

# Coronaviruses (Coronaviridae) Affecting Ruminants, Non-Ruminant Herbivores and Companion Animals Health: A Review

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## ABSTRACT

Coronaviruses have been extensively studied as causative agents for a range of respiratory, enteric and neurological diseases in animals, due to their significant potential impact on animal health and the economy. These viruses infect specific hosts, such as animals and humans, however, certain strains can infect both animals and humans. This article aims to provide detailed information on coronaviruses in ruminants, non-ruminants herbivores and companion animals, as well as to explore the impacts of these viruses on animal health and industries. Different types of published articles and books were reviewed to obtain the information. Extensive studies on various coronaviruses in different animal species, including ruminants, non-ruminant herbivores and companion animals have not only enhanced our understanding of coronavirus biology and ecology but also provided insights into the global distribution and origins of these viruses. Understanding the diverse range of coronaviruses found in both ruminant and non-ruminant herbivores, as well as in companion animals was highlighted in this review. Coronaviruses occurrence and variability are significantly underestimated. Therefore, there is a critical need to expand our knowledge of coronaviruses present in animals, to study their pathogenicity, transmission dynamics and disease prevalence and to effectively respond to the potential future emergence of new coronaviruses, particularly within economically important animal populations.

## KEYWORDS

Coronavirus, ruminants, non-ruminants, companion animals, crown-like spikes

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## INTRODUCTION

Coronaviruses derive their name from the distinctive appearance of "corona" or crown-like spikes on their outer surface. These viruses belong to the subfamily Coronavirinae within the family Coronaviridae and the order Nidovirales<sup>1,2</sup>. Among the four genera, coronaviruses of the *Alphacoronavirus* and *Betacoronavirus* genera affect ruminants, non-ruminants and companion animals<sup>3</sup>, such as bovine coronavirus (BCoV)<sup>4</sup>, rabbit enteric coronavirus (RECV)<sup>5</sup>, equine coronavirus (ECoV)<sup>6</sup>, feline coronavirus (FCoV)<sup>7</sup>, canine respiratory coronavirus (CRCoV)<sup>8</sup> and canine enteric coronavirus (CECoV)<sup>9</sup>.



Table 1. Types of coronaviruses that impact ruminants, non-ruminant herbivores and companion animal

Coronavirus species	Susceptible hosts	Targeted host cells	Tissues/organ
BCoV <sup>4</sup>	Cattle	Epithelial cells, enterocyte	Respiratory and enteric tracts
CECoV <sup>9</sup>	Dog	Enterocyte	Enteric tract
CRCoV <sup>8</sup>	Dog	Pneumocyte	Respiratory tract
ECoV <sup>6</sup>	Horse	Epithelial cells	Respiratory tract
FCoV <sup>7</sup>	Cat	Monocyte, macrophage, enterocyte	Respiratory and enteric tracts, and nervous system
RECV <sup>5</sup>	Rabbit	Enterocyte	Enteric tract

Source: Saif<sup>4</sup>, Descoteaux *et al.*<sup>5</sup>, Pusterla *et al.*<sup>6</sup>, Pedersen *et al.*<sup>7</sup>, Decaro and Buonavoglia<sup>8</sup>, Licitra *et al.*<sup>9</sup>

Coronavirus commonly causes respiratory and enteric diseases in various animals<sup>10</sup>. This review focuses on veterinary and economically important coronaviruses, emphasizing their origins, ecological distribution, genetic diversity, transmission and economic impact. Table 1 provides a list of coronaviruses affecting ruminants, non-ruminants and companion animals.

**Bovine coronavirus (BcoV):** The BCoV causes gastroenteritis, diarrhea and respiratory disease in young and newborn cattle, leading to enormous economic losses worldwide<sup>9</sup>. These financial losses are partly attributed to mortality rate, which can reach up to 69% in beef calves within 2 months of their arrival<sup>11</sup>. Infected calves often exhibit diarrhea, while adult cattle may experience winter dysentery and respiratory disease<sup>12</sup>. Calves can be infected with BCoV through both respiratory and oral routes<sup>13</sup>.

