PERSPECTIVES: GEOLOGY

The 17 August 1999 Izmit Earthquake

Aykut Barka

On 17 August 1999 at 3:02 a.m. local time, northwest Turkey was shaken by a magnitude 7.4 earthquake, catching most people in their sleep. The earthquake's epicenter was located at latitude 41.81°N and longitude 30.08°E, and the earthquake had a depth of around 10 to 16 km. Records of the maximum ground motion varied between 0.3 and 0.4*g*. The earthquake lasted 37 s and consisted of two subevents, the first triggering the second. The nearest major city affected by the earthquake was Izmit at the eastern end of the Marmara Sea, but the earthquake also caused considerable damage in Istanbul, about 70 km away from the earthquake's epicenter. The earthquake's magnitude was initially estimated as 6.7 by the Kandilli observatory, which operates a seismic network in the region. This estimate was considerably lower than the actual magnitude of 7.4 and initially gave a wrong impression of the likely damage to scientists and governmental officials.

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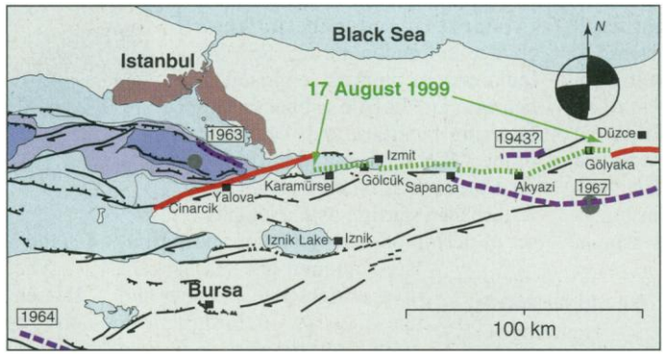
During the first 2 days, the severity and extent of the damage and human loss were seriously underestimated. Rescue operations were delayed because of lack of communications and heavy damage on the major highway connecting Istanbul to Ankara. Alternate roads were blocked by people wanting to reach relatives and friends in the most affected areas because all telephone communication had broken down. Many apartment blocks collapsed completely, causing the death of many people in the earthquake region. A fire in the Tupras oil refineries, which refine 86% of Turkey's oil, threatened to take over other industrial sites and took 5 and a half days to contain and extinguish. The navy base and shipbuilding yard at Gölcük were cut by surface faulting and displaced 4 m to the right, causing considerable damage and the death of at least 400 soldiers and high ranking officers. The total death toll has now reached 15,000, but thousands more are still missing.

The surface rupture caused by the earthquake consists of four segments (see the figure). Three of them—the Gölcük, Izmit-Sapanca, and Arifiye-Akyazi segments in the west—were caused by the first subevent and extend 90 km, trending ~80°NE; they are separated by small offsets (or stepovers) of less than 1 km. The Gölyaka segment at the eastern end, caused by the second subevent, trends ~65°NE and is about 30 km long. The earthquake started at the western end over the first 12 s and, after a pause of 18 s, was followed by rupture in the east for 7 s. The two ends of the surface break to stop at a stepover at the Karamürsel basin in the west, where the fault is offset by about 5 km, and at Eften Lake in the east. The maximum offset along the surface break was measured near Arifiye, east of Sapanca, where the fault displaced a small country road by 5 m dextrally. The displacement caused by the second subevent in the east was less than 1.5 m. The earthquake was almost pure right-lateral strike-slip, that is, the slip on the fault was parallel to Earth's surface, and the fault plane was almost vertical (see the figure). The major aftershocks, with magnitudes ≥4, were located in the Düzce area south of Adapazari and Izmit and the Cinarcik area.



Fault and earthquake map. The map shows the extent of the 1999 rupture (dotted green lines) and the 1943, 1967, and 1964 ruptures (blue, lines) and the segments that received stress increase (red lines) (*7*).

At Degirmendere, a small town near Gölcük at the western end of the rupture zone, the surface breaks cut the toe of the fan delta where the center of the town was located and caused a slump 100 m normal to the coast line. Part of the town slid under the water, including a hotel and several shops and restaurants. At another fan delta east of Gölcük, a 2-m-high normal fault scarp was produced along a fault, connecting two strike-slip segments. This connecting normal fault had an 8-m-high relic scarp, suggesting the occurrence of at least three previous events besides the 1999 event.

