Deer protected from deadly disease by newly discovered genetic differences

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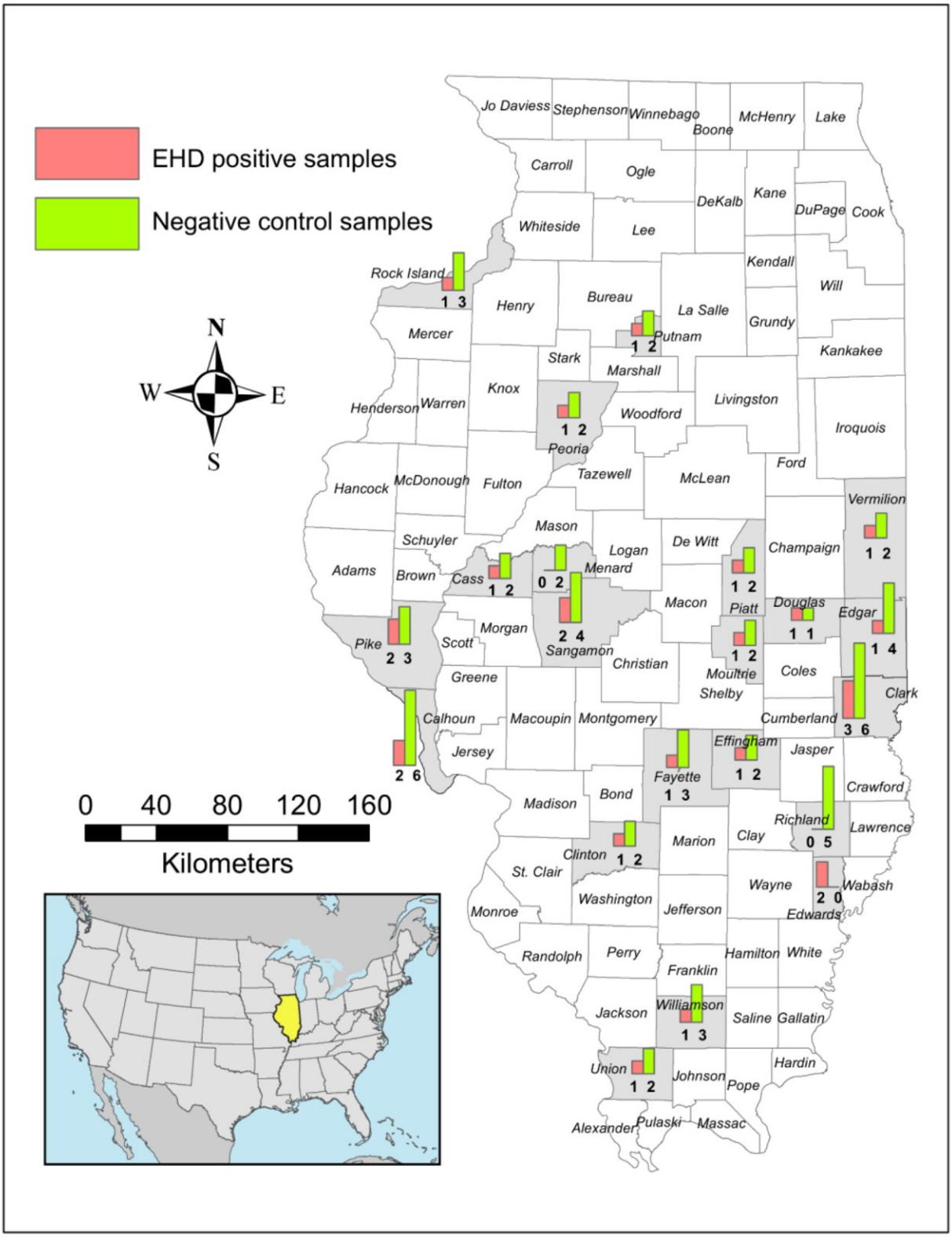

Map showing the provenance of white-tailed deer samples used in this study.

