Vaccination of the young kitten

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ABSTRACT

This paper draws together those diseases of the cat where vaccination is either being practised in the UK or overseas, or is currently being considered. In the UK, vaccination is routinely carried out for feline panleucopenia, feline viral rhinotracheitis (FVR), and feline calicivirus infection, and in quarantine, cats are also vaccinated against rabies. In the USA and some other countries, vaccination is also carried out against feline leukaemia virus infection, feline Chlamydia psitaci infection and routinely against rabies. Attempts are also being made to develop vaccines for feline infectious peritonitis (FIP), although there are a number of problems associated with the development of a successful FIP vaccine which will be referred to later. Factors important in prevention and control of the three diseases feline panleucopenia, FVR, and feline calicivirus infection for which vaccination in the UK is commonly practised will be discussed. For each disease, some background information on the virus and the epizootiology of the disease is given, which it is hoped will lead to greater understanding of the principles involved in prevention and control. A few points will then be made about vaccination in some of those diseases where vaccines are being developed or where it is carried out in other countries.

FELINE PANLEUCOPENIA

Feline panleucopenia was the first of these diseases shown - some 60 years ago - to be viral in origin. It was not until 1964, however, that the virus was isolated and grown in cell culture (Johnson 1964), and this paved the way for the development of safe and highly effective vaccines. The virus is now known to be a parovirus, an interesting group of viruses which have an affinity and requirement for growth in actively dividing cells. Thus, the virus has a predilection for the fetus and newborn where infection may lead to cerebellar ataxia; in older cats, the virus replicates mainly in lymphoid tissue and bone marrow, leading to panleucopenia, and in crypt epithelium of the intestinal mucosa, leading to enteritis (Csiza and others 1971a,b, Gillespie and Scott 1973, Kahn 1978).

Feline panleucopenia is a highly infectious disease and in cat populations which are predominantly unvaccinated, it will almost certainly be endemic. Although any age may be affected, it primarily occurs in young kittens that succumb when their maternally-derived antibody (MDA) has waned. Not all cases show severe signs of disease; in many animals the disease will be mild or subclinical.

Panleucopenia virus is shed in large quantities in all the secretions and excretions of an infected cat, and is also present in blood. Transmission is thought to be mainly oral or intranasal, with initial replication in the oropharyngeal tissues before spread via the bloodstream to the target tissues. Transplacental transmission may also occur (Kilham and others 1967). As virus is present in blood, mechanical transmission via biting insects such as fleas has been suggested, but this seems unlikely to be of significance.

There are several ways by which the virus persists in the cat population. First, it can persist by continuous horizontal spread from acute cases to susceptible animals although for this to occur, there must be a sufficient number of susceptible animals in the population and sufficient opportunities for contact between them. However, few, if any, cat viruses rely solely for their long term survival on continuous epizootic spread, probably because in evolutionary terms there were not enough cats in close enough contact to ensure virus persistence by this system alone. Virtually all cat viruses have another, 'fail-safe' mechanism of survival - either in persistently infected carrier animals, or perhaps because of an ability to persist in the external environment.

In the case of feline panleucopenia, although carriers exist, they are not thought to be of major epizootiological importance, and it is the ability of the virus to persist in the environment which is important. Feline panleucopenia virus is a...
remarkably stable virus, resistant to heat and to many common disinfectants, and may persist on infected premises for up to a year (Johnson 1966, 1969). Only hypochlorite, formaldehyde, and glutaraldehyde have sufficient virucidal activity against the virus (Scott 1980). Hypochlorite is best used with quaternary ammonium compounds to improve cleaning properties. Formalin is toxic and an irritant and should only be used with care to disinfect empty buildings and equipment (Povey 1976).

**Vaccination**

Vaccination against feline panleucopenia has been extremely successful, where it has been carried out. There is only one serotype of the virus, the virus is highly immunogenic and both natural and vaccine-induced immunity is high and long-lived. Feline panleucopenia virus is very similar to mink enteritis virus, and also canine parvovirus, though minor differences may be seen at the DNA level and by using monoclonal antibodies (Parrish and others 1982, Tratschin and others 1982, Appel and Parrish 1987).

