

Delta. The country is home to one of the last populations of the endangered species. PHOTOGRAPH BYCORY RICHARDS

BY LAUREL NEME

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When hundreds of African savanna elephants dropped dead in Botswana's Okavango Delta in 2020, conservationists were alarmed. As the death toll rose-from dozens in March to more than 350 animals by July-their concerns increased, especially because nobody knew what was happening.

A short time later, the mystery deepened when 34 more individuals of the endangered species died across the border in northwestern Zimbabwe in a three-week period, with one more found in November.

"It was very quick," says Chris Foggin, a veterinarian with Victoria Falls Wildlife Trust who examined the elephant carcasses in Zimbabwe. "That was what was so dramatic."

African savanna elephants in the five-country Kavango-Zambezi Transfrontier Conservation Area, or KAZA, represent the majority of what's left of that species, notes Steve Osofsky, director of the Cornell Wildlife Health Center in Ithaca, New York. Around 350,000 African savanna elephants remain, and given the already significant set of threats to their survival, "a new disease could be what tips that last domino towards extinction," says Osofsky, who wasn't involved in the new research.

In September 2020, the Botswana government attributed the deaths of elephants in the Okavango Delta to cyanobacteria in the water that elephants drank, although without data to support it, scientists have questioned this conclusion. Meanwhile, the Zimbabwe carcasses showed no evidence they died from those toxic algae.

And so, the cause of the die-offs continued to elude experts. They ruled out poaching because the tusks were intact, and they found no bullet wounds. Poisoning was unlikely because no other animals, such as vultures feeding on the carcasses, were affected. (Read about the search for answers in Botswana's elephant deaths.)

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Now, new research published recently in Nature Communications points to a different culprit, at least for the Zimbabwe elephants—a bacterium not previously found in elephants of any species, called Bisgaard taxon 45, that causes a massive systemic blood infection called septicemia.

Bisgaard taxon 45 is related to another bacterium, called Pasteurella multocida, that can cause septicemia in cattle and was linked to the death of 200,000 endangered saiga antelope in Kazakhstan in 2015. Yet while it shares many of the same lethal genes, Bisgaard taxon 45 is a separate species.

"It's a disease we know can kill a reasonable number of elephants in a short

space of time," says Foggin, co-leader of the study. And "it has the potential to kill a lot more given the right circumstances."

What those are, however, remains unknown-yet it's pressing to find out. Aerial population surveys in KAZA in 2022 found a high number of new elephant carcasses.



Dozens of elephants have also mysteriously died in Zimbabwe, including this animal. PHOTOGRAPH BY CHRIS FOGGIN

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Finding Bisgaard taxon 45 was no easy task. Scientists examined as many elephant carcasses as they could, first collecting only blood smears because of the possibility of anthrax, a highly communicable bacteria, and then conducting full postmortems on five elephants that had recently died.

The examination revealed the elephants had enlarged livers and spleens, as well as internal bleeding in multiple organs—signs of septicemia. While *P*. multocida was suspected, anomalies in the testing of blood and tissue samples prompted scientists to do additional genetic analysis, leading to the discovery of the different bacteria.

"Doing a postmortem exam on an elephant is tough under any circumstances. But when they're in remote areas, where access is difficult, temperatures are high—it really is heroic work to get the types of diagnostic samples that yield these types of data," Osofsky says.

Scientists don't know how widespread Bisgaard taxon 45 is, or if the bacteria exist as normal flora within elephants and other animals. People bit by captive lions and tigers in the U.S. and U.K. have contracted the bacteria. Scientists have also recorded it in a pet chipmunk in Germany and in healthy captive parrots. (Learn how captive elephants can spread tuberculosis to people.)

"We're assuming it's probably present more frequently than we've known, without causing disease, but we don't have proof of that," Foggin says.

Bacteria often only turn deadly when "something special happens," like the host has a weakened immune system, or it gets into the bloodstream through a wound, explains microbiologist Arnoud van Vliet, a study coauthor and senior lecturer at the University of Surrey's School of Veterinary Medicine in the U.K.

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It's possible environmental stressors, such as heat and lack of food and water, may have led Bisgaard taxon 45 to proliferate and cause the septicemia in the Zimbabwe elephants, says Foggin.

What's next

With much of Africa predicted to get hotter and drier, the bacterium "could cause much worse mortalities down the line," says Foggin. That's why, he says, "it is important to keep monitoring elephant deaths and see if it's responsible."

But understanding where Bisgaard taxon 45 occurs and what triggers it will be tricky. If more incidents occur, scientists can look for trends in environmental conditions and other factors, says study co-leader Laura Rosen, an epidemiologist for the KAZA Animal Health Sub Working Group, which brings livestock and wildlife veterinarians together to manage transboundary animal disease. They can also look to its relative, P. multocida, for clues (See National Geographic's elephant pictures.)

Yet many laboratories in the region aren't equipped with enough resources to discern the differences between P. multocida and Bisgaard taxon 45, she says.

"It is not something anyone would've been looking for before," Rosen says.

Meanwhile, Foggin has already started testing dead or anesthetized elephants on an opportunistic basis, as well as wild lions and other carnivores, for the presence of Bisgaard taxon 45. And he hopes others will do the same.

"It's incumbent on all of us," Osofsky adds, "to build on this study to try to fill in the gaps."



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