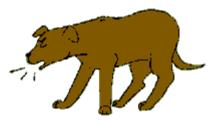


Kennel Cough (Infectious Tracheobronchitis)

What is it?

Kennel cough is an infectious bronchitis characterized by a harsh, hacking cough that most people describe as sounding like "something stuck in my dog's throat." It is analogous to a chest cold for humans and is only a serious condition in special circumstances (see below); in general, it resolves on its own. A dog with kennel cough generally feels active and maintains a normal appetite despite frequent fits of coughing. There is usually no fever or listlessness, just lots of coughing.



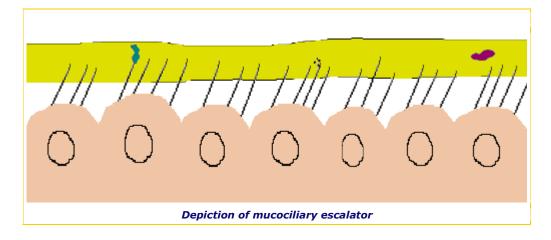
Not sure what a Coughing Dog sounds like?

Dogs can make an assortment of respiratory sounds. Usually a cough is recognizable but it is important to be aware of another sound called a reverse sneeze. The reverse sneeze is often mistaken for a cough, a choking fit, sneezing, retching, or even gasping for breath. In fact, the reverse sneeze represents a post-nasal drip or tickle in the throat. It is considered normal especially for small dogs or dogs and only requires attention if it is felt to be excessive. The point here is to know a cough when you see one. A cough can be dry or productive, meaning it is followed by a gag, swallowing motion, production of foamy mucus (not to be confused with vomiting). Here are some videos that might help.

A coughing dog that has a poor appetite, fever, and/or listlessness should be evaluated for pneumonia.

How Infection Occurs

An infected dog sheds infectious bacteria and/or viruses in respiratory secretions. These secretions become aerosolized and float in the air and can then be inhaled by a healthy dog.



The normal respiratory tract has substantial safeguards against invading infectious agents. The most important of these is probably what is called the mucociliary escalator. This safeguard consists of tiny hair-like structures called cilia that protrude from the cells lining the respiratory tract and extend into a coat of mucus over them. The cilia beat in a coordinated fashion through the lower and more watery

mucus layer called the sol. A thicker mucus layer called the gel floats on top of the sol. Debris, including infectious agents, get trapped in the sticky gel and the cilia move them upward towards the throat where the collection of debris and mucus may be coughed up and/or swallowed.

The mucociliary escalator is damaged by the following:

- shipping stress
- crowding stress
- heavy dust exposure
- cigarette smoke exposure
- infectious agents (viruses such as reovirus, adenovirus, parainfluenza virus, and even the distemper virus can be initiating infections).
- cold temperature
- poor ventilation

Without this protective mechanism, invading bacteria, especially *Bordetella bronchiseptica*, the chief agent of kennel cough, may simply march down the airways unimpeded.

Bordetella bronchiseptica organisms have some tricks of their own as well:

- They are able to bind directly to cilia, rendering them unable to move within 3 hours of contact.
- They secrete substances that disable the immune cells normally responsible for consuming and destroying bacteria.

Because it is common for *Bordetella* to be accompanied by at least one other infectious agent (such as one of the viruses listed below), kennel cough is actually a complex of infections rather than infection by one agent.

Members of the kennel cough complex:

- Parainfluenza virus
- Canine adenovirus type 2
- Canine distemper virus
- Canine herpes virus
- Canine reovirus (type 1, 2, or 3)

Any of these viruses can produce a minor sore throat and cough ultimately allowing a way in for the more toxic *Bordetella bronchiseptica* bacteria.

Classically, dogs get infected when they are kept in a crowded situation with poor air circulation and lots of warm air (i.e., a boarding kennel, vaccination clinic, obedience class, local park, animal shelter, animal hospital waiting room, or grooming parlor). In reality, most causes of coughing that begin acutely in a dog are due to infectious causes and usually represent some form of kennel cough.

THE INCUBATION PERIOD IS 2 TO 14 DAYS

How is Diagnosis Made?

