SEISMOLOGY

Citizen science for studying earthquakes

Seismologist-citizen partnership helped understand the 2021 Haiti earthquake

By Christa von Hillebrandt-Andrade¹ and Elizabeth Vanacore²

t 8:29 a.m. local time [12:29 universal time coordinated (UTC)] on 14 August 2021, the furthest thing from the mind of Haitians was another devastating earthquake. Many had thought that after the 12 January 2010 earthquake [moment magnitude (M_w) 7.0], they would have a respite from this hazard. In the end, the 2021 quake was even more powerful (M_{w} 7.2), releasing ~40% more energy than the 2010 earthquake (1). Tragically, the earthquake killed 2246 people, injured 12,763, left 329 missing, and affected at least 800,000 more people, 650,000 of whom required emergency humanitarian assistance. In addition, water, sanitation, and health facilities were all severely impaired (2). The impact was compounded because of the sociopolitical and economic challenges plaguing the country. On page 283 of this issue, Calais et al. (3) present a case study in the application of citizen science in real-time earthquake monitoring, response, and scientific inquiry.

Haiti is located on the western portion of the island of Hispaniola on the Caribbean plate that is bounded by the North American plate to the North. The Caribbean plate and the North American plate converge obliquely at a rate of ~2 cm/year, with the Caribbean tectonic plate moving east relative to the North American plate (4). The Puerto Rico Trench, which is the deepest part of the Atlantic Ocean and the Caribbean Sea, along with the North Hispaniola Fault and the Septentrional Fault to the north and the Enriquillo Fault to the south (5), accommodate the strain where the plates converge. These fault systems have consistently generated very large earth-

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quakes and tsunamis based on modern seismic monitoring and historical accounts from the past \sim 500 years (4).

As with most earthquakes, the first questions surrounding the 2021 quake were about its strength, its epicenter, and the possibility of a tsunami. In most countries, a national seismic network or tsunami warning center could provide the answers. However, at the time of this earthquake, the Haitian Seismic Network was not operational, and only five seismometers in the entire country recorded the earthquake—specifically, three citizenhosted sensors, one sensor at the US embassy, and one at a local high school.

The alarm system at the Pacific Tsunami Warning Center (PTWC) in Hawaii—the designated tsunami service provider for the Caribbean and adjacent regions—was triggered by seismic signals emanating from stations in Cuba and the Dominican Republic, which were about 200 and 240 km from the epicenter, respectively. Within 10 min of the earthquake, 5 min slower than the goal response time, the PTWC issued the first bulletin with the earthquake epicenter located 120 km west of Port-au-Prince, Haiti. The notification indicated no tsunami threat based on the initially estimated magnitude of 7.0 (*6*).

Nine minutes later, the US Geological Survey (USGS) issued a preliminary analysis of data, which included more regional and global stations, and determined the magnitude of the earthquake to be 7.2 (1). This larger magnitude then triggered the PTWC to issue a tsunami threat message for Haiti at 9:14 a.m. local time (6). By that time, many people had already self-evacuated, given the strong ground shaking. However, in response to the PTWC message, national authorities issued an official tsunami warning, prompting additional evacuations (7). The PTWC measured a tsunami of only 2 cm at Port-au-Prince at 10:00 a.m. local time. It then issued a final threat message at 10:19 a.m. local time (6), after which the warning was canceled (7). Firefighters remove debris in search of survivors after the August 2021 earthquake in Haiti. First responders, such as the ones shown here, will benefit from the improved earthquake monitoring provided by the citizen-science Raspberry Shake network.

Access to nearer field seismic and sea level data could have resulted in a more rapid analysis of the earthquake and an earlier warning of a potential tsunami threat by the PTWC. This was the case for the citizenscience network, which integrated data from its stations closer to the epicenter, as well as regional data, and published the earthquake's size and location within a few minutes (8). The parameters, both location and magnitude, that were calculated by the network were comparable to those of the organizations using regional data.

The citizen-science network included 15 plug-and-play low-cost sensors dubbed Raspberry Shakes (RSs), which are class C sensors according to the US Advanced National Seismic System (8, 9). National and international seismologists established the RS network in Haiti in 2019, given the challenges with the national seismic system (10). The program supplies the sensors to private individuals, who in turn provide the electricity and internet. Along with the RS network, seismologists also developed a data-sharing platform named Ayiti-Séismes that integrates data from the RS network and other national and regional seismic data to automatically calculate and display the location and magnitude of local earthquakes (10).

Smaller earthquakes, called aftershocks, follow large earthquakes. The more sensory stations near an earthquake source, the more sensitive the overall sensory network. A more sensitive network would have a lower magnitude threshold and thus detect and report more earthquakes. Based on the data collected by the RS system, 1031 aftershocks were located within the first 3 weeks after the 2021 mainshock. By comparison, the USGS only reported 37 aftershocks in the same time period.

Aftershocks can also be forecasted, but this depends upon the timely and accurate detection of earthquakes; lowering the magnitude threshold of the data used in the forecast model leads to improved aftershock forecasting. The RS network in Haiti demonstrates that these class C sensors can improve earthquake data catalogs that serve as inputs for aftershock forecasting.

