Chlamydial Infection in Animals: A Review

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SUMMARY

A review of the literature concerning chlamydial infection in birds and animals, particularly domestic animals is presented. Following a general discussion of the agent, the nature of chlamydial infection and diagnostic criteria, information regarding disease is summarized for each species. The possibility of zoonotic transmission is also discussed.

RÉSUMÉ

Une revue des chlamydioses animales
L'auteur présente une revue de la littérature concernant les chlamydioses aviaires et animales, en insistant surtout sur celles des animaux domestiques. Après une discussion générale, relative aux chlamydies, aux chlamydioses et aux critères qui permettent de diagnostiquer ces infections, elle résume, pour chacune des espèces, les renseignements concernant la maladie. Elle commente aussi la possibilité de la transmission de ces infections, des animaux à l'homme.

INTRODUCTION

Chlamydial organisms are members of an exclusive order, Chlamydiales and family Chlamydiaceae. There is only one genus, Chlamydia, in which two species are recognized: *C. trachomatis* and *C. psittaci* (169). Chlamydia resemble bacteria in the composition of the cell wall, in the possession of both RNA and DNA and in multiplication by binary fission. However, unlike bacteria, this replication occurs only within host cells. In this respect, like viruses, the organisms are obligate intracellular parasites. In addition propagation extracellularly has never been achieved, living cells — such as tissue culture, embryonated eggs or laboratory animals are necessary for growth of the organism (70).

Chlamydia trachomatis, the type species for Chlamydia, is recognized by the formation of compact intracytoplasmic microcolonies which stain with iodine compounds. Growth in embryonated eggs is inhibited by sodium sulfadiazine. Principally a parasite of man, it is associated with ocular and urogenital infections and neonatal pneumonia (5,138). One strain is also recognized as the cause of an endemic pneumonitis of mice (169).

Chlamydia psittaci is identified by the formation of dispersed microcolonies, which do not stain with iodine and resist inhibition by sodium sulfadiazine. It can infect most domestic animals, many wild mammals and more than 100 species of wild and domestic birds are also susceptible. In some cases man may be infected by contact with diseased animals (169).

Chlamydial Disease in Animals

Chlamydia psittaci is responsible for a variety of disease syndromes in animals. The organism does not appear to be very host or tissue specific (54, 109, 120). Many strains produce generalized infections in several host species; others localize and cause pronounced inflammation in one or more tissue or organs of a specific host. Although these strains have yet to be adequately characterized, antigenic and pathogenic differences have been demonstrated (94, 109, 156) and definite strain idiosyncracies are recognized (55, 76).

Exceptionally, some animals may experience severe or even fatal disease as a result of chlamydial exposure. A well balanced host-parasite relationship represents the common nature of chlamydial infection. This long-lasting inapparent or "latent" state has been documented in several species: birds (95), cattle (188), guinea pigs (99), sheep (162) and man (60). In some cases chlamydiae are excreted by the latently infected host; in other cases the organisms would be maintained in a noninfectious form. Under circumstances of stress, "carrier" animals may shed organisms in large numbers or may in fact lapse into clinical disease (55, 160).

The intestinal tract is the natural habitat for chlamydiae (141, 160). Inapparent enteric infections are common in ruminants and most avian species (106, 181, 182). Fecal shedding may be the most important mode of transmission; the disease syndromes observed reflect the route of infection (29) and the immune competence of the host animal (141).

Chlamydiae multiply in cells of the reticuloendothelial system, in epithelial cells of the conjunctiva or genital and intestinal tracts, in synoviocytes and in cells if the placenta and fetus (141). Lesions produced depend on the virulence of the causative strain. The syndromes produced may vary from localized conjunctivitis, pneumonia, enteritis, encephalitis, polyarthritis or abortion to generalized disease. Evidently only one of these potential clinical manifestations will be overt in a given animal under any particular set of environmental or physiological conditions (155).

Diagnosis of Chlamydial Infection

Definitive diagnosis of chlamydial infection depends on the isolation and identification of chlamydial agents from discharges, blood, feces, or postmortem tissues of diseased animals. A presumptive diagnosis may be made on the basis of clinical signs and the recognition of elementary bodies or cytoplasmic inclusions in Giemsa stained impression smears, scrapings or exudates.

The isolation of chlamydiae is carried out in embryonated hens' eggs, in mice or in guinea pigs. The yolk sac is usually preferred for culture because it supports the growth of all known chlamydial strains. Criteria used for isolation include the demonstration of elementary bodies and the group antigen in the indicator system. Concurrent infection with bacteria, mycoplasmas or viruses should be ruled out.

