Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Missing information in animal surveillance of MERS-CoV

Chantal Reusken and colleagues[1] have identified neutralising antibodies against Middle East respiratory syndrome coronavirus (MERS-CoV) in dromedary camels, suggesting the role of camels in harbouring the virus and infecting human beings. The study findings suggest that bats and camels jointly play an important part in the persistence and evolution of MERS-CoV, resulting in spillover to the human population.[1,2] Theoretical epidemiologists refer to such combinations of animal hosts as the reservoir community.[3]

To confirm that the two animal species constitute a reservoir community, two conditions should be objectively examined.[3] First, the combination of reservoir hosts should permit persistence of the virus in natural settings (eg, by frequently transmitting the virus to other hosts), which is the necessary condition. Second, in the absence of the reservoir community, transmission cannot be maintained (by definition), which is referred to as the sufficient condition.[3,4] If other animal hosts can also maintain transmission, the reservoir community will have to include these hosts. In view of these conditions, although the identification of antibodies in camels is remarkable, an explicit assessment of the epidemiological role of camels has yet to be made to elucidate the mechanism of emergence in human beings.

How can a complete view of reservoir dynamics be achieved? The series of transmission in a population could be traced with serological techniques. Rather than doing a cross-sectional survey (to take a snapshot of prevalence), the identification of the reservoir host requires understanding of the incidence (ie, rate of new infections) to measure transmissibility. Serial cross-sectional surveys or large-scale follow-up of cohorts (of susceptible camels, not of those already infected) would be required. Alternatively, implementation of a transmission experiment with uninfected camels could also be useful, because the transmissibility of infected camels can be objectively quantified. Even a simple one-to-one transmission experiment could inform about the presence of substantial transmissibility.[5]

Of course, identification of the route of transmission from camels to human beings would also be of utmost importance, and could directly lead to prevention. In addition to such preventive effort, clarification of reservoir dynamics should be given a high priority, because identification of the major route of animal-to-human transmission, without an understanding of the overall picture of persistence and evolution in different hosts, is difficult.

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DPP4-directed therapeutic strategies for MERS-CoV

Christian Drosten and colleagues[1] provided the first complete virological profile of a patient infected with Middle East respiratory syndrome coronavirus (MERS-CoV). Previous study findings have shown that dipeptidyl peptidase 4 (DPP4; also known as CD26) serves as the functional receptor for MERS-CoV.[2] In view of the importance of DPP4 in regulating immune responses,[3] inhibitors of DPP4 binding and activity could modulate the pathogenesis of viral infection and serve as potential therapeutics.

Targeting of the site between the binding domain on the virus surface and the receptor might provide pharmacological action to suppress MERS-CoV infection. Studies have provided new insights into DPP4 interaction with substrates and inhibitors, and numerous inhibitors with varying selectivity have been characterised in DPP4 binding and functional assays.[4] Additionally, on the basis of our work on the use of DPP4 inhibitors as a treatment for autoimmune disease, DPP4 inhibition could suppress the damaging aspects of the body's own antiviral immune response by modulating inflammation.[3] Reversible inhibitors of DPP4 enzymatic activity suppress T-cell proliferation and production of proinflammatory cytokines as well as interleukin 10.[3] As we have shown, DPP4 inhibitor-mediated suppression acts partly through the induction of transforming growth factor β 1 (TGFβ1) production by effector T cells. Consistent with this mechanism, we noted a significant increase of TGFβ1 concentrations in tissue and plasma of mice treated with DPP4 inhibitors.[3] TGFβ1 induction at the site of inflammation could be an additional therapeutic benefit of DPP4 inhibitor treatment,