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COMMENTARY

Earthquake and blood pressure

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Stress is induced by various environmental factors. Although humans prepare for stress by adjusting their internal physiological environment, excess stress impairs the homeostasis of the individual. Environmental factors that induce stress are known as stressors. Stressors affect individuals as perceived stress, which varies in relation to individual differences (for example, genes, development, experience).[1] Perceived stress causes an emotional response (depression, anxiety, anger, hostility), a behavioral response (fight or flight, insomnia, personal behavior, diet, smoking, drinking alcohol, exercise) and consequently a physiological response (Figure 1).

Allostasis is the ability to maintain stability and homeostasis during external changes. Allostatic load, which refers to the bodily compensations made in the pursuit of allostasis, causes physiological and pathophysiological changes. Major allostatic responses involve the sympathetic nerve system and the hypothalamus–pituitary–adrenal (HPA) axis (Figure 1). When these systems are activated, catecholamines are released from the adrenal medulla and from the sympathetic nerves. The allostatic load triggered by stress is normally shut off as soon as the stress has passed, and the plasma levels of both catecholamine and cortisol return to the baseline level. Overexposure to allostatic load can occur either because of hyperresponsiveness or delayed recovery, inducing a hyperactivity of the HPA axis and sympathetic nerves, and ultimately leading to cardiovascular disease on chronic phase (Figure 1).

An earthquake is a typical example of an allostatic model, and associations between exposure to earthquake and cardiovascular disease (for example, coronary artery disease) have been reported in Thessaloniki in 1978, Newcastle in 1991, Northridge in 1994 and in Hanshin-Awaji in 1995.[2–5]

Chen *et al.*[6] showed that all participants in the study demonstrated a nondipper diurnal blood pressure change. This finding is very interesting because it means that overexposure to an allostatic load caused a significantly abnormal circadian rhythm. A natural disaster (for example, an earthquake) can increase the risk of cardiovascular disease through increased blood pressure, an abnormal diurnal change of blood pressure, increased blood viscosity and increased platelet and hemostatic activation. It has also been reported that ventricular arrhythmias were increased more than twofold among patients with implantable cardioverter-defibrillators after the World Trade Center attack on 11 September 2001.[7] These events might be considered as cases of overexposure to allostatic load.

Chen *et al.*'s[6] report showed that blood pressure level increased rapidly in their patients after an earthquake, and remained high until 6 h after the earthquake. Parati *et al.*[8] were the first to report a patient undergoing 24-h ambulatory blood pressure monitoring during an earthquake. Their patient experienced an acute 20% increase in systolic blood pressure and an acute 79% increase in heart rate. Chen *et al.*'s[6] report also showed a markedly increased blood pressure and heart rate. These two earthquakes occurred in the afternoon. This raises a question, what changes in diurnal blood pressure would be observed if 24-h ambulatory blood pressure monitoring were to be recorded during an early morning earthquake, such as the Northridge and Hanshin-Awaji earthquakes? We expect that an earthquake that occurred in the early morning, when sympathetic nerve activity is increased, might have a greater impact on diurnal blood pressure change. In fact, the number of cardiovascular events after the Hanshin-Awaji earthquake was significantly increased in the early morning and night.[9] It has also been reported that there was no statistically significant increase in acute myocardial infarction admissions in the San Francisco Bay Area on the day of the Loma Prieta earthquake in 1989 (at 1704 hours) compared with those on the days before or after the earthquake or compared with those on the same day in 1990. In contrast, there was a >80% increased risk of admission for acute myocardial infarction on the day of the Northridge earthquake in 1994 (at 0431 hours) compared with that on the same date in 1990.[10] Thrombotic factors show a diurnal change, and coronary heart disease deaths (myocardial infarction and sudden death) occurred 1.8 times more often at night (between 2300 and 0500 hours) and 1.4 times more often during the morning (between 0500 and 1100 hours) after the Hanshin-Awaji earthquake.[9,11] The combination of a nondipper blood pressure pattern and diurnal change of thrombotic factors might lead to a cardiovascular event.