Both modified live and inactivated systemic vaccines are available, and although antibody titres may be slightly lower with inactivated vaccines, both types confer entirely adequate immunity. Modified live vaccines probably induce a more rapid onset of protection, and are more likely to be able to overcome low levels of maternally-derived antibody. Inactivated vaccines have the advantage that they may be safely administered to pregnant queens or to kittens less than four weeks of age: modified live vaccines are contraindicated in this situation since feline panleucopenia virus can cross the placenta and induce cerebellar hypoplasia in kittens.

As there is a high degree of correlation between antibody titres in queens and maternally-derived antibody (MDA) levels in kittens, normographs have been used to predict the optimum age of vaccination for an individual litter (Scott and others 1970). However, not all kittens will suckle to the same extent, and therefore the amount of colostrum-derived antibody in an individual kitten may vary. More important, in feline panleucopenia it has been found that such an approach is not usually necessary. In most breeding colonies, where this might be applied, the balance between the virus and host immunity is in a reasonably steady state, because of a long tradition of vaccination coupled with an absence of clinical disease.

In most kittens born to mothers with moderate titres acquired through vaccination, maternally-derived antibody has declined to non-interfering levels by eight to 12 weeks of age. Thus from 12 weeks of age onwards, for most vaccines one dose of vaccine is usually sufficient. Where MDA is likely to be unusually high (for example, if the queen was known to have had the disease, or was vaccinated with live virus just before or during pregnancy) an extra dose should be given at 16 weeks. On the other hand, if MDA is thought to be inadequate (either because the queen was never vaccinated or exposed to field virus, or because the kittens were deprived of colostrum) and kittens are thought to be at risk, then vaccination may be carried out from the age of six weeks onwards, or exceptionally, four weeks. However, additional doses at two to four week intervals are required, ensuring the last dose is at 12 weeks of age or over.

Antibody titres following modified live vaccination have been shown to persist for at least four years (O'Reilly and Hitchcock 1976), and for over one year following inactivated vaccines (Bittle and others 1970, Davis and others 1970). An initial booster at one year of age is, however, advisable, with one to two year re-vaccination thereafter, particularly in high risk situations or where natural boosting is unlikely to occur.

Although feline panleucopenia has been well controlled by vaccination it should be remembered that after an outbreak of disease, vaccination alone should not be relied on for control. Thorough cleansing of premises with an appropriate disinfectant should also be carried out, as the virus is extremely stable, and this can lead to high levels of challenge virus remaining in the environment.

**FELINE VIRAL RHINOTRACHEITIS AND CALICIVIRUS INFECTION**

FVR virus (feline herpesvirus 1) and feline calicivirus are the two main causes of upper respiratory disease in cats and account for approximately 80 per cent of cases (Kahn and Hoover 1976, Gaskell 1985, Gaskell and Knowles 1989). Feline calicivirus infection is generally milder than FVR and is often associated with mouth ulceration. There is only one serotype of FVR virus, but there are a number of strains of feline calicivirus, of slightly varying antigenicity and pathogenicity. Some isolates of calicivirus have been associated with a febrile 'limping' syndrome, unassociated with any respiratory signs (Studdert and others 1970, Pedersen and others 1983).

Cats are mostly infected by direct oronasal or conjunctival contact with an infected animal. Large amounts of virus are shed in oral, nasal and conjunctival secretions; feline calicivirus is sometimes shed in urine and faeces, though this is not thought to be important in the spread of the disease. Indirect transmission via a contaminated environment, equipment, or personnel may also
occur, particularly within the close confines of a cattery. Both viruses are relatively fragile in the external environment, FVR virus surviving for only a day or so at most, and feline calicivirus for about a week. Both are inactivated by a combination of hypochlorite and quaternary ammonium compound. Aerosol transmission of the viruses is not thought to be of major importance, although sneezed macrodroplets can be carried over a distance of 1 to 2 metres.

As both viruses are relatively fragile in the external environment, their alternative, 'fail-safe' mechanism for survival in the inter-epidemic phase is by means of carriers. FVR virus is a typical alphaherpesvirus, the carrier state being virtually the normal sequel to infection and characterised by periods of latency interspersed with episodes of infectious virus shedding, particularly after a stress (Gaskell and Povey 1973, 1977). In cats such stresses include, for example, a change of housing, going into a boarding cattery, going to stud or a cat show, or corticosteroid treatment. Virus shedding generally starts about a week after the stress, and continues for one to two weeks; animals may also show mild clinical signs of respiratory disease at this time. Kittening and lactation may also precipitate shedding: It is worth noting that when only low levels of maternally derived antibody are present, kittens may be protected against disease but not infection, and may become carriers asymptptomatically (Gaskell and Povey 1982).

