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Population-Based Analysis of the Effect of the Northridge Earthquake on Cardiac Death in Los Angeles County, California

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Objectives. **We sought to determine whether a natural disaster affected total cardiovascular mortality and coronary mortality in an entire population.**

Background. **The effect of the January 17, 1994 Northridge Earthquake (NEQ) on all deaths and causes of deaths within the entire population of Los Angeles County is unknown. The purposes of our study were to analyze all deaths in this entire population before, during and after the NEQ and to determine whether the NEQ temporally and spatially altered death due to cardiovascular disease.**

Methods. **We analyzed all death certificate data (n = 19,617) from Los Angeles County during January of 1992, 1993 (control periods) and 1994, using International Classification of Diseases, 9th Revision codes for ischemic heart disease (IHD) and atherosclerotic cardiovascular disease (ASCVD), as well as other causes of death.**

Results. **There was an average of 73 deaths per day due to IHD and ASCVD during January 1 to 16, 1994; this increased to 125 on the day of the NEQ, and then decreased to 57 deaths per day from January 18 to 31 ($p < 0.00001$, before NEQ vs. day of NEQ; after NEQ vs. day of NEQ; and before NEQ vs. after NEQ). The NEQ was associated with an increase in deaths due to myocardial infarction and trauma but not cardiomyopathy, hypertensive heart disease, valvular heart disease, cerebrovascular disease or noncardiovascular causes. Based on plots of daily deaths due to IHD and ASCVD, the decrease in deaths during the 14 days after the NEQ (−144) overcompensated for the increase on the day of the NEQ (+55). Geographic analysis revealed a redistribution of deaths due to IHD and ASCVD toward the epicenter on the day of the NEQ.**

Conclusions. **When an entire population simultaneously experiences a major environmental stress, there is an increase in death due to coronary artery disease (but not other cardiac causes), followed by a decrease that overcompensates for the excess of death. The overcompensation may represent a residual population that is more resistant to stress or a possible preconditioning effect of the stress, or both. This study supports the concept that cardiovascular events within an entire population can be triggered by a shared stress.**

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The Northridge earthquake (NEQ) was associated with some of the greatest ground motion ever recorded and was the most expensive natural disaster in the history of the United States. Our previous studies from the Los Angeles Coroner's Office and coronary care units suggested that the January 17, 1994 NEQ triggered sudden cardiac death and myocardial infarction (1,2). The first study reported unexpected deaths studied in the Coroner's Office, and the second was a study of coronary care units dealing with mainly nonfatal myocardial infarctions. One limitation of a Coroner's Office study is the possibility of bias regarding which cases are examined. Both of these previous studies dealt with selected groups, not the entire population of Los Angeles County. However, whether the NEQ had any impact on total cardiovascular deaths within the entire population of Los Angeles County (8.8 million) has not been previously reported. The purposes of our study were to review all deaths among the entire population of Los Angeles County and to determine whether the NEQ changed the number of deaths due to cardiovascular disease either on the day of the NEQ or during the weeks after the event. In addition, we analyzed whether the NEQ altered the spatial distribution of coronary death.

Methods

Population. We analyzed all daily death certificate data (n = 19,617) from Los Angeles County's Department of Health Services Data Collection and Analysis Unit for January 1994 (the month of the NEQ) and for the control periods January 1992 and January 1993 using the International Classification of Diseases, 9th Revision (ICD-9) codes for ischemic heart disease (IHD) and atherosclerotic cardiovascular disease (ASCVD). We also analyzed deaths due to other forms of cardiovascular disease, including hypertensive heart disease, valvular heart disease and cardiomyopathy, as well as cerebro-

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Abbreviations and Acronyms

ASCVD = atherosclerotic cardiovascular disease
ICD-9 = International Classification of Diseases, 9th Revision
IHD = ischemic heart disease
NEQ = Northridge earthquake

vascular disease, accidents (nonmotor vehicle trauma) and deaths due to noncardiovascular disease and motor vehicle accidents. Maps of Los Angeles County were generated to show the number of deaths due to IHD and ASCVD by location in relation to the NEQ.