Usually the history of exposure to a crowd of dogs within the proper time frame plus typical examination findings (a coughing dog that otherwise feels well) is adequate to make the diagnosis. Radiographs show bronchitis, although severe cases can progress to pneumonia, especially if the canine distemper virus is involved.

How Contagious is it?

Bordetella infection can be picked up by rabbits, guinea pigs, pigs, cats (if they are very young and housed in groups), and other dogs. Bordetella is generally not considered contagious to humans although it is closely related to Bordetella pertussis, the agent of whooping cough. Immunesuppressed humans potentially could be infected.

Among dogs, kennel cough is fairly contagious depending on stress level, vaccination status, and exposure to minor viruses. Dogs shed *Bordetella* organisms for up to 3 months after infection.

Some veterinarians recommend keeping all dogs current on Bordetella vaccinations because you never know when they will be in an unexpected situation.

How is Kennel Cough Treated?

Although most cases will go away on their own, we like to think we can hasten recovery with antibiotics to directly kill the *Bordetella* organism. Kennel cough may be treated with cough suppressants to provide comfort during natural recovery. Alternatively, antibiotics and cough suppressants can be combined.

When is it a Serious Condition?

Very young puppies, especially those with a recent shipping history (i.e., pet store puppies) are especially prone to severe cases of infectious tracheobronchitis that frequently progress to pneumonia.

In dogs where the distemper virus is involved (usually shelter or pet store puppies), there is tremendous potential for serious consequences.

Vaccination Options

There are basically two options for kennel cough vaccination: injectable and intranasal. It is important to realize that not all members of the kennel cough complex have a vaccine. Also, because kennel cough is a localized infection (meaning it is local to the respiratory tract), it is an infection that does not lend itself to prevention by vaccination. Vaccination must be regularly boosted and often vaccination simply muffles the severity of infection without completely preventing it.

Injectable Vaccine

Injectable vaccination is a good choice for aggressive dogs who may bite if their muzzle is approached. For puppies, injectable vaccination provides good systemic immunity as long as two doses are given (approximately one month apart) after age 4 months. Boosters are generally given annually.

There is some controversy over whether previously vaccinated dogs generate better immunity receiving injectable or nasal boosters for kennel cough.

Parainfluenza, adenovirus type 2, and canine distemper, all members of the kennel cough complex, are all covered by the standard DHLPP vaccine, the basic vaccine for dogs. Adenovirus type 2 serum also immunizes against adenovirus type 1, the agent of infectious canine hepatitis.

Nasal Vaccine

Intranasal vaccination may be given as early as 3 weeks of age and immunity generally lasts 10 to 12 months. (Usually this vaccine is boosted annually but if you are expecting imminent exposure as in boarding, competition, or other event where dogs are together, it is optimal to boost if over 6 months have elapsed.) The advantage here is that the local immunity is stimulated right at the site where the natural infection would be trying to take hold.

It takes four days to generate a solid immune response after intranasal vaccination so it is best if vaccination is given at least four days prior to the exposure. Some dogs will have some sneezing or nasal discharge in the week following intranasal vaccination. As a general rule, nasal vaccination provides faster immunity than injectable vaccination.

There is some evidence that young puppies in a high risk environment may benefit from both injectable and nasal vaccination (rather than simply receiving one or the other).

IF A NASAL VACCINE IS ACCIDENTALLY GIVEN AS AN INJECTION, AN ABSCESS CAN RESULT UNDER THE SKIN.

Nasal vaccines cannot be given as injections.

VACCINATION IS NOT USEFUL IN A DOG ALREADY INCUBATING KENNEL COUGH.

If boarding is planned and more than 6 months have passed since the last booster shot, ideally the vaccine should be boosted 5 days or more before the start of boarding.

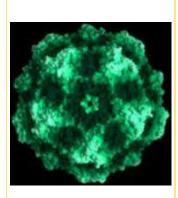
What if Kennel Cough doesn't Improve?

