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# Predicting large tsunamis

*Emile A. Okal*

IN 1992, two catastrophic tsunamis killed more than 2,000 people. These giant sea waves, generated by earthquakes or submarine landslides, can travel across the oceans and bring destruction to faraway shores: in 1960, an earthquake in Chile led to the deaths of more than 100 people in Japan. In the case of the Nicaraguan earthquake on 2 September last year, the death toll was high because the ground tremors felt by the coastal populations were deceptively mild. On page 714 of this issue[1], Kanamori and Kikuchi take a hard look at the seismic data available for this earthquake. The long-period seismic waves measured at the time should, they say, have conveyed the much-needed warning.

In the Nicaraguan event, there was an alarming disparity between the fairly low conventional magnitude (reported to be as low as $m_b$ = 5.3) and the size of the tsunami generated: in some sections of the coast, the earthquake was not even felt by the local people who were swept away by the waves a few minutes later. The other major tsunami of 1992, on 12 December, completely destroyed the village of Riangkrok on Flores Island, Indonesia (see the accompanying box). The waves at some locations reached 26 metres in height, whereas along most of the shoreline the maximum amplitudes were only 3 to 4 metres.

Because tsunami waves cross the ocean comparatively slowly (250 m $s^{-1}$, or roughly the speed of a jetliner), early warning should be possible. The risk could be assessed either from reports of the size of sea waves at shorelines closer to the epicentre, or by evaluating the parent earthquake, whose surface waves travel about 15 times faster. Not surprisingly, tsunami generation is often directly related to the earthquake's size — in layman's terms, its magnitude. But the concept of magnitude was developed at a time when there was little theoretical understanding of the propagation and generation of seismic waves, or of the physical nature of the forces required to describe an earthquake source. The chief shortcoming of the conventional 'Richter' magnitude scale, traditionally based on the magnitude at a period of 20 seconds, is that it saturates around $M$ = 8, just where tsunami generation can become substantial. (The body wave magnitude, $m_b$, is measured at a period of about 1 second, and saturates still earlier.) Far better as a measure of earthquake size is the seismic moment, a *bona fide* physical quantity associated with the earthquake source, and measured in physical units (dyn cm). This can be used to extend the concept of magnitude to much lower wave frequencies, thus avoiding the problem of saturation.

Thanks to developments in broadband seismic instrumentation in the 1980s, we can now estimate the seismic moment of a parent earthquake in quasi-real time. Distant coastal populations can then be warned if tsunamis seem likely. Several algorithms exist[2–4], one of which has been successfully tested down to a distance of 1.5° (about 170 km)[5]. If the quake is closer than this, however, there may be only a few minutes in which to assess the earthquake and deliver a warning. Many coastal communities in earthquake-prone areas rely on public education, which can be summarized as "If it shakes really strongly, run for the hills at once".

Enter the ominous 'tsunami earthquakes'. This term was coined by Kanamori in the 1970s to describe events whose tsunami was much larger than expected from their seismic waves[6]. Examples include the 1896 earthquake in Sanriku, Japan; the 1946 Aleutian earthquake (which unleashed possibly the strongest tsunami of the century in the Pacific, despite a Richter magnitude of only 7.2), and smaller events in the Kuriles (1963, 1975) and Peru (1960).

Part of the problem with tsunami earthquakes is that their size may be underestimated if the seismic energy is released exceedingly slowly. A slow source is inefficient at generating the high frequencies that rock buildings and alert their inhabitants (0.2 Hz and above) and even those used in conventional magnitude scales (0.05 Hz and above). But the vibrations can still interfere constructively at the mantle-wave

## Disaster on Flores Island

**THREE weeks after the tsunami hit Flores Island, Indonesia (see Okal's article above), an international tsunami survey team of scientists and engineers from five countries visited the location. They found that, at the site of the village of Riangkrok, located on the northern tip of the easternmost peninsula, the waves had caused devastation at enormous heights above sea level. The maximum height reached was measured as 26.0 m on the south hillside slope; the average of four different measurements was 19.6 m from sea level at the time of the tsunami onslaught. There is now no trace of the village at Riangkrok. It was destroyed and completely washed away. Almost all the coconut trees have been knocked down and washed away, leaving only their root marks still visible on the beach. The area engulfed by the tsunami is unmistakably identifiable as brown, bare ground. The size of the debris and coral rocks (dragged up from the sea floor) can be seen by comparison with the person to the right of the rock in the picture. This sort of destruction is very different from that observed at other tsunami-damaged locations in Flores Island, where the waves ran up to heights of only 3 to 4 m above sea level, and also at the sites of the Nicaraguan tsunamis, where most of the trees survived the tsunami flows, despite being swamped. All the signs indicate that the tsunami flow forces at Riangkrok must have been fiercely strong, as they ripped away everything that had stood in the area. At this small rural village, 137 people lost their lives to the tsunami.** Harry Yeh

*Harry Yeh is in the Department of Civil Engineering, University of Washington, Seattle, Washington 98195, USA.*

frequencies (5 mHz) and at those typical of tsunami waves (1 mHz). Kanamori and Kikuchi have exceptionally clear evidence that the Nicaraguan earthquake arose from a very slow rupture. Nevertheless, its true size (or moment) was accurately given within the hour following the event, as was the case for the Indonesian shock three months later.