The virus is responsible for both respiratory and enteric diseases in cattle<sup>4</sup>. As a result, the virus can be detected in diarrheic fecal samples and respiratory droplets from calves and cattle, regardless of whether they have respiratory symptoms<sup>14</sup>. The BCoV replicates on the surface epithelial cells, particularly in the distal half of the villi, leading to cell death, sloughing off the dead cells and replacing by immature cells, which results in stunting and fusion of adjacent villi<sup>13</sup>. This deterioration in the intestinal surface area reduces the digestive and absorptive capacity of the intestine. Additionally, high mortality rates in BCoV-infected cattle are attributed to severe gastroenteritis manifested by bloody diarrhea in the feces, resulting from damage to the intestinal villi<sup>13</sup>. Moreover, BCoV infection can lead to severe or fatal disease in adult cattle when combined with other factors such as mucosal immunity deficiency, stress from transportation or co-infection with other secondary respiratory pathogens<sup>15</sup>.

The origin of BCoV remains uncertain. However, analysis of its genome has revealed a remarkably high nucleotide identity of 96% with human coronavirus OC43 (HCoV-OC43), suggesting a zoonotic transmission from bovines to humans<sup>16</sup>. There have been reports of BCoV being detected in a 6-year-old child with diarrhea, indicating a possible interspecies transmission to humans<sup>17</sup>.

Similar to other betacoronaviruses, BCoV displays high variability in both the structure and sequences of its genomes. Notably, a distinguishing genetic feature of BCoV is the presence of a hemagglutinin-esterase (HE) protein, which is not found on the surface of other betacoronaviruses<sup>18</sup>. The HE protein acts as the secondary viral attachment protein alongside the S protein<sup>15</sup>. Consequently, HE may contribute to the broad host range observed in betacoronavirus species<sup>15</sup>.

Vaccines targeting BCoV are available to protect against respiratory and enteric disease in cattle and young calves<sup>18</sup>. Successful disease control has been reported in numerous countries through the parenteral vaccination of pregnant cows. A protective approach for the calves involves vaccinating pregnant cows in their third trimester will trigger the production of crucial maternal immunity required for the early-stage immunity of the calves<sup>18</sup>.

**Rabbit enteric coronavirus (RECV):** Coronavirus has been reported in commercial rabbitries<sup>19</sup>. Two distinct pathological forms of coronavirus infection have been observed in rabbits, namely; i) enteric disease (local form) and ii) pleural effusion and cardiomyopathy (systemic form)<sup>19</sup>. The enteric disease

rising from RECV infection exhibits characteristic lesions and symptoms consistent with enteritis<sup>20</sup>, while pleural effusion and cardiomyopathy were reported in a laboratory experiment<sup>19</sup>. The RECV is transmitted through the fecal-oral route. The virus primarily attaches to and replicates in the small intestine<sup>21</sup>.

Young rabbits aged between 3 and 10 weeks are particularly susceptible to RECV infection, although the virus may also be present in clinically healthy adult rabbits<sup>22</sup>. Clinical manifestations of RECV infections in rabbits include watery diarrhea, abdominal distension, anorexia and sudden death, although asymptomatic cases can also occur<sup>23</sup>. Morbidity rates can range from 40 to 60%, with mortality rates may exceed 75% among affected rabbits<sup>24</sup>. Microscopic examination may reveal intestinal villous blunting, crypt hypertrophy, complete M-cell necrosis and necrosis of villous epithelial cells overlying the gut-associated lymphoid tissue (GALT) in the small intestine<sup>25</sup>.

According to recent research, various management strategies aimed at preventing RECV infections, such as delayed weaning and dietary modifications, have been considered<sup>19</sup>. Nevertheless, it has been found that the utilization of broad-spectrum antibiotic treatment and vaccination has not proven to be effective in managing and preventing the morbidity and mortality of rabbits<sup>26</sup>.

**Equine coronavirus (ECoV):** The ECoV is the only strain of coronavirus that is known to infect and cause disease in horses. The virus was first isolated in North Carolina, USA in 1999<sup>27</sup> and since then, sporadic ECoV infections have been reported in various parts of the world<sup>28</sup>. However, there is limited information available on the epidemiology and evolution of ECoV.