Statistics. The Z statistic was used to determine if there were differences in number of deaths due to various causes during the three periods (before NEQ [January 1 to 16], day of NEQ [January 17] and days after NEQ [January 18 to 31]). Assuming that the proportion of heart-related deaths occurring before, during and after the NEQ follow a trinomial distribution, we used a normal approximation to test for differences in the average daily proportion of deaths before versus during, after versus during and before versus after the NEQ. All statistics were analyzed by Research Triangle Institute, North Carolina.

Results

The total number of deaths reported in Los Angeles County in January of 1992, 1993 and 1994 were 6689, 5995 and 6933, respectively; and the total number of deaths due to IHD and ASCVD were 2089, 1886 and 2094, respectively. There was an increase in the total number of deaths on the day of the NEQ (Fig. 1). The numbers of daily deaths due to IHD and ASCVD during January of 1992, 1993 and 1994 are shown in Figure 2. There was an average of 73 deaths per day due to IHD and ASCVD during January 1 to 16, 1994; this increased to 125 on January 17, 1994 (day of NEQ) and then decreased to 57 deaths per day from January 18 to 31 ($p < 0.00001$ for before vs. day of NEQ, for after vs. day of NEQ and for before vs. after NEQ) (Fig. 2). By January 31, 1994, the number of deaths had returned toward baseline. There were no similar increases during the control years of 1992 and 1993.

Based on our previous study (1), we tested the null hypothesis that during the 2 weeks after the NEQ there was a compensation for the excess of deaths due to IHD and ASCVD on the day of the NEQ. This was tested against the alternative hypothesis that during the 2 weeks after the NEQ there was an overcompensation for the excess IHD and ASCVD deaths on the day of the NEQ. Plots of deaths due to IHD and ASCVD per day during January of 1992, 1993

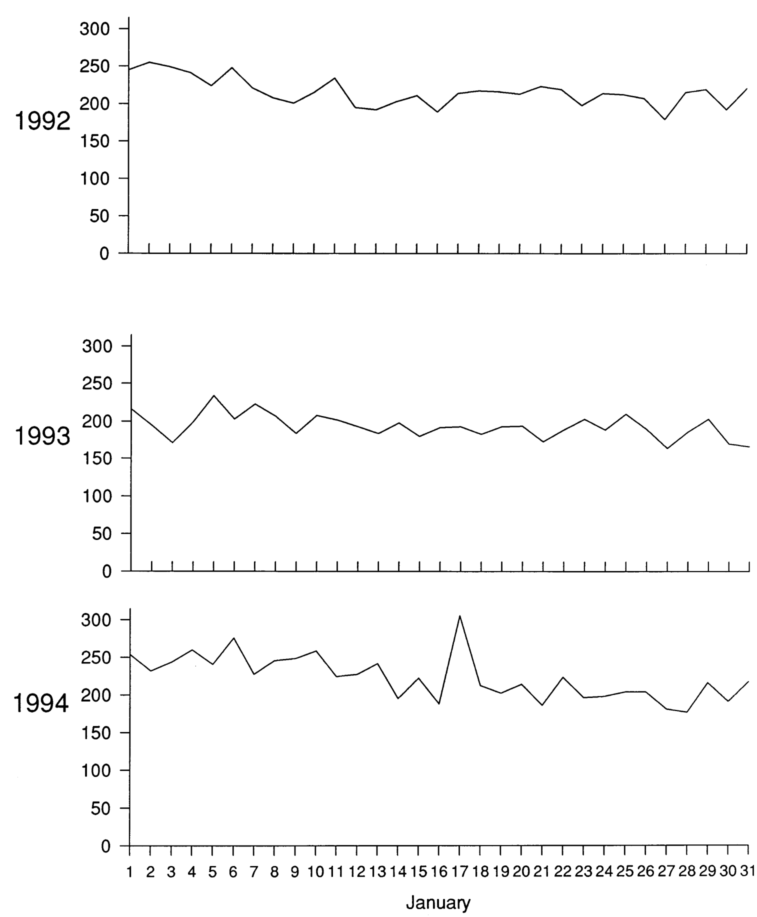


Figure 1. Total daily deaths in Los Angeles County for January 1992, 1993 and 1994, showing an increase on January 17, 1994—the day of the NEQ.

