

Transmission of antibiotic resistant *E. coli* mapped in wild giraffe social networks

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Escherichia coli. Credit: Rocky Mountain Laboratories, NIAID, NIH

A team from the University of Minnesota has shown that antibiotic-resistant *Escherichia coli* bacteria in wild giraffes most likely come from anthropogenic sources, such as local cattle herds and humans. The

research is published in *Applied and Environmental Microbiology*.

"We found that the majority of antibiotic resistance [genes](#) identified in giraffe *E. coli* had been previously identified in *E. coli* from both humans and domestic cattle in East Africa," said corresponding author Elizabeth A. Miller, Ph.D., Postdoctoral Research Associate, College of Veterinary Medicine, University of Minnesota, St. Paul. There was little evidence that the bacteria were being transmitted between giraffes via social interactions, she said.

Surprisingly, giraffes three months of age or younger were more likely to harbor antibiotic resistant *E. coli* than other age groups. "This is particularly surprising as giraffe neonates nurse from their mothers exclusively and interact minimally with other group members, leading us to predict they would have low exposure to [resistant bacteria](#) and residual [antibiotics](#) in their environment," said Dr. Miller. "These results suggest there may be competition between antibiotic resistant and susceptible *E. coli* strains in the giraffe neonatal gut, with resistant *E. coli* having a selective advantage.

Just why antibiotic resistant *E. coli* outcompete the susceptible strains is a matter of pure speculation at this point, said Dr. Miller. "One intriguing theory refers to iron acquisition genes that have been observed to occur on the same plasmid as resistance genes," she said. "Bacteria with these extra iron acquisition genes might have a selective advantage in this iron-depleted environment, and any [antibiotic resistance genes](#) associated with the iron resistant genes would be carried along for the ride," she said, noting that the theory had previously been suggested in a 2004 article in this journal.

The research is contributing to scientific understanding of how resistance genes are spread in natural ecosystems, said Dr. Miller. "This research also highlights the potential importance of host age as a

predictor of harboring antibiotic-resistant bacteria—at least in giraffes. Further, the fact that resistant *E. coli* were more frequently identified in neonatal giraffes suggests that the neonate gut may represent a site of complex competition dynamics between microbe members. While similar age-related patterns have been observed in humans and some domesticated animals—especially cattle—to our knowledge, this study is the first to show this striking pattern in a wild mammal."

Coauthor Kimberly VanderWaal, then a Ph.D. student at the University of California, Davis, conducted the first part of the research in a wildlife reserve in Kenya that integrates commercial cattle ranching with wildlife conservation, as part of her Ph.D. thesis. She conducted the behavioral observations from a research vehicle, to determine social group membership and the giraffes' movements within the reserve. Her focus at the time was on how social interaction influences disease transmission. That research, which was novel, and which resulted in several papers as well as the thesis, inspired Dr. Miller to conduct a similar study in baboons, for her Ph.D. thesis.

Following receipt of her Ph.D., Dr. VanderWaal became Assistant Professor, Department of Veterinary Population Medicine, College of Veterinary Medicine, at the University of Minnesota, bringing with her the frozen samples of *E. coli* that she had collected in Kenya. When Dr. Miller found out that Dr. VanderWaal was looking for a postdoctoral researcher, she immediately applied.

Once installed at the University of Minnesota, Dr. Miller used standard antibiotic susceptibility testing to test Dr. VanderWaal's *E. coli* for resistance to six antibiotics and used whole genome sequencing to identify the resistance genes likely responsible for the observed resistance phenotypes. She then conducted social network analyses on the data Dr. VanderWaal had collected, to investigate how these resistance genes might be spreading within the [giraffe](#) population.

"It is important to note that we really do not know what—if any—the risk of clinically resistant bacteria in wildlife is for humans and domestic animals," said Dr. Miller. "While it is theoretically possible for these resistant bacteria to spread into human populations, given they likely originated in humans and/or domestic animals, the risk to us is probably minimal. However, to fully understand how antibiotic resistance spreads at both the local and global level, we need to have an understanding of all reservoirs of antibiotic [resistance](#) and the potential transmission dynamics among them."

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