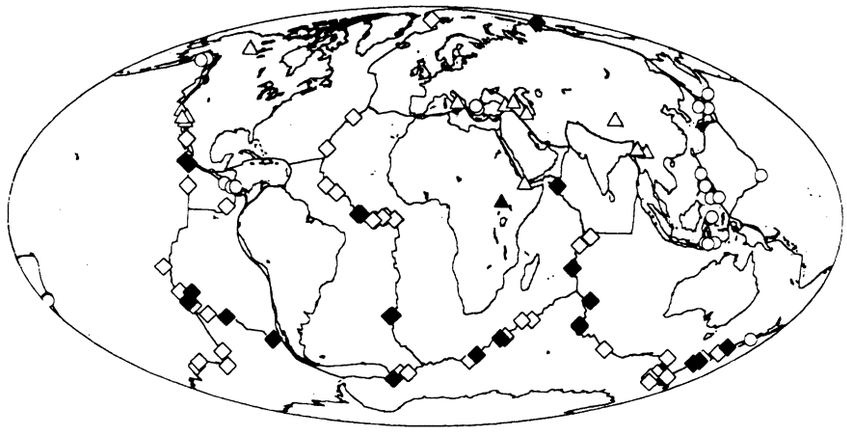
Before the quake: Detecting the slow groan

Earth typically gives some clue before unleashing its fury. People threatened by hurricanes, volcanic eruptions, and other natural disasters receive anywhere from a few minutes to several days of warning, often enabling them to seek safety. Yet the planet extends no such courtesy in the case of earthquakes, which catch victims completely unawares and have thus far eluded scientific attempts at prediction.

A pair of seismologists now reports finding evidence that some large earthquakes start off slowly, building up for several minutes before letting loose violent vibrations. While the discovery does not provide a direct means of forecasting tremors, it yields new and puzzling information about the birth of earthquakes — a topic scientists must address before they can even hope to predict damaging shocks.



Ihmlé and Jordan/SCIENCE

On a map of Earth's tectonic plates, shaded diamonds show 19 oceanic earthquakes that started slowly. A 1990 jolt in southern Sudan (shaded triangle) was the only continental earthquake that displayed similar precursory features.

"We're looking at something new in terms of physics. With the data we have, we're facing some new type of earthquake dynamics that is not understood," says Pierre F. Ihmlé of the Institut de Physique du Globe in Paris. Ihmlé and Thomas H. Jordan of the Massachusetts Institute of Technology described their earthquake search in the Dec. 2 SCIENCE.

Ihmlé and Jordan studied the low-frequency vibrations recorded from 107 shallow-depth earthquakes around the world. In 20 cases, they found indirect evidence of large energy releases — called slow earthquakes — starting hundreds of seconds before the jerky, high-frequency vibrations that traditionally define the start of an earthquake. Scientists have identified isolated examples of slow precursors in the past, but this is the first systematic search, Jordan says.

If seismologists could catch slow precursors in action, they could — in theory — alert endangered populations minutes before the start of damaging quakes. But the feasibility of such a scenario remains unclear.

Because all but 1 of the 20 slow earthquakes occurred on oceanic faults, Ihmlé and Jordan cannot tell how their findings relate to continental earthquakes, which pose the most risk to people. If the slow precursors arise from unusual properties of oceanic rock, they may not typically precede tremors on land. In fact, scientists have found no evidence of such slow deformation prior to well-monitored earthquakes in California.

Ihmlé and Jordan call their conclusions tentative because they have not been able to see the slow precursors directly on seismograms of the 20 earthquakes they studied. In fact, they delayed reporting their results for several years in order to rule out alternative interpretations of the data.

William L. Ellsworth of the U.S. Geological Survey in Menlo Park, Calif., describes these findings as tantalizing. "It reinforces the possibility that there are short-term processes that are occurring immediately before the earthquake propagates down the fault," says Ellsworth. But he adds that "seismologists are always going to be cautious until they can see things in the actual seismograms."

— R. Monastersky

Researchers spot another blinding gene

Investigators have scored again in their efforts to understand the genetic causes of macular dystrophies, eye disorders that damage the central portion of the retina. These diseases include age-related macular degeneration, a leading cause of blindness.

Because the disease strikes late in life, getting enough family members together for a genetics study can prove difficult, says Edwin M. Stone of the University of Iowa in Iowa City. Parents have usually died by the time the disorder strikes their children. So to understand the genetics of the eye disorder, researchers often study closely related diseases.

In this latest discovery, scientists find that two mutations on the gene for an enzyme called tissue inhibitor of metalloproteinases-3 (TIMP3) may lead to Sorsby's fundus dystrophy (SFD), which usually strikes adults before age 50, report Bernhard H. F. Weber of the Julius Maximilians University in Würzburg, Germany, and his colleagues.

"It's a very, very significant contribution," says Stone, who coauthored earlier studies describing a gene associated with a similar macular disorder (SN: 7/18/92, p.37).

Weber's team found different mutations in TIMP3 in two unrelated families with SFD. The investigators could not detect mutations in healthy family members or in 176 other volunteers.

"To our knowledge, this is the first report providing strong evidence that mutations in a member of the TIMP gene family are causative of a degenerative human disease, namely SFD," Weber's group reports in the December NATURE GENETICS.

The TIMP3 mutations may promote excessive growth of blood vessels through Bruch's membrane, which lies below the retina, they speculate. As a result, the membrane thickens or bleeds, disrupting vision. How the mutations cause this growth remains a mystery, but it occurs more often in SFD patients than in those with other macular dystrophies.

Researchers don't know whether these findings will ever lead to gene therapy for macular disorders. Stone argues that too many people develop macular degeneration for the disease to be a candidate for gene therapy.

"You're probably not going to go in there and replace the TIMP3 gene in the retina in a bunch of 70-year-olds. What you're going to do, hopefully, is understand why the blood vessels [grow excessively] and come up with some drug or something that can shut that down."

The retinal degeneration-slow–peripherin gene, the only other specific gene implicated in macular dystrophy, helps maintain the structure of the photoreceptors in the eyes. Some mutations in the gene appear to lead to a degenerative retinal condition known as retinitis pigmentosa, while others cause macular disease, Stone says.

"It remains to be shown . . . how exactly all of these mutations cause these diseases. . . . That's going to take a whole bunch more work," Stone points out.

— T. Adler