SARS-CoV-2 Transmission in Livestock Industry and Agro-environment

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Definition

The outbreak of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a public health emergency that turns the year 2020–2021 into annus horribilis for millions of people across international boundaries. The interspecies transmission of this zoonotic virus and mutated variants are aided by exposure dynamics of infected aerosols, fomites and intermediate reservoirs. The spike in the first, second and third waves of coronavirus confirms that herd immunity is not yet reached and everyone including livestock is still vulnerable to the infection. Of serious concern are the communitarian nature of agrarians in the livestock sector, aerogenous spread of the virus and attendant cytocidal effect in permissive cells following activation of pathogen recognition receptors, replication cycles, virulent mutations, seasonal spike in infection rates, flurry of reinfections and excess mortalities that can affect animal welfare and food security. As the capacity to either resist or be susceptible to infection is influenced by numerous factors, identifying coronavirus-associated variants and correlating exposure dynamics with viral aerosols, spirometry indices, comorbidities, susceptible blood types, cellular miRNA binding sites and multisystem inflammatory syndrome remains a challenge where the lethal zoonotic infections are prevalent in the livestock industry, being the hub of dairy, fur, meat and egg production. This entry provides insights into the complexity of the disease burden and recommends precision smart-farming models for upscaling biosecurity measures and adoption of digitalised technologies (robotic drones) powered by multiparametric sensors and radio modem systems for real-time tracking of infectious strains in the agro-environment and managing the transition into the new-normal realities in the livestock industry.

1. Comorbidity of Coronavirus Infections across Species

Viruses are obligate intracellular parasites having ribonucleic acid (RNA) or deoxyribonucleic acid (DNA) with single or double strands enclosed within a protective protein shell (capsid). The capsid has oligomeric structural subunits constructed with helical, icosahedral or complex symmetry. It shields the genome from lethal agents. Viruses are acellular, lacking enzyme machinery for metabolism and ribosome for protein synthesis, but carry their own genome during transmission [1]. They depend on host cells for replication, genome transcription and translation of mRNA transcripts into proteins. Viruses are enveloped, filamentous, isometric (icosahedral) or head–tail shaped. RNA viruses are less stable than DNA viruses [2]. Both have divergent genome sizes of diameter 20–400 nm. Using genomic sense or polarity system, RNA viruses belong to two groups: positive-strand and negative-strand viruses [1]. Most ssRNA viruses have ~6–14 positive charges on sequence adjacent to the interior of the capsid at the amino or C-terminal ends. CoVs are enveloped RNA viruses and have been a major public health emergency for decades. Approximately 70% of viruses are ribonucleic acid (RNA) viruses and are responsible for almost 44% of emerging infectious diseases under various ecological zones worldwide. Incidence of lethality has remained high since 1931 when the first human coronavirus (HCoV-229E) was discovered, and enteric CoV was isolated from turkeys in 1951 [1]. The novel coronavirus (SARS-CoV-2) exhibits bulbar projections produced from the spike of glycoprotein (peplomers) on the capsid. Its zoonotic nature implies that both humans and animals are vulnerable to infection. The susceptibility of nonhuman species has been confirmed by sequencing oral and nasopharyngeal swab specimens from Syrian hamsters, bats, cats, dogs, ferrets, lions, mink and a few companion animals. Analysis of samples from the upper respiratory tract of ferrets equally confirms the prevalence of SARS-CoV-2 infection, but weak replication of the same strain was found in chickens, dogs, ducks and pigs. However, the high nucleotide similarity index (96.2%)

of SARS-CoV-2 with coronavirus RaTG13 shows that horseshoe bats (Rhinolophus spp.) harbour the same viral strain [1][3][4].