During the earthquake, local people observed a ball of flame and the sound of an explosion in the gulf area. There were also reports of an abundance of dead crabs and jellyfish 2 days before the earthquake in Degirmendere. The former observation can be explained by the release of methane gas, trapped in the gulf on the delta swamp, by the friction on the fault during the rupture. The second observation is probably related to the release of radon gas in the sea. For several days after the earthquake, balloons of strong light coming out of the sea were seen and recorded over the gulf and the northeastern Marmara Sea. Such lights have previously been reported after earthquakes elsewhere (*1*). Similar phenomena have also been recognized by my students in contemporary illustrations of the 1509 and 1556 central Marmara Sea earthquakes published by Ambraseys and Finkel (*2*, *3*). They might be related to the release of radiactive gases from the rupture zone.

The 17 August 1999 earthquake was the seventh in a sequence of earthquakes migrating westward along the North Anatolian fault and caused a 1000-km rupture. The time interval between these westward migrating earthquakes varied from 3 months to 32 years, including the 1999 event. The first earthquake in the sequence occurred in 1939 and caused rupture along a 360-km segment of the fault, with maximum horizontal displacements of up to 7.5 m (*4*). The 17 August 1999 earthquake increased the earthquake risk on the Yalova segment, that is, the western continuation of the northern strand toward the Marmara Sea, and the Düzce-Bolu segment at the eastern part of the Düzce-Hendek fault. The slip at the western end of each previous rupture segment was about 4 m, triggering the buildup to the subsequent event (*4*). The 1999 earthquake also terminated with a 4 m offset at the western end at the Karamürsel basin.

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During the 20th century, only the 1943 Hendek earthquake with a magnitude of 6.4 affected the rupture zone of the 1999 earthquake. However, earlier earthquakes in 1719, 1754, 1878, and 1894 occurred in the Gulf of Izmit (*3*). The 1719 and 1754 earthquakes caused the death of 6000 and 2000 people, respectively, in the Gulf of Izmit, Istanbul, and Adapazari region (*3*). There is little information about the 1878 earthquake, which caused considerable damage and loss of life in the Sapanca and Adapazari regions. The 1894 earthquake caused damage and loss of life (1400 people) from Istanbul to Adapazari. The rupture zone for this earthquake is believed to be located either on the Yalova segment or the Cinarcik basin. Among these earlier earthquakes, the 1719 earthquake is perhaps most similar in magnitude and location to the 1999 earthquake, although it may have occurred closer to Istanbul. The remaining two earthquakes, in 1754 and 1878, probably occurred in the area between eastern part of the Gulf of Izmit and Adapazari.

Recent measurements with the Global Positioning System have indicated that the northern strand of the North Anatolian fault, which goes through the Gulf of Izmit and the northern Marmara Sea, has a slip rate of about 15 mm per year (*5*). This suggests that the recurrence interval for a 4 to 5 m displacement is about 300 years. Modeling of the 1939 to 1967 earthquake sequence (*6*) illustrates that during this period, stress has increased in the Gulf of Izmit region by a few bars. Given the estimated slip rate, information from historical earthquakes, and the modeling results, the location and severity of this earthquake should not have come as a surprise.

References and Notes

1. T. Rikitake, *Earthquake Prediction* (Elsevier, Amsterdam, 1976).
2. N. N. Ambraseys and C. F. Finkel, *Terra Nova* **2**, 167 (1990).
3. _____, *ibid.* **3**, 527 (1991).
4. A. A. Barka, *Bull. Seismol. Soc. Am.* **86**, 1238 (1996).
5. C. Straub, thesis, Eidgenössische Technische Hochschule, Zürich (1996).
6. R. S. Stein, A. A. Barka, J. H. Dietrich, *Geophys. J. Int.* **128**, 594 (1997).
7. The focal mechanism for the ruptures was taken from Harvard CMT.

