Canine Parvovirus:

A historical analysis of the development of canine parvovirus from its origin of feline parvovirus (FPV) its vaccine development, and the current understandings of its treatment around the world

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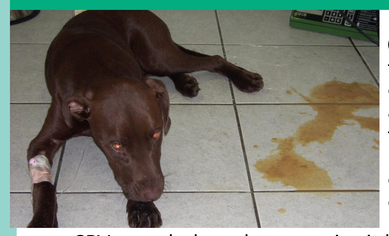
Overview



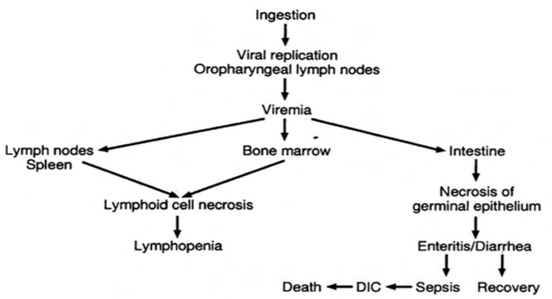
Canine Parvovirus (CPV) infect canines through the lymphoid cells. Parvovirus spreads across populations typically through feces, but it can travel through external wear on the dogs, owners, or ground matter.

CPV spreads throughout a canine is by replicating in the S phase of mitosis. In older animals, it will be limited to just the lymphoid or the small intestines, leading to lymphopenia. However, in neonatal and young puppies, the virus replicates throughout all tissues, leading to inflammation of the heart. .

Parvovirus is easily diagnosed by the mucoid diarrhea, or watered down and runny feces, and the constant vomiting of bile. Eventually, the canine will experience myocardia, or a heart attack, and then the dog will enter shock and most likely die.

Parvovirus is highly contagious between canines, but the current vaccines work efficiently as prevention. However, puppies cannot be fully vaccinated until they are 12 weeks of age, so they are very susceptible to contracting it after ten days post birth.

(Picture below; pathogenesis of canine parvovirus)



Origin

There are multiple theories as to the origin of CPV. Typically, the theory is held that it directly mutated from feline panleukopenia virus (FPV). However, another argument is for CPV being an adaptation from non-domestic carnivore diseases, such as blue fox parvovirus and mink enteritis virus (Truyen 2006). The main change between these diseases is the adaption of a canine transferrin receptor, the binding mechanic that allows it to infect different host cells based on their specialization. This receptor is also antigenic, and the mutations here allows it to be recognized differently between species' immune systems.

CPV did not develop from recombination, but instead through increased rates of mutations and the increased selection ability of the canine transferrin receptor. Most of these mutations came from single nucleotide substitutions, leading to missense mutations. Interestingly, though, is that these mutations occur at rates of RNA virus mutations, despite these changes being present in DNA strands. This rate is in comparison to those of HIV and human influenza, leading to its high infectivity despite constant vaccine development

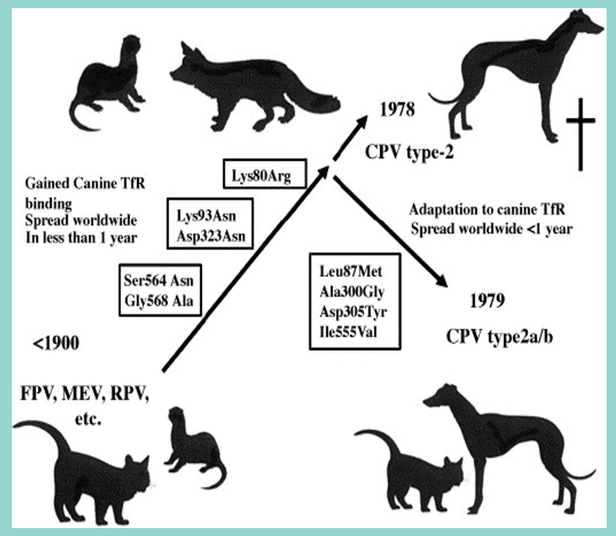
(Pictured right; proposed evolutionary path of CPV)

Historical Development

Canine Parvovirus was first diagnosed in the United States in 1978, but it was theorized that it first developed in Greece in 1974, Europe in 1976, and then it traveled to America. Initially, the disease itself was not recognized as a viral infection until mass amounts of canines became fatally ill. It was initially seen as just a gastrointestinal and respiratory disease and was called the minute virus of canines. But, as the symptoms were discovered, the connection to other parvoviruses was made, leading to the naming of it as Canine Parvovirus 1. When it was first diagnosed, CPV was considered to by non-pathogenic, leading to a delayed response in developing treatments for it. The first treatment for CPV was a serum from FPV infected cats. The serum would help with symptoms, yet did not fully heal the patient, proving the connection between the two diseases.