Aside from COVID-19, Ebola, severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) are common RNA viral diseases that confer burdens of illnesses across species [5]. Complications emanating from such infections are heterogeneous across gender and age groups but correlate with morbidity indices. Comorbidity is the coexistence of one or more diseases simultaneously with a primary medical condition in a patient. Most patients that died of COVID-19 had comorbid complications that worsened their chances of survival. While elderly people with underlying conditions are prone to higher risks, children infected with the virus manifest mild or no symptoms. The majority of patients in intensive care units or under invasive mechanical ventilation exhibiting a high prevalence of comorbidities (46–86.2%) due to extreme obesity (BMI ≥ 30), chronic renal failure, asthma, hypertension, type 2 diabetes, ischemic heart disease and chronic obstructive pulmonary disease did not survive [6]. General feeling of malaise, fever, dry cough, anorexia, pneumonia, abnormal heartbeats, shortness of breath, lymphopenia and high viral loads linked with cytokine activation and histopathological changes are concomitantly detected during clinical tests [7]. Loss of smell or taste and acute confusional state (delirium) coupled with disorientation, psychomotor changes and clouding of consciousness are newly detected COVID-19 symptoms. Recent findings affirm that patients suffering from severe SARS-CoV-2 infection have elevated levels of C-reactive protein, ferritin and D-dimer; high neutrophil-to-lymphocyte ratio; and increased levels of inflammatory cytokines and chemokines [8]. The overlapping effects of these conditions impact comorbidity–polypharmacy score, cumulative illness rating and mortality. Many people and livestock animals are still vulnerable to coronavirus infection. To ensure animal welfare and efficient biosecurity measures in the agro-environment, it is necessary to use novel biosensors for viral detection, tracking immune response and identifying high-risk zones to minimise exposure to viral aerosol and microdroplets from humans, livestock, fomites or passive vectors.

2. Viral and Host Receptor Interaction

Cellular receptors of human coronaviruses (HCoVs) belong to the membranous ectopeptidase family [9][10]. Receptors are macromolecules (glycoproteins) mediating biological processes to bind signaling molecules (ligands) intracellularly or extracellularly for initiating cellular responses. Intracellular receptors exist within the cytoplasm and bind to hydrophobic ligands roving across the plasma membrane. Cell-surface receptors (transmembrane receptors) are membrane-anchored proteins that bind to ligands on the surface of the cell to perform signal transduction functions [9][11]. Receptors have a transmembrane domain and an extracellular domain with a ligand binding (allosteric binding) site. The binding of receptors to ligands occurs at agonistic (ligand-binding) and antagonistic (ligand-blocking) sites. These include ligand-gated channels, G-protein-coupled receptors and enzyme-associated receptors. An enzyme-linked receptor is an integral membrane protein performing catalytic and receptor functions. Receptors undergo conformational changes to elicit responses by actively binding to or dissociating from ligands in cellular signaling or gated ion channels. Ligand-gated ion channels or ionotropic receptors are transmembrane ion-channel proteins that open to allow passage of Na^+ K^+, Ca^{2+} and/or Cl^- ions through the membrane in response to the binding neurotransmitter. G-protein-coupled receptors such as 7T-transmembrane domain receptors, heptahelical receptors, serpentine receptors and G-protein-linked receptors detect molecules extracellularly and activate internal signal transduction pathways and cellular responses [12].

Viruses use multiple molecular species as receptors for infecting target cells. These receptors consist of sialic acid ($C_{11}H_{19}NO_9$), glycosaminoglycans (negatively charged polysaccharides) and cellular adhesion molecules (intercellular adhesion molecules, vascular cell adhesion molecule-1), acetylcholine receptors, chimeric antigen receptors, interleukin-2 (IL-2) receptors, complementary receptors, growth factor receptors and neurotransmitter receptors [13][14][15]. Genome encoding occurs after viral invasion to elicit gene expression, accessory proteins and adaptation to the host. SL-CoV and SARS-CoV share similar genomic sequences and highly conserved gene products in the receptor-binding domain of the N-terminal region of S-proteins. A virus is unlikely to establish and replicate itself in the host without evasion or

antagonism. As the mechanisms of attachment, intracellular trafficking, uptake and incursion into cytosol are affected by the bond between virus and receptor, the synergy between them even heightens the probability of infection occurring and activity of spike protein in the process. Whilst host cells exert defense mechanisms for rapid detection of infection or suppression of immune response, viruses have ways of evading them through pattern recognition receptors (PRRs). Upon infection, PRRs sense nucleic acids in the cytoplasm or nucleus to activate antiviral innate immunity. RNA viruses use non-primer-dependent mechanisms for replication, distortion of RNA recognition and antiviral signaling. SARS-CoV-2 exploits the angiotensin-converting enzyme-2 (ACE-2) and cellular protease TMPRSS2 receptors to penetrate the host through the ACE-2 pathway [14]. ACE-2 is a zinc metalloenzyme and transmembrane receptor expressed in the glandular cells of gastric, duodenal and rectal epithelium; pneumocytes of the lungs; enterocytes of the small intestine, heart, kidney, liver and testis; and vascular endothelium cells. ACE-2 serves as an entry point into cells for HCoV-NL63, SARS-CoV and SARS-CoV-2. It coincidentally acts as a receptor for severe acute respiratory syndrome. The density of ACE-2 in each tissue correlates with severity of disease [13][15].

