Short-Term Effect of Natural Disasters on Coronary Heart Disease Risk Factors

Maurizio Trevisan, Egidio Celentano, Costantino Meucci, Eduardo Farinara, Fabrizio Jossa, Vittorio Krogh, Dante Giumetti, Salvatore Panico, Antonio Scottoni, and Mario Mancini

In this analysis of the data from a longitudinal study on coronary heart disease risk factors, it was found that participants screened a few weeks after a major disaster (earthquake) had a higher heart rate, serum cholesterol levels, and serum triglyceride levels than matched participants that were screened shortly before the catastrophic event. The two groups of participants did not differ with regard to their characteristics at the baseline examination carried out 5 years previously. The lack of difference in blood pressure between exposed and nonexposed participants could be explained by the lag-time between the earthquake and the blood pressure measurements. We conclude that the acute stress associated with major disasters can influence risk factors for coronary heart disease. Permanent elevation of these risk factors due to the disruption of the social environment of the individuals affected by major disasters might be responsible for the apparent long-term adverse effects on cardiovascular mortality discussed previously in the literature. (Arteriosclerosis 6:491–494, September/October 1986)

The study of short- and long-term effects of major disasters (natural or man-made) on the health status of persons affected by them has been the focus of attention for a long time. The improvement in living conditions and in health care techniques occurring in the Western world in the last century has reduced the risk of communicable disease epidemics. The scientific community has continued to wonder about the effects of disasters on major chronic diseases such as cancer, cardiovascular disease, and psychiatric disorders. To date, scant data are available on the short- and long-term effects of major disasters on coronary heart disease risk factors. In this retrospective study, we were able to analyze the relationship between a major earthquake and some of the major risk factors for coronary heart disease.

Methods

The Olivetti Study is a longitudinal investigation of risk factors for arteriosclerosis. The baseline examination was carried out in 1975–1976, and the 5-year follow-up examination was carried out in 1980–1981. The participants in this study represent the entire male working population of the Olivetti factory in Naples, where 91% of the entire working force of the factory are men. The number of participants screened during the 1980–1981 examination totaled 1400.

During both examinations, participants were seen in the morning after overnight fasting. Blood samples were drawn for the determination of total serum cholesterol and triglycerides. Trained observers measured blood pressure with a standard mercury sphygmomanometer according to a standardized protocol. In brief, blood pressure was measured after the participant had been seated, without talking, for at least 5 minutes. The right arm was used, and two measurements were taken 2 minutes apart. The first and fifth phases of the Korotkoff sounds were recorded. The values indicated here represent the average of the two readings. Heart rate was calculated from a 12-lead electrocardiogram that was administered as part of the standard examination. Weight and height were measured on a beam balance scale with participants shoeless and wearing indoor clothing.

The 5-year follow-up examination was interrupted on November 27, 1980, by an earthquake that shook Southern Italy. The magnitude of the quake was 6.8 on the Richter scale and affected a large area of Southern Italy. The official statistics reported were: 4441 deaths, more than 50,000 injured, and 280,000 homeless. The total damages are estimated in U.S. dollars at approximately $13,265,000,000. The City of Naples is located 130 km from the epicenter of the quake, and the amount of earthquake damage was approximately $375,000,000, with 118 deaths and approximately 100,000 homeless.

The examinations were resumed after 2 weeks and lasted for 6 more weeks. Participants who were screened after
the resumption of the study were defined as "exposed" (n = 277); participants who were screened before the earthquake were defined as "nonexposed" (n = 1,123). Participants known to be on dietary or pharmacological treatment for hypertension, hyperlipidemia, or diabetes were excluded. In addition, to control for the possible confounding effect of seasonal variation in risk factors, only participants screened within 2 months before or after the catastrophic event were selected. Exposed and nonexposed participants were then closely matched by age (±1 year) and weight (±1 kg), and only subjects with baseline data (1975–1976) were selected, resulting in 96 exposed and 96 nonexposed participants. No detailed information was available on the individual involvement of the participants in the disaster. We ascertained that none of these participants experienced death in the family or any major property loss or damage, and no one had to be relocated because of the quake.

