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EDITORIAL

Perspectives Concerning SARS-CoV-2 Transmission for the Application of the Livestock Breeding

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Viral transmission between animals and humans has been defined as Zoonosis and zooanthroponosis. The vaccine has been claimed to be the best tool to prevent viral epidemics. However, as learned from SARS-CoV-2, vaccines cannot be the true answer to prevent viral infection for everyone. Some vaccinated persons are still reported to get infected. Viral mutation has been principally postulated to explain immune evasion. Questionable, why the mutated viral strain does not evade the immunity of everyone who has been vaccinated? Mutated viral strains cause various symptoms, non-symptomatic to morbidity and mortality, in different individuals with more or less the same ratio as the original SARS-CoV-2. Approximately, 25-35% of the SARS-CoV-2 detected individuals are asymptomatic, while 15-20% developed severity and about 2-5% have critical symptoms [1,2]. Logically, the viral mutation could keep mutating in any part of its genome. The new variant might maintain infectivity in the same person and might develop to infect another person who once has not been susceptible to the original strain. Thus, the dynamics of viral infection could change from time to time. This requires a better explanation to lead us in the right direction to prevent the emergent virus either now or in the future.

A virus is an obligated intracellular agent therefore it requires entering the susceptible host cell for replication. This is unlike most bacteria which are extracellular organisms and can proliferate regardless of entering into any cell of the infected host. Actually, the virus needs susceptible cellular molecule(s) to play a role as its receptor and co-receptor for attachment and penetration. Each virus uses its receptor-binding domain (RBD)

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to bind a different cellular molecule for this process. For example, SARS-CoV-2 has been known to use angiotensin-converting enzyme 2 (ACE-2) as its receptor and transmembrane protease serine2 (TMPRSS2), and furin as its co-receptors. Many studies found that the cellular molecules of individuals are polymorphism including ACE-2, TMPRSS2, and furin. The association of these cellular molecule variants and the severity of SARS-CoV-2 infection has been suggested in many studies [3-5]. In other words, individuals must have a susceptible variant for viral attachment and penetration. Individuals who were reported to be positive with the viral genome do not mean that they are truly infected. They could be just exposed (invaded), not infected. SARS-CoV-2 has the ability to persist outside the cell for a week in a suitable condition which becomes its advantage for finding a susceptible host for easier transmission than other viruses.

On the other hand, like any foreign substance, a viral agent that is exposed to individuals could induce adaptive immunity to prevent and eradicate the virus. The major immune cells are helper T (Th) cell, cytotoxic T (Tc) cell, and B cell which plays a role to synthesize antibodies. The adaptive immunity could respond to the invading viruses regardless of whether the viruses cause infection or not. To make a story short, the induction of the compatible Th and Tc is dependent on the existence of the major histocompatibility complex (MHC) alleles of the individuals [6,7]. To produce effective adaptive immunity, individuals require compatible MHC alleles to induce the associated Th and Tc cell clones. Subsequently, Th cell clones play the essential role to promote the activated Tc and B cell to be the memory Tc and B cell for effective and long-lasting immunity. Individuals who have incompatible MHC alleles cannot generate the susceptible Th and/or Tc cell clones. Tc is a key to eliminating the viral infected cell while antibody plays a role to neutralize the viral agent for preventing its entry into a target cell. To produce the memory B cell clones that are absolutely specific to the virus's RBD, the compatible Th must be activated. The memory B cell clones can be differentiated to be an antibody-secreting B cell to produce IgG and IgA which are the effective isotype of antibody to prevent the future invading virus [8,9]. Without compatible Th, the individuals can produce only IgM which usually is less effective and disappear sooner. This later group of individuals cannot produce immunity to prevent themselves from the future invading virus [8,9]. This explains why some individuals cannot prevent viral infection although they have been vaccinated. In addition, the vaccinated individuals who have been detected to be reactive to the SARS-CoV-2 might not be truly infected but invaded. It should find a suitable clinical practice to differentiate and handle the situation.

Many viruses, including SARS-CoV-2, do not cause any pathogenesis directly to the host. It is the host immune cells that timeously produce various kinds of cytokines for responding to foreign substances that invade a body regardless of whether they are pathogens or not. The side-effect of pro-inflammatory cytokines subsequently becomes the cause of illness to the host. In the virally infected hosts, the generation of the viral progeny induces unlimited amounts of pro-inflammatory cytokines, so-called cytokine storms, to cause severe pathogenesis [10,11]. In the viral invaded host, on the other hand, the pro-inflammatory cytokines can also be produced but in a limited amount because of no production of the viral progeny but can still cause possible mild symptoms.

Accordingly, as shown in Table 1, it could be proposed

Table 1. Classification of the population based on the existences of susceptible variant(s) of receptor/co-receptor of the particular virus and the compatible MHC alleles

Population group	Susceptible variant(s) of Receptor/co-receptor	Compatible MHCs alleles	Appearances
1	+	+	cause infection and likely to show severe symptoms, however adaptive immunity should be able clear the viral agent within 1-2 weeks
2	+	-	cause infection without any compatible and effective immunity. High potential to cause mortality. Note: to avoid false positive effectivity of the candidate drug, the study should be processed with only this group, not others.
3	-	+	No infection (no viral replication), could be either non or mild symptomatic can produce the entire adaptive immunity against the invaded virus Note: these individuals could be a good source of passive immunity for group 2.
4	-	-	No infection (no viral replication), could be either non or mild symptomatic can produce IgM antiviral antibody, but no memory B and T cells

to classify individuals into four groups based on their genetics of the cellular variants for the susceptibility to the viral infection and the compatible MHC alleles to produce the viral immunity.

Since mammal livestock have similar properties of cellular functions and immune systems as humans, based on this perspective, selective breeding cannot be applied in humans but could be done with mammal livestock. Mammal livestock breeders should have the characteristics as in the population group 3 and their MHC alleles should be heterozygous to generate more compatible MHC alleles for interaction to more varieties of the antigenic epitopes of the emergent viruses. This can bring sustainability to the mammal livestock industry worldwide. In addition, it might be a good way to prevent viral zoonosis. It should be noted that the characteristics of the breeders in group 4 should be avoided since they could be the source of the viral reservoir. This issue could be discussed at another opportunity.

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