As previously noted, this infection is generally self-limiting. It should be at least improved partially after one week of treatment. If no improvement has been observed in this time, a re-check exam (possibly including radiographs of the chest) would be a good idea. Failure of kennel cough to resolve suggests an underlying condition. Kennel cough can activate a previously asymptomatic collapsing trachea or the condition may have progressed to pneumonia. There is also another respiratory infection called canine influenza, which seemed to be a racing greyhound issue exclusively until late 2005. This infection produces fever and pneumonia but starts looking like a routine kennel cough. This particular infection is much more severe, highly contagious, but for now seems to be uncommon.

If you have questions about a coughing dog, do not hesitate to bring them to your veterinarian, or use the Ask A Vet feature on the home page of Veterinary Partner.

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What is Parvo?



Canine parvovirus.
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JY. Sgro UW-Madison

Parvoviruses are a large group; almost every mammal species (including humans) seems to have its own parvovirus. Fortunately, each virus is specific for which animal species it can infect (i.e. the pig parvovirus will not infect people, the canine parvovirus will not infect cats, etc.) The canine parvovirus will affect most members of the dog family (wolves, coyotes, foxes etc.).

Parvoviruses are smaller than most viruses and consist of a protein coat (a capsid) and a single strand of DNA inside. It is hard to believe that such a simply constructed organism could be so deadly; however, this virus has proved especially effective at infecting rapidly dividing host cells such as intestinal cells, bone marrow cells, cells of the lymph system, and fetal cells. Parvoviruses are not enveloped in fat the way many other viruses are. This makes parvoviruses especially hardy in the environment and difficult to disinfect away.

While the parvoviruses of other species have been well known for decades, the canine parvovirus is a relative newcomer. The original canine parvovirus, discovered in 1967 and called **CPV-1** did not represent much of a medical threat except to newborn puppies but by 1978, a new variant, **CPV-2** appeared in the U.S. This newer version seems to represent a mutation from the feline parvovirus (which is more commonly known as the feline distemper virus). Because this virus was (and is) shed in gigantic numbers by infected animals, and because this virus is especially hardy in the environment, worldwide distribution of the virus rapidly occurred. At this time, the virus is considered to be ubiquitous, meaning that it is present in **EVERY ENVIRONMENT unless regular disinfection is applied.**

Attempting to shield a puppy from exposure is completely futile.

In 1978, no dog had any sort of immunity against this virus. There was no resistance and the epidemic that resulted was disastrous. To make matters worse, a second mutation creating **CPV-2a** had occurred by 1979, and it seemed to be even more aggressive. Vaccine was at a premium and many veterinarians had to make do with feline distemper vaccine as it was the closest related vaccine available while the manufacturers struggled to supply the nation with true parvo vaccines.

Over thirty years have passed since then. The most common form of the virus is called CPV-2b. Virtually all dogs can be considered to have been exposed to it at least to some extent, which means that most adult dogs, even those inadequately vaccinated, can be considered to have at least some immunity. It is also worth mentioning the new particularly virulent strain of parvovirus: CPV-2c, which is rapidly becoming the second most common form of canine parvovirus. CPV-2c was discovered in the year 2000 and is able to infect cats. Cats vaccinated against feline distemper can be considered protected. Currently available vaccines cover all variants of canine parvo including CPV-2c as do all the commercially available diagnostic test kits.

For more specific information about Canine Parvovirus-2c, see the American Veterinary Medical Association's FAQ.

Parvoviral infection has become a disease almost exclusively of puppies and adolescent dogs.

Parvoviral infection must be considered as a possible diagnosis in any young dog with vomiting and/or diarrhea. With proper hospitalization, survival rates approach 80%. Still, there are many myths and misunderstandings about this virus, how it is spread, and how to prevent it. The purpose of this web site is to clear up these misconceptions and provide the public with an accurate information source.

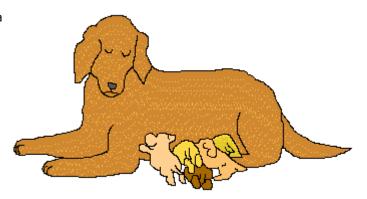
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Parvo Vaccination Options/Prevention



Maternal Antibody: Our Biggest Obstacle

The biggest problem in protecting a puppy against this infection ironically stems from the natural mechanism of protection that has evolved. As mentioned previously, puppies obtain their immunity from their mother's first milk, the colostrum, on the first day of life. This milk contains the mother's antibodies against parvovirus, and until these antibodies wane to ineffective levels they will protect the puppy.