2.1. Viral Tropism and Immunogenicity of Coronaviruses

By definition, viral tropism is the capacity of viral strains or isolates to cause infection in cells or tissues and induce syncytial formation as acute or chronic infections [16]. Acting as a macrophage (M), T cell or dual tropic, tropism is a major factor in pathogenesis and persistence of infection in the peripheral motor nerves and central nervous system. Viral tropism develops in stages in a receptor-dependent or receptor-independent manner. All the developmental stages culminate with the synthesis of progenies [14]. Replication of RNA viruses within the host cell depends on enzymes in the virion to synthesise its mRNA. Viral RNA also serves as its own mRNA. Most RNA viruses complete their replication in the cytoplasm, while others are transcribed in the nucleus. Several infectious pathogens go through causative and reactive pathways to reach their targets. As viral titres are highest at the early stage of infection, the viral kinetics varies between symptomatic, asymptomatic and paucisymptomatic among patients [17]; a virion with glycoprotein (GPX) enters the host and then targets the cell with GPX receptors and fuses with it to initiate reverse transcription and synthesis of viral proteins plus ribonucleic acid followed by an assemblage of viral particles to advance its tropism. Pathogenesis of coronaviruses conforms to either intestinal or respiratory infection model. Almost 80% of COVID-19 symptomatic patients develop mild conditions; 15% develop severe hypoxaemia, dyspnoea and tachypnoea conditions; and 5% become critically ill with septic shock and/or multiorgan dysfunction [18].

Another pertinent issue is the immunogenicity of coronaviruses. Coronaviruses encode accessory proteins likely involved in immune antagonism or pathogenesis. Immunogenicity is the capacity of foreign substances (antigens) to induce humoral or cell-mediated immune response in the host. Innate immune response acts as the first line of defence against pathogenic agents by detecting pathogen-associated molecular patterns [19]. Respiratory viruses thrive in a condition where the immune system is either depressed or underdeveloped. There is a wide variation in terms of immune response elicited by different viral strains. Whilst every cell has an intrinsic capacity to restrict viral infection, most respiratory viruses can suppress innate immune responses to efficiently replicate and induce it [20]. When the immune system is infected by pathogens, it concentrates in the epitopes, which allow host cells to differentiate between closely related foreign invaders. The early stage of innate response is critical in shaping the downregulation of the adaptive immune response. While some victims develop lifetime immunity against certain viral infections, others are extremely susceptible. For instance, the likelihood of reinfection of a person who has previously suffered from chicken pox is low, but reinfection with haemagglutinin-neuraminidase (H1N1) flu strain might occur after 10 years. Again, infants and adolescents who have recovered from mild COVID-19 infection soon become critically ill by reason of exaggerated immune response, known as multisystem inflammatory syndrome in children (MIS-C) [19][21][22].

As shown in **Table 1** and **Table 2**, some nonhuman outbreaks such as bovine coronavirus (BCoV) from cattle, infectious bronchitis virus (IBV) from chickens, porcine respiratory coronavirus (PRCoV) plus

porcine haemagglutinating encephalitis virus (PHEV) from pigs, feline coronavirus (FCoV) from cats, canine respiratory coronavirus (CRCoV) from carnivores and bat-SL-covzc45/bat-SL-covzxc21 from bats exhibit respiratory tropism with varying complications or outcomes [1][23]. The chain of infection across species suggests that foraging animals and nomadic farmers in an agrarian environment are vulnerable to COVID-19 infection. In response to viral infection, most cell types produce interferons for release into extracellular fluid in the host. Interferons (IFN) are naturally occurring glycoproteins from the helical cytokine family. IFNs have molecular weights of 16,776–22,093 dalton. Interferon is obtainable from leukocytes (IFN-α), fibroblasts (IFN-β) or lymphocytes (IFN-γ) of human cells. The IFN system is a major frontline defence against viral infection. IFNs are type I (IFNα and IFNβ) and type III (IFNλ) cytokines. They are secreted in response to viral infections and biological inductions. Early control of viral replication by type I interferons, complement proteins and innate immune mediators limits the spread of viruses in the early phase of infection. Expression of IFN genes occurs downstream of the double-stranded RNA-sensing by host RIG-I-like receptors for coronavirus infection.

