Living in areas with different levels of earthquake damage and association with risk of cardiovascular disease: a cohort-linkage study

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Summary

Background Cardiovascular disease rates are known to increase immediately after a severe earthquake. However, less is known about the magnitude of this increase over time in relation to the amount of housing damage. We assessed the effect of area housing damage from a major earthquake sequence in Christchurch, Canterbury province, New Zealand, on cardiovascular disease-related hospital admissions and deaths.

Methods For this cohort-linkage study, we used linked administrative datasets from the Statistics New Zealand Integrated Data Infrastructure to identify individuals aged 45 years or older living in Christchurch from the date of the first earthquake on Sept 4, 2010. Individuals were assigned the average damage level for their residential meshblock (small neighbourhood generally comprising 10–50 dwellings) using the insurance-assessed residential building damage costs obtained from the Earthquake Commission as a proportion of property value. We calculated the rates of cardiovascular disease-related hospital admissions (including myocardial infarction) and cardiovascular disease-related mortality and rate ratios (adjusted for age, sex, ethnicity, small-area deprivation index, and personal income) by level of housing damage in the first year and the 4 subsequent years after the earthquake. The rate ratio association between earthquake housing damage and cardiovascular event was examined by Poisson regression, and linear test of trends across damage categories was done by regression modeling.

Findings We identified 179 000 residents living in the earthquake-affected region of Christchurch, of whom 148 000 had complete data. For the first 3 months after the Feb 22, 2011 earthquake, the Poisson regression-adjusted rate ratio (RR) for cardiovascular disease-related hospital admissions for residents from areas that were most damaged (compared with residents from the least damaged areas) was 1·12 (95% CI 0·96–1·32; test for linear trend p=0·239). In the first year after the earthquake sequence, for residents from areas that were most damaged (*vs* the least damaged areas), Poisson regression-adjusted RRs were 1·10 (1·01–1·21; test for linear trend p=0·068) for cardiovascular disease-related hospital admissions, 1·22 (1·00–1·48; p=0·036) for myocardial infarction-related hospital admissions, and 1·25 (1·06–1·47; p=0·105) for cardiovascular disease-related mortality, corresponding to an excess of 66 (95% CI 7–125) cardiovascular disease-related hospital admissions, including 29 (0–53) additional myocardial infarction-related hospital admissions and 46 (13–73) additional deaths from cardiovascular disease. In the 4 subsequent years, we found no evidence of an association of these outcomes with earthquake damage.

Interpretation Rates of cardiovascular disease and myocardial infarction were increased in people living in areas with more severely damaged homes in the first year after a major earthquake. Policy responses to reduce the effect of earthquake damage on cardiovascular disease could include pre-earthquake measures to minimise building damage, early wellbeing interventions within the first year to address post-earthquake stress, and enhanced provision of cardiovascular disease prevention and treatment services.

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Introduction

Several studies have reported increased incidence of cardiovascular disease events in the weeks following earthquakes and other major natural disasters.[1–6] The 1994 earthquake in Northridge, CA, USA, was followed by a 35% increase in the incidence of acute myocardial infarctions in the week after the earthquake compared with the week before.[1] An even greater increase in myocardial infarction was seen after the 1995 Hanshin-Awaji earthquake in Japan[2] and this increase was associated with the percentage of houses destroyed in a region.[3] In the 6 weeks after the 1999 earthquake in Taiwan, the rate of hospital admission with myocardial infarction increased significantly compared with the same period in the previous year.[4] In the first 3 weeks after the Great East Japan Earthquake in 2011, the

Research in context

Evidence before this study
Most earthquake studies examine the effect of earthquakes on cardiovascular disease immediately after the impact and show an acute increase in the incidence of cardiovascular disease such as myocardial infarction and Takotsubo cardiomyopathy. Few earthquake studies have examined cardiovascular disease outcomes by level of housing damage. We searched Google Scholar using the terms "earthquake" and ("cardiovascular" or "heart" or "cardiac" or "myocardial") and ("damage" or "housing") for articles published in any language between database inception and April 27, 2017. We also examined reference lists from review articles.

Added value of this study
Comprehensive population-wide linked data on hospital admissions and mortality, and insurance-assessed damage on housing allowed us to do a long-term analysis of the effect of earthquake-related housing damage on cardiovascular disease in a major earthquake sequence in Christchurch, Canterbury province, New Zealand. We found increased rates of hospital admissions related to myocardial infarction and cardiovascular disease, and increased cardiovascular disease-related mortality in the first year after earthquake onset but no evidence of an increase in cardiovascular events after the first year, despite the ongoing psychologically stressful process of insurance claim settlement, rebuilding, and relocation.

Implications of all the available evidence
Mitigation of the effect of major earthquake damage on cardiovascular disease risk could include improved building standards, early interventions to address post-earthquake stress, and enhanced provision of cardiac services.

incidence of acute coronary syndrome and cardiopulmonary arrest increased significantly, with an increased incidence of heart failure for more than 6 weeks.[5]

An earthquake is a largely random, external shock that is experienced differently by individuals with different levels of exposure to loss and stress. Recovery from damage caused by a significant earthquake can take years, with disruption to housing and infrastructure, displacement, and loss of amenity affecting everyday functioning.[7] However, few follow-up studies of cardiovascular disease rates after an earthquake have investigated the effect of chronic stress on cardiovascular outcomes by level of property damage and few have done this beyond 6 months. The risk of cardiovascular disease by distance from the epicentre has been examined;[1] however, distance is not always linked to the amount of earthquake damage (figure 1) because damage is also related to building features and the natural landscape. After a devastating earthquake in 1988 in Armenia (with 25 000 estimated fatalities), employees of the country's ministry of health (n=35 043) were interviewed for up to 4 years after the earthquake, revealing a higher probability of self-reported or family-reported heart disease in individuals more affected by loss of material possessions.[8] Our study provides the power (148 000 adults aged 45 years and older) to objectively examine cardiovascular disease outcomes after a less severe earthquake event in Canterbury province, New Zealand. We use national-level data held on hospital admissions for all people in the earthquake and non-earthquakes areas, and high-resolution geocoded data on earthquake impact using insurance-assessed residential damage costs. We examine cardiovascular disease outcomes in the first year and the second to fifth years in view of the prolonged period of physical and social disruption.

