Journal of Bacteriology & Parasitology

OPEN ACCESS Freely available online

Commentary

A Commentary on the Corona Virus before the Next Pandemic

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ABSTRACT

Making it to the next host goes to the heart of virus fitness. There are in this regard two broadly opposing virus life history strategies. Acute viruses tend to be replicative and swiftly transmit to the next host. Instead, a persistent virus may be shed in minor quantities, if at all, keeping virus replication costs and host damage to a minimum.

Keywords: Virus; Corona virus; SARS-CoV-2

DESCRIPTION

There is a third dimension. A study describing the transmission ecology of the world main livestock viruses, 36 in total, revealed that the acute-persistent and the antagonism-mutualism gradients track with an outer- to inner-body line-up of organ systems [1]. Respiratory viruses are swiftly transmitted to the next host, followed by enteric viruses. Skin viruses may be acute or persistent. Virus establishing in the distal urogenital tract mucosa are also latently present in the sacral ganglia. More infiltrative viruses tend to lifelong establish in the reproductive organs system, the immune and circulatory systems, or just the blood circulation.

From the body outside to the inside, horizontal transmission modes give way to vertical modes and transmission involving hematophagous arthropods. The nature of the virus-host relationship shifts towards ever more intimacy. Vertical transmission diminishes the divide between virus and host from one host generation to the next, progressively so. Virus establishment in the immune system may yield immune-tolerance and sustained virus circulation in the bloodstream. From outer to inner-body the main virus groups concern, respectively, myxo-viruses, pox viruses, herpes viruses, and retroviruses. Coronaviruses establish in the enteric tract, respiratory tract, or, also, in the reproductive organs.

There are at least two additional dimensions to be considered in conjunction with the above. First, the host domain, by plotting natural against anthropogenic host environments [2]. Second, the evolutionary timescale, by contrasting long- and short-term ecological dynamics. For hundreds of millions of years viruses have co-evolved with arthropods, fishes, amphibians, reptiles, birds, and mammals [3]. Given the largely a-clinical infection-transmission dynamics today present in wildlife, the least damaging virus transmission mechanisms were evidently selected for [2].

Evolution of pathogenic fitness probably gained in importance starting from the Neolithic [4]. The domestication of plants and animals and the subsequent expanding human settlements presumably paved the way for the evolution of crowd agents, bacterial and viral [5]. This development must have been very gradual, in pace with the human and livestock demographics [5].

For example, phylogenetic evidence indicates that the measles virus evolved from the rinderpest virus between the 11th and 12th centuries [6]. In the process, the virus shifted from the enteric bovine to the human respiratory tract. The evolution of pandemic and panzootic disease agents took more time still. During the 14th century Black Death, caused by Yersinia pestis, ravaged civilizations across the Old World [7]. During the 18th century rinderpest swept through Europe and, late 19th century, across the African continent [8]. The emergence of the Peste des petits ruminantsVirus (PPRV), from the rinderpest virus, dates back to the 1910s [9]. Similarly, a suit of novel, aggressive respiratory and enteric viruses newly emerged over the past 150 years in poultry and pig production [2].

Today, the new emergence of human and livestock viruses appears to result mainly from two distinct sets of drivers. First, animal mass rearing may attract or select for opportunistic, hyper virulent respiratory or enteric viruses [1,2]. Second, ecological perturbation, in particular in the form of human ecosystem encroachment, may explain why deep-rooted viruses, including HIV and Ebola viruses, switched host [2]. The emergence of SARS-CoV-2 could relate both to drivers in the food and agriculture realms and ecosystem fragility.

Importantly, the above narrative on animal virus ecology and evolution does not imply that pathogenic virus fitness solely evolves as a result of human action. Virus host radiation has been in place for long. Species jumps, shifts in the virus organ system tropism, and in viral virulence, frequently operate in concert, as a result of a

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Received: February 11, 2021; **Accepted:** February 25, 2021; **Published:** March 04, 2021

Citation: Slingenbergh J (2021) A Commentary on the Corona Virus before the Next Pandemic. J Bacteriol Parasitol. 12: 390.

combination of natural and human factors. Still, the weight of the anthropogenic elements appears on the increase.

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