Logarithmic transformation of triglycerides was used throughout the analyses. Student's t test analysis was performed for comparison of the average values between exposed and nonexposed participants. Adjustment for differences in weight gain during the two screenings between the exposed and nonexposed subjects was carried out by analysis of covariance. During the baseline examination, serum cholesterol was determined by the method of Abell et al.2 and serum triglycerides, by the method of Pantulu et al.3 During the follow-up examination, serum cholesterol and triglyceride levels were determined by an enzymatic procedure.4 During both examinations, quality control for the laboratory was carried out according to the guidelines of the World Health Organization laboratory in Prague.

A systematic difference was described between the methods used during the baseline and follow-up examinations.5 The values expressed here for the follow-up examination have been adjusted to take into consideration the described difference between the two methods.

Results

Table 1 summarizes the characteristics of the total number of exposed and nonexposed participants at the 1980–1981 examination. To control for possible confounding effects, exposed and nonexposed participants with baseline values were successfully matched with regard to age and weight, as shown in the Table 2. No difference was detected between exposed and nonexposed persons with regard to systolic and diastolic blood pressure. However, exposed participants showed, on the average, significantly higher heart rates, serum cholesterol, and serum triglycerides. The results were similar for all participants (Table 1) and for the matched groups (Table 2).

To control for possible confounding due to different baseline characteristics of the exposed and nonexposed, we looked at the average values of the participants' characteristics at the examination carried out in 1975–1976 (Table 3). No significant difference was detected between exposed and nonexposed subjects with regard to the variables under investigation, even though the exposed persons were on the average 1 kg lighter than nonexposed subjects. To further investigate the possible confounding

<table>
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<tr>
<th>Characteristic</th>
<th>Exposed</th>
<th>Nonexposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>96</td>
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<tr>
<td>Age (yrs)</td>
<td>41.5±6.3</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>26.1±2.8</td>
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<td>Weight (kg)</td>
<td>73.7±8.3</td>
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<td>SBP (mm Hg)</td>
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<td>129.7±19.8</td>
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<tr>
<td>DBP (mm Hg)</td>
<td>81.9±9.4</td>
<td>82.2±12.0</td>
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<tr>
<td>Heart rate (bpm)</td>
<td>75.0±12.0</td>
<td>71.8±11.1*</td>
</tr>
<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>213.9±40.4</td>
<td>201.3±45.4*</td>
</tr>
<tr>
<td>Serum triglycerides (mg/dl)</td>
<td>164.7±94.9</td>
<td>137.1±82.1*</td>
</tr>
</tbody>
</table>

Values are means ± sd. Data are from the 1980–1981 examination of individuals who did (exposed) and did not (nonexposed) experience the 1980 earthquake.

* p < 0.01.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Exposed</th>
<th>Nonexposed</th>
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</thead>
<tbody>
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<td>96</td>
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<td>Age (yrs)</td>
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<td>Heart rate (bpm)</td>
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<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>189.4±59.2</td>
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</tr>
<tr>
<td>Serum triglycerides (mg/dl)</td>
<td>146.2±67.2</td>
<td>136.6±82.3</td>
</tr>
</tbody>
</table>

Values are means ± sd.
In this retrospective analysis of the Olivetti longitudinal study, participants screened after experiencing a major disaster (exposed) had significantly higher heart rates, serum cholesterol levels, and triglyceride levels than participants who were screened before the catastrophic event (nonexposed). No difference was detected between exposed and nonexposed subjects with regard to systolic and diastolic blood pressure.

In recent years, investigators have been focusing their attention on the effects of catastrophic events on chronic diseases. Unfortunately, the number of reports published to date is very limited. Faich and Rose, analyzing the effects of a blizzard in Rhode Island on the health of the inhabitants of the affected area, found an increase in ischemic heart disease mortality as early as during the first 5 days after the blizzard, compared to mortality rates for the same area during the same period of time in previous years. The authors believed that this increase in ischemic heart disease was due to the increase in both physical and psychosocial stress that occurred as a result of the blizzard. This excess in cardiovascular mortality seems to be present even in the long-term, as indicated by the 10-year follow-up of the residents of Donora County in Pennsylvania who were affected by an acute air pollution episode in 1948. Two other longitudinal studies in Great Britain and in the United States indicate long-term increases in total and cancer mortality in areas affected by major disasters compared to control areas.

