PREPARE-EU

Connecting European Expertise for Pandemic Preparedness

ABSTRACT BOOK & PARTICIPANT LIST

presented by

PCR4ALL

KU LEUVEN

LEUVEN ONE HEALTH

BE READY

COMPREHEND

FORTIFIEDx

LEAPS

Triple A COAT

DURABLE
Research Network against Epidemics

B-PATH

SUNRISE

ESCAPE
CONNECTED · SCALABLE · PREPARED

One BAT

DECIPHER

UniHealth
POINT-OF-CARE INNOVATION

PREPARE-EU Abstract Book – Table of Contents

Poster 21: Veterinary medicine in human pandemics: Feline Infectious Peritonitis compared to Long Covid

N. Schneider* and M. Spanoghe* **

*Long Covid Belgium Association

**Laboratory of Biotechnology and Applied Biology, HEPH Condorcet, Ath, Belgium

In cats, there is a fatal disease called Feline Infectious Peritonitis (FIP), caused by the mutation of a coronavirus (FCoV) in the small intestine [1]. As a result, the viral persistence depletes the regulatory T cells in the intestinal lymph nodes [2]. Their physiological function is to suppress plasma cells that produce antibodies directed against the body or without sense. In the context of FIP, the destruction of misdirected plasma cells does not take place. This results in a massive production of senseless antibodies in stressed infected cats, causing generalized vasculitis with fatal consequences [3]. During the course of FIP, atypical amyloid structures are formed [4], the ends of which have an antigenic structure. The latter induce the formation of the erroneous antibodies. A significant difference from Long Covid (LC) in humans is the absence or near absence of misdirected antibodies. What is surprising, however, is the trigger of autoimmune diseases in Long Covid patients, such as lupus, diabetes 1, Hashimoto's thyroiditis, Bell's palsy, atypical myasthenia gravis amongst others. The clinical picture of LC is highly variable and multisystemic, with many patients being diagnosed with autoimmune diseases that they did not express before their SARS-CoV-2 infection. Moreover, some of these diseases have low incidences (AIED)[5].

One possible explanation for the high incidence of these orphan diseases, which are often hereditary and do not appear spontaneously at an advanced age, lies in the pathomechanism of antibody formation described in cats. Some of them, especially those directed against nerves, are more common than others. An important hypothesis regarding the development and maintenance of disease symptoms in the LC results from a complex disorder of the immune system, which is well known in veterinary medicine. Therefore, we believe that a convergence between the knowledge acquired by scientists in veterinary medicine, especially in zoonoses, should be much more involved in human pandemics.

References

1 – Hartmann, K. (2005). Feline infectious peritonitis. *Veterinary Clinics: Small Animal Practice*, *35*(1): 39 – 79

2 – Vermeulen BL, Devriendt B, Olyslaegers DA et al. (2013). Suppression of NK cells and regulatory T lymphocytes in cats naturally infected with feline infectious peritonitis virus. V*eterinary Microbiology, 164(*1 – 2), 46 – 59

3 – Myrrha, L. W., Silva, F. M. F., Peternelli, E. F. D. O., Junior, A. S., Resende, M., & Almeida, M. R. D. (2011). The paradox of feline coronavirus pathogenesis: a review. *Advances in Virology* (1)109849

4 – Charnley, M., Islam, S., Bindra, G.K. et al. (2022). Neurotoxic amyloidogenic peptides in the proteome of SARSCOV2: potential implications for neurological symptoms in COVID-19. *Nat Commun* 13, 3387. https://doi.org/10.1038/s41467-022-30932-1

5 – Ruckenstein, M.J. (2004). Autoimmune inner ear disease. *Current Opinion in Otolaryngology & Head and Neck Surgery 12*(5), 426 - 430