Another interesting question is how long the nondipper blood pressure pattern lasts after an earthquake. Stress continues after an earthquake because of the fear of an aftershock. We previously reported three individuals who had white-coat hypertension and who underwent 24-h ambulatory blood pressure monitoring before and after the Hanshin-Awaji earthquake. All developed sustained hypertension, which persisted for 2 months after the earthquake. The mean values of 24-h blood pressure, day blood pressure and night blood pressure, which were increased 2 months after the earthquake, decreased 8–11 months after the earthquake to values close to those before the earthquake, and sustained hypertension did not continue.[12] We observed an 18 mm Hg increase in systolic blood pressure, and an 8 mm Hg increase in diastolic blood pressure in hyper-

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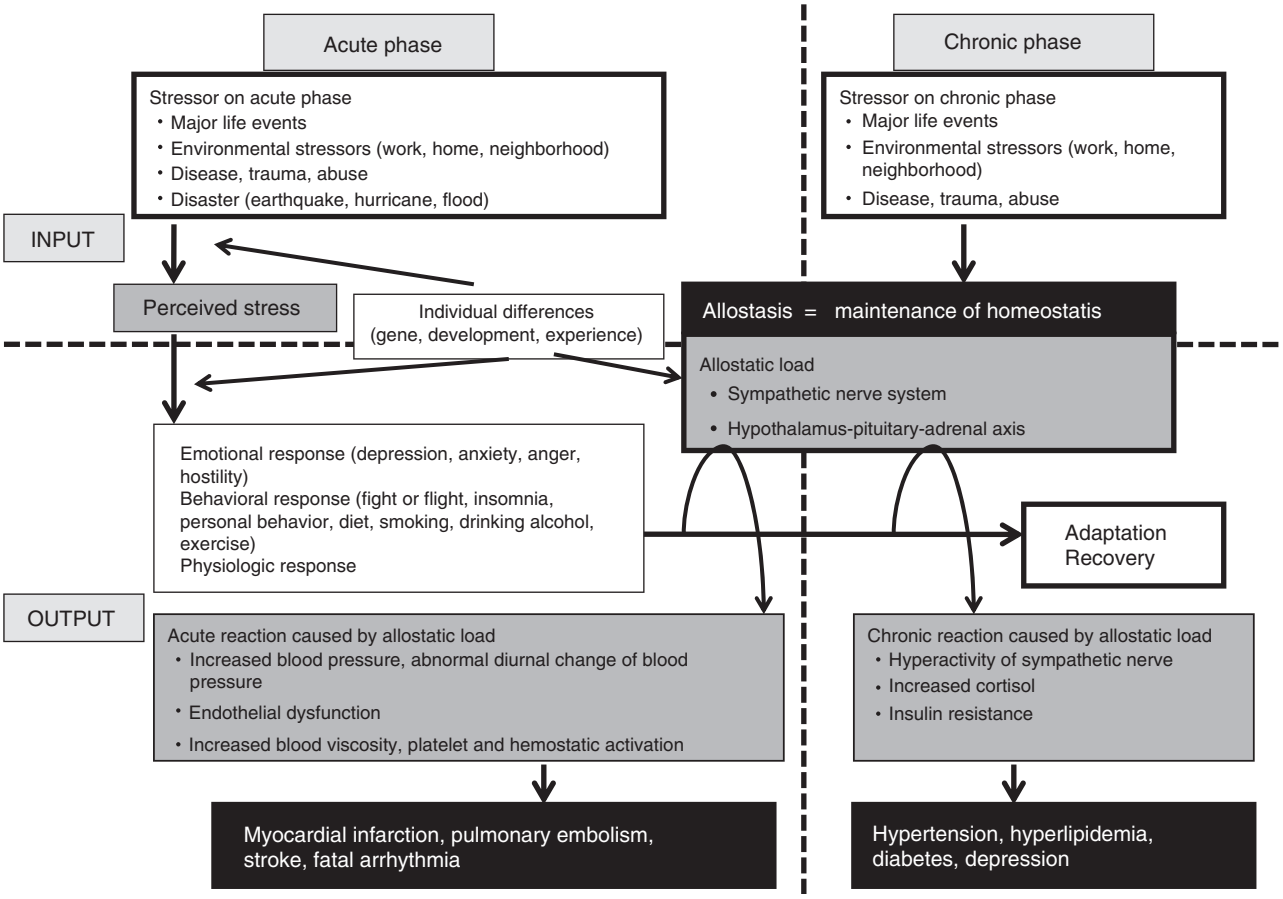

Figure 1 Stressor, allostatic load and cardiovascular disease.

tensives 2 weeks after the Hanshin-Awaji earthquake,[13] and the earthquake-induced blood pressure increase was prolonged in patients with microalbuminuria for at least 2 months after the earthquake.[14] Kamoi *et al.*[15] showed that the mean systolic morning home blood pressure and the median urinary albumin excretion rate increased significantly within 3 months and returned to the pre-earthquake level at 6 months, and increased systolic and diastolic morning home blood pressures were significantly associated with urinary albumin excretion rate increase in type II diabetic patients after the 2004 Mid Niigata Prefecture earthquake. Urinary albumin excretion has an important function in the progression of hypertension after an earthquake, and the effect of this parameter is greater than that of other hypertensive organ damage. We also showed that the plasma levels of a fibrin degradation product (D-dimer) in well-controlled hypertensives increased between 1 and 2 weeks after the Hanshin-Awaji earthquake. Individual differences in physiological recovery from earthquakes remain unclear.