Both acute and chronic stress can precipitate cardiovascular disease.[9–11] Acute stress has a triggering effect on myocardial infarction via transient vasoconstriction and thrombotic effects that lead to plaque disruption and thrombosis,[12] and acute stress is also a trigger for Takotsubo cardiomyopathy (broken heart syndrome).[13] Sudden stress leads to increased heart rate, blood pressure, myocardial oxygen demand, activation of the sympathetic nervous system, inflammatory cytokines, and platelet aggregation.[12] Furthermore, chronic anxiety and depression are consistently associated with heart disease[10,14–16] and poorer outcomes for patients with cardiovascular disease.[17] Psychological distress is a key pathway by which earthquake housing damage can increase the risk of cardiovascular disease. Studies from Canterbury show the effect of the earthquakes on mental health.[18,19] In a birth cohort study,[19] participants aged 35 years at the time of the earthquakes who were exposed to the earthquakes were 1·4 times (95% CI 1·1–1·7) more likely to have mental health disorders than participants not exposed, 20–24 months after the earthquake onset (accounting for 11–13% of the overall prevalence of mental health disorders in the cohort).

The 2010–11 Canterbury earthquakes provide a modern natural experiment to examine the effect of the amount of housing damage and subsequent associated psychological stress on the incidence of hospital admissions due cardiovascular disease. The Canterbury earthquake sequence began with a magnitude 7·1 earthquake early on the morning of Sept 4, 2010, which caused widespread damage. The worst damage occurred from the magnitude 6·3 earthquake centred on Christchurch, occurring at lunchtime Feb 22, 2011. The sequence of earthquakes caused 185 deaths, injured at least 6000 people, and damaged infrastructure and properties,[20] resulting in 170 000 residential building damage claims equating to about NZ$30 billion of damage.[21] In the first year

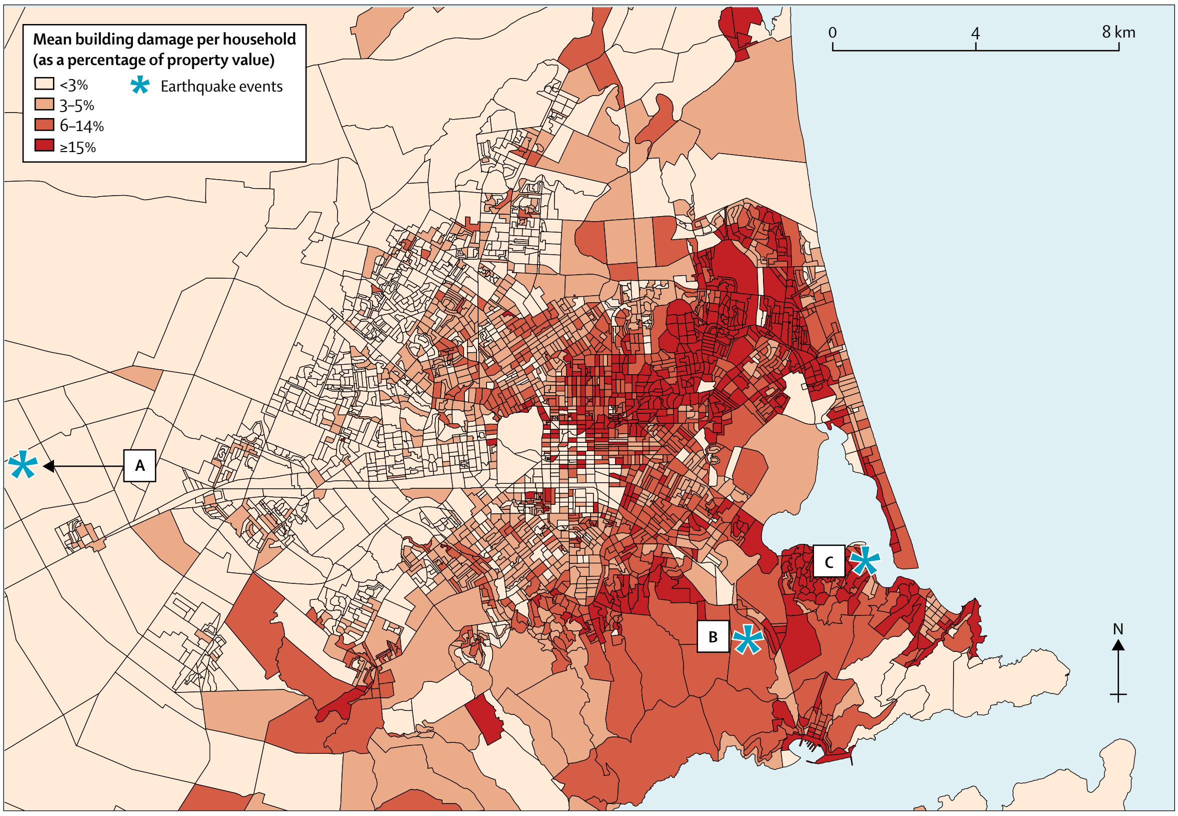

Figure 1: **Earthquake damage from the Canterbury earthquake sequence**
Map shows mean assessed-damage costs per household as a proportion of property value, summarised by meshblock using data from the Earthquake Commission and the 2006 census number of households (scale 1:125 000). Stars indicate the three most damaging earthquake events in the first year of the Canterbury earthquake sequence: (A) occurred on Sept 4, 2010, with a magnitude of 7·1 M_W and was centred off the map in Darfield, 40 km west of Christchurch, Canterbury province, New Zealand; (B) occurred on Feb 22, 2011, with a magnitude of 6·3 M_L; and (C) occurred on June 13, 2011, with a magnitude of 6·3 M_L.

approximately half of the assessed building damage was from the Feb 22, 2011 event and two-fifths from the Sept 4, 2010 event (Earthquake Commission data). The major loss from the Canterbury earthquakes was from damage to buildings and infrastructure. The earthquakes were followed by a prolonged recovery period with ongoing aftershocks (June and December, 2011) and a complicated insurance settlement process.[22] Insurance settlement after the Canterbury earthquakes has been slower than that for major earthquakes in Japan and Chile, and after 5 years residential insurance claims have not been fully settled.[21] For these reasons, we hypothesised that the effect of the Canterbury earthquakes on major adverse cardiovascular events might also be prolonged, extending beyond the initial post-earthquake period, with worse cardiovascular outcomes for people living in the most damaged areas.

The short-term effect of the 2011 Christchurch earthquake on cardiovascular disease has previously been reported by examination of Christchurch hospital data to identify year-to-year trends. In the first 2 weeks after the September, 2010 earthquake, overall cardiac hospital admissions and ST-elevation myocardial infarctions significantly increased in people admitted to Christchurch Hospital compared with rates reported 3 weeks before and 1 year before.[6] After the February, 2011 earthquake, 21 people were admitted to hospital for Takotsubo cardiomyopathy, compared with six people after the first earthquake.[6] Our research question was whether the level of residential earthquake damage in the area in which someone lived was associated with a greater number of cardiovascular events in the 3 months, 1 year, and 2–5 subsequent years after the earthquake.