The problem is that they will also inactivate vaccine.

Vaccine is a solution of inactivated virus, either live and weakened (attenuated or modified) or killed. This virus is injected into the puppy. If there is still adequate maternal antibody present, this vaccine virus will be destroyed just as if it were a real infection. There will be a period of about a week when there is not enough maternal antibody to protect the puppy but too much to allow a vaccine to work. (This period is called the window of vulnerability.) After this period, vaccine can be effective.

The next problem is that the age at which vaccine can be effective is different for each individual puppy.

To get around this, we vaccinate puppies in a series, giving a vaccine every 2 to 4 weeks until age 16 weeks. By age 16 weeks, we can be certain that maternal antibodies have waned and vaccine should be able to take. It should be recognized that some individuals, especially those of well-vaccinated mothers, must be vaccinated out to 20 weeks unless a high titer vaccine is used.

After a puppy is born, maternal antibody levels drop by half approximately every 10 days.

Puppies that were born first or were more aggressive at nursing on the first day will get more maternal antibody than their littermates.

Mother dogs vaccinated at approximately the time of breeding will have the highest antibody levels to pass on to their puppies.

REMEMBER, the more maternal antibody a puppy has, the less likely a vaccine is to work and the longer one must wait for antibody to wane and for vaccination to be effective.

Should Live or Killed Vaccine Be Used?

Killed vaccine is the least effective at penetrating maternal antibody. It is also associated with more vaccine reactions since more stabilizing chemicals are used in a killed vaccine. I recommend using live parvo vaccine only unless there is any question about the immunologic competence of the dog to be vaccinated and the dog is an adult. Killed vaccine should probably not be relied upon for puppies.

What is a High Titer Vaccine?

In the mid-1990s a new innovation in parvo vaccination was developed: the high titer vaccine. The term "high titer" refers to the amount of virus in the dose of vaccine and means that there is a great deal more virus than in the standard vaccines. When the puppy is vaccinated, maternal antibody binds the virus present. If a high titer vaccine is used, there is still virus left over after all the maternal antibody has been used up. This extra virus can then stimulate the puppy's own immune system. High titer vaccines commonly produce full protection by age 12 weeks (though I recommend carrying vaccination out to age 16 weeks to be certain - an especially good idea for breeds predisposed to infection such as the Rottweiler, Doberman pinscher, and American pit bull terrier).

At this point virtually all commercially available live vaccines are of the high titer type.

It should be noted that giving vaccine more frequently than every 2 weeks will cause interference between the two vaccines and neither can be expected to be effective. This includes giving vaccines for different infections. Vaccines should be spaced 2 to 4 weeks apart.

It is commonly held that puppies need a certain number of vaccines for protection to be achieved (usually either 3 or 4 is the magic number). The number of vaccines given has nothing to do with protection. In order for protection to be achieved, vaccine must be given when it can penetrate maternal antibody.

A vaccine FAQ can answer common vaccination questions. LINK

Vaccinating Adult Dogs

Classically, parvovirus vaccine has been administered annually to all dogs. Vaccine against canine parvovirus has been included in the distemper combination vaccine (the DHLPP, "6 in one," etc.)

There has also been some thought that annual vaccination is not necessary, especially for a disease where adult dogs are considered low risk. Many university teaching hospitals have switched to a 3-year schedule for adult dogs, plus the American Animal Hospital Association recommends that parvo vaccination be given to adult dogs on a 3-year schedule. There is still controversy regarding this practice, especially given possible financial impact to most veterinary hospitals. Do not be surprised if your veterinarian has chosen to follow the university and begins recommending a 3-year vaccination protocol for this virus.

Read the AAHA vaccination guidelines.

What is the Meaning of a Vaccine Titer?

A vaccine titer is a blood test that measures the antibody level a dog is carrying against a certain virus. There are two methods of measuring parvovirus antibody titer: hemagglutination inhibition and serum neutralization. The value refers to how diluted the dog's serum (blood) must be for antibody to still be detectable. Based on work at Cornell University, the following titer levels are generally considered protective:

- Hemagglutination inhibition titer of 1:80 or more
- Virus neutralization titer of 1:20 or more

The virus neutralization titer is felt to be the most accurate representation of protection.