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In general, cases become agent negative before they become serologically negative. For this reason, serology plays an important role in the diagnosis of latent or chronic infection, as well as in the confirmation of ongoing disease. The most efficient serological method of diagnosis in most mammals, including man, is the complement fixation test (CF test). This test is group specific and therefore, utilizes antigen that can be prepared from virtually any chlamydial strain. Except in prevalence studies it is usually necessary to test paired sera to establish a diagnosis of current infection. However, the CF test is relatively insensitive in some infections, especially localized conjunctival or venereal diseases, with at best positive results in only 50% of proven cases. The newly developed microimmunofluorescence test appears to be more sensitive than the CF test in such cases in humans. In addition several serotypes of chlamydia may be identified by using specific antigens or serums. This newer method offers considerable, yet unexplored, potential in the diagnosis of chlamydial disease in animals (70, 80, 141, 160).

AVIAN CHLAMYDIOSIS

Chlamydial infection is recognized worldwide in at least 130 species of birds (92, 93). Originally chlamydiosis, observed in man and psittacine birds was called psittacosis (98). The term ornithosis was introduced to differentiate the disease in wild and domestic fowl from that in psittacine birds (91). This distinction is artificial, since except for epizootiological variations, the diseases are essentially the same. *Chlamydia psittaci* isolated from either psittacine or nonpsittacine birds can be used interchangeably to reproduce an identical disease in birds of either group (109).

Avian chlamydiosis is principally a disease of colonially nesting wild birds. Latent inapparent infection is the predominant state. Overt disease is induced by stress such as poor nutrition, overcrowding, bacterial or protozoal disease, shipping, racing or migration (43, 46). Infective chlamydia are shed in lacrimal and nasal secretions, but mostly in feces where they remain viable for several months. Transmission occurs primarily through aerosols of fecal dust. Transmission between wild birds and domestic fowl has been demonstrated (57, 65, 171). Respiratory exposure is probably the most common route of infection, oral infection being second (108). Attempts to demonstrate egg transmission in pigeons and turkeys have failed (8, 27). However, this has been shown to occur in chickens (27), ducks (134), seagulls (65) and psittacines (96). The epizootiological significance of this is unknown since most infected eggs fail to hatch. Turkey feather mites and chicken lice have also been shown to harbour infective organisms (31). Important factors in maintaining chlamydial infection are those which permit contact of susceptible birds with infected excreta. Large flocks of wild pigeons in urban areas, trade in psittacine birds, racing programs with pigeons and migrations of wild birds such as waterfowl, seagulls, finches and sparrows all contribute to the dissemination of chlamydia throughout the avian population.

The salient features of chlamydiosis in different species of birds have been discussed in several texts (11, 117, 160). Clinical disease is typical of many systemic avian infections: salmonellosis, mycoplasmosis, pasteurellosis, aspergillosis and influenza. Postmortem lesions — serositis with yellowish-grey exudate, hepatomegaly, splenomegaly and bronchopneumonia are also not specific enough for positive diagnosis (160). It is necessary to demonstrate elementary bodies in impression smears of infected organs and preferably isolate chlamydia in embryo-nated eggs or mice to confirm chlamydiosis (44, 92, 110). Serological examination (indirect complement fixation test) of a flock can confirm suspected infection with avian chlamydia or confirm an isolation made from a sporadic case detected at necropsy. In flocks where the disease is enzootic, morbidity and mortality are highest among the young, but losses do not exceed 10-20%. In contrast, the infection in a disease-free flock causes morality as high as 90% in birds of all ages.

Chlortetracycline is the drug of choice for treatment to eliminate clinical disease and fecal shedding. Infected birds should receive 0.5% chlortetracycline in cooked mash or pelletted feed continuously for at least 45 days (4). However, the virulence of the strain causing an epornitic determines the success of treatment (8).

CHLAMYDIAL INFECTION IN DOMESTIC RUMINANTS

Intestinal Infection

Chlamydial organisms have been isolated in the feces of normal cattle (158, 181, 189), sheep (152, 153, 182) and goats (105). Up to 60% of the animals in a particular herd may shed organisms for several years, in levels that vary from minimally detectable to 10^4-10^6 infectious units per gram of feces (162). The epidemiological significance of this is undetermined. Chlamydiae isolated from fecal material are capable of producing pneumonia after intratracheal inoculation (29, 123) and abortion after parenteral infection (153). Schachter and associates (142, 143) have demonstrated two distinct chlamydial serotypes in sheep and cattle: type 1 isolated from abortion, pneumonia or enteric infection and type 2 associated with polyarthritis, encephalitis or conjunctivitis. These two groups do not crossreact with each other or with avian isolates. However, within each type, isolates from cattle and sheep are antigenically alike. Therefore, inapparent intestinal infection may play an important role in the maintenance and pathogenesis of ruminant pneumonia and abortion but be less significant in other chlamydial diseases.