Table 1. Characterisation of genome and natural reservoirs of coronaviruses among avian species.

Host Species	Phylogenetic Genera of Coronaviridae Family	Designation of Viral Isolate/Prototype	Implication of Infection
Domestic fowl (Gallus gallus domesticus) Family/Order: Phasianidae/Pangalliformes	Chicken coronavirus (γ-coronavirus)	Infectious bronchitis virus (IBV) strain; chicken-dominant coronavirus (CdCoV).	Replication of virions in the epithelial layers weaken the immune response, causes nutrient malabsorption, enterotropism and poor welfare.
Duck (Anas platyrhynchos) Family/Order: Anatidae/Anseriformes	Duck coronavirus (γ-coronavirus)	Infectious bronchitis virus (IBV), DdCoV/GD/2014	Fatal, rapidly spreading viral infection of young ducklings.
Domestic geese (Anser anser) Family/Order: Anatidae/ Anseriformes	Goose coronavirus (unclassified γ-coronavirus)	Goose coronavirus (GCoV); infectious bronchitis virus (IBV)	Precociously infected geese exhibit respirotropism, retarded growth, abnormal growth of feathers.
Pheasant (Phasianus colchicus) Family/Order: Phasianidae/ Galliformes	Pheasant coronavirus (γ-coronavirus)	Ph/UK/27/B287-4/99; Ph/UK/24/B114-4/99; Ph/UK/24/B307-12/98; Ph/UK/24/B88-4/99; γCoV/ph/China/I0623/17 (I0623/17), γCoV/ph/China/I0710/17 (I0710/17)	Distortion of respiratory tract and renal blot, nephritis, visceral gout, air sacculitis, conjunctivitis, sinusitis, splenomegaly, poor hatchability, excess mortality.
Domestic Pigeon (Columba livia domestica) Family/Order: Columbidae/ Columbiformes	Pigeon coronavirus	N/A	Ruffled feathers, dyspnoea and excessive mucus from the beak, high susceptability to secondary infections.
Guinea fowl (Numida meleagris) Family/Order: Numididae/ Galliformes	Guinea fowl coronavirus (GfCoV)	GfCoV/FR/2011; GfCoV/2014	Neonatal respiratory distress syndrome, enteritis, low feed intake, poor flock performance, excess mortality.
Turkey (Meleagris gallopavo) Family/Order:	Turkey coronavirus (TCoV) γ-coronavirus	N/A	Bluecomb (enteric) disease and diarrhoea, poult enteritis complex or intestinal disorders at starter phase, anorexia, emaciation, morbidity/mortality (5–100%), poor egg quality (shell deformation, albumen thinning).

Table 2. Characterisation of natural reservoirs of coronaviruses among selected mammalian species.

