

Transmissions between mammals demonstrate the serious dangerousness that mutations of avian influenza viruses might lead to an epidemic or pandemic for humans

Münster, Germany, 08.05.2023. Public debate, press articles and, most recently, scientific publications also have alerted to a potentially threatening development related to the avian flu virus currently in circulation around the globe.

After an outbreak in Peru highly pathogenic avian influenza (HPAI) killed 3,500 South American sea lions, and is raising fears that humans could become more vulnerable, because there are concerns that bird flu could be getting better at crossing over to mammals after the disease.

In the past year the virus has spread rapidly among poultry and wild birds, but it is not thought to have been spreading between mammals. However, the virus responsible for an outbreak of HPAI at a Spanish mink farm in which over 50,000 American animals were killed was found to have acquired a mutation that allowed it to replicate faster in mammals. This spread of avian influenza into and between wild mammals has raised concerns that viruses could be getting better at adapting to humans as well. This has only increased by the recent infection of two people in Cambodia in February - one of whom died later.

Most HPAI strains can't infect humans and the ones which can in the past only bound to receptors that are uncommon in the upper airways of mammals, making infections less likely.

The reason is that the HPAI virus use hemagglutinin - a protein on the surface of the virus- like a "key" to open the "lock" of a (e.g. bird) host cell in order to enter this cell.

But this "key" is only fitting if the host cell displays a certain sugar structure on its receptor protein the so called α 2,3-linked sialic acid.

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Such sugar structures are primarily found in the entire body of birds and only in minor amounts in the lower airways in mammals like humans, ferrets, seals and a variety of domestic farm animals and wild animals. Mammals as opposed to birds display mainly a so called α 2,6-linked sialic acid attached to the receptor in cells of their tissue. Structural analysis by scientists of the hemagglutinin molecule of avian influenza virus strains showed that a point mutation that changes the conformation might lead to an increase in affinity for α 2,6-linked sialic acid. This alteration in binding enables the virus to adhere to nasal turbinates which are known to express α 2,6-linked sialic acid. In consequence this might enable the virus to infect and be transmissible between humans and other mammals.

Moreover other important avian influenza virus genes can have point mutations (e.g., NS1, polymerase, NA) and can result in critically pathogenesis in humans and other mammals or adapt to humans. In any way such mutations in the avian influenza virus enable the virus efficiently to attach to the upper respiratory tissues of humans and proliferate with subsequent release/aerosolization of virus particles. This may lead to the severe event that the highly pathogenic avian influenza virus in the future may be transmitted via the airborne route between mammals - an event we all know from the severe corona pandemic.

Sources:

<https://www.nhm.ac.uk/discover/news/2023/march/bird-flu-kills-thousands-south-american-sea-lions-outbreak-continues.html>

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