Although adult animals rarely develop enteric disease as a result of intestinal infection, chlamydial enteritis has been reported in calves under ten days of age (106, 162, 188). This shift in host-parasite balance in favor of the organism may be secondary to deficient local immunity in the gut. The disease has been reproduced experimentally in colostrum deprived calves (166, 168). Even so, the role of chlamydiae in the neonatal calf diarrhea complex is undetermined. The syndrome produced experimentally is clinically similar to that attributed to bacteria or viruses. Mucoid, watery and bloody diarrhea have been observed.

Catarrhal enteritis and occasionally fibrinous peritonitis are observed at postmortem (2). Histological lesions — dilatation of crypts, epithelial slough and leukocytic infiltration are nonspecific. Chlamydial inclusions can be demonstrated by fluorescent antibody staining in the cytoplasm of the mucosal epithelial cells of the ileum and jejunum of infected calves (34).

Correction of acid-base imbalance

and dehydration is essential in treatment. Specific antibiotic therapy with chlortetracycline is indicated whenever chlamydiae are recovered from feces, in spite of the high incidence of isolates unassociated with disease and even in the face of significant *Escherichia coli* isolation (160, 168).

Respiratory Infection

Chlamydial agents have been isolated from cases of clinical pneumonia and have experimentally reproduced this disease in sheep (29, 84), goats (66, 102) and cattle (115, 184).

Ovine pneumonia is observed in feeder lambs soon after stress, often in association with shipping. It is characterized by fever, dyspnea and anorexia (151). More dramatic signs, such as heavy mucopurulent nasal discharge, frothing and death, occur as a result of secondary bacterial invasion in which *Corynebacterium pyogenes, Pasteurella multocida, Pasteurella hemolytica* and mycoplasma are frequently implicated (13, 29). Goats affected with chlamydial pneumonia have symptoms similar to sheep (60).

Chlamydial pneumonia in calves may be subclinical, detected incidentally at slaughter, or severe with prostration, marked dyspnea and diarrhea. Most cases are of moderate severity characterized by fever, mucoid or mucopurulent nasal discharge, coughing and depression (106, 115, 160). Chlamydia have been implicated in the shipping fever syndrome. However, the concurrent presence of bacteria, reovirus or parainfluenza virus is necessary to experimentally reproduce the disease (45, 118, 177).

Infection occurs by inhalation of infected nasal discharges or fecal dust. Exudative bronchopneumonia is typical with interstitial leukocytic inflitration (71). Histological changes may be apparent only in acute or peracute cases (184).

When assessing the significance of chlamydial respiratory infection, it should be remembered that although chlamydiae have been incriminated as the cause of enzootic pneumonia in ruminants, the presence of other agents appears to be essential for the development of severe disease. It is possible that chlamydial infection conditions the lung to attack by bacteria common in pneumonia (13). In this respect chlamydia might play a primary role in pathogenesis, even though by themselves they induce relatively mild signs (160).

Ocular Infection

Chlamydial agents have been associated with keratoconjunctivitis (28) and follicular conjunctivitis (22, 165) in sheep. The frequent occurrence of these conditions in conjunction with polyarthritis or pneumonia may represent a manifestation of systemic infection or concurrent local infection, probably by aerosol (63, 79).

Chlamydiae have been isolated from cattle with keratoconjunctivitis (30). These organisms reproduced acute lesions after subconjunctival inoculation of experimental animals. Conjunctivitis has also been reported in cattle afflicted with pneumonia and polyarthritis (160).

Polyarthritis

Chlamydemia following oral infection in calves and sheep may result in subsequent localization of organisms in joints (36). These organisms are antigenically distinct from those normally isolated in ruminant feces, but may produce transient diarrhea where fecal shedding occurs early in the course of the disease (80, 142, 143).

Polyarthritis has been observed in both feedlot lambs and those on ranges primarily in the midwestern United States. Morbidity is high (80%), especially in larger lambs, but mortality is less than 1% (26). The disease is characterized by fever, stiffness, lameness, anorexia and frequently conjunctivitis. Enlargement of joints is not usually apparent except in longstanding cases (157). Most lambs will "warm out" of their stiffness with forced exercise (147). Tetracycline, 150-200 mg/day, given prophylactically to feedlot lambs will decrease the incidence of disease, but will not prevent it (121).