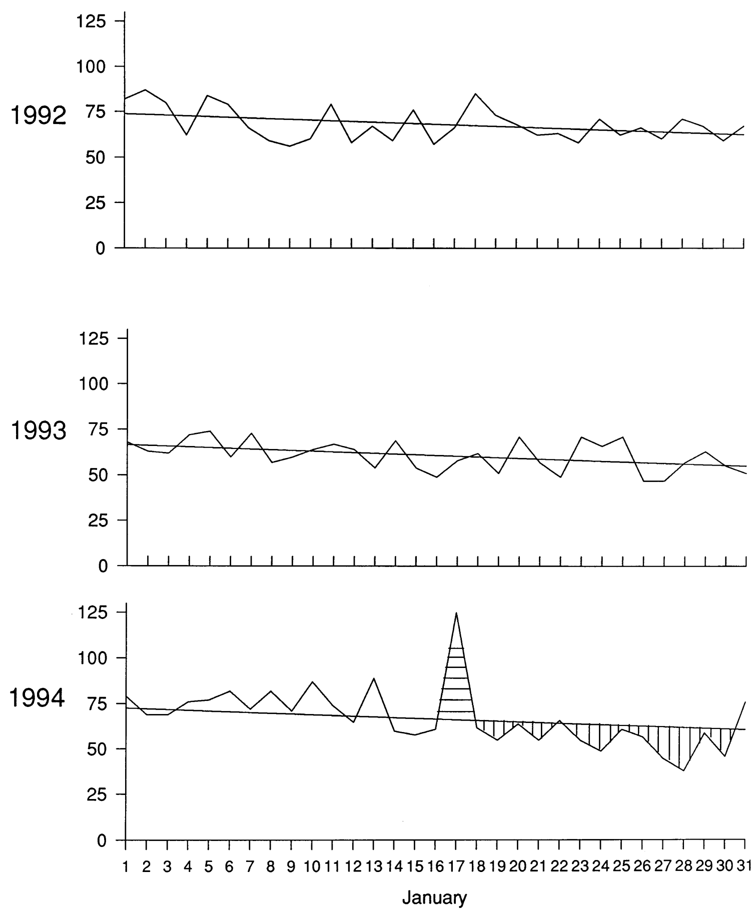


Figure 2. Daily deaths in Los Angeles County due to IHD and ASCVD during January of 1992, 1993 (control periods) and 1994. There was an increase in these deaths on the day of the NEQ (January 17, 1994), followed by a decrease over a 2-week period, with eventual recovery. During the 2 weeks after the NEQ, there was a decrease in deaths due to IHD and ASCVD, which overcompensated for the increase on the day of the NEQ. The **horizontal bars** represent the excess number of deaths on the day of the NEQ. The **vertical bars** represent the decrease in deaths during the 14 days after the NEQ.

and 1994 (Fig. 2) suggested that the slopes for the control years and that part of the quake year (1994) before the NEQ were not different, although the intercepts were different. This was confirmed by statistical analysis ($p = 0.6789$ for a test of slopes, $p = 0.0006$ for a test of intercepts). In choosing a model that assumes equal slopes and different intercepts for the relation between IHD and ASCVD versus time, we determined that the best model was a simple linear model in which the slope was -0.36764 and the intercepts in 1992, 1993 and 1994 were 73.3, 66.7 and 76.3, respectively. Fits to the logarithm and square root transformed data did not improve the fit of the model. As shown in Figure 2, and using this model, on the day of the NEQ there were 55 excess deaths due to IHD and ASCVD; during the next 14 days there were 144 fewer deaths than would have been predicted. This represents a significant decrease in the number of deaths that *overcompensated* for the increase in deaths on the day of the NEQ by 89 ($144 - 55 = 89$). Thus, during this 2-week period, there were on average 6.38 fewer daily deaths than expected, even after compensating for the increase on the day of the NEQ ($Z = 1.75$, one-sided $p = 0.039$).