There is a great deal of controversy regarding whether or not a certain level of antibody can be considered tantamount to protection. Many veterinarians do not feel it is useful to run titers until this issue is resolved (i.e., there is more to protection than an antibody level; there is an entire immune system involved and there is no simple way to assess the entire immune system). Other veterinarians find it cost ineffective to recommend titers prior to vaccination; it costs a great deal more to run the titer than to simply give the vaccination. If the titer is adequate, the worst possible outcome is that the vaccine will be ineffective. Other veterinarians question whether or not it is harmless to annually give vaccinations when there is already adequate immunity. At this time there is no single answer to this issue and we recommend trusting your veterinarian's educated opinions regarding these issues.

Protection after Infection

A puppy that has recovered from a parvovirus infection can be expected to have strong immunity. This has been tested out to 20 months after infection and immunity is believed to be lifelong; because this is unproven, continued vaccination is commonly recommended.

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Distemper (Q&A below)

Most of us have heard of distemper infection for dogs and gather it is bad. The basic vaccine for dogs is "the distemper shot," which vaccinates against distemper, parvovirus and some minor kennel cough agents. Luckily, this is all most people ever hear of distemper. If you are reading this, however, you probably have a dog that is suspected of having this dreaded infection.

The typical distemper suspect is a rescue or pet store dog or puppy, usually with questionable vaccination history or an as yet incomplete vaccination series. The dog or puppy has been housed with other rescue dogs.



Symptoms begin with:

- Gooey eye and nose discharge
- Fever, which often comes and goes unnoticed
- Poor appetite
- Coughing and development of pneumonia

The virus is attacking interfaces of the body with the environment (the mucous membranes) and starts with the respiratory tract, hence the pneumonia, but it does not stop there. The virus moves on to produce:

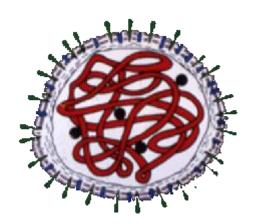
- Vomiting and diarrhea
- Callusing of the nose and foot pads (hence one of the old names for distemper hard pad disease).

After completing what is called the mucosal phase of infection where environmental interfaces are attacked (as described by the above GI and respiratory disease), the virus proceeds to the central nervous system for its neurologic phase leading to:

- Seizures, classically starting with snapping or tremors of the jaws that progress to convulsions of the whole body. This distemper classic sign is called a chewing gum fit.
- Seizures are not the only distemper sign by any means. Tremors, imbalance, and limb weakness all may occur. Signs may progress to death or may become non-progressive and permanent. Recovery is also possible.

This means that the dog appears to recover only to break with neurologic disease 1 to 3 weeks later. Younger puppies or individuals with weak immunity often die during the mucosal phase while stronger individuals may have relatively mild mucosal signs and not appear ill until the neurologic phase strikes.

The Virus Itself



The canine distemper virus is closely related to the human measles virus and in years past puppies were immunized for distemper with the vaccine against measles. It has been said that a child in the home of a dog vaccinated with live distemper virus vaccine will become exposed to the virus and immunized against the measles (we do not recommend such experiments at home).

The distemper virus consists of a single strand of RNA, encased in a protein coat that is again encased in a fatty envelope. This sounds esoteric but the fatty envelope makes all the difference in the world. The fatty envelope is easily disrupted in the environment, which makes it impossible for

infectious virus to persist in the environment. Because an intact fatty envelope is required for infection, virus transmission must involve dog to dog contact or at least contact with extremely fresh (less than 30 minutes old at 60 degrees and up to 3 hours old at room temperature) infected body secretions. As with other viruses, living virus happily freezes and can survive for years if kept frozen and protected from light. Routine disinfection and cleaning readily kills the distemper virus in a kennel setting.