The most commonly observed clinical signs associated with ECoV infections include fever, anorexia and lethargy<sup>29</sup>. Only about 10% of gastrointestinal diseases and 3% of neurological disorders in infected horses are reported. Mortality has been reported in severe cases of ECoV infection<sup>30</sup>.

Transmission of ECoV occurs through the fecal-oral route, with horses becoming infected by ingesting contaminated feed and water<sup>31</sup>. Fecal samples are commonly used for diagnosing ECoV in both diarrheic and non-diarrheic horses<sup>32</sup>.

At present, there is no vaccine available for ECoV. However, several studies are exploring experimental vaccines against ECoV<sup>33</sup>. The most effective way to control and prevent the spread of ECoV is through strict biosecurity measures and isolating sick horses in quarantine. Therefore, the only treatment for infected horses is supportive care to manage the symptoms until recovery<sup>33</sup>.

**Feline coronavirus (FCoV):** The FCoVs primarily affect the intestinal tract of cats, but they can also lead to a fatal immune-mediated disease<sup>34</sup>. The FCoVs belong to the genus *Alphacoronavirus* and the species *Alphacoronavirus 1*, alongside TGEV, PEDV, CCoV, HCoV-229E and HCoV-NL63<sup>35</sup>. There are two biotypes of FCoV: Feline infectious peritonitis virus (FIPV) and feline enteric coronavirus (FECV). The FIPV causes feline infectious peritonitis (FIP), while FECV is responsible for feline enteric disease.

**Two serotypes of FCoV existed:** Serotype I and serotype II<sup>36</sup>. Serotype I accounts for 80 to 95% FCoVs infections, while serotype II represents about 25%<sup>37</sup>. The FIP is a progressive, immune-augmented and fatal disease<sup>38</sup>. The FIP is characterized by the presence of fibrinous and granulomatous serositis, which is an inflammation of the serous membranes, a protein-rich serous effusion in body cavities and the formation of granulomatous lesions<sup>39</sup>. This disease presents in two main forms: wet or effusive (exudative) and dry or non-effusive (proliferative)<sup>7</sup>. The FIPV primarily attaches and replicates in the epithelial cells of the pharynx or the jejunum of the intestinal tract<sup>39</sup>. Acute infections with FIPV lead to viremia and rapid virus spread in the thorax and abdomen, often resulting in mortality<sup>40</sup>.

Symptoms of FIPV infection in cats include anorexia, chronic fever, malaise and in some cases, neurological or ocular diseases<sup>41</sup>. In addition, FIP is a major cause of death among young domestic cats, with the highest incidence occurring in cats aged 3 months to 3 years<sup>41,42</sup>. However, FIP can affect cats of any age, particularly those with suboptimal immune function<sup>43</sup>.

The FECV is identified as a virulent biotype of FCoV and is considered the hypervirulent biotype precursor of FIPV<sup>44</sup>. Studies reported that approximately 5% of persistently infected cats with FECV may develop FIP<sup>45</sup>. This suggests that FIPV evolves from FECV through specific mutations in the viral genomes of individually infected cats<sup>45</sup>. Unlike FIPV, FECV typically leads to mild enteritis<sup>46</sup>, with many infections being subclinical. As a result, the virus can be detected in the feces a few days after infection and can persist for several months<sup>47</sup>.

Cats are usually infected with FCoV through oro-nasal exposure by coming into contact with the virus in feces or with contaminated fomites<sup>48</sup>. It is believed that the sharing of litter boxes among cats contributes to virus transmission<sup>49</sup>.

The treatment of FCoV infections primarily aims to reduce the inflammatory and hyperimmune response. Moreover, various antivirals and immunosuppressants are being investigated for their potential efficacy in managing FIP cases<sup>50</sup>. As a preventive measure, vaccines against FCoV are available and may provide some level of protection to cats that have not previously been exposed to FCoV<sup>50</sup>.