For the ICD-9 code for acute myocardial infarction, the average daily deaths were reported as 20 for January 1 to 16, 1994; 38 on the day of the NEQ; and 17 for January 18 to 31, 1994 ($p = 0.004$ for before vs. day of NEQ; $p = 0.0005$ for after vs. day of NEQ; $p = 0.02$ for before vs. after NEQ).

For all heart disease, the average daily deaths were 82 for January 1 to 16, 1994; deaths increased to 134 on the day of the NEQ and then decreased to 63 for January 18 to 31, 1994 ($p < 0.00001$ for before vs. day of NEQ, for after vs. day of NEQ and for before vs. after NEQ).

Figure 3 shows daily deaths during January of 1992, 1993 and 1994 for hypertensive heart disease, cardiomyopathy, valvular heart disease and cerebrovascular disease. There were no increases in deaths due to these other causes of heart disease or cerebrovascular disease on the day of the NEQ, nor decreases after the NEQ. As expected, there was an increase in deaths due to accidents (nonmotor vehicle trauma) on the day of the earthquake (32 vs. 3 to 10 during control periods). There were no increases in death due to other noncardiac causes or automobile accidents on the day of NEQ.

Figures 4 and 5 are maps of Los Angeles County by area showing the average number of daily deaths due to IHD and ASCVD during the days before the NEQ and on the day of the NEQ. Note the redistribution of daily deaths near the epicenter on the day of the NEQ compared with before the NEQ.

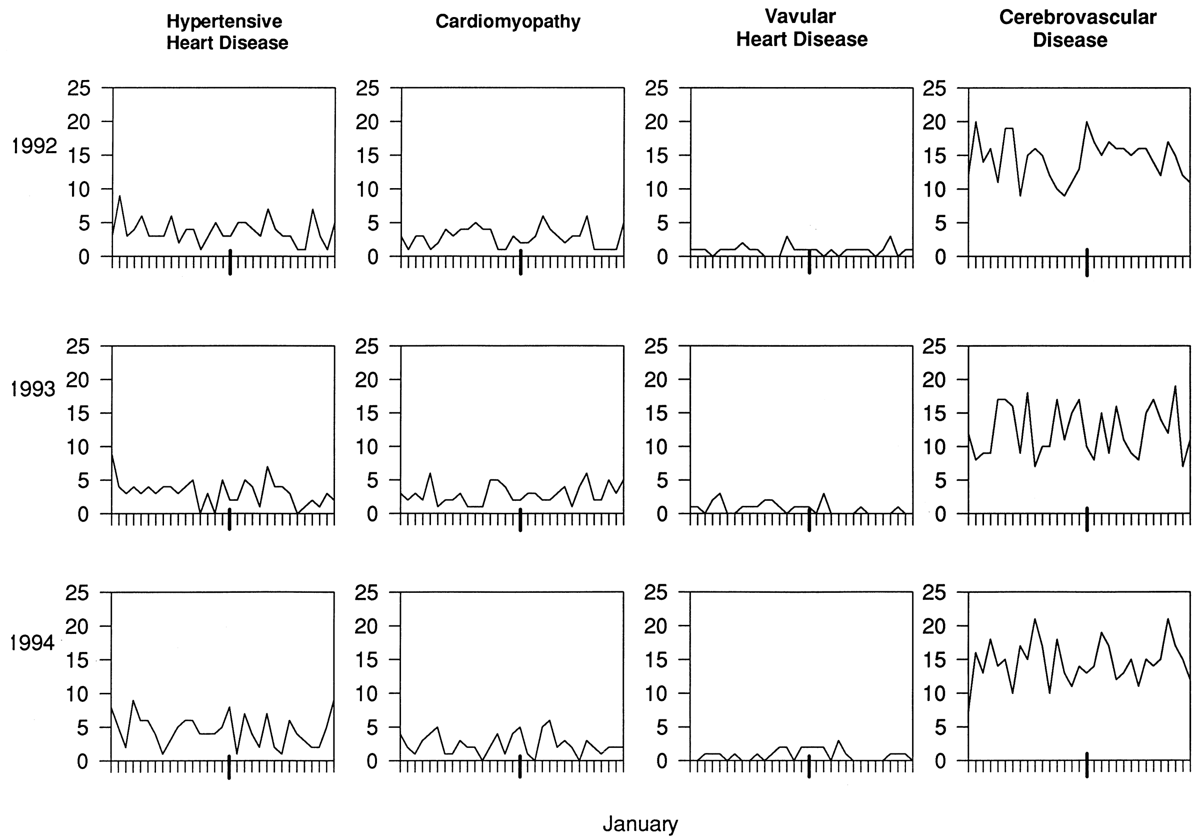


Figure 3. Deaths from hypertensive heart disease, cardiomyopathy, valvular heart disease and all cerebrovascular disease during January of 1992, 1993 and 1994.