Transmission and Infection

The infected dog typically infects other dogs via coughing infected respiratory secretions though the virus is shed in most other body secretions, including urine. The virus enters the new host via the nose or mouth and promptly begins to replicate. The virus is engulfed by cells of the immune system called macrophages. The idea is that the virus will be engulfed, walled off within the cell, and then destroyed by enzymes. Unfortunately for the new host, this process does not damage the virus as intended; instead, the virus is able to use the macrophage as a means of transportation through the host's body. Within 24 hours, the virus has traveled to the lymph nodes of the lung. By the 6th day, the virus has migrated to the spleen, stomach, small intestine, and liver. Fever is developing at this point.

By day 8 or 9 an important crux is reached in the timetable of infection. The host is mounting an immune response during this time and the outcome depends on how fast and how well this is accomplished. A strong immune response begins to clear the virus at this point and has eliminated all traces of virus with no symptoms of illness by Day 14. A weak immune response allows the virus to reach the epithelial cells, the cells that line every interface the body has with the outside world. The tender epithelial cells lining the chambers of the brain are infected as well. The host begins to get sick as the virus spreads, but as the host's immune response grows symptoms wane. This phenomenon accounts for the wide variability in symptoms; some dogs get only a few mild symptoms while others get a full lethal combination.

After clearing from most internal organs, the virus is able to hide out for long periods of time in the nervous system and skin. Because of this phenomenon, callusing of skin or - much worse - seizures may occur long after the infection was thought to be cleared.

Most cases in the U.S. involve puppies. The colostrum suckled in the first day or so of life will provide them with a solid reflection of their mother's immunity. This immunity will have waned by age 16 weeks, leaving the puppy vulnerable if vaccines have not been administered for further protection. In our society most mother dogs will have received some form of vaccination and thus be able to pass on at least some immunity and will have some ability to protect herself. In societies where vaccination is not common, distemper attacks

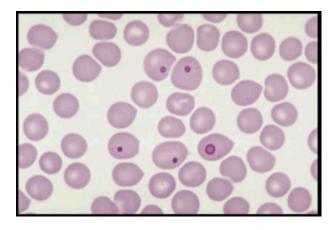
dogs of all ages.

Confirming the Distemper Infection

As if it is not bad enough that this infection has a poorly defined endpoint so one never knows for sure if the dog is out of the woods, it is almost impossible to confirm a distemper diagnosis. Because of this, distemper is a clinical diagnosis, which means that rather than confirming infection with a test that is negative or positive, the veterinarian must look at the whole picture: what symptoms are there, is the history typical, etc. The virus itself remains elusive so that positive test results are meaningful in confirming the infection, but negative results do not rule it out. The following are tests that can be used:

Distemper Inclusion Bodies

Distemper inclusion bodies are clumps of virus that are visible under the microscope within infected cells. Post-mortem inclusion bodies are readily visible in the urinary bladder tissue, thus making confirmation of distemper after death relatively easy. In the living patient, we typically have ready access to blood cells and cells of the eye's conjunctival membranes (the pink park of the eye socket). To enhance the visibility of inclusion bodies, immunocytology is used. In this technique, antibodies against distemper virus are tagged with fluorescent markers. The antibodies bind to virus, if it is present, effectively dying the inclusion body with glow-in-the-dark fluorescent color. The presence of inclusion bodies confirms distemper infection. The lack of detectable inclusion bodies does not rule out distemper infection as inclusion bodies ultimately become coated with the host's own antibodies, which in turn block the fluorescent-tagged antibodies used in the test.



If callusing of the footpads or nose is evident, a biopsy of this tissue can be tested for inclusion bodies fairly late in infection.

Distemper Antibody Levels

Distemper titers (another word for antibody level) of either the IgM type (produced in early stages of infection) and the IgG type (produced in later phases of infection) can be checked. The problem is that distemper vaccination induces these same antibodies, and often distemper suspects have recently been vaccinated. A high IgM titer indicates recent infection or recent vaccination, but there is no way to tell which.

PCR Testing

PCR testing involves amplification of DNA so as to allow detection of very small amounts of virus. Since the distemper virus is an RNA virus, not a DNA virus, a test called reverse transcriptase PCR must be used but the amplification concept is the same. Vaccination will interfere with PCR testing for approximately 2 weeks (i.e. the modified virus from the vaccine will be detected creating a false positive).