Changes in diurnal blood pressure after an earthquake, which constitutes a major stressor, show individual differences. Chen *et al.*'s[6] report is very important in this regard because individual differences in diurnal blood pressure after the Wenchuan earthquake were observed in 11 participants. Although the mid/long-term changes in blood pressure after an earthquake remain unclear, it is important that blood pressure control be decided by not only basing it on clinic blood pressure but also referring to home/shelter blood pressure to prevent earthquake-related cardiovascular events.[16] Although the efficacy of hypertensive medications (for example, β blockers) in ameliorating the hyperactivity of sympathetic nerves after an allostatic load remains unclear, a sudden and marked increase in blood pressure and heart rate presents a risk of cardiovascular events, even if the change is transient. Therefore, physicians should control blood pressure by reference to changes in clinic/home blood pressure chronologically during and after the disaster.

1 Kario K, McEwen BS, Pickering TG. Disasters and the heart: a review of the effects of earthquake-induced stress on cardiovascular disease. *Hypertens Res* 2003; **26**: 355–367.
2 Katsouyanni K, Kogevinas M, Trichopoulos D. Earthquake-related stress and cardiac mortality. *Int J Epidemiol* 1986; **15**: 326–330.
3 Dobson AJ, Alexander HM, Malcolm JA, Steele PL, Miles TA. Heart attacks and the Newcastle earthquake. *Med J Aust* 1991; **155**: 757–761.
4 Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. *N Engl J Med* 199; **334**: 413–419.
5 Suzuki S, Sakamoto S, Koide M, Fujita H, Sakuramoto H, Kuroda T, Kintaka T, Matsuo T. Hanshin-Awaji earthquake as a trigger for acute myocardial infarction. *Am Heart J* 1997; **134**: 974–977.
6 Chen Y, Li J, Xian H, Li JB, Liu S, Liu GJ, Lin JN, Han J, Zeng Z. Acute cardiovascular effects of the Wenchuan earthquake: ambulatory blood pressure monitoring of hypertensive patients. *Hypertens Res* 2009; **32**: 797–800.
7 Steinberg JS, Arshad A, Kowalski M, Kukar A, Suma V, Vloka M, Ehlert F, Herweg B, Donnelly J, Philip J, Reed G, Rozanski A. Increased incidence of life-threatening ventricular arrhythmias in implantable defibrillator patients after the World Trade Center attack. *J Am Coll Cardiol* 2004; **44**: 1261–1264.
8 Parati G, Antonicelli R, Guazzarotti F, Paciaroni E, Mancia G. Cardiovascular effects of an earthquake: direct evidence by ambulatory blood pressure monitoring. *Hypertension* 2001; **38**: 1093–1095.
9 Kario K, Ohashi T. Increased coronary heart disease mortality after the Hanshin-Awaji earthquake among the older community on Awaji Island. Tsuna Medical Association. *J Am Geriatr Soc* 1997; **45**: 610–613.
10 Brown DL. Disparate effects of the 1989 Loma Prieta and 1994 Northridge earthquakes on hospital admissions for acute myocardial infarction: importance of

superimposition of triggers. *Am Heart J* 1999; **137**: 830–836.
11 Kario K, Ohashi T. After a major earthquake, stroke death occurs more frequently than coronary heart disease death in very old subjects. *J Am Geriatr Soc* 1998; **46**: 537–538.
12 Kario K, Matsuo T, Shimada K. Follow-up of white-coat hypertension in the Hanshin-Awaji earthquake. *Lancet* 1996; **347**: 626–627.
13 Kario K, Matsuo T, Kobayashi H, Yamamoto K, Shimada K. Earthquake-induced potentiation of acute risk factors in hypertensive elderly patients: possible triggering of cardiovascular events after a major earthquake. *J Am Coll Cardiol* 1997; **29**: 926–933.
14 Kario K, Matsuo T, Shimada K, Pickering TG. Factors associated with the occurrence and magnitude of earthquake-induced increases in blood pressure. *Am J Med* 2001; **111**: 379–384.
15 Kamoi K, Tanaka M, Ikarashi T, Miyakoshi M. Effect of the 2004 Mid-Niigata Prefecture earthquake on home blood pressure measurement in the morning in type 2 diabetic patients. *Clin Exp Hypertens* 2006; **28**: 719–729.
16 Kario K, Shimada K, Takaku F. Management of cardiovascular risk in disaster: Jichi Medical School (JMS) Proposal 2004. *Jpn Med Assoc J* 2005; **48**: 363–376.