Age and gender data are shown in the Table 1. Women with IHD and ASCVD died at an older age (early 80s) than did men (early to mid 70s) across all periods. This did not appear to be affected by the NEQ.

Discussion

Effect of NEQ on cardiac events. The present study showed that when the entire population of Los Angeles County simultaneously experienced the major stress of the NEQ, there was an increase in death due to IHD and ASCVD, followed by a significant decrease in these deaths. The exact cause of the decrease is not known, but there are several possibilities. One is that a portion of the decrease was a compensation for the increase on the day of the NEQ. In other words, some people at risk of dying of IHD and ASCVD may have died early due to the trigger of the NEQ-related stress. However, because the decrease in the present study *overcompensated* for the increase, other people actually may have developed some type of protection after the initial stress. The NEQ may have triggered death among susceptible patients, leaving a population that was more resistant than usual to triggers of cardiovascular events. Perhaps the stress of the NEQ preconditioned a population of people. Preconditioning is the phenomenon whereby brief episodes of ischemia, catecholamines or other stresses impart cardiac protection (3,4). For example, in experimental models, transient coronary artery occlusions of 5 to 10 min before longer occlusions of 40 to 60 min reduce infarct size (3) and in some small animal models actually reduce arrhythmias (5,6). Similar observations have been observed with injections of catecholamines before coronary artery occlusions (4). There have now been a series of recent clinical studies suggesting that the stress of preinfarct angina improves in-hospital outcome, and in some studies this includes in-hospital survival (7,8). Finally, could the decrease in coronary deaths after the NEQ have been due to people moving out of the area? Although this is a possibility, it is unlikely because there was no decrease in death due to other cardiac causes or cerebrovascular disease after the NEQ. Also, death due to IHD and ASCVD during the first week of February 1994 increased to 63 per day from 55 per day during the last week in January.

This study also suggested that the increase in deaths on the day of the NEQ was primarily due to trauma and IHD and ASCVD, rather than hypertensive heart disease, cardiomyopathy, valvular heart disease, cerebrovascular disease and non-