The feline calicivirus carrier state is characterised by more-or-less continuous virus shedding, but unlike FVR, carriers may spontaneously recover and suddenly eliminate the virus. In some groups of infected cats, most will still be shedding virus from the oropharynx at 30 days after infection. Generally there is then an exponential decline in the proportion of animals that remain carriers, with approximately 50 per cent eliminating the virus by 75 days after infection, and a small proportion probably continuing to shed for the rest of the animal's life (Wardley and Povey 1977, Gaskell and others 1982). Feline calicivirus carriers may be divided into high, medium and low level shedders, each shedding a fairly constant amount of virus that fluctuates around a mean for that individual cat (Wardley 1976); to detect low level shedders a series of swabs taken over several weeks may be needed, since sometimes the amount of virus shed falls below the level of sensitivity of the test.

Feline calicivirus carriers are very common, despite vaccination. Before vaccines were introduced, in the early '70s, surveys showed that approximately 8 per cent of household pets, 25 per cent of show cats and 40 per cent of colony animals were shedding the virus (Wardley and others 1974). Recently, we have found that approximately 20 per cent of Liverpool University Hospital cases and general practice cases, referred for reasons other than that of respiratory signs were shedding calicivirus (Knowles and others 1989). In the same study we also found that approximately 85 per cent of cats with chronic stomatitis were feline calicivirus carriers, and approximately 80 per cent had antibody to feline immunodeficiency virus. The significance of this in the pathogenesis of the disease is not known and awaits further study.

Vaccination

Vaccination against the two main respiratory viruses of cats has now been practised for a number of years, and where vaccination is carried out, it has been relatively successful in controlling the disease. However, milder forms still sometimes occur and problems can arise, for example, in stray cat refuges, and also in young kittens in catteries as they lose their maternally-derived antibody (MDA). This is because both these respiratory viruses are extremely widespread in the cat population and clinically healthy carriers are common, thus ensuring that there is plenty of exposure.

Another problem with cat respiratory virus vaccines has been that although there is only a single serotype of FVR virus, there is antigenic variation among feline caliciviruses. Most are closely related and strains selected for vaccine use have broad antigenicity (Povey 1974, Kahn and others 1975, Kalunda and others 1975). Nevertheless, some strains occur which are not protected for by current vaccines (Pedersen and others 1983, Knowles 1988), and widespread use of particular vaccines may encourage selection for these. Traditionally both viruses have been isolated in approximately equal frequency from cases of feline respiratory disease, but recently, at the University of Liverpool, we have noted a slightly higher isolation rate than expected of feline calicivirus compared to FVR virus (Knowles and others 1989). This may be explained in part by the antigenic diversity among FVC isolates and the consequent relative efficacy of the two vaccines.

Modified live and inactivated systemic vaccines are available, and a modified live intranasal. The FVR virus component of the current inactivated vaccine available in the UK is a subunit glycoprotein fraction, designed to avoid an apparent hypersensitivity reaction noted with whole virus preparations by that particular manufacturer. In previously healthy, unexposed cats, all types of vaccine induce reasonable protection against disease, though not necessarily against infection, and all may be used with reasonable confidence in routine vaccination programmes.

Most vaccines marketed are modified live
systemic, and in general, these are quite satisfactory. However, they should be administered carefully as there are occasional reports that if they are inadvertently given via the respiratory route (e.g., if a cat licks the injection site or an aerosol is made with the syringe) then respiratory signs can develop (Povey 1977). Thus in completely virus-free colonies an inactivated vaccine might be preferable. Where rapid onset of protection is required (for example, during an outbreak of disease, or in a stray cat home) then the intranasal route is probably indicated. Complete protection against challenge has been shown four days after intranasal vaccination, and partial protection after two days. The disadvantage is that intranasal vaccine may induce mild sneezing and occasionally other signs but this usually resolves in a few days without treatment.

The optimum age for vaccination of young kittens can be difficult to determine. In general, kittens should be vaccinated initially with systemic vaccines at nine weeks of age, when in most cases MDA has declined to non-interfering levels, with the second dose three to four weeks later. However, the duration of maternally-derived antibody in individual kittens can be quite variable; for FVR, two to 10 weeks (Edwards and others 1977, Gaskell and Povey 1982), and for feline calicivirus, antibody may last up to 10 to 14 weeks (Johnson and Povey 1983). In addition, little work has been done in relating maternally-derived antibody levels to either protection or interference with vaccination. As this is an age at which apparent vaccine reactions are common, this is an area which should be looked at further.