**Canine respiratory coronavirus (CRCoV):** The CRCoV belongs to the genus Betacoronavirus and shares genetic similarities with BCoV and HCoV<sup>51</sup>. The CRCoV can infect dogs of all ages and breeds<sup>52</sup>.

Dogs usually get infected with CRCoV through airborne exposure to the bio-aerosols of respiratory secretions from infected dogs<sup>52</sup>. In addition, studies reported the presence of the virus in air samples, water troughs, toys in pens and on surfaces such as dog kennels, food and water bowls<sup>53</sup>. The risk of CRCoV infection is heightened in environments where a large numbers of dogs are kept in close confinement, such as boarding kennels, shelter facilities and dog show kennels<sup>54</sup>.

The virus induces acute or subacute respiratory disease in dogs, potentially leading to severe pneumonia and fatality. Common pathologic findings of CRCoV infection in dogs include mild inflammatory changes in the upper respiratory tract, particularly in the nares and the trachea<sup>55</sup>. Infected dogs typically exhibit clinical signs such as cough, sneezing and nasal discharge. In addition, there is observed deterioration of the trachea surface cilia<sup>56</sup>, which commonly occurs in CRCoV infections in dogs<sup>57</sup>. This damage facilitates deeper penetration of the airways by co-infection with secondary respiratory pathogens, leading to more severe clinical cases<sup>58</sup>.

Unfortunately, there is no commercially available vaccine for preventing CRCoV or reducing the clinical disease<sup>52</sup>. Moreover, no specific treatment for CRCoV infections can be applied. The only treatment approach involves supportive therapy based on the clinical signs. Stringent biosecurity measures, such as isolating sick dogs in quarantine, changing clothes and practicing thorough hand washing after handling the dogs become an alternative to curbing the spread of CRCoV<sup>57</sup>.

**Canine enteric coronavirus (CECoV):** The CECoV belongs to the genus *Alphacoronavirus* within group 1 coronavirus species and is distinct from the group 2 CRCoV, sharing 69 and 21% identity in nucleotide and an amino acid sequences, respectively<sup>9</sup>. Notably, genetic similarities analysis associates CECoV with other group 1 coronaviruses such as FCoV and TGEV<sup>57</sup>. The CECoV typically induces mild, self-limiting diarrhea in dogs, particularly in young puppies<sup>59,60</sup>. Transmission of the virus can occur through the fecal-oral route<sup>60</sup>. A study reported a high prevalence of CECoV infections in dogs living in densely populated environments like shelters or kennels<sup>61</sup>.

The CECoV infection dogs often lead to high morbidity but low mortality<sup>54</sup>. Clinical manifestations of CECoV-infected dogs include vomiting, lethargy, loss of appetite and diarrhea<sup>61</sup>. Studies have indicated that the virus primarily attaches and replicates in the epithelial cells of the small intestine<sup>61</sup>. Gastroenteritis is a common clinical sign of CECoV infection, with severe cases potentially leading to mortality due to diarrhea<sup>59</sup>. In addition, the virus has been observed to occasionally infect the respiratory tract in natural conditions<sup>62</sup>.

Currently, there is no specific treatment for CECoV infections. However, implementing strict biosecurity measures and providing supportive nursing care to infected dogs are recommended approaches<sup>60</sup>.

## CONCLUSION

This review highlights the diverse range of coronaviruses found in both ruminant and non-ruminant herbivores, as well as in companion animals. However, their occurrence and variability are significantly underestimated. Therefore, there is a critical need to expand our knowledge of coronaviruses present in animals, to comprehensively study their pathogenicity, transmission dynamics and disease prevalence and to effectively respond to the potential future emergence of new coronaviruses, particularly within economically important animal populations.

## SIGNIFICANCE STATEMENT

This study on eco-biology, transmission and disease caused by the diverse range of coronaviruses in ruminants, non-ruminants and companion animals provides valuable insights into their impact on animal health and the economy. Understanding of these attributes is essential for preparing and managing effective diagnosis and control strategies. Moreover, this study will contribute to expanding the knowledge of coronaviruses in animals and facilitate proactive measures to address the potential future emergence of new coronaviruses, particularly among economically valuable animal populations.