Methods

Data management

This cohort-linkage study links insurance claim information (by small area, meshblocks) to administrative data on health outcomes for the residential population at earthquake onset. The study design was a cohort analysis followed up for 5 years. The cohort was the usual resident population aged 45 years or older in the affected region (Christchurch City, Waimakariri, and Selwyn Districts) and the rest of New Zealand, identified using an individual's most recent address recorded for the day before the onset of the earthquakes (Sept 3, 2010) in a database of linked administrative datasets from the Statistics New Zealand Integrated Data Infrastructure.

This study was approved by the University of Otago Human Ethics Committee (HD16/002) on Feb 9, 2016.

This study was made possible by a centralised approach to earthquake insurance in New Zealand. We created an earthquake damage exposure measure using the average level of residential building damage in the small area in which each individual resided (ie, census meshblocks with most comprising 10–50 dwellings) using apportioned building damage costs from the Earthquake Commission. The Earthquake Commission is a New Zealand Crown entity that managed claims for damage caused by the earthquakes and provided insurance cover of up to $100 000 to all households with private home and contents insurance (which was about 90% of all residential dwellings).

Residential damage was summarised by meshblock, and assigned by the date the damage was caused. We solely used damage data from the Feb 22, 2011 earthquake in the 3 month analysis, but for the 1 year follow-up and 2–5 years follow-up periods we also included damage costs sustained from Sept 4, 2010, in the 1 year following the onset of the earthquake sequence (to allow for multiple claims to the Earthquake Commission and difficulty in apportioning the damage to one event). We defined area housing damage as a ratio between the total value of building damage claims assessed by the Earthquake Commission at an occupied dwelling and the property's value (based on valuations done for allocating local council rates). If the Earthquake Commission had no record of building damage claims the ratio was zero. We selected this ratio as an exposure of housing damage because it is less dependent on an individual's socio-economic position because it is a measure of damage proportional to the total value of the property. In each meshblock the average level of building damage per dwelling was assigned to individuals according to the meshblock they resided in before the start of the earthquake sequence. We used the 2006 census data as the denominator for the number of dwellings in each meshblock. We selected four levels of building damage to account for a wide distribution of earthquake damage while maintaining similar numbers in each group. Residential building damage claim data from the Earthquake Commission are available for all properties that have house insurance (around 90%). Additionally, the distribution of the Earthquake Commission damage claims is similar to the patterns of other measures of exposure to earthquake impact used in earlier health impact studies, suggesting these claims are a good measure of property damage.[23]

In view of the wide range of cardiovascular disease diagnoses associated with natural disasters,[5,13,24] we defined cardiovascular disease incidence broadly as first post-earthquake hospital admission to any public hospital in New Zealand with a primary diagnosis of cardiovascular disease according to the International Classification of Diseases, 10th revision (ICD-10; see appendix for ICD-10 codes), irrespective of previous admissions for the same outcome of interest before the earthquake. Myocardial infarction was a subset of this broader definition (ICD-10 codes I210-I214, I219-221, I228-229). We also classified mortality according the same ICD-10 codes; however, mortality data were available only to the end of 2013 (3 years follow-up). We predetermined that results would be reported for 3 months after the Feb 22, 2011 earthquake, and 1 year and 2–5 years after the onset of the earthquake sequence on Sept 4, 2010. Individuals were excluded from follow-up after they had the outcome of interest, for any time they were outside of the country, and if they died. The model was limited to individuals who were in the Integrated Data Infrastructure spine (data refreshed in September, 2016). The spine includes individuals with data on New Zealand tax, births, or specific types of visa records. Statistics New Zealand did the probabilistic data linkage using QualityStage version 8.5, using a combination of name, sex, and date of birth variables to calculate the probability of a match.[25] The proportion of unique individuals within the ministry of health dataset who were linked to the spine (ie, the link rate) was 84%, with a false-positive rate of 1·4%. Among individuals who were successfully linked, complete data for the full-regression model were available for 81–83% of residents. About one in ten people had missing income (including no benefits or superannuation), which could have occurred if their income was solely from self-employment or investment income.

See Online for appendix

Data analysis

We calculated age-standardised hospital admission rates (per 100 000 person-months) using the WHO world standard population and age-standardised rate ratios (RR). We then examined the RR association between area housing damage and cardiovascular events using a Poisson regression analysis to adjust for potential confounding from age (10 year age groups at time of earthquake onset), ethnicity (total ethnicity; Māori *vs* non-Māori, Pacific *vs* non-Pacific), small neighbourhood deprivation (quintiles of NZDep06,[26] a national index of socioeconomic deprivation), and individual income (1 year before Sept 4, 2010). The deprivation index is calculated at the meshblock level using nine variables in the 2006 census, covering dimensions of income, owned home, support, employment, qualifications, living space, communication, and transport. The reference category was people living in Christchurch areas with the lowest levels of area housing damage, and outcomes in this group were compared with people living in areas of greater earthquake damage and the rest of New Zealand. We did a linear test of trend across these categories by regression modelling earthquake damage categories as a linear variable for Christchurch residents. We tested for effect modification by sex for all outcomes. For cardiovascular outcomes that were significantly associated with earthquake damage, we also tested for effect modification