For some earthquakes, no clue that large tsunamis were imminent can be sifted from the seismic records. For such events as the 1946 Aleutian tsunami (which destroyed the city of Hilo, Hawaii), it has been proposed that there was massive failure of the sea floor in the form of underwater slumping of sedimentary masses during or after the rupture[7]. Precedents can be found on land (such as the explosion of Mount St Helens in 1980), or even at sea, in the case of the large earthquake off the Grand Banks of Newfoundland in 1929[8].

Kanamori and Kikuchi reconcile the two kinds of tsunami earthquakes, at least in part, by suggesting that sedimentary structures are crucial in both situations. On the one hand, they can account for the slowing down of the seismic rupture, when the latter is forced into mechanically inferior materials such as subducted sedimentary layers; rupture in sedimentary layers does enhance tsunami generation[9]. This mechanism could explain the large Nicaraguan tsunami, which took place in an area where the sparse sedimentary cover of the young plate is subducted straight into the trench without being scraped and piled up as an accretionary structure. On the other hand, where large sedimentary structures exist on the ocean floor, a sizeable earthquake can lead to their total failure in an episode of slumping, providing exceptional energetic coupling with the water column, and throwing up a large tsunami wave.

Local slumps are also thought to be important in smaller earthquakes, such as the 1989 Loma Prieta shock in California. Despite its epicentre being on land, this shock generated a small tsunami in Monterey Bay through a small landslide triggered by strong underwater motion[10]. It is too early to say for sure what caused the exceptional tsunami amplitudes on 12 December at Riangkrok, but the earthquake certainly resulted in subaerial landslides. One may shiver at the thought of what was perhaps the grand-daddy of all Pacific tsunamis, when prodigious underwater slumps, each involving up to 5,000 $km^3$ of sediment, allegedly took place a few million years ago along the Hawaiian chain[11].

The goal of research in tsunami warning can be stated simply as the eradication of tsunami earthquakes, if we follow Kanamori's definition of these as events whose tsunamis we do not understand on the basis of their seismic waves. Kanamori and Kikuchi have made a considerable step forward by digging deeper into the nature of the seismic source and the origin of its low-frequency components. We may realistically hope that slow earthquake components will in the future be identified in time to give coastal residents the signal to run for the hills. □

*Emile A. Okal is in the Department of Geological Sciences, Northwestern University, Locy Hall, 1847 Sheridan Road, Evanston, Illinois 60208, USA.*

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VIRAL TRANSACTIVATION

# Pleiotropy and henchman X

*Michael Gilman*

STANDARD dime-novel detective practice is to tail the henchman; eventually, he will lead you to the master criminal. So it is with virologists, who have reasoned that studying regulatory proteins, especially those of small viruses heavily dependent on host-cell functions, will lead them to the master regulatory machinery of the cell. But these proteins have not yielded their secrets easily, in large part because their activity can result in a variety of consequences. This is pleiotropy, and disentangling the many functions of pleiotropic activators has been the principal challenge facing virologists.

The report by Kekulé *et al.* on page 742 of this issue[1] may lead to the unravelling of pleiotropy for a particularly perplexing viral transactivator, the X protein of hepatitis B virus. It seems that X (denoted HBx in the paper) activates protein kinase C, a key component of cellular signal transduction; in doing so, it achieves a pleiotropic effect on many transcription factors that respond to extracellular signals, and presumably prepares the host cell for the task of replicating the viral genome. Moreover, by targeting protein kinase C, X mimics the activity of tumour promoters, which may explain the virus's unusual oncogenic properties.

Hepatitis B virus (HBV) is a small DNA virus with a genome of 3.2 kilobases which sports a mere four open reading frames. One of these encodes the regulatory protein X, with a predicted relative molecular mass of 17,000. The gene for X is conserved in hepadnaviruses and, where it can be examined, seems to be required for viral replication. The X protein is a transcriptional activator of both the HBV genome and a panoply of cellular promoters[2–4]. In particular, it activates reporter genes under the control of signal-responsive elements such as AP-1/CRE sites and κB elements[5–7].

Kekulé *et al.* now show that activation of AP-1 by X is blocked by several specific inhibitors of protein kinase C (PKC) and by downregulation of the enzyme. They also find that transient expression of the X gene causes production of the membrane-associated lipid activator of PKC, diacylglycerol, and rapid translocation of PKC from cytosol to membrane, a hallmark of PKC activation. Thus, under these conditions, X appears to act (directly or indirectly) by triggering a phospholipase, mimicking an activity of growth factors and hormones.

Although this observation can neatly explain how X can activate so many promoters, the literature remains replete with conflicting results. Two groups have reported that X can function as a transcriptional activator when targeted to a promoter by fusion to a heterologous DNA-binding domain[5,8]. And the protein appears to interact directly with members of the CREB/ATF transcription factor family[9]. Together these data suggest that X has a rather more orthodox transactivation function, one that would not seem to involve changes in phospholipid metabolism. Clearly, however, nearly all the transcription factors affected by X are also responsive to cellular signal transduction pathways, so the involvement of the protein in signal transduction may be the common thread that links many of these observations.

What, then, is the direct target of protein X action? It could be a phospholipase, but this is not sufficient to account for all of the protein's reported activities. More likely, X is itself pleiotropic, either because it directly modifies several target proteins, perhaps through an associated protein kinase activity[10], or because, like a growth-factor receptor, it activates a number of cellular signal-transduction pathways. This latter possibility is attractive because it could account for the observation that X can