Host Species	Phylogenetic Genera of Coronaviridae	Designation of Viral Isolate/Prototype	Implication of Infection
Bats (Miniopterus spp.) Family/Order: Microchiroptera/Therapsid	Bat coronavirus (α-coronavirus)	Bat-CoV/China/A515/2005; Bat-CoV/P, Bat-CoV/133/2005, BM48-31/BGR/2008, HKU4, HKU5, Bat-CoV-273/2005, RsSHC014; Bat-CoV/HKU9–1/China/2007	Diminishing bat genetic conservation. Reducing annual crop pollination, seed dispersal and pest control.
Cattle Family/Order: Bovidae/ Artiodactyla	Bovine coronavirus (BCoV)	Isolate Alpaca, AH187, E-AH187, E-AH187-TC, E-AH65, E-AH65-TC, R-AH187, R-AH65, R-AH65-TC	Severe diarrhoea in neonate calves, winter dysentery in cattle, respiratory infections in calves. Silvopastoral grazing/ranching restriction, tacit weight loss, morbidity, emergency culling, low meat and milk yield.
Dromedary Camel	Camel coronavirus (α-coronavirus)	DcCoV UAE-HKU23; MERS-like CoV	Source of zoonotic Middle East respiratory syndrome (MERS-CoV) infecting unciliated bronchial epithelial cells, type II pneumocytes.
Feline Family/Order: Felidae/ Carnivora	Feline enteric coronavirus (FeCoV)	Feline infectious peritonitis (FIP), UU4-54; feline APN, feline infectious peritonitis (FIP), virulent	Asymptomatic carriers experience seroconversion among cats. Biotypes replicate in macrophages, causing severe and lethal disease.
Giraffe (Giraffa camelopardalis)	Giraffe coronavirus (GiCoV)	CoV (GiCoV-OH3) US/OH3/2003, US/OH3-TC/2006	Weight loss, malabsorption of nutrients and water due to diarrhoea, decline in tourism and economic outcomes for hospitality industry.
Human Family/Order: Hominidae/Primates	Human coronavirus (β-coronavirus)	Human CoV-OC43, HCoV-229E, HKU1, HCoV-NL63	Induces acute respiratory distress syndrome, cytokine storm and multiple complications in immunocompetent adults and infants.
Mink (Neovison vison or Mustela lutreola) Family/Order: Mustelidae/Carnivora	Mink coronavirus	WD1127, WD1133, MV1-Lu, NB3 SARS-CoV-2, NB7 SARS-CoV-2	Raises secondary viral host, respiratory disease, emergency culling, high mortality.
Murine Family/Order: Muridae/ Rodentia	Murine coronavirus	Murine hepatitis virus: MHV-1, MHV-3, MHV-JHM.IA, RA59/R13, RA59/SJHM, RJHM/A, SA59/RJHM	Receptor (CEACAM1) binds MHV S-protein to activate virus–cell membrane fusion A59 strain. It infects mice liver and brain, demyelinating disease peaking at about 1 month postinfection.
Pig	Porcine coronavirus (Δ-coronavirus, unsegmented)	Porcine transmissible gastroenteritis virus (TGEV), porcine respiratory coronavirus (PRCoV); porcine haemagglutinating encephalomyelitis coronavirus (PHEV)	Infects ciliated bronchial epithelial cells and type II pneumocytes causing swine acute diarrhoea syndrome (SADS-CoV) enteritis among piglets or neonates, mortality.
Rabbit (Oryctolagus cuniculus/ Family/Order: Lagomorpha	Rabbit coronavirus (β-coronavirus)	RbCoV-HKU14	Infects upper respiratory tract, sparing the lungs. Shortage of wool, meat, gourmet products.

By interacting with their specific heterodimeric receptors on the cell surface, interferons initiate an array of signals that induce cellular antiviral activities, modulate inflammatory responses, inhibit or stimulate cell growth and apoptosis and modulate other components of immune system [21]. Stimulation of interferon (IFN)-dependent antiviral response at the early stage of infection is vital for monitoring immune

response. Host sensing of viral double-stranded RNA triggers the activity of IFNs [24]. IFNs initiate innate immune responses by exerting direct antiviral effects on interferon-stimulated genes [25]. IFN invasive strategies involve avoidance, suppression of IFN induction and signaling. By avoidance, a virus masks itself or by-products from being recognised by the host sensors that activate the IFN system. Viruses can suppress IFN induction by inhibiting sensors in the host or downstream signaling molecules to prevent initiation. By suppressive means, gene products in the viral gene block signaling events or downregulation of type I IFN receptors to inhibit the activation of an antiviral state in the infected cell or enhancement of IFN response by activating late type I IFN genes [26].

Of five groups of immunoglobulins (IgG, IgA, IgM, IgD and IgE) with diverse amino acid sequences, antibodies exist in soluble and membrane-bound forms. Antibodies and antigens interact by spatial complementarity of lock-and-key mechanism. High specificity and affinity is the most striking feature of antigen–antibody interaction. Antibodies are secreted by β-cells in the adaptive immune system or plasma cells. Each antibody unit has a minimum of two antigen-binding sites bound bivalently or multivalently. Bonding of an antibody with an antigen forms an immune complex functioning as an entity. The immune system releases protein messengers for regulating the immune defence of host cells. The collective term for these messengers is cytokines. Cytokines are glycoproteins acting nonenzymatically on target cells of picomolar to nanomolar concentrations. The physiology of cytokines is complex and diverse. Cytokine secretion can stimulate the release of different cytokines to express the same functions [27]. It mediates the actions of interleukins, interferons and tumour necrosis and growth factors. The majority are secreted by more than one type of immune system, fibroblasts and endothelial cells. Cytokine interleukin-2 influences the function of virtually every cell in the immune system. Over-secretion of immune cells may trigger cytokine release syndrome or cytokine storm [28]. Under multiple respiratory viral infections, cytokine storm causes virus-induced tissue destruction, extreme inflammation and mortality. Several complications at the advanced stages of severe COVID-19 infection have been linked to cytokine release syndrome. Evidence has shown that the immune response of COVID-19 patients under critical condition may be as vicious as the virus responsible for the illness due to comorbidities and hyperinflammatory immune response [29].

