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Zoonotic potential of emerging animal diseases

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Commentary: Zoonotic potential of emerging animal diseases

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Palmer and colleagues have proposed an algorithm for early qualitative risk assessment of the emerging zoonotic potential of animal diseases,[1] a vital problem since more than half of all new or emerging infectious diseases agents in humans are zoonotic in origin.[2] Human infections due to agents such as the coronavirus responsible for severe acute respiratory syndrome (SARS), avian influenza A viruses, and HIV pose enormous problems because they are (*a*) difficult to manage clinically, (*b*) prohibitively expensive to treat in resource-poor areas, (*c*) capable of rapid global spread, (*d*) virtually impossible to eliminate once stable transmission among humans has been established, or (*e*) capable of inducing fear and substantial economic losses. Therefore, prior knowledge and public health preparedness are essential for their prevention and control. The bottleneck for this control effort lies in discovering and characterising these agents. Once achieved, these should be followed by systematic analyses of the risk of the agents causing human diseases.

Using porcine hepatitis E virus, porcine circovirus, bovine norovirus, Borna disease virus, and *Clostridium difficile* as examples, Palmer and colleagues systematically analysed the available scientific and clinical data on the microbes and the microbe-host interactions, and gave recommendations on the level of confidence of their risk of zoonotic transmission.[1] Their work exposed the fact that current knowledge is often insufficient to exclude the possibility of human infections.

In many disease syndromes where the aetiological agents cannot be defined or when the syndrome is conventionally regarded as idiopathic, clinicians often fail to explore the history of animal exposure and do not order microbiological tests for zoonotic agents. Many of these tests are not routinely available in local hospital laboratories, and serological tests for animal diseases are generally not standardised for testing human samples.

When zoonotic transmission occurs, some mutations might have occurred which could impair the sensitivity of rapid tests such as nucleic acid amplification. Most scientists agree that the mechanisms of interspecies jumping between viruses are poorly understood. It takes only a single amino acid change to alter the receptor binding specificity of the haemagglutinin of influenza A H5N1 virus, allowing the virus to be transmitted from chicken to human rather than just chicken to chicken.[3] Similarly, the substitution of one amino acid in the surface spike protein of SARS coronavirus changes its specificity from civets to human.[4]

Thus, any preceding epidemiological, clinical, and microbiological analysis may not be able to foretell such events—with potentially catastrophic consequences. As a result, it would not be possible to make confident recommendations for public health decisions or risk communications to the public without continuous research effort and a comprehensive surveillance programme. Continuous serological and disease monitoring for workers with frequent animal exposure could be a sentinel system for this surveillance programme.

The search for new microbial agents in animals is equally important because the simian immunodeficiency virus and bat SARS coronavirus were discovered shortly after human infections with SARS coronavirus and HIV were noted.[5 6] The human coronavirus OC43 is believed to have been acquired after an interspecies jump from bovine coronavirus in the 19th century.[7] It is theoretically possible that such precursor or related viruses could be discovered once a family of virus is known to exist by a comprehensive virological search in animals. This should, of course, be followed by regular monitoring of its evolution and spread in animals. Coupled with the surveillance in occupationally exposed people, it might give us a better idea of the zoonotic risk of these agents.

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