

Analogies between Porcine Respiratory Coronavirus 1990's Outbreak and COVID-19 Pandemic

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Abstract

This commentary article focuses on biological parallels between Porcine Respiratory Coronavirus and Severe Acute Respiratory Syndrome Coronavirus-2 pandemics. Emphasis is on spread, antibody (humoral) immunity and need for development of highly accurate, virus-specific antibody assay capable to differentiate between antibodies induced by newly emerged COVID-19 coronavirus and those induced by other, closely related coronaviruses.

Keywords: Coronavirus COVID-19; SARS-CoV-2; Antibody; Spike; Glycoprotein; Protective immunity

Abbreviations: COVID-19: 'CO' stands for corona, 'VI' for virus, and 'D' for disease (Formerly, this disease was referred to as '2019 novel coronavirus' or '2019-nCoV. '); SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2 of the genus Betacoronavirus that is the causative agent of COVID-19; SARS: Severe Acute Respiratory Syndrome; MERS: Middle East Respiratory Syndrome; PRCV: Porcine Respiratory Coronavirus; TGEV: Transmissible Gastroenteritis Virus; S protein: Spike protein; RNA: Ribonucleic acid; WHO: World Health Organization

Commentary

As the entire world is experiencing high level of anxiety linked to Coronavirus Disease 2019 (COVID-19), i.e., Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) pandemic, it brings me back to Wooster, Ohio, where I worked at the Ohio Agricultural Research and Development Station during 1995-1999 as the graduate student earning PhD, performing laboratory and animal experiments with two coronaviruses of veterinary importance: Porcine Respiratory Coronavirus (PRCV) and Transmissible Gastroenteritis Virus (TGEV). As the names imply, first virus has the respiratory tract tropism while second one causes gastrointestinal disease. Both PRCV and TGEV are well recognized pathogens of farm pigs but other mammalian species can occasionally also get infected - mildly or asymptotically. Interesting and human health translatable fact is that TGEV and PRCV share evolutionary ancestor, similar like SARS and SARS-CoV-2 do [1-3]. In fact, PRCV is the natural deletion mutant (with affected Spike or S glycoprotein epitope D) of TGEV which means that PRCV appeared sometime during late 1980s as the novel virus with entirely changed tissue tropism from its ancestral virus TGEV (gut to lungs), and pathogenicity (deadly to asymptomatic-mild). After its spontaneous emergence, PRCV spread worldwide within several years in all countries with intensive swine industry while inducing the herd immunity, i.e., antibody-driven protection against its much deadlier relative TGEV [4].

Back during my years in Wooster, we used to joke with fellow graduate students that nobody cares about coronaviruses because

they are not deadly enough and only cause mild respiratory or gastrointestinal illnesses. This view was applicable also for coronaviruses of human health importance [5]. However, because of unique nature of coronavirus evolution (high rate of coronavirus ribonucleic acid (vRNA)-driven mutations), there always was a possibility that new coronavirus strains could evolve not only towards less deadly but also more pathogenic forms. Few months ago, such scenario came into fruition in conditions of central China. It is not coincidental that SARS-CoV-2 emerged in a place where evolution of RNA viruses occurs much faster than in rest of the world. It is predominantly due to high density populations of humans and animals living in close proximities and prone for emergence and transmission of novel viruses [6].

Such state took place by the end of 2019 in Wuhan with appearance of SARS-CoV-2 but also in 2002/2003 in Hong-Kong with emergence of COVID-19's close relative SARS [7]. It is important to remember that viruses spread within susceptible populations not necessarily to wipe them out but to gain advantage over new evolutionary niches and to spread further. On the positive note, viral outbreaks are inevitably followed by herd immunity and eventual decrease in number of new cases. In case of coronaviruses, herd immunity is dependent on "virus-neutralizing" antibodies recognizing the virus spike (S) surface glycoprotein epitopes. It is of interest to emphasize that many of the currently available COVID-19 commercial antibody assays are based on antibodies (polyclonal or monoclonal) that are not capable to measure the host immune responses induced exclusively by COVID-19. This is because

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polyclonal and many monoclonal antibodies recognize epitopes that are shared among closely related coronaviruses. In case of human respiratory coronaviruses these are represented by SARS-CoV-2, SARS, Middle Eastern Respiratory Syndrome (MERS), common cold coronavirus, and possibly others. Therefore, the new and more specific SARS-CoV-2 exclusively reactive antibodies need to be developing for production of highly accurate commercial diagnostic serological assays. Only in this way, it will be possible to determine levels of convalescent antibodies correlating specifically with COVID-19 immune protection.

Conclusion

In conditions of high-density populations and more than one susceptible species, emergence of another unique virus is always only a question of time. It is likely that the new COVID-XY virus will be more pathogenic than COVID-19. That is where the World Health Organization (WHO) plays its unique role. WHO should have its resources ready and available for continuous epidemiological monitoring of Wuhan-like hot spots. As demonstrated by annual vaccinations against influenza, newly emerging viruses can successfully be abated although not eliminated. Notwithstanding, COVID-19 vaccine and/or treatment are still months away. Therefore, it is likely that naturally rather than passively acquired (vaccine) protection will start to slow down the current pandemic. In countries like United States or Sweden however, where implementation of social distancing policies varies widely among different states and socio-economic groups, we will see not one but likely multiple Pandemic peaks. Considering this, we can only hope that the race for discovery, evaluation, production and distribution of efficacious

vaccine will be faster than the COVID-19's capability to evolve into more virulent and/or pathogenic form.

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