Typically, the capacity of the body to either resist infection or be susceptible is influenced by a number of factors such as comorbidities, blood type, nutrient cycling and intactness of immune system coupled with a repertoire of polygenic makers influencing sociogenomics and behavioural stereotypes across species (**Figure 1**). Most of the elderly people that died of COVID-19 infection had underlying conditions and, presumably, shortened telomeres [30]. Rapid immune response occurring in the infected host during acute viral infection indicates the effect of telomere shortening on immune depression. Symptoms of telomere syndromes vary but depend on the patient’s telomere length. While some patients have few or no symptoms, others exhibit bone marrow failure; pulmonary fibrosis; liver cirrhosis; and gastrointestinal, skin and mucosal abnormalities. Again, it is not clear if the body of victims can synthesise inhibitory antibodies against viral epitopes and establish antigen-binding sites against SARS-CoV-2 molecules. As cases of reinfection with SARS-CoV-2 and its mutant variants are emerging, novel biosensors with robust sero-surveillance strategy can be exploited as a requisite standard for identifying group risks, estimating antibody levels and trends of population immunity and predicting response to outbreaks.

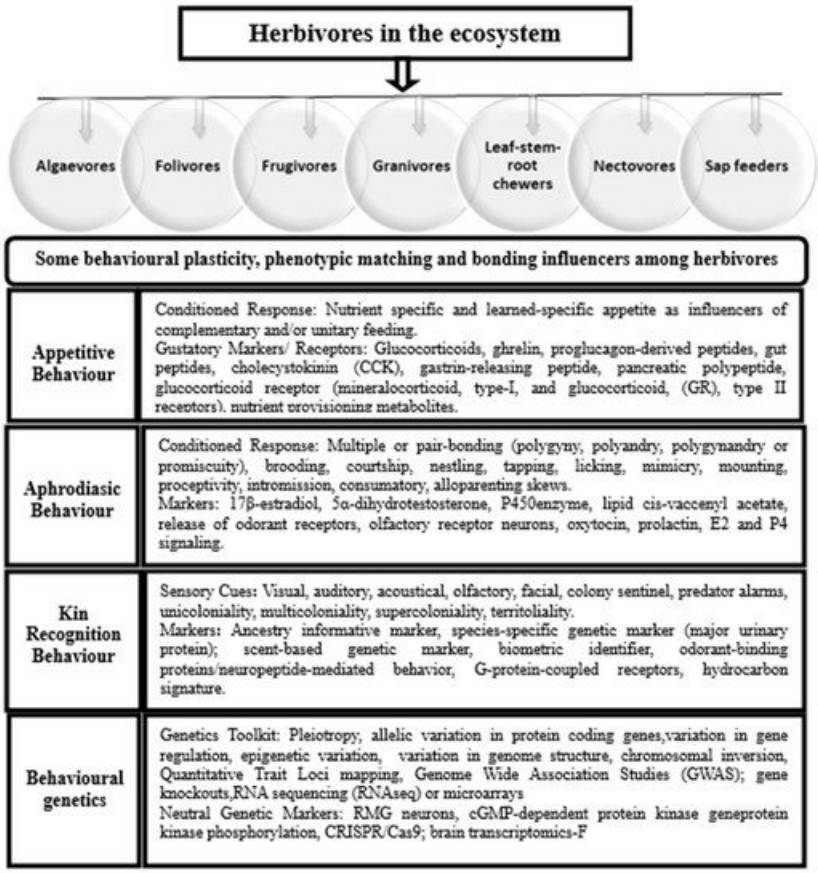

Figure 1. Repertoire of polygenic markers influencing sociogenomics and behavioural stereotypes among vertebrates on pasture land.