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## REFERENCES

1. Woo, P.C.Y., R.J. de Groot, B. Haagmans, S.K.P. Lau and B.W. Neuman *et al.*, 2023. ICTV virus taxonomy profile: *Coronaviridae* 2023. *J. Gen. Virol.*, Vol. 104. 10.1099/jgv.0.001843.
2. Ismail, M.I., 2021. Coronaviruses of the veterinary and socio-economic importance: Classification, pathogenicity, transmission, and evolution of the coronaviruses. *J. Trop. Resour. Sustainable Sci.*, 9: 93-102.
3. Perlman, S., 2020. Another decade, another coronavirus. *N. Engl. J. Med.*, 382: 760-762.
4. Saif, L.J., 2010. Bovine respiratory Coronavirus. *Vet. Clin. North Am. Food Anim. Pract.*, 26: 349-364.
5. Descôteaux, J.P., G. Lussier, L. Berthiaume, R. Alain, C. Seguin and M. Trudel, 1985. An enteric coronavirus of the rabbit: Detection by immunoelectron microscopy and identification of structural polypeptides. *Arch. Virol.*, 84: 241-250.
6. Pusterla, N., R. Vin, C. Leutenegger, L.D. Mittel and T.J. Divers, 2016. Equine coronavirus: An emerging enteric virus of adult horses. *Equine Vet. Educ.*, 28: 216-223.
7. Pedersen, N.C., J.W. Black, J.F. Boyle, J.F. Evermann, A.J. McKeirnan and R.L. Ott, 1984. Pathogenic Differences Between Various Feline Coronavirus Isolates. In: *Molecular Biology and Pathogenesis of Coronaviruses*, Rottier, P.J.M., B.A.M. Zeijst, W.J.M. Spaan and M.C. Horzinek (Eds.), Springer, Boston, Massachusetts, ISBN: 978-1-4615-9373-7, pp: 365-380.
8. Decaro, N. and C. Buonavoglia, 2008. An update on canine coronaviruses: Viral evolution and pathobiology. *Vet. Microbiol.*, 132: 221-234.