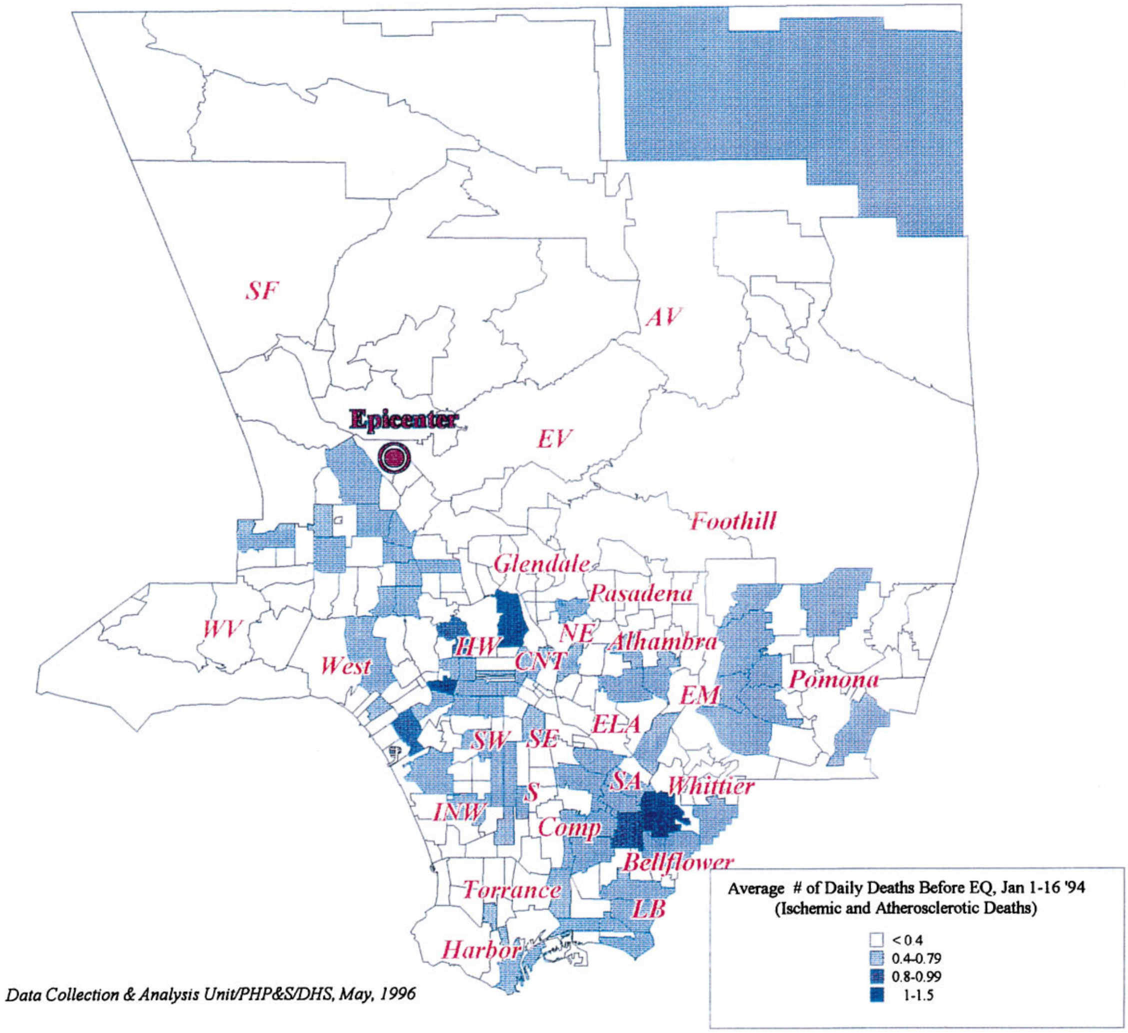
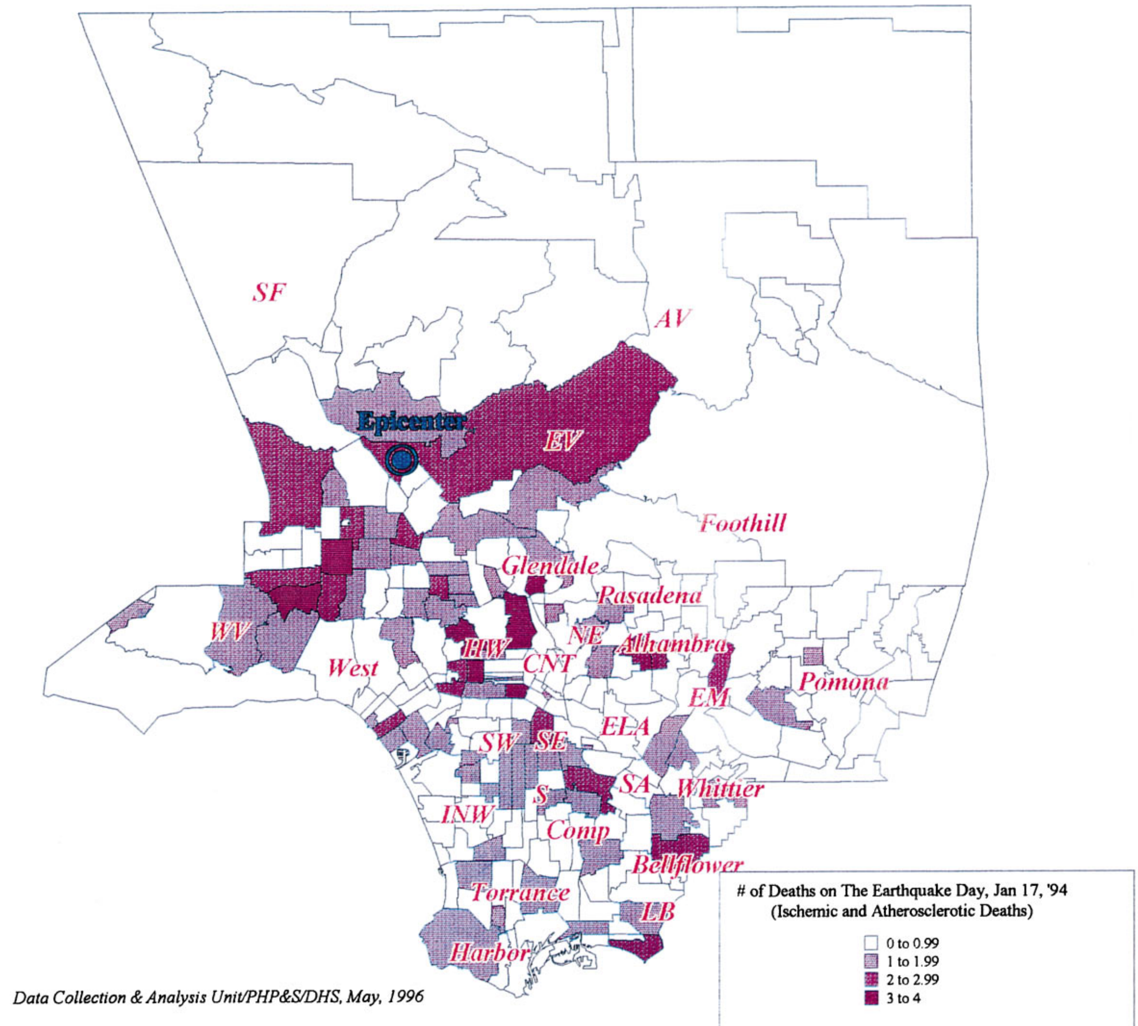