	Christchurch Area*		Rest of New Zealand	
	Men	Women	Men	Women
Total				
Resident population (aged ≥45 years)				
Total	83 500	95 500	693 000	773 000
Number with complete data†	69 500 (83%)	78 500 (82%)	559 000 (81%)	625 000 (81%)
Follow-up				
3 months (Feb 22–May 21, 2011)				
Person-months	236 000	271 000	1 960 000	2 200 000
Cardiovascular disease-related hospital admissions	726	570	5600	4350
First year (from Sept 4, 2010, to Sept 3, 2011)				
Person-months	949 000	1 093 000	7 890 000	8 880 000
Cardiovascular disease-related hospital admissions	2460	2070	19 700	15 800
Myocardial infarction-related hospital admissions	558	405	4400	2640
Cardiovascular disease-related deaths	555	687	3890	4250
Second to fifth years (from Sept 4, 2011, to Sept 3, 2015)				
Person-months	3 440 000	4 050 000	28 600 000	32 900 000
Cardiovascular disease-related hospital admissions	6810	5450	57 100	44 400
Myocardial infarction-related hospital admissions	1850	1150	14 300	8580
Cardiovascular disease-related deaths (2 years up to Dec 31, 2013, only)‡	687	864	5130	5510
Earthquake damage in first year				
Housing damage costs (NZ$), neighbourhood average§				
$0–24 999	35 500 (43%)	40 400 (42%)	NA	NA
$25 000–49 999	19 800 (24%)	22 800 (24%)	NA	NA
$50 000–99 999	13 500 (16%)	15 500 (16%)	NA	NA
≥$100 000	14 700 (18%)	16 800 (18%)	NA	NA
Housing damage costs relative to property value (NZ$), neighbourhood average§				
<3%	34 000 (41%)	38 200 (40%)	NA	NA
3–5%	22 400 (27%)	26 400 (28%)	NA	NA
6–14%	14 900 (18%)	16 800 (18%)	NA	NA
≥15%	12 300 (15%)	14 100 (15%)	NA	NA
Potential confounders				
Age group				
45–54 years	31 400 (38%)	33 500 (35%)	263 000 (38%)	281 000 (36%)
55–64 years	24 500 (29%)	26 300 (28%)	203 000 (29%)	215 000 (28%)
65–74 years	15 800 (19%)	17 700 (19%)	134 000 (19%)	146 000 (19%)
75–84 years	9080 (11%)	12 000 (13%)	72 500 (10%)	90 300 (12%)
≥85 years	2790 (3%)	5900 (6%)	20 400 (3%)	41 000 (5%)
Ethnicity¶				
Māori	4060 (5%)	3860 (4%)	70 400 (10%)	78 900 (10%)
Pacific	1260 (2%)	1190 (1%)	31 400 (5%)	33 600 (4%)
European	74 300 (89%)	86 000 (90%)	546 000 (79%)	615 000 (80%)
Missing	1110 (1%)	1660 (2%)	10 300 (1%)	13 700 (2%)
Personal income quintile				
Lowest income	11 100 (13%)	13 300 (14%)	108 000 (16%)	121 000 (16%)
Second quintile	19 700 (24%)	23 700 (25%)	159 000 (23%)	185 000 (24%)
Middle quintile	11 200 (13%)	24 900 (26%)	90 600 (13%)	186 000 (24%)
Fourth quintile	13 800 (16%)	13 900 (15%)	103 000 (15%)	106 000 (14%)
Highest income	19 400 (23%)	8730 (9%)	152 000 (22%)	81 600 (11%)
Missing	8390 (10%)	11 000 (11%)	81 300 (12%)	93 800 (12%)

(Table 1 continues on next page)

	Christchurch Area*		Rest of New Zealand	
	Men	Women	Men	Women
(Continued from previous page)				
Deprivation				
Least deprived	25 600 (31%)	28 000 (29%)	160 000 (23%)	173 000 (22%)
Second quintile	18 400 (22%)	21 800 (23%)	145 000 (21%)	161 000 (21%)
Middle quintile	16 900 (20%)	20 500 (21%)	136 000 (20%)	154 000 (20%)
Fourth quintile	12 700 (15%)	14 700 (15%)	132 000 (19%)	152 000 (20%)
Most deprived	9870 (12%)	10 500 (11%)	119 000 (17%)	131 000 (17%)
Missing	0%	0%	1140 (0%)	2120 (0%)
Living in a different area after 1 year‖				
Did not move	71 100 (85%)	81 300 (85%)	604 000 (87%)	676 000 (87%)
Moved	12 400 (15%)	14 200 (15%)	89 300 (13%)	97 000 (13%)

Baseline characteristics of the study population were taken on Sept 3, 2010. Values are based on New Zealand administrative data for residents aged 45 years and older by sex and earthquake-affected region. Percentages are relative to the total number of people with complete data in each category, shown at the top of each column. NA=not applicable. *Canterbury Area includes Christchurch City, Waimakariri, and Selwyn Districts, Canterbury province, New Zealand. †Complete data on sex, residence, age, ethnicity, personal income, and deprivation; this was the subgroup used for all regression analyses. ‡Mortality data were only available until Dec 31, 2013. §The mean damage variables are calculated as a mean of each dwelling in the meshblock in which a person lived on Sept 3, 2010; the total number of dwellings in a meshblock was the number recorded in the 2006 census. ¶Total ethnicity (missing data included in the denominator). ‖Living in a different meshblock according to the residential address in the Integrated Data Infrastructure database.

Table 1: **Baseline characteristics, earthquake damage claims, and cardiovascular disease events**

by ethnicity, deprivation, and whether or not someone had moved to a different meshblock after 1 year. Regression analyses were done on the dataset with complete data for all the variables in the final model. Our primary analysis categorised people by residence on the day before the first earthquake, and kept them in this category even if they moved elsewhere. We calculated attributable risk in the first year of follow-up to estimate the number of cases of cardiovascular disease due to the exposure among the group living in the areas of greatest damage. All statistical analyses were done using SAS version 9.4 and maps were produced in ArcGIS 10.4.1.

Role of funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. This study was done in collaboration with Statistics New Zealand and within the confines of the New Zealand Statistics Act, 1975. Access to the data used in this study was provided by Statistics New Zealand under conditions designed to give effect to the security and confidentiality provisions of the Statistics Act, 1975. The results presented in this study are the work of the authors, not Statistics New Zealand or the funder. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

We identified 179 000 residents living in the earthquake-affected region of Christchurch, of whom 148 000 had complete data. Among residents in the earthquake-affected region of Christchurch, 1296 individuals were admitted to hospital with a primary diagnosis of cardiovascular disease during the first 3 months after the earthquake (corresponding to 0·5 million person-months follow-up), 4530 were admitted in the first year (2·0 million person-months), and 12 260 were admitted in the second to fifth year (7·5 million person-months; table 1). The Christchurch population had fewer Māori and Pacific people, fewer people living in the most deprived areas, and a slightly older population than the population in the rest of New Zealand, all of which were adjusted for in the multivariate analysis.

For the first 3 months after the most damaging earthquake on Feb 22, 2011, the age-standardised RR for cardiovascular disease-related hospital admissions for residents from areas with 15% or more damage compared with residents from areas with less than 3% damage was 1·15 (0·98–1·34; figure 2; appendix). In the first year after the earthquake sequence, for residents from areas with 15% or more damage (*vs* <3% damage), age-standardised RRs were 1·13 (1·04–1·24) for cardiovascular disease-related hospital admissions, 1·25 (1·03–1·51) for myocardial infarction-related hospital admissions, and 1·28 (1·09–1·52) for cardiovascular disease-related mortality (appendix). No dose-response pattern was seen in the age-standardised rates of cardiovascular disease in the second to fifth years after the earthquake sequence.