9. Licitra, B.N., G.R. Whittaker, E.J. Dubovi and G.E. Duhamel, 2014. Genotypic characterization of canine coronaviruses associated with fatal canine neonatal enteritis in the United States. *J. Clin. Microbiol.*, 52: 4230-4238.
10. Ismail, M.I., 2023. A review of veterinary and economically devastating coronaviruses: Emphasising poultry and pigs. *BioSci. Rev.*, 5: 63-75.
11. Azizzadeh, M., H.F. Shooroki, A.S. Kamalabadi and M.A. Stevenson, 2012. Factors affecting calf mortality in Iranian Holstein dairy herds. *Preventive Vet. Med.*, 104: 335-340.
12. Gagea, M.I., K.G. Bateman, T. van Dreumel, B.J. McEwen and S. Carman *et al.*, 2006. Diseases and pathogens associated with mortality in Ontario beef feedlots. *J. Vet. Diagn. Invest.*, 18: 18-28.
13. Lotfollahzadeh, S., O. Madadgar, M.R. Mohebbi, M.R.M. Dezfouli and D.G. Watson, 2020. Bovine coronavirus in neonatal calf diarrhoea in Iran. *Vet. Med. Sci.*, 6: 686-694.
14. Clark, M.A., 1993. Bovine coronavirus. *Br. Vet. J.*, 149: 51-70.
15. Chae, J.B., J. Park, S.H. Jung, J.H. Kang, J.S. Chae and K.S. Choi, 2019. Acute phase response in bovine coronavirus positive post-weaned calves with diarrhea. *Acta Vet. Scand.*, Vol. 61. 10.1186/s13028-019-0471-3.
16. Franzo, G., M. Drigo, M. Legnardi, L. Grassi and D. Pasotto *et al.*, 2020. Bovine coronavirus: Variability, evolution, and dispersal patterns of a no longer neglected betacoronavirus. *Viruses*, Vol. 12. 10.3390/v12111285.
17. Vijgen, L., E. Keyaerts, E. Moas, I. Thoelen and E. Wollants *et al.*, 2005. Complete genomic sequence of human coronavirus OC43: Molecular clock analysis suggests a relatively recent zoonotic coronavirus transmission event. *J. Virol.*, 79: 1595-1604.
18. Zhang, X.M., W. Herbst, K.G. Kousoulas and J. Storz, 1994. Biological and genetic characterization of a hemagglutinating coronavirus isolated from a diarrhoeic child. *J. Med. Virol.*, 44: 152-161.
19. Decaro, N., M. Campolo, V. Mari, C. Desario and M.L. Colaianni *et al.*, 2009. A candidate modified-live bovine coronavirus vaccine: Safety and immunogenicity evaluation. *New Microbiol.*, 32: 109-113.
20. Varga, M., 2014. Infectious Diseases of Domestic Rabbits. In: *Textbook of Rabbit Medicine*, Varga, M. (Ed.), Butterworth-Heinemann, Oxford, United Kingdom, ISBN: 9780702049798, pp: 435-471.
21. Zappulli, V., S. Ferro, F. Bonsembiante, G. Brocca and A. Calore *et al.*, 2020. Pathology of coronavirus infections: A review of lesions in animals in the one-health perspective. *Animals*, Vol. 10. 10.3390/ani10122377.
22. Kerr, P.J. and T.M. Donnelly, 2013. Viral infections of rabbits. *Vet. Clin. North Am.: Exot. Anim. Pract.*, 16: 437-468.
23. Edwards, S., J.D. Small, J.D. Geratz, L.K. Alexander and R.S. Baric, 1992. An experimental model for myocarditis and congestive heart failure after rabbit coronavirus infection. *J. Infect. Dis.*, 165: 134-140.
24. Oglesbee, B.L. and B. Lord, 2020. Gastrointestinal Diseases of Rabbits. In: *Ferrets, Rabbits, and Rodents: Clinical Medicine and Surgery*, Quesenberry, K.E., C.J. Orcutt, C. Mans and J.W. Carpenter (Eds.), W.B. Saunders, Philadelphia, Pennsylvania, ISBN: 9780323484350, pp: 174-187.
25. Descôteaux, J.P. and G. Lussier, 1990. Experimental infection of young rabbits with a rabbit enteric coronavirus. *Can. J. Vet. Res.*, 54: 473-476.
26. DiGiacomo, R.F. and C.J. Maré, 1994. Viral Diseases. In: *The Biology of the Laboratory Rabbit*: Manning, P.J., D.H. Ringler and C.E. Newcomer (Eds.), Academic Press, Cambridge, Massachusetts, ISBN: 9780124692350, pp: 171-204.
27. Krogstad, A.P., J.E. Simpson and S.W. Korte, 2005. Viral diseases of the rabbit. *Vet. Clin. North Am.: Exot. Anim. Pract.*, 8: 123-138.
28. Guy, J.S., J.J. Breslin, B. Breuhaus, S. Vivrette and L.G. Smith, 2000. Characterization of a coronavirus isolated from a diarrheic foal. *J. Clin. Microbiol.*, 38: 4523-4526.
29. Miszczak, F., V. Tesson, N. Kin, J. Dina, U.B.R. Balasuriya, S. Pronost and A. Vabret, 2014. First detection of equine coronavirus (ECoV) in Europe. *Vet. Microbiol.*, 171: 206-209.
30. Fielding, C.L., J.K. Higgins, J.C. Higgins, S. McIntosh and E. Scott *et al.*, 2015. Disease associated with equine coronavirus infection and high case fatality rate. *Vet. Intern. Med.*, 29: 307-310.