Because of the variable and sometimes short duration of MDA in kittens, especially in FVR, and because of the high prevalence of carriers in colonies where the disease is endemic, respiratory disease often occurs in young kittens in such colonies before they have been successfully vaccinated. Earlier vaccination schedules can be used in an attempt to reduce this immunity gap, but management measures are often also necessary (such as early-weaning and isolation) to ensure kittens are not already incubating the disease at the time of vaccination, or perhaps are already carriers (Gaskell 1985). Systemic vaccines may be used from three to four weeks of age, vaccinating at three to four week intervals until 12 weeks old.

Although intranasal vaccination should normally be initiated at 12 weeks of age and is not licensed in the UK for earlier use, in some circumstances it can be useful for preventing disease in very young kittens, particularly in colonies where the disease is endemic, because it is not interfered with by maternally-derived antibody. As intranasal vaccine itself can sometimes induce mild clinical signs, vaccination of very young kittens should only be attempted where there is a high and unavoidable risk of exposure. The age at which the vaccine should be given depends to some extent on when clinical signs in young kittens have been occurring; for example, if disease has been occurring at four to five weeks of age, then vaccination should be performed at three weeks of age, and then probably at six and 12 weeks.

In general, immunity in feline viral respiratory disease is not particularly high or long lived. Annual boosters are usually recommended for vaccines, but in some circumstances (for example, a stud cat, or an old cat going for the first time into a boarding cattery) then six-monthly boosting is advisable.

Vaccine reactions and breakdowns

Probably the most common reason why apparent vaccine reactions occur (i.e., respiratory signs which appear within a week or so of vaccination) is because the cat is already incubating the disease at the time of vaccination. This is particularly true in young kittens, which are generally vaccinated just as their MDA wanes. The incubation period of the feline respiratory viruses is generally two to 10 days, but just occasionally, it may be longer, up to two to three weeks. The second point is that the cat might already be a field virus carrier. Both FVR and feline calicivirus carriers are widespread in the population, and both may show persistent or recurrent signs related to damage from the original infection. In addition, FVR carriers may shed and show signs as a result of stress, and it has been suggested that vaccination and the attendant disruption of routine may sometimes initiate this. It is most unlikely that vaccination will 'cure' carriers, though it is just possible that in FVR it may reduce the severity of the shedding episodes.

The modified live systemic vaccines should not induce disease if administered correctly, although, as already discussed, they may still induce signs if they inadvertently reach the respiratory route. Recently, we have noted the apparent localisation to the oropharynx of a live systemic feline calicivirus vaccine when given to previously immune animals; the epizootiological significance of this is unclear (Bennett and others 1989). Clinical signs that may occur following intranasal vaccination have already been discussed. Generally these take the form of mild sneezing some five to nine days later in up to 60 per cent of cats, though more severe signs may sometimes be seen (Orr and others 1980). In general, vaccine reactions attributable to live virus vaccines are uncommon, though it should be remembered that differences in microbial flora and any intercurrent disease, especially with the immunosuppressive cat diseases such as feline leukaemia virus infection or feline immunodefi-
ciency virus infection, may lead to signs that might not otherwise be seen.

Apparent vaccine breakdowns may also be explained in a number of ways. Assuming the vaccine was potent and had been stored and given correctly, even under ideal conditions, protection is not necessarily complete in all animals. In the field, there may be intercurrent disease, or overwhelming infection, or MDA may have interfered with the original vaccination programme. Other respiratory pathogens may be involved, such as Chlamydia psittaci or vaccine resistant strains of feline calcivirus. In colonies, respiratory disease may occur in young kittens as previously discussed, because of the ‘immunity gap’ and because of high risk of exposure from carriers.

It should also be noted that although previously unexposed, systemically vaccinated cats are protected against disease, they are not necessarily protected against infection: such animals can become carriers and be a source of infection to other cats (Orr and others 1978, Gaskell and others 1982). Intranasally vaccinated cats may become carriers of the, albeit attenuated, calcivirus vaccine component, but there is some evidence that intranasal vaccination may protect against the development of the FVR carrier state, at least in the short term (Orr and others 1980).