The increased incidence of coronary heart disease mortality and morbidity could be explained by an increase in risk factors for such conditions. The first reports indicating a relationship between massive stressful situations and hypertension date back to the 1940s. Various authors, presenting evidence for an increase in blood pressure in frontline soldiers during the Second World War. Gelstein (cited in reference 10) and Ehrstrom reported a high prevalence of hypertension in Finnish and Russian frontline soldiers. In both reports, the prevalence of hypertension in these combat soldiers was higher than the prevalence of hypertension in their comrades who were stationed away from the battle zone. In a third report, Graham found a high proportion of hypertensive soldiers in a battalion engaged in long-term mobile warfare in Africa. Blood pressure levels returned to normal in the majority of the soldiers after a few weeks of rest away from the battle zone.

In 1948, Ruskin et al. reported an elevation of arterial pressure in the victims of the Texas City disaster. This elevation was usually temporary, and blood pressure returned to normal within 2 weeks after the acute episode. In a more recent report focusing on the effects of the Wyoming Valley flood on the health of the residents of Buffalo Creek, Logue et al. reported an association between incidence of hypertension and the amount of damage resulting from the flood in a group of women followed for 5 years.

Numerous studies have presented evidence for an association between stressful situations and an increase in serum lipids. These reports have found an increase in serum lipids in medical students undergoing tests, middle-aged men self-reporting to be under stress, tax accountants during tax return deadlines, and workers undergoing job loss. In particular, the studies by Kasl et al. and report an increase in both serum cholesterol and blood pressure in relation to job loss. Increases in these
risk factors did not occur simultaneously, however; the increase in blood pressure seemed to take place before the increase in serum cholesterol.

In the present report, we could not detect any increase in blood pressure in the exposed as compared to the nonexposed participants. The apparent contrast between our study and the previously cited reports could be explained by both the lag time occurring in our study between the event and the blood pressure measurement in the exposed, and the fact that the impact of the event on the exposed was less serious than for many of those experiencing the disaster more directly. None of the participants experienced deaths in the family or major property damage. Furthermore, none of the workers in the factory lost their job as a result of the earthquake.

It is most likely that what we were observing was the carry-over effect of the acute stress due to the earthquake on the risk factors of these participants, and by the time we made our measurements, the participants' blood pressure had already returned to baseline values. This hypothesis is supported by the report by Ruskin et al. on the Texas City disaster victims. The authors observed that the elevation in blood pressure took place as early as 1 hour after the explosion and that for the majority of the victims, blood pressure returned to "normal" within 2 weeks after the disastrous event. In both the Texas City disaster and in our study, the catastrophic event consisted of an acute event with no long-term major repercussions on the social environment of the persons involved.

The retrospective nature of the present study does not allow the exclusion of the possibility that the observed differences between exposed and nonexposed subjects are due to reasons not considered in the present report rather than to the stress associated with the earthquake. Nevertheless, the higher heart rates in the exposed participants compared to the nonexposed, despite similar heart rate levels at the examination 5 years earlier, support the hypothesis that participants screened after the earthquake were at higher stress levels than participants screened before the catastrophic event. By the same token, the differences observed between exposed and nonexposed persons in serum lipids do not seem to be related to previous baseline characteristics of these participants. In conclusion, our findings support the hypothesis that the acute stress resulting from major disasters is associated with a short-term elevation in the levels of coronary heart disease risk factors. This effect is still detectable 2 weeks after the event with regard to serum lipids, but not blood pressure.

If a catastrophic event results in the disruption of social and economic life (e.g., damage or loss of property, deaths or major illness in the family, relocation of the persons involved in the event with disruption of both social and family ties), the long-term stress associated with this situation could result in a permanent elevation of blood pressure and serum lipids, resulting in higher morbidity and mortality from cardiovascular disease.

Further studies need to be planned in order to better characterize this association between major disasters and cardiovascular diseases, and to further identify individual characteristics that would help to select persons at higher risk. The identification of individuals at higher risk of becoming ill is the essential first step for launching preventive programs aimed at reducing the adverse effects of major disasters on health.

References


Index Terms: cholesterol • blood pressure • triglycerides • earthquake • disaster • arteriosclerosis
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Arterioscler Thromb Vasc Biol. 1986;6:491-494
doi: 10.1161/01.ATV.6.5.491

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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