Cerebrospinal Fluid Antibody Levels

In neurologic distemper cases, cerebrospinal fluid is often tapped and distemper antibody levels checked. Distemper antibodies in cerebrospinal fluid are highly indicative of distemper infection as vaccine-induced antibodies do not cross the blood-brain barrier into the CSF fluid.

Treatment for Distemper

Many bizarre protocols have emerged over time as we grope for meaningful anti-viral therapy. The fact remains that recovery from distemper is all about immunity and the only real treatment is supportive care while the patient mounts an immune response. If the patient has pneumonia, antibiotics are used on the secondary bacterial infections. Airway dilators are used as needed. Physical therapy is used to promote coughing. If the patient has diarrhea, intravenous fluids are used to prevent dehydration.

Distemper is extremely variable in its ability to produce symptoms and recovery occurs at any stage. This has led to assorted therapies being credited with effect when what was more likely witnessed was the natural removal of the infection by the host's immune system.

Neurologic distemper is particularly difficult to treat. Still, it is possible for dogs to recover with livable deficits even from neurodistemper; euthanasia is best left for progressive, incapacitating neurologic symptoms.

Preventing Infection

If confirming diagnosis and therapy are the pitfalls of distemper, prevention is the easy part. Effective distemper vaccination has been available since the 1950s. Prior to widespread vaccination, distemper was the scourge of the canine community, wiping out entire towns of pet dogs. Today, distemper is a rare disease except in the shelter, rescue, and pet store world.

The "distemper shot" is the basic immunization for dogs. It is generally combined with vaccine for canine parvovirus as well for parainfluenza, adenovirus 2, leptospirosis, and sometimes coronavirus. Puppies are vaccinated beginning at age 6 to 8 weeks, and then every 2 to 4 weeks thereafter until age 16 weeks. The next vaccine is one year later. After that subsequent vaccination boosters are given every 1 to 3 years or based on antibody levels depending on the policy of the supervising animal hospital.

Vaccine is available in the traditional modified live virus format, where distemper virus is modified to induce immune-response but not illness. Vaccine is also available in the recombinant format where a live harmless virus (not the distemper virus at all modified or otherwise) is used to carry the portion of the distemper virus that generates the immune-response. The benefit of the recombinant format is that it is completely impossible for distemper or distemper encephalitis to occur as the result of vaccination. These complications are exceedingly rare but still possible with modified live virus vaccine.

The use of the human measles virus to vaccinate against canine distemper is largely passé nowadays. Immunity obtained this way does not last as long and is not as successful as that obtained with a modified live or recombinant distemper vaccine.

Q&A FAQ

It seems that here we have a disease for which a lethal outcome is possible yet there is no test to confirm the infection and no way to determine if the infection is truly over. The owner of a distemper suspect will have numerous questions and here we attempt to answer questions that may not have been overtly covered in the above text.

Q. I own a young dog with badly stained and pitted teeth. I was told this might indicate she had distemper as a puppy. How would distemper have caused this?

A.



The distemper virus attacks epithelial cells. These cells line the interfaces with the environment, including the mouth. In puppyhood the buds of the permanent teeth are still developing from epithelial cells. The distemper infection and associated fevers can leave these tooth buds permanently damaged so that the adult teeth come in with stained and pitted enamel. This is called enamel hypoplasia.

Q. Is there any way to predict whether a dog with distemper will progress to the neurologic phase?

A. Not really. The phase of infection that precedes the virus entering the central nervous system is the phase where the skin is attacked. Callusing of the

nose and foot pads tends to be associated with the development of neurologic distemper. As a general rule about 50% of dogs that recover from the mucosal phase will progress to neurodistemper. Of the dogs that develop neurodistemper, it has been said that about 50% will do so within a month or two of the mucosal phase. Our impression is that 50% is probably a bit of an over-estimation. The risk of progressing to neurodistemper is less in adult dogs as they tend to mount more effective immune responses than puppies do.

Q. We owned a dog that died and was suspected of having distemper. How should we disinfect our home before a new dog is introduced?

A. One of the few positive aspects of distemper is that the virus cannot live without fresh secretions; it is inactivated in minutes outside the living host's body. Minimal disinfection is necessary.