31. Sanz, M.G., S. Kwon, N. Pusterla, J.R. Gold, F. Bain and J. Evermann, 2019. Evaluation of equine coronavirus fecal shedding among hospitalized horses. *Vet. Intern. Med.*, 33: 918-922.
32. Vin, R., N. Slovis, P.J. Henney, U.B.R. Balasuriya and C.M. Leutenegger, 2012. Equine coronavirus, a possible cause for adult horse enteric disease outbreaks. *J. Equine Vet. Sci.*, 32: S44-S45.
33. Prutton, J.S.W., S. Barnum and N. Pusterla, 2020. Evaluation of safety, humoral immune response and faecal shedding in horses inoculated with a modified-live bovine coronavirus vaccination. *Equine Vet. Educ.*, 32: 33-36.
34. Pusterla, N., R. Vin, C. Leutenegger, L.D. Mittel and T.J. Divers, 2017. Equine Coronavirus Infection. In: *Emerging and Re-Emerging Infectious Diseases of Livestock*, Bayry, J. (Ed.), Springer, Cham, Switzerland, ISBN: 978-3-319-47426-7, pp: 121-132.
35. Myrrha, L.W., F.M.F. Silva, E.F. de Oliveira Peternelli, A. Silva Jr., M. Resende and M.R. de Almeida, 2011. The paradox of feline coronavirus pathogenesis: A review. *Adv. Virol.*, Vol. 2011. 10.1155/2011/109849.
36. de Groot, R.J., S.C. Baker, R. Baric, L. Enjuanes and A.E. Gorbalenya *et al.*, 2012. Family-Coronaviridae. In: *Virus Taxonomy: Ninth Report of the International Committee on Taxonomy of Viruses*, King, A.M.Q., M.J. Adams, E.B. Carstens and E.J. Lefkowitz (Eds.), Elsevier, Amsterdam, Netherlands, ISBN: 9780123846846, pp: 806-828.
37. Amer, A., A.S. Suri, O. Abdul Rahman, H.B. Mohd, B. Faruku, S. Saeed and T.I.T. Azmi, 2012. Isolation and molecular characterization of type I and type II feline coronavirus in Malaysia. *Virol. J.*, Vol. 9. 10.1186/1743-422X-9-278.
38. Benetka, V., A. Kübber-Heiss, J. Kolodziejek, N. Nowotny, M. Hofmann-Parisot and K. Möstl, 2004. Prevalence of feline coronavirus types I and II in cats with histopathologically verified feline infectious peritonitis. *Vet. Microbiol.*, 99: 31-42.
39. Pedersen, N.C., 2014. An update on feline infectious peritonitis: Virology and immunopathogenesis. *Vet. J.*, 201: 123-132.
40. Kipar, A. and M.L. Meli, 2014. Feline infectious peritonitis: Still an enigma? *Vet. Pathol.*, 51: 505-526.
41. Kipar, A., J. Kremendahl, D.D. Addie, W. Leukert, C.K. Grant and M. Reinacher, 1998. Fatal enteritis associated with coronavirus infection in cats. *J. Comp. Pathol.*, 119: 1-14.
42. Sherding, R.G., 2006. Feline Infectious Peritonitis (Feline Coronavirus). In: *Saunders Manual of Small Animal Practice*, Birchard, S.J. and R.G. Sherding (Eds.), Saunders, Philadelphia, Pennsylvania, ISBN: 978-0-7216-0422-0, pp: 132-143.
43. Hartmann, K., 2005. Feline infectious peritonitis. *Vet. Clin. North Am.: Small Anim. Pract.*, 35: 39-79.
44. Andrew, S.E., 2000. Feline infectious peritonitis. *Vet. Clin. North Am.: Small Anim. Pract.*, 30: 987-1000.
45. Barker, E.N., S. Tasker, T.J. Gruffydd-Jones, C.K. Tuplin and K. Burton *et al.*, 2013. Phylogenetic analysis of feline coronavirus strains in an epizootic outbreak of feline infectious peritonitis. *J. Vet. Intern. Med.*, 27: 445-450.
46. Chang, H.W., H.F. Egberink and P.J. Rottier, 2011. Sequence analysis of feline coronaviruses and the circulating virulent/avirulent theory. *Emerg. Infect. Dis.*, 17: 744-746.
47. Vogel, L., M. van der Lubben, E.G. Te Lintelo, C.P.J. Bekker and T. Geerts *et al.*, 2010. Pathogenic characteristics of persistent feline enteric coronavirus infection in cats. *Vet. Res.*, Vol. 41. 10.1051/vetres/2010043.
48. Sykes, J.E., 2014. Feline Coronavirus Infection. In: *Canine and Feline Infectious Diseases*, Sykes, J.E. (Ed.), W.B. Saunders, Philadelphia, Pennsylvania, ISBN: 9781437707953, pp: 195-208.
49. Almeida, A.C.S., M.V. Galdino and J.P. Araújo Jr., 2019. Seroepidemiological study of feline coronavirus (FCoV) infection in domiciled cats from Botucatu, São Paulo, Brazil. *Pesqui. Vet. Bras.*, 39: 129-133.
50. Addie, D., S. Belák, C. Boucraut-Baralon, H. Egberink and T. Frymus *et al.*, 2009. Feline infectious peritonitis: ABCD guidelines on prevention and management. *J. Feline Med. Surg.*, 11: 594-604.
51. Drechsler, Y., A. Alcaraz, F.J. Bossong, E.W. Collisson and P.P.V.P. Diniz, 2011. Feline coronavirus in multicat environments. *Vet. Clin. North Am.: Small Anim. Pract.*, 41: 1133-1169.
52. Szczepanski, A., K. Owczarek, M. Bzowska, K. Gula and I. Drebot *et al.*, 2019. Canine respiratory coronavirus, bovine coronavirus, and human coronavirus OC43: Receptors and attachment factors. *Viruses*, Vol. 11. 10.3390/v11040328.