Another important question to answer is what fault or faults are responsible for the shaking. With the earthquake locations obtained using the RS data, Calais et al. determined the source of the activity to be an eastwest fault zone ~80 km long with seismicity concentrated in two clusters. They also determined that the cluster to the east, which included the mainshock, was associated with vertical motion along the Enriquillo Fault. Meanwhile, the second cluster further to the west along the Ravine du Sud Fault was associated with lateral motion. The identification of the clusters was possible thanks to the lower magnitude detection threshold of the citizen-science network, thus demonstrating that low-cost sensors can also provide valuable scientific information.

The use and expansion of the low-cost class C sensors will not replace the need for national and regional seismic networks but do provide an avenue to expand network coverage in regions with logistical, economic, geographical, or other challenges that limit possible installation of class A and B sensors. The science-public partnership and the expanded use of RSs or similar instruments, such as in Haiti, also provide a possible avenue to expand earthquake-monitoring capabilities to underserved communities to foster disaster risk reduction.

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METABOLISM

Complex regulation of fatty liver disease

Hepatic lipogenesis is fine-tuned by mechanistic target of rapamycin (mTOR) signaling

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onalcoholic fatty liver disease (NAFLD) is an umbrella term for hepatic abnormalities, including steatosis (fat accumulation, NAFL) and nonalcoholic steatohepatitis (NASH), which is NAFL plus hepatic injury, inflammation, and fibrosis (1). NAFLD has a prevalence of $\sim 25\%$ worldwide and results from the inability of the liver to maintain lipid homeostasis, leading to accumulation of triglyceride (TG), the major energy-storage molecule in mammals. Obesity, insulin resistance, and diabetes mellitus are drivers of NAFLD, so it is not surprising that mechanistic target of rapamycin (mTOR), which sits at the crossroads of nutrient signaling (2), plays a critical role in its etiology. It was also expected that the role of mTOR would be complex, but the extent of this complexity seems endless. On page 364 of this issue, Gosis et al. (3) present evidence that selective inhibition of a noncanonical arm of mTOR complex 1 (mTORC1) signaling inhibits hepatic de novo lipogenesis (DNL) and protects mice from NAFLD.

The normal liver contains between 15 and 75 g of hepatic TG (1 to 5% of an ~1500-g total liver weight). In NAFL, liver fat may increase to 20 to 30% of a 2000-g liver, or ~500 g of hepatic TG. Steatosis can lead to substantial hepatic pathology, including cirrhosis and hepatocellular carcinoma. Four major metabolic processes regulate hepatic TG amounts. The major driver of TG accumulation, which accounts for 65 to 70% of hepatic TG, is delivery of plasma albumin-bound fatty acids (FAs), which are derived mainly from adipose tissue (4). The second pathway for accumulation is DNL, which is the synthesis of TG from acetyl-coenzyme A derived mainly from metabolism of glucose in the mitochondria. DNL can account for 5 to 30% of hepatic TG (4, 5). The two pathways responsible for "disposing" of hepatic TG and maintaining normal hepatic TG content are oxidation of

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FAs and secretion of TG in very-low-density lipoprotein (VLDL). All of these pathways, which are altered in individuals with insulin resistance, obesity, and diabetes mellitus, are regulated, at least in part, by mTOR.

mTOR was identified in the mid-1990s as a protein kinase that was the target of the immunosuppressive drug rapamycin, when in complex with 12-kDa FK506-binding protein (FKBP12) (2, 6, 7). Subsequently, the involvement of mTOR in many central cellular functions beyond immunosuppression was identified, as were two key regulatory components. mTOR exists in two distinct complexes: regulatory-associated protein of mTOR (RAPTOR) "defines" mTORC1, and rapamycin-insensitive companion of mTOR (RICTOR) defines mTORC2. There is a detailed understanding of the regulation of each mTORC by numerous proteins as well as the many downstream processes regulated by each, depending on signals from hormones, nutrients, and energy-producing pathways (2). The number of molecules involved, as well as the many autoregulatory feedback loops, suggests that there are more molecules and pathways left to be discovered, as in the case of NAFLD and DNL.

A link between insulin signaling and the sterol-regulatory element binding proteins (SREBPs), particularly SREBP-1c, was demonstrated in the late 1990s (8, 9). Subsequent studies showed that insulin signaling, through its hepatic receptor, is required for the proteolytic processing and transport to the nucleus of SREBP-1c, where it transcriptionally activates several genes required for DNL. Further studies generated inconsistent and sometimes conflicting data regarding the regulation of DNL by mTORC1. For example, studies indicated that deletion of Raptor. which reduced mTORC1 activity, or deletion of tuberous sclerosis complex 1 (Tsc1) or Tsc2, which activated mTORC1, both resulted in reduced DNL and protection from hepatic steatosis in mice (10, 11). Gosis et al. attempted to clarify these conflicting data. They identified a noncanonical pathway involving the protein folliculin (FLCN) that, when depleted in livers of mice, results in suppressed SREBP-1c activity and DNL, with protection against NAFLD.

^{10.1126/}science.abo5378



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Science, 376 (6590), • DOI: 10.1126/science.abo5378

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