2.2. Evolution and Mutability of SARS-CoV-2

RNA viruses have short generation times and fast evolutionary rates. The fastest evolution corresponds to single-stranded RNA and reverse-transcribing viruses, followed by double-stranded RNA and single-stranded DNA viruses, whereas double-stranded DNA viruses evolve more slowly on average. Viral evolutions are mostly identified from open reading frames. A reading frame is a set of nonoverlapping triplets of three consecutive nucleotides. An open reading frame (ORF) is a section of the reading frame with no stop codon which is a sequence of three adjacent DNA or RNA nucleotides corresponding with amino acid during protein synthesis [31]. It should be noted that proteins cannot be synthesised if RNA transcription stops before reaching the stop codon. A gene encoding a protein has an ORF that can be translated into an amino acid sequence (AAS). To start with, the AAS or nucleotide sequence (NTS) must be obtained from the same DNA sequence. Since the AAS is the final product under evolutionary trend, multiple alignments of protein-coding genes use AAS during alignment with NTS. Coronaviruses contain specific genes in the ORF downstream regions that encode proteins for replication, nucleocapsid and spike formation. These viruses have overlapping ORFs (ORF1a, ORF1b) which are a continuous stretch of codons for gene prediction to identify regions of genomic DNA encoding protein, RNA genes and regulatory regions. The ORF7a gene creates an accessory protein for viral infection and replication in the host. If the domain of the ORF is known, the rates of mutation can be modified as wobble base pairing can permit higher mutation rates in the third nucleotide of a given codon without affecting the genetic code [32].

Genomes in RNA viruses have the highest mutation rates to circumvent the immune system of the hosts [33]. This is premised against the circulation of mutant spectra and replication by viral quasispecies. A minor alteration in NTS or AAS can annul the activity of a receptor due to errors during replication, deletion or insertion of DNA segments. The term mutation rate (MR) refers to the frequency of new variants created per site per genome replication or average number of errors produced in genomes of progeny per base per replication cycle (mut/nuc/rep) [32][33][34]. MR is affected by base composition,

environment, size of gene and position in the genome. RNA viruses use encoded RNA-dependent RNA polymerase for genome replication to exploit mutation and escape antibody and cytotoxic T lymphocyte responses. The mutation rate is a critical parameter for understanding viral evolution [35]. Mutations might be induced via random errors during replication or genetic shuffling (recombination) among an infected population with weaker immunity. Mutation influencing spike protein alters amino acid (614) by substitution from aspartic acid (D) to glycine (G) at a genome domain encoding the spike protein (D614G). MR of RNA viruses occurs at rates of six orders of magnitude above that of the hosts. Mutability boosts the adaptation of RNA viruses to varied environments. MR can be calculated as follows:

$$\mu = [(r_2/N_2) - (r_1/N_1)] \times \ln (N_2/N_1) = (f_1 - f_2) \times \ln (N_2/N_1)], \quad (1)$$

where r_1 = observed number of mutants at time point 1; r_2 = observed number of mutants at the next time point; and N_1 and N_2 = numbers of cells at time points 1 and 2, respectively.

3. Conclusions

The outbreak of SARS-CoV-2 adds to the scale of disease burden and challenges the livestock industry is facing vis-a-vis the menace of infectious diseases. Due to recurrent surges in viral waves across agro-ecological zones, it is uncertain what the devastating effects of these infections will be in the nearest future. Another worrisome issue for the livestock sector is the potential of a wide range of mammalian and avian species being natural reservoirs for different strains of infectious zoonotic diseases such as coronavirus with its highly transmissible variants. Knowing that herd immunity is not yet attained in many climes and that livelihood partly depends on food security from animal products, indiscriminate exposure of farmers and livestock during routine and periodic activities to environmental variables harbouring coronaviruses may pose a serious threat to public health and food production. Thus, neglecting the impacts of viral aerosols on agrarian communities will unavoidably have devastating effects on animal welfare and food security. Therefore, effective use of smart precision livestock models, biosensor-powered digital technology and Internet of Things (IoT) may enhance catch-all and efficient monoplex or multiplex platforms for rapid-response and high-throughput screening of livestock and the agro-environment to limit transmissibility of zoonotic infections along food chains. A combination of digitalised technologies can be introduced into communal farming systems and rebranded with a modernised sense of “cooperative smart livestock farming” to help the resource-poor farmers mitigate transitioning into the new-normal realities in the livestock industry for realising sustainable and high production efficiency of animal products.

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Keywords

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