Counties in Illinois from which samples were collected are shaded in gray. The number of EHD-positive deer sampled is indicated by a red bar in each county. Control sample numbers are indicated by a green bar in each county. Numbers are shown under each bar. Credit: *Genes* (2023). DOI: 10.3390/genes14020426

It was the height of summer 2022 when the calls started coming in. Scores of dead deer suddenly littered rural properties and park preserves, alarming the public and inconveniencing landowners. According to officials at the Urbana Park District, it was Epizootic Hemorrhagic Disease (EHD), a midge-borne viral illness that pops up in white-tailed deer populations around the state every few years. And when susceptible deer are infected, they die within days.

Now, University of Illinois scientists have found gene variants in deer associated with the animals' susceptibility to EHD.

"This is the first time this gene has been sequenced completely in white-tailed deer. This is important because without the sequences, there's no starting point to do any kind of research," says study co-author Alfred Roca, professor in the Department of Animal Sciences, part of the College of Agricultural, Consumer and Environmental Sciences (ACES) at U of I.

The team sequenced the gene for Toll-Like Receptor 3 (TLR3), a protein that spans membranes of intracellular organelles in immune cells and helps recognize double-stranded RNA (dsRNA) viruses. When a dsRNA virus, such as the one that causes EHD, enters the cell, TLR3 activates the host's first immune defenses, triggering inflammation and priming the rest of the immune system.

When the team sequenced TLR3 from EHD-infected and uninfected

deer, they found dozens of variable sites in the DNA known as single-nucleotide polymorphisms (SNPs). Two of the SNPs were significantly more common in uninfected deer.

"Because we found mutations in TLR3 more frequently in EHD-negative animals, we think deer with these mutations are less susceptible to EHD," says co-author Yasuko Ishida, research scientist in the Department of Animal Sciences at U of I.

That conclusion is rooted in the probability that many white-tailed deer in Illinois are exposed to EHD in their lifetimes, but only some will die from the disease.

In many areas, outbreaks occur every 3-5 years, when environmental conditions favor the life cycle of midges that carry the virus. The midges spend their larval stages in mud under ponds and puddles where deer drink during drought conditions. As those water sources dry up, usually during late summer, the midges' muddy habitat is exposed and the adult flies emerge to bite and infect deer. The cycle can be interrupted locally by a soaking rain or a cold snap, which is why outbreaks don't happen every year.

The researchers emphasize that EHD is not transmissible to humans or pets through midge bites or consumption of infected deer meat.

Although there's not much wildlife managers can do to disrupt the cycle and prevent outbreaks in natural habitats, the team says it's still helpful to understand the genetic underpinnings of the disease. Theoretically, deer in captive herds could be sampled to characterize the level of vulnerability to EHD, and wild herds could be sampled during the hunting and EHD-outbreak seasons, informing managers and the public of future risk.

"The value of this research is that it helps inform the public about EHD. It helps them to understand not only what the disease will look like, but potentially the severity of an outbreak in a particular area. Sometimes there's value in knowing what to expect," says study co-author Nohra Mateus-Pinilla, wildlife veterinary epidemiologist at the Illinois Natural History Survey, with adjunct appointments in the Department of Animal Sciences, the Department of Natural Resources and Environmental Sciences in ACES, and the Department of Pathobiology.

Considering the disease's episodic nature, it's not likely to present as a severe outbreak again in Urbana parks anytime soon. But it is an increasing threat to the state's northern regions, including Chicagoland. Another recent study by Mateus-Pinilla, Roca, and others published in *Pathogens* shows the disease has been slowly but steadily moving northward in Illinois. The researchers don't know whether that's due to climate change or greater reporting, but it's clear EHD isn't restricted to rural parts of Illinois.

"It's very complicated to respond to an outbreak of EHD because there are often large numbers of deer found dead near water. People don't know what to do when that happens, but we encourage the public to report potential EHD outbreaks to their local IDNR wildlife biologist for the surveillance and future study of the disease," says Jacob Wessels, who completed the research as part of his master's degree and now serves as a conservation police officer with the Illinois Department of Natural Resources.

The article, "The Impact of Variation in the Toll-like Receptor 3 Gene on Epizootic Hemorrhagic Disease in Illinois Wild White-tailed Deer (Odocoileus virginianus)," is published in *Genes*.

More information: Jacob E. Wessels et al, The Impact of Variation in the Toll-like Receptor 3 Gene on Epizootic Hemorrhagic Disease in

Illinois Wild White-Tailed Deer (Odocoileus virginianus), *Genes* (2023). DOI: 10.3390/genes14020426

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