By 1980, CPV had spread worldwide through the travel of infected canines. Most patients infected were asymptomatic, leading to difficulty tracking the spread of infection. The pathogenesis was not fully clarified, leading to mass pandemonium on how to treat the new disease. At the end of 1981, herd immunity was reached, and CPV cases lowered. However, in 1984, CPV mutated into CPV-2a, creating the new symptom of shock. This strain infected both vaccinated and unvaccinated dogs, and the onset of death typically occurred without any other symptoms arising first. The new CPV had exponential growth, doubling its infected population size every year (Shackelton 2005).It is still the most common strain of CPV present today., vaccine development had led to CPV being under control by this mutation.

By 2005, CPV had evolved again to spread to other animals, including felines. It can also still be found in wild carnivores such as foxes and skunks. Many dog parks and recreational areas began implementing stronger requests of cleaning up feces after pets to prevent contamination of their properties. But, developing vaccine application led to prevention of parvovirus in dogs above 3 months of age. However, in recent years, a push against vaccines has led to a reemergence of CPV within older dogs, but the support of animal vaccinations is on the rise again.



Vaccine Studies

Initially, researchers responded to the CPV pandemic through vaccinations of live samples of FPV and Mink enterititis viruses in 1979. The assumption was their relation would heighten the canine's immune system without causing symptoms. Following these attempts were vaccines of inactivated initial CPV, modified CPV-2a (mutated version), and commercial mink enteritisis virus vaccines. However, none of these vaccines were effective and failed to prevent transmission between canines , but they were sold at mass my veterinarians, leading to large profits.

With these failed vaccines, the public began to distrust vaccines for their dogs. Many puppies were vaccinated with live versions of CPV with poor effacing results due to the rapid mutation of the disease. However, in 1984, after the development of the lyme disease vaccine for canines and its proven success, the public became more open to CPV vaccines again. As CPV mutated, the vaccines developed in response, leading to a stronger vaccine with little failure rates currently.

The current vaccinations consist of high-titer, meaning the amount of virus per vaccine dose, and low passage CPV, where the virus can infect cells but not cause disease. There are 3 does of vaccine that are given to puppies at 6, 9, and then 12 weeks of age, leading to full vaccination at 3 months old.

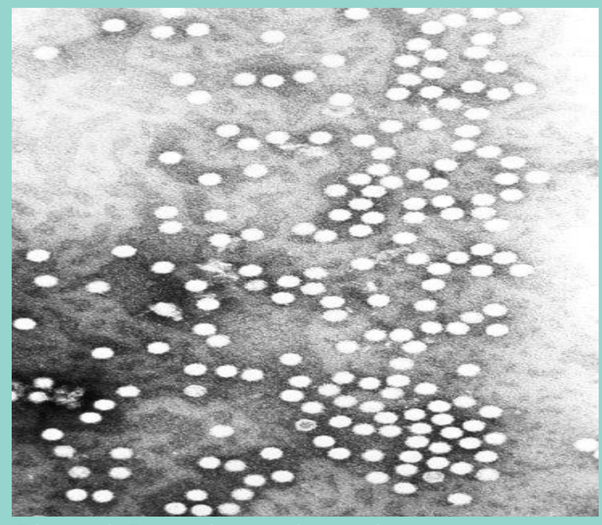
There is current debate on whether canines should receive yearly boosters to support their immune systems. Those who have recovered from a CPV infection are likely to have immunity for life. For those vaccinated, 93.7% have strong, healthy immune responses for two years (Goddard, 2010). A current concern is the practice of overvaccination leading to resistant strains of CPV developing. So, current veterinarians base their recommendation of a booster vaccination based on the antibodies present in serological results.

Current State

Although CPV is not as rampant as when it first developed, it is still a dangerous infection that requires careful practices to prevent. CPV-2a can exist of surfaces for up to 5 months, and many disinfectants do not destroy the virus. Common household beach is effective, but it must be left on the surface for an hour to inactivate CPV (Goddard 2010).

For puppies under the age of full vaccination, special procedures are followed to treat them without the risk of CPV exposure. Many veterinary clinics have a separate kennel room to house infant patients or those infected. In specialized clinics, such as surgery or oncology, veterinarians may deny treatment until the canine is fully vaccinated as to not risk exposing their highly vulnerable patients.

Even though CPV is under control of vaccination and sterilization in the United States, other countries are experiencing new waves of CPV that are becoming epidemics. In India in 2021, a new wave of Canine Parvovirus arose tha led to quick deaths of the infected animals (R.A. 2021). This new strain not only targets puppies but specifically rottweilers, but this infection pattern is not fully understood yet. This occurrence, though, has led to a push against the purchase of foreign dogs as to not spread mutant strains of CPV to other countries.



Pictured: Parvovirus under a microscope

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