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Vanessa Barrs is Professor of Feline Medicine & Infectious Diseases in the Sydney School of Veterinary Science and the Marie Bashir Institute. She is also a registered Specialist in Feline Medicine, and was pivotal in the establishment and ongoing success of the Valentine Charlton Cat Centre, which celebrated its 10th anniversary in 2015. Vanessa is a one-health infectious diseases researcher particularly interested in pathogens of animals and humans, including fungi and viruses. In addition to over 100 refereed publications and book chapters, Vanessa is President of the International Society of Companion Animal Infectious Diseases.

Dr Mark Kelman will be presenting on behalf of Professor Barrs.

Abstract

Halting parvovirus outbreaks in Australian cats

Parvoviruses are highly contagious, environmentally resilient viruses that are often deadly to unvaccinated cats and dogs. They cause severe enteritis, dehydration, sepsis and death. Feline parvoviruses (FPVs) cannot infect dogs, but canine parvoviruses (CPVs) can jump from dogs to cats and vice-versa. Since 2013 there have been several large outbreaks of parvoviral disease in shelter-housed cats around Australia resulting in hundreds of deaths and temporary closure of some shelters.

Results will be shared from a study that was conducted to determine which viruses (CPVs or FPVs) have caused these outbreaks in shelter-housed cats and what factors have contributed to the "perfect storm" that has been brewing to result in the re-emergence of this preventable disease. The results of these study will inform the development of vaccination and biosecurity strategies in shelters to prevent further outbreaks.

Full Paper

Introduction

Feline panleukopenia, also known as feline infectious enteritis is a highly contagious and often fatal disease of cats caused by feline panleukopenia virus (FPV) or canine parvovirus (CPV), which are closely related strains of *Carnivore protoparvovirus* 1.
Transmission of parvoviruses is usually indirect ingestion or inhalation of virus particles in fomites (e.g. infected body fluids, faeces). These small DNA viruses are resistant to many disinfectants and have the ability to survive harsh environments for 12 months or more. Reservoirs of virus in the environment are likely maintained by cats that are subclinically infected (asymptomatic) and recovered infected cats that shed virus in their faeces for several weeks after recovery.

Over 90% of cases of feline panleukopenia are caused by feline panleukopenia virus (FPV). However, CPVs have been responsible for some outbreaks of disease in cats in South East Asia and for individual cases of disease in Europe. Clinical signs of disease occur after a 2 to 10 day incubation period. FPV and CPV need to use the host’s cellular enzymes to replicate successfully (cellular DNA polymerases in rapidly multiplying cells). They first replicate in the lymphoid tissues of the oropharynx 18 to 24 hours post-infection. This is followed by a viraemia 2 to 7 days later with widespread dissemination of the virus in lymphoid tissue, bone marrow and intestinal crypt epithelium. Leukopenia (depletion of white blood cells) occurs primarily due to viral replication in white blood cell precursors in the bone marrow.

When very young kittens (six weeks old or less) are infected, sudden death due to severe dehydration and overwhelming sepsis (secondary bacterial infection) is common. In older kittens and adult cats the first signs of infection are usually lethargy and loss of appetite, which progresses to vomiting and/or diarrhoea, which can be haemorrhagic. In contrast to parvovirus infection of dogs, diarrhoea of infected cats is less commonly bloody. Common complications that result in death include severe dehydration and circulatory shock, septicaemia and disseminated intravascular coagulation (clotting abnormalities).

Infected queens can abort (early pregnancy) or give birth to kittens with neurological defects that affect co-ordination and vision, depending on the stage of pregnancy at which infection occurs.

Effective vaccinations against FPV have been available since the 1960s. Studies have shown that FPV vaccines afford protection against canine parvoviruses. Clinical disease was rare in Australian cats from the mid-1970s until 2014, when an outbreak was reported to a national online disease surveillance reporting tool.

The aim of our study was to determine the strains of *Canine protoparvovirus 1* (FPV or CPV) and epidemiological factors involved in outbreaks of feline panleukopenia reported in Australia since 2014.

**Materials and Methods**

Veterinarians and shelter owners were contacted to arrange site-visits, collect samples and obtain information about animal movements, biosecurity and vaccination protocols for qualitative analysis. DNA was extracted from faeces or tissues collected at post-mortem of cats with clinical signs of panleukopenia from each outbreak.

DNA sequencing of the VP2 gene was performed. Phylogenetic analysis of viruses from the outbreak and from CPV-like and FPV-like VP2 sequences available on GenBank was performed using the maximum likelihood method.

**Results**

Three outbreaks causing over 350 fatalities were identified in; (A) 2014, Melbourne; (B) 2015, Melbourne and Mildura, a city 540 km from Melbourne; (C) 2016, Melbourne and Sydney. Outbreaks in Mildura and Melbourne were caused by identical, or closely related, FPV genotype(s), while the Sydney outbreak was caused by a different FPV genotype. Most cases occurred in cats from council pounds or charitable or private shelters.
Shelters with the highest number of fatalities did not perform routine vaccination. In shelters that did administer vaccines, disease occurred in incompletely-vaccinated cats (e.g. kittens that had received only one vaccine) or cats that had not been vaccinated because of respiratory disease or other illnesses.