In contrast, mortality is extremely high in young calves affected with chlamydial polyarthritis, but the disease tends to be sporadic (154). Affected calves are born weak, become reluctant to move and develop stiff gait. Some have fever and mild diarrhea. Joints, grossly enlarged, are painful on palpation. Navel swelling does not occur. Death follows two to ten days after the appearance of signs (163, 168).

Chlamydial inclusions may be found in Giemsa stained smears of synovial cells. Isolation of chlamydial organisms may be made from joint fluid or plasma in early cases (71, 80, 147, 157, 164).

Encephalitis

Sporadic bovine encephalomyelitis (SBE) was first recognized in Iowa in 1940 (90). Subsequent reports established its worldwide distribution (9, 43, 104). The disease tends to be enzootic in a herd; outbreaks occur when new animals are introduced, severe cases occur sporadically.

Initial depression and inactivity progress to anorexia and fever. Excessive salivation, dyspnea, nasal discharge and mild diarrhea are characteristic. Some animals may recover at this stage but most develop nervous signs. Stiff gait, circling and staggering are common. Progressive weakness leads to paralysis and occasionally opisthotonus. Death occurs in 60% of cases, ten to 14 days after the onset of signs. Survivors are slow to recover and suffer marked loss of condition (16, 61, 81, 160).

Sporadic bovine encephalomyelitis results from generalized chlamydial infection (79, 104, 106,). Lesions in various organs are secondary to inflammation of the vascular endothelium and mesenchymal tissue. Serositis involving all three body cavities is usual. Hyperemia and edema, evenly distributed throughout the brain, lead to the nervous signs which dominate the clinical disease (16, 79, 160). Definitive confirmation of SBE can only be achieved by isolation of chlamydia from the brain and occasionally, spleen or liver (80).

Chlamydial encephalitis in sheep has not been reported as a separate entity, but does occur rarely in conjunction with polyarthritis (79).

Intrauterine Infection and Abortion

The ability to establish placental and fetal infection is a property of many chlamydial strains, in several animal species, regardless of the type of placentation (160). An episode of chlamydemia is necessary for infection of the placenta and fetus. In experimental infections this occurs during a secondary blood infectious phase following replication of the organisms in somatic organs. The uterine infection progresses independently; organisms appear to be eliminated from other organs soon after multiplication (12, 128, 159). The fetus becomes infected

hematogenously, although uterine fluids may contain organisms after rupture of fetal membranes or from fetal excretions. Chlamydemia is observed in the dam subsequent to abortion and organisms may be shed in her feces (114).

Enzootic abortion of ewes was first described in Scotland in 1936 (56). Now recognized in many parts of the world (14, 190) including Canada (23, 112), the syndrome has also been identified in goats (83). Chlamydial infection of pregnant ewes may result in abortion, stillbirth or the delivery of weak lambs. Most abort in the last month of gestation. On initial exposure the incidence of abortion may be as high as one-third of the flock. Once enzootic, approximately 1-5% abort annually. These are usually young ewes although animals of all ages are susceptible (83, 86, 190). A fever occurs one or two days after initial exposure, but there are no overt signs of infection until abortion occurs. Most ewes recover uneventfully; a small proportion retain placentas, a few may die with complicating sequelae. Fertility and subsequent pregnancies are not affected (160, 172).

Epizootic bovine abortion occurs suddenly in a herd. There is no clinical evidence of disease prior to abortion, usually in the seventh to ninth month of gestation. Occasionally infection results in the delivery of dead calves at term or the birth of weak calves which die later. The placenta is commonly retained and milk production drops in dairy cows but overall there is little adverse effect on the dam. Seasonal occurrences observed by some authors appear to reflect breeding practices (78, 159, 161). A sylvatic cycle involving rodents, ticks and fleas has also been suggested (33).

Ascites, hepatopathy and enlarged edematous lymph nodes are characteristic in aborted calves. Petechia occur on the oral mucosa, conjunctiva, subcutis, thymus, salivary glands and lymph nodes. Pathology in fetal lambs is less dramatic. The liver may be congested, with pinpoint grey foci on the cut surface; a small proportion have swollen lymph nodes. Petechiation is not visible on exposed mucosal surfaces but can be found in the subcutis, thymus, salivary glands and occasionally lymph nodes. Granulomatous inflammation (diffuse focal reticuloendothelial hyperplasia) is specific for intrauterine chlamydial infection which apparently induces chronic disease in the fetus (71, 78, 161, 172).