For the first 3 months after the February, 2011 earthquake, the RR for cardiovascular disease-related hospital admissions, after adjusting for age, sex, ethnicity, personal income, and deprivation index using Poisson regression, for residents from areas with 15% or more damage compared with residents from areas with less than 3% damage was 1·12 (95% CI 0·96–1·32; test for linear trend p=0·239; table 2). In the

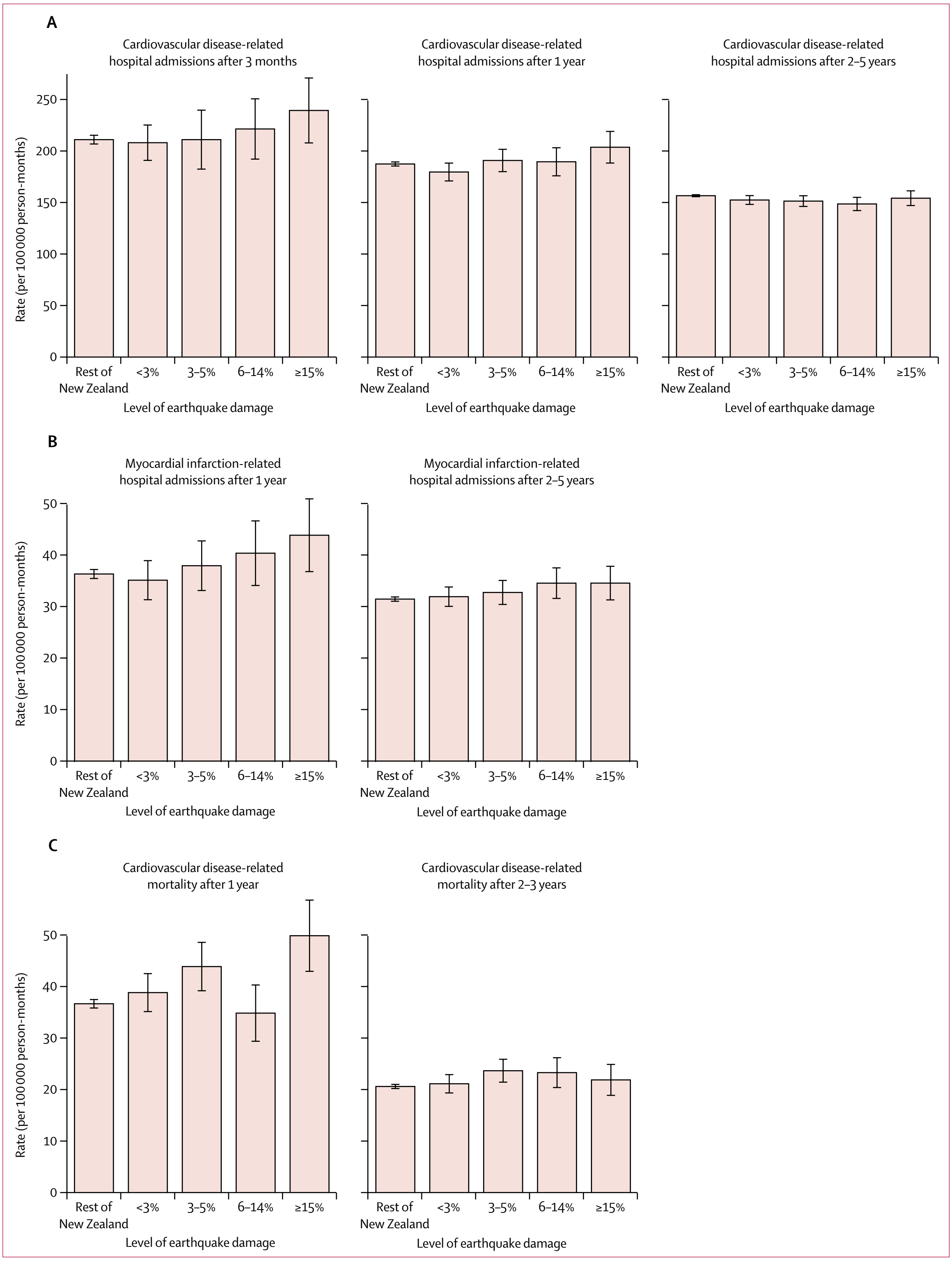

***Figure 2:* Age-standardised incidence rates of cardiovascular disease-related hospital admissions, myocardial infarction-related hospital admissions, and cardiovascular disease-related mortality**
Shown are data taken from the area affected by the earthquake sequence in Canterbury and from the rest of New Zealand, by the level of earthquake damage, for rates in men and women aged 45 years or older. The damage for the 3 months follow-up was limited to the damage that occurred from the earthquake on Feb 22, 2011, not cumulative damage from throughout the first year as it was for the other follow-up periods. Error bars represent 95% CIs. Damage is the Earthquake Commission assessment of residential building damage as a percentage of a property's valuation, and is averaged by meshblock.

first year after the earthquake sequence, for residents from areas with 15% or more damage (*vs* <3% damage), Poisson regression-adjusted RRs were 1·10 (1·01–1·21; test for linear trend p=0·068) for cardiovascular disease-related hospital admissions, 1·22 (1·00–1·48; p=0·036) for myocardial infarction-related hospital admissions,