Figure 4. Map of Los Angeles County showing average number of daily deaths due to IHD and ASCVD (period before NEQ, January 1 to 16, 1994) by location. AV = Antelope Valley; CNT = Central; Comp = Compton; ELA = East Los Angeles; EM = El Monte; EV = East Valley; HW = Hollywood; INW =Inglewood; LB = Long Beach; NE = Northeast; S = South; SA = San Antonio; SE = Southeast; SF = San Fernando; SW = Southwest; WV = West Valley. (Abbreviations refer to health districts designated by the Los Angeles County Department of Health Services.)

cardiac causes. This study is in agreement with two previous studies from our group (1,2) showing an increase in sudden cardiac death from the Los Angeles Coroner's Office and an increase in acute myocardial infarction reported by coronary care units on the day of the NEQ. It extends these studies by showing that environmental stress can have an impact on cardiovascular deaths within an entire population and can alter these patterns both temporally as well as spacially, as suggested by the maps in Figures 4 and 5.

Previous studies. Trichopoulas et al. (9) reviewed death certificates after the Athens earthquake of 1981 and observed an increase in the number ASCVD deaths during the 5 days after the quake—from an average of 2.6 deaths per day before the quake to 5.4 per day after. In contrast to our study, the highest frequency of cardiac deaths in the study by Trichopoulas et al. (9) was on the third day after the earthquake. Our study also differs from this and previous ones (10) in that it analyzed a much larger population and number of deaths and examined the forms of cardiac death. Recent reports from the Great Hanshin Earthquake in Japan have also suggested that this event was associated with an increase in myocardial infarction (11) as well as electrocardiographic abnormalities, such as deep negative T waves and sympathetic activation within the heart assessed by metaiodobenzylguanidine imaging (12). Finally, our data



lend strong support for the concept that stress can trigger cardiovascular events, as proposed by Muller and Willich and their coworkers (13–16). It is likely that emotional and physical stress associated with natural disasters can be

Figure 5. Map showing average number and location of deaths due to IHD and ASCVD on the day of the NEQ (January 17, 1994). There is a redistribution of these cardiac deaths toward the epicenter. Abbreviations as in Figure 4.