How long is a recovered dog contagious?

A recovered dog may shed virus up to 2 to 3 months. It is important to keep this in mind when taking a recovered pet anywhere where there are other dogs. The most intense viral shedding occurs in the first 2 weeks of infection.

Q. What is old dog encephalitis?

A. The condition called old dog encephalitis refers to a chronic brain inflammation that can occur in a dog that had distemper many years before. These brain lesions are identical to those in dogs that progress to a chronic neurologic distemper. For some reason, in some individuals the dog lives nearly all its life as a distemper survivor only to break with neurodistemper in old age.

Q. What is vaccinial distemper?" Can a dog actually get distemper from its vaccine?

A. Vaccinial distemper refers to the development of neurodistemper 10 to 21 days after administration of a modified live distemper vaccine. It is not possible to have this reaction when a recombinant vaccine is used.

Q. Can humans get infected with the canine distemper virus?

A. Humans can get infected with the virus, meaning the virus seems able to replicate in the human body, but no illness results. At one time, multiple sclerosis was thought to be associated with exposure to the canine distemper virus but further research suggests that it is actually the human measles virus (a close relative of canine distemper) that may be the culprit. It does not appear that there is any human hazard in the canine distemper virus.

Q. Should a recovered dog continue to receive distemper vaccinations?

A. Technically, if a dog has recovered from the distemper virus the resulting immunity should be lifelong and vaccination is unnecessary. That said, the "distemper vaccine" is actually a combination vaccine covering up to seven infections, including canine parvovirus. You may not want to skip these other important vaccinations. Further, as discussed, confirmation of distemper infection can be problematic. This begs the question as to whether there is any harm to vaccinating with a product that includes a possibly unnecessary distemper vaccine. In fact, if a dog has recovered from distemper, there will be a rapid antibody response against the distemper vaccine live virus and it will be inactivated just as a natural infection would be. So the answer to this question is that while a recovered dog does not need distemper vaccination, there is no harm in continuing to give the vaccine and reaping the benefit of the additional infections typically covered in the product.

If you have a distemper question, we would love to add it to this FAQ as others may benefit. Feel free to use the Ask a Vet feature on the Veterinary Partner home page.

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Dog Rabies: A Serious and Contagious Disease

Dogs > Vaccines > Dog Rabies: A Serious and Contagious Disease



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Dog Rabies

Dog rabies is one of the most well-known viral diseases affecting dogs. Most communities require a rabies vaccination by law because dog rabies is transmissible to humans and many other animals. There is no treatment for rabies and it is deadly. Transmission to humans is not common in the United States, but people who work closely with animals, especially wildlife, and people who travel

frequently are considered at high risk and urged to get a rabies vaccination. **Dog rabies vaccination** is widely available and usually affordable. Transmission of dog rabies usually occurs through contact with wildlife. Infection occurs when a rabid animal bites a non-rabid animal, or a human. A dog rabies vaccine is essential because your dog could easily cross paths with a rabid raccoon, fox, skunk or other animal.

Rabies Symptoms

Rabies symptoms often occur in a period of stages. Once in the body, the virus spreads to the brain and incubates for 3-8 weeks. In the first or prodromal stage, a rabid dog displays apprehension, change in personality, and fever. This stage last for two or three days. In the second, or furious, phase, a rabid dog becomes irritable and restless. He may appear oversensitive to lights and noises. A rabid dog may become aggressive and attack other animals or people. Finally, a rabid dog suffers disorientation, seizures, and death. Dog rabies symptoms may include an intermediate paralytic stage, which affects the head and throat. A rabid dog in this stage may drool excessively due to his inability to swallow. Labored breathing and a dropped jaw may ensue. The paralytic stage leads to death from respiratory failure.

Rabies Diagnosis and Rabies Vaccine

Rabies diagnosis involves removal of the brain, so it can be inspected under a microscope. Rabies is incurable, but humans who have been exposed may receive preventative post-exposure vaccinations. Luckily dog rabies is easily preventable. **Rabies shots** can be procured from your vet and from many local humane associations. Rabies shots are inexpensive and are even made more available during public vaccination events.

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