	Men	Women	Combined
Hospital admissions for cardiovascular disease			
3 months* (Feb 22–May 21, 2011)			
Rest of New Zealand	0·95 (0·84–1·07)	0·92 (0·81–1·05)	0·94 (0·86–1·02)
<3% housing damage	1	1	1
3–5% housing damage	1·01 (0·81–1·26)	1·01 (0·79–1·28)	1·01 (0·86–1·19)
6–14% housing damage	1·05 (0·85–1·30)	0·92 (0·72–1·17)	0·99 (0·84–1·16)
≥15% housing damage	1·06 (0·86–1·32)	1·20 (0·95–1·51)	1·12 (0·96–1·32)
p value test for trend†	0·535	0·282	0·239
First year (from Sept 4, 2010, to Sept 3, 2011)			
Rest of New Zealand	0·98 (0·91–1·05)	0·95 (0·88–1·02)	0·96 (0·92–1·02)
<3% housing damage	1	1	1
3–5% housing damage	1·05 (0·95–1·17)	1·05 (0·94–1·18)	1·06 (0·98–1·14)
6–14% housing damage	1·06 (0·94–1·19)	0·99 (0·87–1·13)	1·03 (0·94–1·12)
≥15% housing damage	1·10 (0·97–1·25)	1·10 (0·96–1·26)	1·10 (1·01–1·21)
p value test for trend†	0·120	0·315	0·068
Second to fifth year (from Sept 4, 2011, to Sept 3, 2015)			
Rest of New Zealand	0·96 (0·92–1·00)	0·99 (0·95–1·04)	0·97 (0·94–1·00)
<3% housing damage	1	1	1
3–5% housing damage	0·96 (0·91–1·03)	1·00 (0·93–1·07)	0·98 (0·94–1·03)
6–14% housing damage	0·94 (0·88–1·02)	1·02 (0·94–1·11)	0·98 (0·93–1·03)
≥15% housing damage	0·98 (0·91–1·06)	1·02 (0·94–1·12)	1·00 (0·95–1·06)
p value test for trend†	0·327	0·526	0·738
Hospital admissions for myocardial infarction			
First year (from Sept 4, 2010, to Sept 3, 2011)			
Rest of New Zealand	0·98 (0·84–1·13)	0·86 (0·72–1·02)	0·93 (0·83–1·04)
<3% housing damage	1	1	1
3–5% housing damage	1·10 (0·88–1·37)	1·07 (0·82–1·38)	1·09 (0·92–1·29)
6–14% housing damage	1·06 (0·83–1·37)	1·15 (0·85–1·54)	1·10 (0·91–1·33)
≥15% housing damage	1·23 (0·95–1·58)	1·20 (0·89–1·62)	1·22 (1·00–1·48)
p value test for trend†	0·124	0·158	0·036
Second to fifth year (from Sept 4, 2011, to Sept 3, 2015)			
Rest of New Zealand	0·89 (0·82–0·96)	0·96 (0·86–1·06)	0·91 (0·86–0·97)
<3% housing damage	1	1	1
3–5% housing damage	0·98 (0·87–1·11)	1·04 (0·89–1·21)	1·01 (0·91–1·10)
6–14% housing damage	1·00 (0·88–1·15)	1·20 (1·01–1·42)	1·07 (0·96–1·19)
≥15% housing damage	0·95 (0·82–1·10)	1·08 (0·89–1·30)	1·00 (0·89–1·12)
p value test for trend†	0·651	0·109	0·511
Cardiovascular mortality			
First year (from Sept 4, 2010, to Sept 3, 2011)			
Rest of New Zealand	0·90 (0·78–1·04)	0·83 (0·73–0·95)	0·87 (0·78–0·95)
<3% housing damage	1	1	1
3–5% housing damage	1·07 (0·86–1·33)	1·12 (0·93–1·36)	1·11 (0·97–1·28)
6–14% housing damage	1·00 (0·77–1·29)	0·78 (0·60–1·01)	0·88 (0·74–1·06)
≥15% housing damage	1·24 (0·97–1·59)	1·25 (1·00–1·55)	1·25 (1·06–1·47)
p value test for trend†	0·152	0·392	0·105

(Table 2 continues on next page)

and 1·25 (1·06–1·47; p=0·105) for cardiovascular disease-related mortality (table 2, figure 3).

Subsequently, in the second to fifth year after the earthquake sequence onset, we found no significant association between earthquake damage and hospital admissions related to cardiovascular disease or myocardial infarction, which occurred at almost identical rates by level of damage (table 2). We also found no association between earthquake damage and cardiovascular disease-related mortality in the second to third years (table 2).

Regression-adjusted myocardial infarction-related hospital admissions and cardiovascular disease-related mortality rates in the least damaged areas of Christchurch tended to be greater than for individuals living in the rest of New Zealand, consistent with a generalised increase in cardiovascular disease rates in Christchurch after the earthquakes compared with the rest of New Zealand. This pattern was similar for all cardiovascular disease outcomes and across all time periods of follow-up (table 2, figure 3).

We estimate that in the first year for the residents aged 45 years or older living in the most damaged areas of Christchurch (n=26 900), an excess of 66 (95% CI 7–125) cardiovascular disease-related hospital admissions occurred, including 29 (0–53) additional myocardial infarction-related hospital admissions and 46 (13–73) additional deaths from cardiovascular disease, compared with what would have been expected had these residents lived in the areas of Christchurch with the least damage.

We found no statistical evidence that sex was an effect modifier of the association between earthquake damage and any of the cardiovascular disease outcomes on a multiplicative scale (ie, interaction term in the fully adjusted Poisson regression model; appendix). In the first year, for hospital admissions related to cardiovascular disease and myocardial infarction (outcomes for which we found statistical evidence of a dose response), we tested interactions between earthquake damage and each variable of Māori ethnicity, deprivation, and moving homes, and found no statistical evidence of interactions on a multiplicative scale.

We also analysed results by the absolute costs of damage (as compared with our default percentage damage measure of earthquake damage; appendix), which suggested a dose-response between damage and cardiovascular disease events in the first year for cardiovascular disease-related mortality (p=0·066), but the picture was less clear for cardiovascular disease-related hospital admissions.

Discussion

We found increased rates of hospital admissions related to myocardial infarction and cardiovascular disease, and cardiovascular disease-related mortality among residents from areas most damaged by the earthquakes versus those from the least damaged areas in the first year after the earthquake sequence. Myocardial infarction comprised about a third (8·7 of 24) of the increased incidence of cardiovascular disease-related hospital admissions in the most damaged areas in Christchurch (figure 2, appendix). This means that other cardiovascular diseases are contributing to the excess cardiovascular disease-related hospital admissions and these probably include Takotsubo cardiomyopathy, unstable angina, arrhythmias, heart failure, and stroke.[5,6,24]

Despite the challenging and prolonged progress to recovery, we found no evidence of persistently increased cardiovascular disease rates proportionate to area housing damage in the second to fifth years. This finding suggests that the acute stress pathway is probably more important for the relation between area housing damage and cardiovascular disease. For example, our findings are consistent with the 23% increase in psychological disorders in the first year post earthquake for people living in the most earthquake-affected areas compared with those living in the rest of the city.[18] Chronic stress from the earthquake could also affect cardiovascular disease but might be less closely linked to where someone lived at the start of the earthquake and more closely associated with differing rates of neighbourhood recovery not examined in this Article. Chronic stress might be modified by factors such as home ownership, employment consequences, insurance settlements, resilience, and whether someone stays or moves away from the city. Both home owners and tenants in highly damaged areas were faced with relocation, limited housing options, and rapidly increasing rental prices due to a shortage of housing.

