Surface mutation lets canine parvovirus jump to other species

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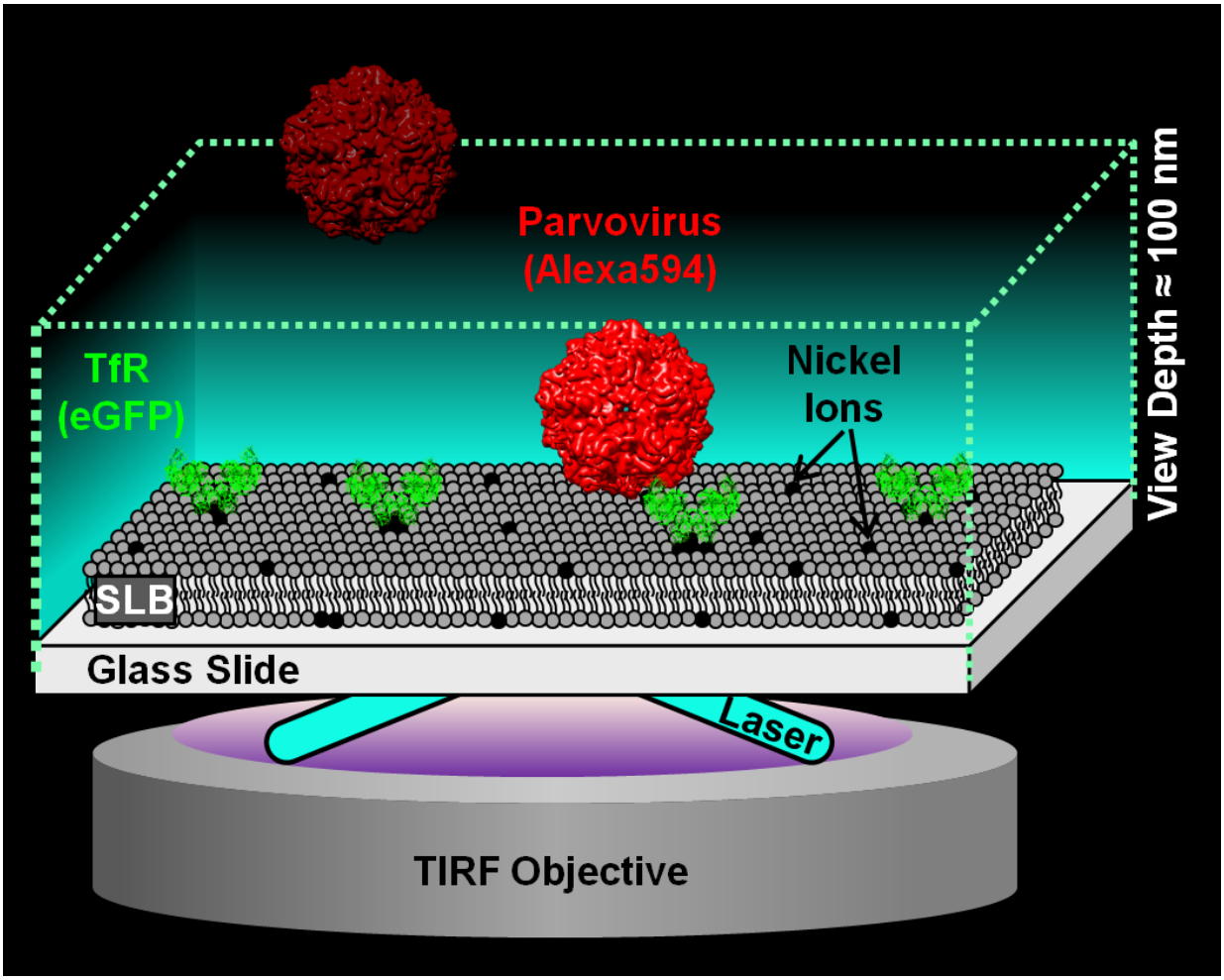

A schematic of the single-particle tracking binding as say used in the research in to parvovirus. Credit: Cornell University

Canine parvovirus, or CPV, emerged as a deadly threat to dogs in the late 1970s, most likely the result of the direct transfer of feline panleukopenia or a similar virus from domesticated cats.

CPV has since spread to wild forest-dwelling animals, including raccoons, and the transfer of the virus from domesticated to wild carnivores has been something of a mystery.

"The underlying issue is, how do viruses jump from one animal to another and what controls viral host range?" said Colin Parrish, the John M. Olin Professor of Virology and director of the Baker Institute for Animal Health at Cornell University.

Parrish co-authored a research paper, published in the *Journal of Virology*, with Susan Daniel, associate professor in Cornell's Robert Frederick Smith School of Chemical and Biomolecular Engineering, which contends that a key mutation in the protein shell of CPV—a single amino acid substitution—plays a major role in the virus' ability to infect hosts of different species.

"That was a critical step," he said. "It took a lot of changes to allow that to happen."

He said another key factor in CPV's infectivity is adhesion strengthening during TfR binding.

"There's an initial attachment, which is probably relatively weak," he said. "The thing just grabs on and holds on a little bit, sort of like using your fingertips. And then it looks like there's a second attachment that is much stronger, where it's like you grab on and hold on with both hands and won't let go."

"We think that the second event, this structural interaction that occurs in

a small proportion of the binding cases, seems to be critical," he said. "We think that it actually causes a change in the virus, that it triggers a small shift in the virus that actually makes it able to infect successfully."

One of Daniel's specialties is the investigation of chemically patterned surfaces that interact with soft matter, including biological materials such as cells, viruses, proteins and lipids. Her lab has pioneered a method called single-particle tracking - placing artificial cell membranes into microfluidics devices, fabricated at the CNF, to study the effect of single virus particles on a variety of membrane host receptors, in this case from both dogs and raccoons.

"The nice thing about these materials is that we can design them to have all different kinds of chemistries," she said. "So in this particular study, we can put the receptor of interest in there, isolated from everything else so we can look at the specific effect of that receptor on a particular virus interaction."

Daniel's lab also developed the precision imaging devices used in the study. "Another piece of this paper is how the parvovirus actually sits down and binds even stronger over time with that receptor," Daniel said. "That was kind of a new result that came out of the technique itself, being able to look at individual binding events."

"When this virus infects a young animal, it can be fatal," Parrish said. "It's very unpleasant, and if you own a puppy or a kitten, that's why you should vaccinate."

Provided by Cornell University

https://phys.org/news/2016-04-surface-mutation-canine-parvovirus-species.html