Movement of unvaccinated kittens or cats from council pounds or private shelters to networks of private foster carers was identified in all outbreaks, including between Melbourne and Mildura in outbreak B. The median age of cats at diagnosis was 8 weeks. All outbreaks occurred from summer to autumn, coinciding with peak shelter intakes of kittens. Suboptimal biosecurity protocols were also common.

**Discussion**

Two important factors were identified as contributing to the recent feline panleukopenia outbreaks in Sydney and Melbourne; absent or incomplete vaccination and suboptimal biosecurity.

**Vaccination and FPV**

Shortly after birth, kittens ingest colostrum from their mothers. Colostrum contains antibodies that protect kittens against infections that the queen has been exposed to or vaccinated against. These antibodies, known as maternally-derived antibodies (MDA), gradually decline over time. By 16 weeks of age, MDA are no longer detectable in most kittens. As MDA levels fall, a point is reached where MDA no longer protect against infection BUT the antibodies interfere with vaccination, making it ineffective. For example, MDA levels fail to protect against FPV infection in most 8 to 12 week-old kittens but they interfere with vaccination, so vaccination at 8 to 12 weeks will fail to confer vaccinal immunity (Figure 1). This window is known as the “immunity gap”.

![Figure 1](image.png)

**Figure 1.** The immunity gap is the period when maternal immunity no longer protects the kitten from infection by feline panleukopenia virus, but still interferes with the development of vaccinal immunity. Figure adapted from ABCD guidelines on prevention and management of feline panleukopenia.4
The World Small Animal Veterinary Association (WSAVA) recommends that kittens be vaccinated from 6 weeks of age every 3 to 4 weeks until they are 16 weeks of age or older. A booster vaccine is recommended at the age of 6 months or 1 year with subsequent booster vaccinations every three years thereafter.\(^5\)

**In situations where FPV has broken out in a shelter, kittens should be vaccinated from 4 weeks of age, using modified live virus (MLV) vaccines.** Cats of unknown vaccine status should not be housed together. Vaccination should be repeated every 2-3 weeks until 16 weeks of age. MLV vaccines are recommended because they induce a more rapid onset of immunity than inactivated vaccines. In one study 64 eight-to-ten week old kittens were vaccinated with one dose of inactivated or MLV FPV vaccine. Protective antibody titres were present 14 days post-vaccination in 31% of kittens receiving inactivated FPV versus 85% of kittens receiving MLV, respectively.\(^6\)

In the Australian FPV outbreaks high numbers of fatalities occurred in council and private shelters where vaccination against FPV was not practiced. Currently in NSW the Animal Welfare Code of Practice No.5 – Dogs and cats in animal boarding establishments (http://www.dpi.nsw.gov.au/animals-and-livestock/animal-welfare/general/welfare-of-dogs/aw-code-5) states that:

"1.3 Establishments which provide commercial boarding services, Council Pound services and veterinary hospital services must comply with the standards of this code.

6.1.2 For cats, vaccination against feline infectious enteritis (feline panleukopenia) and feline respiratory disease is required. A current vaccination certificate (certifying that vaccination was done within the preceding 12 months) must be produced for each cat before admission."

Unfortunately, under the regulation, the code is only applicable to fee for service boarding establishments. A new code of practice specifically for council pounds and animal shelters is under development. Two council pounds involved in this study in NSW and Victoria did not practise vaccination of cats prior to the outbreak of FPV. One has since implemented vaccination of cats in their care.

In the author’s opinion stringent legislation is urgently required mandating vaccination of cats in animal shelters or animal holding facilities on admission to the shelter.

**Disinfection and FPV**

No disinfectants are effective against parvoviruses (FPV or CPV) unless the organic material in which they are contained (e.g. faeces, vomit) has been first cleaned using detergent. The presence of organic material inactivates disinfectants. In general disinfectants need a minimum contact time to render virus particles non-infectious, and surfaces that have been disinfected should be rinsed before cats are exposed to them as ingestion of disinfectants, or contact with paws and skin, can cause severe ulcers and burns.

There are several disinfectants that are effective against parvoviruses including:

1. **F10\(^\text{TM}\)** – this is a combination of two classes of disinfectant, a biguanide and a quaternary ammonium compound (benzalkonium chloride), neither of which are effective against FPV by themselves. F10 disinfectant is effective at a concentration of 1:100 with a minimum contact time of 15 minutes.

2. **Potassium peroxymonosulfate (Virkon\(^\text{TM}\))**

3. **Sodium hypochlorite (household bleach)** – is effective using a 1:32 dilution of a 5 to 6% solution of sodium hypochlorite. Bleach is corrosive and can cause damage to non-stainless steel cages.
Biosecurity protocols
Preventing the spread of infection within a shelter environment and requires strict adherence to biosecurity protocols that should be discussed and established by shelter staff together with their veterinarian.

Looking to the future
Once an FPV outbreak occurs there is an ongoing risk of future, seasonal outbreaks in the same area coinciding with peak kitten season, since kittens are most vulnerable to infection. This has been seen in the recent Melbourne outbreak. Rapid responses to FPV outbreaks and implementation of vaccination protocols using modified live vaccines and more frequent and younger vaccination of kittens as well as best-practice infection control practices are essential to prevent further spread of disease.

References


