stress disorder, current smoking and binge drinking.

4. Discussion

In this study of persons exposed to the WTC disaster, we observed a strong relationship between GERD and the occurrence of myocardial-infarction. After adjustment for confounders (race/ethnic group, obesity, WTC disaster exposure, PTSD, smoking and alcohol consumption), there was still a 70 % increased risk of myocardial-infarction given a history of GERD and GERS over 18 years of observation.

These results have implications for 9/11 related conditions since GERD is highly prevalent among WTC Health Program clients. Among the WTC Health Program covered conditions, GERS has been the second and currently third after cancer following chronic rhinosinusitis (Daniels et al., 2021). GERD is also a predominant condition in a 9/11 condition complex referred to as aero-digestive conditions including obstructive airway disease (asthma, chronic bronchitis, chronic obstructive pulmonary disease/emphysema), and upper respiratory diseases (*e.g.* chronic rhinosinusitis) (Santiago-Colon et al., 2020). Most previous reports on the association of GERD with heart disease are consistent with this study. Longitudinal studies using the same source data reported significant hazard ratios for GERD and cardiovascular disease outcomes including acute coronary syndrome (AHR = 1.62), angina (AHR = 1.49), and chronic ischemic heart disease (AHR = 1.47) (Chen et al., 2016) and for acute myocardial-infarction (AHR = 1.48) (Lei et al., 2017). A separate cross-sectional study reported significant associations for self-reported GERD and myocardial-infarction (adjusted odds ratio (AOR) = 1.2), angina (AOR = 1.9), and stroke (AOR = 1.3) (Jansson et al., 2008). There was one study in the United Kingdom using General Research Practice data that found no association between GERS and myocardial-infarction after identifying patients with GERS and myocardial-infarction and controlling for other co-morbid disease risk factors, including hypertension, diabetes, hyperlipidemia, and obesity (Johanson et al., 2003). Nonetheless a recent report by Sun et al. (2022) in a Mendelian randomization approach included a multitude of risk factors and still found a statistically significant association between GERS and heart disease. GERD is also associated with these other risk factors for coronary disease: hypertension (Wei et al., 2024), diabetes (Sun et al., 2015) and obesity (Freidenberg and Xanthopoulos, 2008), although the directionality of these associations is unclear.

Several mechanisms have been postulated to explain the effects of GERD on heart function. First, the esophagus and heart share common visceral pain receptors in the spinal cord and vagal nerve innervation. Also, there is esophago-cardio reflex where if the esophagus is stimulated by acid reflux, the blood flow in the heart is reduced. It has been found that this did not occur in heart transplant patients, who have denervated hearts (Dobrzycki et al., 2005).

Second, chronic GERD could increase the risk of coronary heart disease and subsequent acute myocardial-infarction through the release of cytokines and other pro-inflammatory agents in common with arterial

inflammatory agents that cause damage to endothelial layer of the coronary arteries (Gries et al., 2023). Key inflammatory mediators between the esophageal distress and heart ischemia include IL-6, IL-8, platelet activating factor and other cytokines, which are produced in severe chronic esophageal reflux (Altomare et al., 2013). The role of IL-6 was implicated by showing that medicinal reduction of IL-6 resulted in less risk of coronary heart disease (Swerdlow et al., 2012).

Angina pectoris (angina) which in some cases is linked with reflux disease (Smith and Papp, 1962) and which also has been established neurologically as the gastro-cardio reflex (Dobrzycki et al., 2005) was evaluated in this study as a concomitant outcome to myocardial-infarction. Approximately 30 % of the myocardial-infarction cases had angina in their diagnostic history with 33 % of these with the same year of diagnosis. When all reported angina cases were removed from the analysis the hazard ratio for GERD/GERS and myocardial-infarction remained significant indicating that the association was independent of the presence of angina.

Strengths and limitations. The major strength of this study is that we had longitudinal cohort data from which we could assess the likelihood that self-reported myocardial-infarction increased due to GERD. The study also had a long time period of 18 years to use for assessing these relationships and was able to parse this period in order to evaluate continuity of the hazard. However, there are a number of evident limitations in this observational study. First, this study cannot rule out other unmeasured factors even though an attempt was made to control for confounders. Second, the study relied on self-report of gastro-esophageal symptoms and myocardial infarction including recall of physician imparted diagnosis and year when this diagnosis was first delivered to the subject. The reliability of self-report may also be differential between the conditions included in this analysis. For instance, it is more likely that a person will recall the experience and diagnosis of a myocardial-infarction and the year it occurred *versus* other more common conditions such as GERD. Third, we may be underestimating the risk of myocardial-infarction among those with GERD, because persons who died because of myocardial-infarction would not have had an opportunity to report a myocardial-infarction in a follow-up wave. Thus, this paper is limited to an evaluation of the association between GERD and myocardial infarction using available self-report. Further clinically based studies would be done to verify any causal relationship. In any case, our results for GERD and myocardial-infarction are collaborated by other reports, some of which used clinical data to define GERD as well as other risk factors, in addition to myocardial-infarction outcome.

5. Conclusion

The finding of a two-fold association between GERD and myocardial-infarction suggests it is important for WTC Health Programs to take note of this relationship because of the high prevalence of GERD and GERS among their client population. It is recommended that persons with GERD be monitored by their primary care physicians more closely for indicators of impending myocardial infarction, especially among those with existing risk factors.

CRediT authorship contribution statement

Robert Brackbill: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Ayda Ahmadi:** Software, Methodology, Formal analysis. **Howard Alper:** Writing – review & editing, Methodology. **Jiehui Li:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization. **Shengchao Yu:** Writing – review & editing, Methodology, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

The data that has been used is confidential.

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