Zoonotic risk of SARS-COV-2

At the time of writing, the number of cases of SARS-COV-2 in animals is increasing, but there remains no evidence that these infected animals are a zoonotic risk. Animals that have tested positive seem to have contracted the infection from people (reverse zoonosis), although it would be incredibly difficult to prove, on an individual case basis, whether a cat was infected by the owner or vice versa as many infections in animals are clinically silent.

Any animal has the potential to be a fomite, although accurate information about how long coronavirus is viable on the coat and what sort of infectious dose could be delivered from an animal from an infected household with virus on the coat is lacking. Currently reasonable advice seems to be hand washing which should almost completely negate the risk apart from aerosolising virus during stroking.

What is evident is that a number of animal groups are at risk: chiroptera (bats), felids, mustelids (such as ferrets and badgers), rodents and primates. Experimental evidence suggests that cats can be infected intranasally (all be it with a relatively large viral dose) and they will excrete for a short time and can infect other cats housed in close proximity. However, the duration of contact needed for productive transfer of infection from cat to cat has not been examined. Most infected cats showed mild or no clinical signs. One study suggested that subadult cats were more susceptible and showed more severe pathology. Based on these data, reasonable advice would suggest that animals should not be allowed to lick faces and that potentially pets from COVID-19 positive households should be kept confined as much as practicable.

Of the animals other than cats, mustelids seem particularly susceptible and there have been outbreaks on mink farms in the Netherlands with the mink showing respiratory signs. The mink were believed to be infected by an employee.

Currently species at risk of infection seem to be those whose angiotensin-converting enzyme (ACE)-2 receptor is similar to that of humans. The ACE-2 receptor mediates the entry of a number of coronaviruses into cells, allowing infection to be productive. ACE-2 receptors are widely expressed in the heart, vessels, gut, lung, kidney, testes and brain. ACE-2 is mostly bound to cell membranes and only small amounts are present in the circulation in a soluble form.

The ACE-2 receptor acts as a counterbalance to ACE. ACE cleaves angiotensin I to angiotensin II which acts primarily as a vasoconstrictor but also acts to promote collagen synthesis and transforming growth factor-β, tending to promote inflammation and thrombosis. ACE-2 hydrolyses angiotensin II into the vasodilator angiotensin. With viral infection ACE-2 receptor expression is downregulated, which may be critically important in individuals whose ACE-2 receptor levels are already low such as people with hypertension or diabetes as they are at risk of unopposed action of angiotensin II. From this we might suspect that animals with similar pathologies to people at risk may show more severe clinical signs. It also raises the question as to whether ACE inhibitors or receptor blockers may have value in therapy.

Undoubtedly there is much more to learn about SARS-COV-2 and from a veterinary perspective the role, if any, that animals have in the epidemiology of infection in people. For now, the best evidence suggests that zoonotic risks are very low but we need to remain vigilant as new evidence evolves. CA

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