Table 1. Age and Gender Data of People Who Died of Atherosclerotic Cardiovascular Disease and Ischemic Heart Disease

	January 1–16			January 17, 1994			January 18–31		
	No. Died	Age (yr) Mean	SD	No. Died	Age (yr) Mean	SD	No. Died	Age (yr) Mean	SD
1992									
Men	498	73.6	12.1	41	73.9	10.8	458	73.3	12.5
Women	593	81.9	11.5	25	83.9	10.6	474	81.7	11.5
1993									
Men	512	74.1	13.0	37	74.4	12.8	395	73.8	12.8
Women	498	81.0	12.4	21	82.5	10.5	423	81.3	12.4
1994									
Men	533	74.2	13.2	74	72.4	13.7	383	74.0	13.5
Women	638	82.7	11.5	51	82.6	11.0	415	81.6	11.3

added to the list of other factors, such as circadian rhythms, that precipitate cardiac events.

Conclusions. Our study suggests that when an entire population simultaneously experiences a severe environmental stress, there is an increase in deaths due to ischemic and ASCVD within the entire population, followed by an initial fall that overcompensates for the excess in deaths. The overcompensation may represent a residual population that is more resistant to stress or a possible preconditioning effect of stress, or both. These findings were not observed for other types of cardiovascular disease, cerebrovascular disease or noncardiac disease.

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References

1. Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. N Engl J Med 1996;34:413–9.
2. Leor J, Kloner RA. The Northridge Earthquake as a trigger for acute myocardial infarction. Am J Cardiol 1996;77:1230–2.
3. Murry CE, Jennings RB, Reimer KA. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation 1986;75:1124–36.
4. Bankwala Z, Hale S, Kloner RA. Alpha-adrenoceptor stimulation with exogenous norepinephrine or release of endogenous catecholamines, mimics ischemic preconditioning. Circulation 1994;90:1023–8.
5. Hagar JM, Hale SL, Kloner RA. Effect of preconditioning ischemia on reperfusion arrhythmias after coronary artery occlusion and reperfusion in the rat. Circ Res 1991;68:61–8.
6. Li Y, Whittaker P, Kloner RA. The transient nature of the effects of ischemic preconditioning on myocardial infarct size and ventricular arrhythmia. Am Heart J 1992;123:346–53.
7. Kloner RA, Shook T, Przyklenk K, et al., for TIMI 4 Investigators. Previous angina alters in-hospital outcome in TIMI 4: a clinical correlate to preconditioning? Circulation 1995;91:37–45.
8. Anzai T, Yoshikawa T, Asakura Y, et al. Preinfarct angina as a major predictor of left ventricular function and long-term prognosis after a first Q wave myocardial infarction. J Am Coll Cardiol 1995;26:319–27.
9. Trichopoulos D, Katsouyanni K, Zavilsanos X, Tzonou A, Dalla-Vorgia P. Psychological stress and fatal heart attack: the Athens (1981) earthquake natural experiment. Lancet 1983;1:441–4.
10. Dobson AJ, Alexander HM, Malcolm JA, Steele PL, Miles TA. Heart attacks and the Newcastle earthquake. Med J Aust 1991;155:757–61.
11. Suzuki S, Sakamoto S, Miki T, Matsuo T. Hanshin-Awaji earthquake and acute myocardial infarction. Lancet 1995;345:981.
12. Yamabe H, Hanaoka J, Funakoshi T, et al. Deep negative T waves and abnormal cardiac sympathetic image (^{123}I-MIBG) after the great Hanshin earthquake of 1995. Am J Med Sci 1996;311:221–4.
13. Muller JE, Abela GS, Nesto GH, Tofler GH. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. J Am Coll Cardiol 1994;23:809–13.
14. Muller JE, Ludmer PL, Willich SN, et al. Circadian variation in the frequency of sudden cardiac death. Circulation 1987;75:131–8.
15. Muller JE, Tofler GH. Triggering and hourly variation of onset of arterial thrombosis. Ann Epidemiol 1992;2:393–405.
16. Willich SN, Maclure M, Mittelman M, Arntz HR, Muller JE. Sudden cardiac death: support for a role of triggering in causation. Circulation 1993;87:1442–50.