This is the first study to our knowledge that has linked insurance assessments of housing damage in small geographical areas to cardiovascular disease rates. Our findings relate specifically to housing damage in a high-income country, but were similar to the Armenia study,[8] which reported an association between material loss and loss of family members and self-reported heart disease mortality and morbidity, with a dose-response between greater loss and greater heart disease risk (odds ratio 2·6, 95% CI 1·5–4·7; no test for trend; adjusted for age, sex, education, and body-mass index). Our findings for myocardial infarction rates in the first year showed a significant dose–response relation with area housing damage at the small area level. This finding corresponds to results from the 1995 Great Hanshin-Awaji earthquake in Japan (6000 fatalities), in which myocardial infarction was weakly associated with the percentage of houses that were completely destroyed in the broader region (n=16, r=0·530, p=0·062).[3] The Canterbury earthquakes resulted in many fewer injury-related fatalities (n<200) than earthquakes in Japan and Armenia; however, our results still show a consistent increase in cardiovascular disease-related hospital admissions and mortality by level of earthquake damage, resulting in 66 excess cardiovascular disease-related hospital admissions. This increase in admissions occurred in a group of people living in areas with relatively low levels of housing damage (ratio of >15% property value) and suggests that earthquake-related cardiovascular events are possible even in a moderate earthquake event.

We found no evidence that ethnicity, deprivation quintile, and moving homes modified the association between area housing damage and hospital admissions; however, the possibility that these factors might modify the results cannot be ruled out in view of the limited power of this study to detect interactions.

	Men	Women	Combined
(Continued from previous page)			
Second to third year (from Sept 4, 2011, to Dec 31, 2013)			
Rest of New Zealand	0·89 (0·78–1·01)	0·91 (0·81–1·03)	0·90 (0·83–0·99)
<3% housing damage	1	1	1
3–5% housing damage	1·02 (0·84–1·23)	1·26 (1·07–1·49)	1·16 (1·02–1·31)
6–14% housing damage	1·02 (0·81–1·27)	1·11 (0·91–1·37)	1·07 (0·92–1·25)
≥15% housing damage	0·93 (0·73–1·18)	1·08 (0·87–1·33)	1·01 (0·86–1·18)
p value test for trend†	0·654	0·454	0·767

Data are rate ratio (95% CI), unless stated otherwise. Data relates to people aged 45 years or older living in areas affected by the 2010–11 Canterbury earthquake sequence. Housing damage is the residential building damage assessed by the Earthquake Commission as a proportion of property valuation, averaged by meshblock. Follow-up person-time was censored for first event, censored for mortality, and censored to time out of the country. Data adjusted for age, sex, ethnicity, personal income, and deprivation index. *The damage for the 3 months follow-up was limited to the damage that occurred from the earthquake on Feb 22, 2011, not cumulative damage from throughout the first year as it was for the other follow-up periods. †p value is the test for linear trend examining whether damage is a predictor of cardiovascular events in the model adjusted with the variables listed above but for which damage categories are modelled as a linear variable and the rest of New Zealand is excluded.

Table 2: **Poisson regression rate ratios of incidence of cardiovascular outcomes by housing damage relative to property value**

This study uses linked administrative datasets, including national hospital admissions data, to identify Christchurch residents affected by the earthquake sequence and examine their cardiovascular disease outcomes. Up to 5 years of complete follow-up data were available for hospital admissions wherever in the country an individual might have moved to. We used residential building damage insurance assessment data from a national agency (the Earthquake Commission) to compare cardiovascular disease outcomes across small geographical areas with different levels of earthquake-damage. Although this method precluded the examination of cardiovascular disease outcomes for individual residences and house-to-house damage can vary within a meshblock, the nature of earthquakes means the damage is often similar in localities and we used a very small area unit comprising an average of about 30 dwellings. People's health might also be affected by small-area contextual or spill-over effects of small area damage, such as from damage to neighbourhood infrastructure and facilities. Any measurement error from this approximation and any misassignment of address or housing damage assessment costs probably would have biased the effect size towards no effect (ie, lowered the RRs towards one).

The linkage percentage of health data to the spine (84%) and incomplete data on some variables in the regression model (81–83% complete) might have affected our results; however, a vastly different association between housing damage and cardiovascular disease outcomes would have to be present in the excluded groups to bias the results in this study. We