PERSPECTIVES: BIOMEDICINE

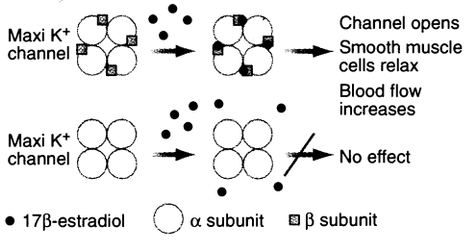
Beating the Odds with Big K

Shai D. Silberberg and Karl L. Magleby

When it comes to longevity, women have an advantage over men because they are less susceptible to cardiovascular disease, at least until the onset of menopause (*1*). This advantage is due largely to the beneficial effects of their estrogen hormones on blood vessels (*2*). Estrogen crosses the plasma membrane of vascular endothelial and smooth muscle cells and binds to specific intracellular receptors. The ligand-receptor complex then alters gene expression, which results in protection of blood vessels from injury and atherosclerosis. In addition to these long-term protective effects, estrogen induces rapid dilation of blood vessels without altering gene expression. However, it is not clear whether estrogen mediates this effect by binding to novel receptors in the plasma membrane, or by activating intracellular nongenomic pathways. In a study on page 1929 of this issue, Valverde *et al.* (*3*) seek to resolve this conundrum. They report that a potassium ion (K^+) channel known to participate in the rapid regulation of blood vessel tone is directly activated by 17β-estradiol, the major circulating estrogen in premenopausal women.

Contraction of vascular smooth muscle decreases the diameter of blood vessels, and thus controls blood flow and blood pressure. Graded changes in the voltage across the plasma membrane of smooth muscle cells in the blood vessel wall result in graded muscle contraction. When the intracellular potential becomes more positive (depolarization), voltage-dependent Ca^{2+} channels in the plasma membrane are activated. The entry of Ca^{2+} into smooth muscle cells through these channels then leads to muscle contraction. This process can be reversed by the opening of K^+-selective channels. The increased efflux of K^+ from muscle cells induces the membrane potential to become more negative, which closes the Ca^{2+} channels, resulting in muscle relaxation (*4*). The realization that modulating the activity of K^+ channels can be used to control blood pressure has sparked considerable interest in identifying K^+ channels in vascular smooth muscle cells and in developing drugs to modulate them (*5*).

One class of K^+ channel that participates in the relaxation of smooth muscle is the large-conductance, calcium-activated K^+ channel (*4*), also affectionately referred to as the Maxi K^+ or Big K channel because of its unusually large conductance. Maxi K^+ channels differ from most other K^+ channels in that their activation is under dual control—switched on by either depolarization or by an increase in intracellular Ca^{2+} (*6*). This dual (often synergistic) activation is possible because of the Maxi K^+ channel's structure. Each of the four α subunits that assemble to form a functional Maxi K^+ channel (*7*) can be divided into two parts: a core (which is similar to that in other voltage-activated K^+ channels) complete with a voltage sensor, and an extended tail that houses an intracellular Ca^{2+} binding domain (*8*). In addition to the pore-forming α subunits common to all Maxi K^+ channels, those in vascular smooth muscle have an auxiliary β subunit that combines with α subunits in a one-to-one stoichiometry (see the figure) (*9*). The β subunit has profound effects on Maxi K^+ channel activity, decreasing by 5- to 10-fold the concentration of intracellular Ca^{2+} required to keep the channel open 50% of the time compared with the β subunit-deficient Maxi K^+ channels in skeletal muscle (*9*, *10*). That the activation of Maxi K^+ channels in vascular smooth muscle leads to rapid dilation of blood vessels raises the question of whether estrogen binding to the β subunit is involved in this process.



Maximizing the benefits of K^+ channels. The Maxi K^+ channel of vascular smooth muscle cells is composed of both α and β subunits (**top**), whereas that of skeletal muscle cells is composed of α subunits alone (**bottom**). 17β-Estradiol binds to and increases the activity of Maxi K^+ channels with β subunits. The resulting efflux of K^+ from the vascular smooth muscle cells results in closure of Ca^{2+} channels and relaxation of the muscle in the blood vessel wall.

Valverde *et al.* (*3*) address this question by expressing human Maxi K^+ channels in *Xenopus* oocytes and examining the effects of estrogen on channel activity. They found that 17β-estradiol increased the activity of Maxi K^+ channels composed of both α and β subunits, but had no effect on channels composed of α subunits alone. When estrogen was coupled to bovine serum albumin (which prevents the hormone from crossing the plasma membrane), the Maxi K^+ α/β channel

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