# Wallabies and bats harbor 'fossil' genes from the most deadly family of human viruses

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Research by Derek Taylor has shown that modern marsupials harbor a "fossil" copy of a gene that codes for filoviruses, which cause Ebola and Marburg hemorrhagic fevers and are the most lethal viruses known to humans. Credit: University at Buffalo

**Modern marsupials may be popular animals at the zoo and in children's books, but new findings by University at Buffalo biologists reveal that they harbor a "fossil" copy of a gene that codes for filoviruses, which cause Ebola and Marburg hemorrhagic fevers and are the most lethal viruses known to humans.**

Published this week in the online journal *BMC Evolutionary Biology*, the paper ("Filoviruses are ancient and integrated into mammalian genomes") demonstrates for the first time that mammals have harbored filoviruses for at least tens of millions of years, in contrast to the existing estimate of a few thousand.

It suggests that these species, which maintain a filovirus infection without negative health consequences, could have selectively maintained these so-called "fossil" genes as a genetic defense.

The work has important implications for the development of potential human vaccines, as well as for the modeling of disease outbreaks and the discovery of emerging diseases, including new filoviruses.

"This paper identifies the first captured 'fossil' copies of filovirus-like genes in mammalian genomes," says Derek J. Taylor, PhD, associate professor of biological sciences in the UB College of Arts and Sciences and co-author. "Our results confirm for the first time that several groups of mammals, including groups such as marsupials that never colonized Africa, have had an association with filoviruses."

The UB co-authors say that if the rarely captured genes represent antiviral defenses or genomic scars from persistent infections, then the work opens up new possibilities for identifying reservoir species for filoviruses, which harbor the virus but remain asymptomatic.

"The reservoir for filovirus has remained a huge mystery," says Jeremy A. Bruenn, PhD, UB professor of biological sciences and co-author. "We need to identify it because once a filovirus hits humans, it can be deadly."

When the UB researchers studied samples from the fur of a wallaby at the Buffalo Zoo and a brown bat caught on the UB campus, they found that the genomes of both animals as well as some other small mammals contain "fossil" copies of the gene for these deadly viruses, and thus could be candidate reservoir species for them.

"Who knew that the bats in the attic as well as modern marsupials harbored fossil gene copies of the group of viruses that is most lethal to humans," asks Taylor.

The research also demonstrates a new mechanism by which different species of mammals can acquire genes, through non-retroviral integrated RNA viruses, which the UB scientists had previously identified in eukaryotes but was unknown in mammals.

The UB scientists note that it is well-known that RNA retroviruses, like HIV-AIDS, can be integrated into mammal genomes.

"But because filoviruses infect only the cytoplasm of cells and not the nucleus and because they have no means of making DNA copies that might be integrated into the genome -- as retroviruses do -- it was never thought gene transfer could occur between non-retroviral RNA viruses and hosts," says Bruenn. "This paper shows that it does and it may prove to be a far more general phenomenon than is currently known."

The research also reveals that existing estimates that filoviruses originated in mammals a few thousand years ago were way off the mark.

"Our findings demonstrate that filoviruses are, at a minimum, between 10 million and 24 million years old, and probably much older," says Taylor. "Instead of having evolved during the rise of agriculture, they more likely evolved during the rise of mammals."

Provided by University at Buffalo

"Wallabies and bats harbor 'fossil' genes from the most deadly family of human viruses." July 2nd, 2010. www.physorg.com/news197298768.html