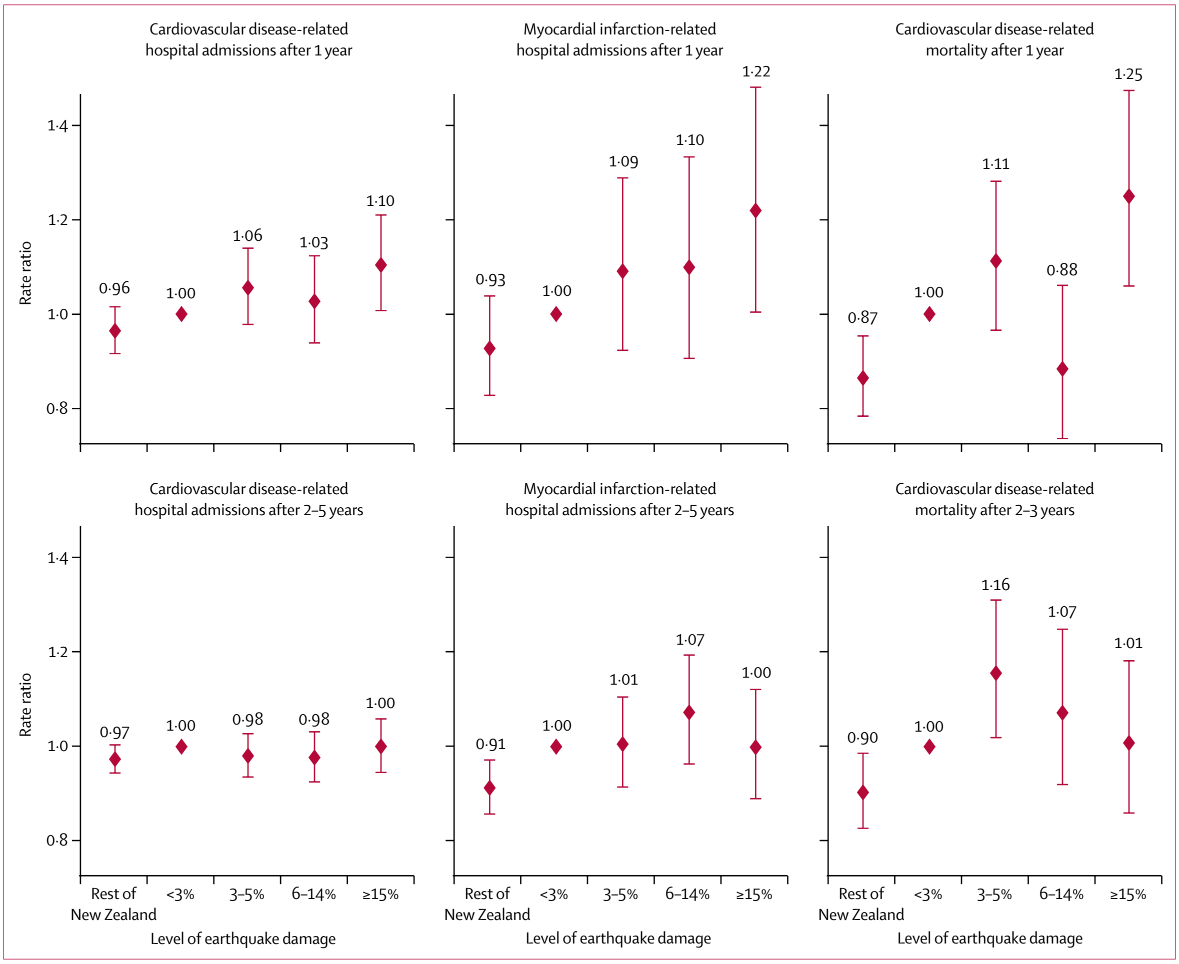

Figure 3: **Poisson regression-adjusted rate ratios of cardiovascular disease-related hospital admissions, myocardial infarction-related hospital admissions, and cardiovascular disease-related mortality**
Shown are data taken from the area affected by the September, 2010 Canterbury earthquake sequence and from the rest of New Zealand, by the level of earthquake damage, for rates in men and women aged 45 years or older. Housing damage was measured as a proportion of property value and averaged by meshblock. Results were adjusted by Poisson regression for potential confounding from age, ethnicity, deprivation, and personal income. Error bars represent 95% CIs.

were also unable to account for earthquake damage in homes with no insurance cover, but this is probably a small proportion (approximately 10%).

Although earthquake damage might be expected to be independent of cardiovascular disease risk pre-earthquake (a random event), in this study we found greater levels of area housing damage in areas ranked as more highly deprived than in those ranked least deprived. Our final results might be affected by unmeasured confounding such as from cardiovascular disease risk; however, adjustment for age, sex, ethnicity, income, and neighbourhood deprivation index (measured at the level of about 100 people) only slightly reduced the RR for damage with cardiovascular disease (appendix). If unmeasured cardiovascular disease risk factors were the cause of spurious association of damage with cardiovascular disease event, a larger reduction in the RR would be expected when adjusting for ethnicity and socioeconomic position because they are associated with cardiovascular disease risk factors.[27] Furthermore, if confounding was an issue, we would have expected an increased association of damage with cardiovascular disease events in the subsequent 2–5 years of follow-up.

Finally, despite the size of the population in Christchurch, the power of this study to examine dose-response associations and interactions was limited, as shown by the width of some 95% CIs (figure 3).

There are several ways to increase resilience to earthquake shocks and thereby reduce earthquake-related cardiovascular disease events. First, pre-earthquake measures to minimise housing damage in an earthquake are likely to reduce the psychological stress experienced in a natural disaster by limiting the time and cost of repair and loss of homes. These measures include policies and standards for building construction, insurance policies and processes, and local government planning. In New Zealand, earthquake building standards are high, but there is potentially room for improvement to better recognise seismic conditions in land use planning, as seen already in Christchurch.[22]

Second, some cardiovascular disease events might be prevented by addressing earthquake-related stress; for example, through enhanced provision of counselling and psychological support services after a major natural disaster. However, more research is needed in this area. Psychological preparedness is recognised as an important need and should be developed and integrated with other public education and community engagement readiness and recovery strategies.[28] Interventions to improve mental health were introduced in Christchurch, including the "All Right?" wellbeing campaign, which was started a full 2 years after the February, 2011 earthquake. Self-reported wellbeing significantly improved in Christchurch between 2012 and 2015, and fewer people reported struggling to deal with earthquake consequences (28% *vs* 46%).[29] However, the null relationship between area housing damage and cardiovascular disease in the second to fifth years probably occurred due to the tapering off of earthquake-related cardiovascular disease events.

Finally, cardiovascular disease-related mortality after an earthquake might be reduced by pre-event planning to ensure adequate health-care resources are available to address cardiovascular disease risk in primary care and to promptly manage an excess number of hospital admissions related to cardiovascular disease and myocardial infarction, particularly within the first year in the early recovery period.

Administrative data sources show the association between living in an area of earthquake damage and increased incidence of cardiovascular disease in the year after the onset of the Canterbury earthquake sequence. Aspects of preparedness, resilience, and recovery should all be considered to better understand how to address the effect of earthquake housing damage on cardiovascular disease risk.

Contributors

VC proposed the concept for this study. TB and AMT were the principal investigators. All authors contributed to the study design, data interpretation, editing, and approved the final version of the manuscript. AMT and VC did the scientific literature review. AMT managed access to the data, designed the figures, and wrote the first draft of the manuscript. AMT did the data analysis with support from TB.

Declaration of interests

We declare no competing interests.

Acknowledgments

The results in this Article are not official statistics. They have been created for research purposes from the Integrated Data Infrastructure (IDI), managed by Statistics New Zealand (NZ). The opinions, findings, recommendations, and conclusions expressed are those of the researchers, not Statistics NZ or MBIE. Access to the anonymised data used in this study was provided by Statistics NZ under the security and confidentiality provisions of the Statistics Act 1975. Only people authorised by the Statistics Act 1975 are allowed to see data about a particular person, household, business, or organisation, and the results in this Article have been confidentialised to protect these groups from identification and to keep their data safe. Careful consideration has been given to the privacy, security, and confidentiality issues associated with using administrative and survey data in the IDI. Further detail can be found in the Privacy impact assessment for the IDI available from www.stats.govt.nz. We thank the Earthquake Commission for sharing property damage information and assisting with its interpretation; Chris Bowie (Opus, Lower Hutt, New Zealand) for advice on the management and interpretation of Earthquake Commission data; Statistics NZ, Sheree Gibbs, June Atkinson, and the Virtual Health Information Network for assistance with the use of the IDI; and Ichiro Kawachi for comments on the final draft of this manuscript.

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