FELINE CHLAMYDIA PSITTACI INFECTION

Chlamydia psittaci infection in cats is associated with a persistent conjunctivitis and sometimes mild respiratory signs (Wills and Gaskell 1985). It should therefore be considered as a differential diagnosis in the feline respiratory disease complex, especially when the respiratory virus vaccines appear to be unsuccessful, and conjunctivitis is the predominant sign. C. psittaci is also susceptible to some antibiotics, notably oxytetracycline, but unless prolonged therapy for several weeks of all the cats in a household is carried out, re-infection is common.

In some countries, vaccination against C. psittaci infection has been performed for a number of years, although there has been some debate as to its efficacy. However, some newer vaccines seem to induce reasonable protection against disease, though not necessarily against infection (Mittel and Strating 1977, Shewen and others 1980). In one recent study, vaccinated cats, although showing reasonable protection against clinical signs, shed the organism for longer than the unvaccinated controls (Wills and others 1987). This may have been a reflection of the small numbers of animals used, or it may have been a feature of the immune response to this vaccine: in general, such vaccines are probably worth using as an adjunct to control.

FELINE LEUKAEMIA VIRUS INFECTION

Although FeLV has now been known for 25 years, and an enormous amount of research has been carried out over that time, effective vaccination has been difficult to achieve. Killed vaccines appear to be ineffective and although some success has been achieved with live virus vaccines, safety considerations preclude their use. A further complicating factor has been that FeLV contains an immunosuppressive protein p15E which can make vaccinated cats more susceptible to disease after challenge than non-vaccinates (Olsen and others 1977, Mathes and others 1979). In some countries a vaccine against FeLV is marketed which contains viral antigens shed into the cell culture medium from virus-infected tumour cells (Lewis and others 1981, Lewis and others 1988). The vaccine seems to stimulate antibody against the viral proteins gp70 (the major envelope protein), p27 (the core protein), and p15E. The reason why the p15E in the vaccine is not immunosuppressive is not clear, possibly it is in a precursor form. This vaccine has been reported to be about 70 to 80 per cent effective in protecting cats against virus challenge, although a much lower efficacy has recently been reported by others (Pedersen 1987).

However, such a vaccine obviously offers some protection against infection and disease, and as such, it may be worth using in previously unexposed cats that may be at risk or in colonies where a test and eradication programme has been carried out. The vaccine does not, of course, cure persistently infected cats and kittens born to persistently infected queens will already have become infected before vaccination can be carried out. In virus-free colonies it is also important to realise that while incoming cats may be vaccinated, they are not necessarily free from infection.

This currently available vaccine is still a first generation vaccine and it is likely that second generation vaccines using recombinant DNA technology or subunit vaccines using improved adjuvants such as ISCOMS (Osterhaus and others 1985) will become available in the future.

FELINE INFECTIOUS PERITONITIS

Feline infectious peritonitis is a difficult disease with respect to developing a vaccine. The main reason is that it appears to be immunemediated in that pre-existing antibody actually appears to enhance the disease following challenge (Pedersen and Boyle 1980, Weiss and Scott 1981). Initial attempts to develop a vaccine confirmed that, except with low doses, vaccination
rendered cats more susceptible to disease (Pedersen and Black 1983). Recent work has explored the possibility of using recombinant viruses carrying specific FIP genes which it is hoped may be important in eliciting a protective rather than an aberrant immune response, but no real success has so far been reported (Spaan and others 1989).

Recently, a temperature sensitive mutant attenuated strain of FIP virus has been described in North America, which appears to induce reasonable protection and no immune enhancement following challenge (Gerber and others 1989). However, since the pathogenesis and epizootiology of FIP remains unclear, such a vaccine will require extensive testing to ensure its stability and safety, especially when interacting with a number of possible feline coronaviruses in the field.

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ABSTRACT

Horner's syndrome in dogs and cats

CAUSES of Horner's syndrome included trauma of the head, neck and chest, chronic otitis, cranial thoracic mass and injury. Specific causes included falls from high-rise flats and iatrogenic induction after cleaning the external ear. Twenty-seven animals were affected on the left side and 22 on the right. A specific cause was found in all 16 cats but in only 15 out of 33 dogs. Complete reversal of signs occurred in 36 animals, nine were lost to follow-up and one cat with mediastinal lymphosarcoma was euthanased. Signs persisted in three dogs. Mean recovery for both species was 7-7 weeks. Results of pharmacological testing and various treatments did not affect the outcome.

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