53. Priestnall, S.L., J. Brownlie, E.J. Dubovi and K. Erles, 2006. Serological prevalence of canine respiratory coronavirus. *Vet. Microbiol.*, 115: 43-53.
54. Erles, K. and J. Brownlie, 2005. Investigation into the causes of canine infectious respiratory disease: Antibody responses to canine respiratory coronavirus and canine herpesvirus in two Kennelled dog populations. *Arch. Virol.*, 150: 1493-1504.
55. Yeşilbağ, K., Z. Yilmaz, S. Torun and A. Pratelli, 2004. Canine coronavirus infection in Turkish dog population. *J. Vet. Med.*, 51: 353-355.
56. Mitchell, J.A., H.W. Brooks, B. Szladovits, K. Erles, R. Gibbons, S. Shields and J. Brownlie, 2013. Tropism and pathological findings associated with canine respiratory coronavirus (CRCoV). *Vet. Microbiol.*, 162: 582-594.
57. Stavisky, J., G. Pinchbeck, R.M. Gaskell, S. Dawson, A.J. German and A.D. Radford, 2012. Cross sectional and longitudinal surveys of canine enteric coronavirus infection in kennelled dogs: A molecular marker for biosecurity. *Infect. Genet. Evol.*, 12: 1419-1426.
58. Buonavoglia, C. and V. Martella, 2007. Canine respiratory viruses. *Vet. Res.*, 38: 355-373.
59. Le Poder, S., 2011. Feline and canine coronaviruses: Common genetic and pathobiological features. *Adv. Virol.*, Vol. 2011. 10.1155/2011/609465.
60. Stavisky, J., G.L. Pinchbeck, A.J. German, S. Dawson, R.M. Gaskell, R. Ryvar and A.D. Radford, 2010. Prevalence of canine enteric coronavirus in a cross-sectional survey of dogs presenting at veterinary practices. *Vet. Microbiol.*, 140: 18-24.
61. Radford, A.D., D.A. Singleton, C. Jewell, C. Appleton and B. Rowlingson *et al.*, 2021. Outbreak of severe vomiting in dogs associated with a canine enteric Coronavirus, United Kingdom. *Emerg. Infect. Dis.*, 27: 517-528.
62. Tennant, B.J., R.M. Gaskell, D.F. Kelly, S.D. Carter and C.J. Gaskell, 1991. Canine coronavirus infection in the dog following oronasal inoculation. *Res. Vet. Sci.*, 51: 11-18.