The predominant pathological change in chlamydial abortion of ewes is placentitis. While the fetal surface of the placenta is smooth and edematous, the maternal surface is granular and pinkish-yellow with hyperemia and hemorrhage. Some cotyledons are necrotic. Clay coloured exudate accumulates in intercotyledonary areas. Bovine placentas from early abortions are brown-yellow, edematous and necrotic; later they resemble ovine placentas except that there may be patchy distribution of lesions (77, 101, 159). Some have noted a superficial resemblance to *Brucella abortus* placentas (18). Histological lesions, ulcerative endometritis and placentome necrosis occur differently in ewes and cattle. Chlamydial agents have a predilection for the hilar region of the ovine placentome, but attack the interplacentome regions of the bovine placenta first (71, 77, 101).

Elementary bodies or inclusions may be demonstrated in smears of infected placenta or fetal organs. Fetal brain, lungs, liver, spleen, kidney, stomach contents and lymph nodes as well as pleural and peritoneal fluid should be used in isolation attempts (77, 80, 111, 161). Virus, mycoplasma and bacteria culture should be performed. Samples from ovine abortion may yield *B. abortus* or *Vibrio fetus* as well as chlamydia (40). A diagnostic rise in CF titer can be demonstrated in dams in paired serum samples taken at the time of abortion and two or three weeks later (159, 161, 172).

To control chlamydial abortion all pregnant animals should be segregated during calving and lambing seasons. Infected fetuses and placentas should be carefully disposed of and pens disinfected. Chlortetracycline (2.5-5 g orally or 350 mg IV daily) is effective only if given prior to secondary chlamydemia. Since this cannot be predicted unless the onset of infection is known, its clinical usefulness is questionable (85). Immunization studies have produced variable results. An apparently effective vaccine used in the U.K. is not available in Canada and the U.S. (12, 88, 89, 111).

Although the ovine and bovine diseases appear to flourish independently, the causative agents are antigenically identical (41, 142, 143). In sheep flocks the aborting ewe is considered the source of infection for other ewes at earlier stages of gestation. Organisms are shed in uterine fluids, the aborted fetus, the placenta and feces. The significance of inapparent intestinal infection in aborting dams and others in the flock is unknown. Ongoing intestinal infection does not inhibit superinfection of ewes with abortion agents (153) and fecal isolates will produce abortion if given parenterally (162). It is assumed that transmission in cattle occurs similarly; however, this has not been verified. Genital infection in bulls and rams has been reported but the role of venereal transmission is unexplored.

Other Genital Infections

Chlamydial agents are now considered a major cause of nongonococcal venereal infection in humans (62, 69, 119, 129, 144, 175). Natural and experimental genital infections in bulls and rams have been reported by several authors. Epididymitis, orchitis, seminal vesiculitis and infections of other accessory glands have been identified (7, 87, 133, 167). The agent isolated cannot be differentiated from that of chlamydial abortion (37, 160), in fact, many of the reported cases occurred in herds where abortion was endemic. Experimentally inoculated bulls and rams excrete organisms in semen, but the semen is usually of unsatisfactory quality for fertilization (35, 170). Whether male animals are infected incidentally in a herd where abortion occurs, or whether they are essential in the maintenance and dissemination of the agent remains to be established.

EQUINE CHLAMYDIAL INFECTION

Chlamydia have been implicated in, but not isolated from, cases of ophthalmia in horses (160). Experimental pneumonia has been induced in horses using the agent of goat pneumonitis (135). In one case chlaymdiae were isolated from the blood of young foals affected with bronchopneumonia; however, attempts to reproduce the disease were not reported (124). There are no confirmed reports of chlamydial infection leading to other equine disease syndromes. Although this species appears peculiarly resistant to infection, it is possible that this scarcity of information more accurately reflects the failure to consider chlamydiae in diagnosis.

CHLAMYDIAL INFECTION IN SWINE

Chlamydial agents infect pigs under natural conditions, but their role in the pathogenesis of disease has not been extensively investigated (160). Kolbl (75) detected intestinal chlamydial infection in normal pigs and Wilson and Plummer (183) demonstrated chlamydial antibodies in a high proportion of pigs that did not have specific signs of disease. Chlamydiae have been isolated from cases of keratoconjunctivitis (116), polyserositis (59) and enzootic pneumonia (125, 148). In addition, bronchopneumonia has been produced in pigs infected experimentally with the agents causing chlamydial pneumonia in goats (136) and sheep (113). One group of SPF pigs developed fatal systemic chlamydial disease after being exposed to "normal" conventional piglets (178). Chlamydiae have also been identified in herds experiencing enzootic abortion and orchitis (17, 125, 174) and in association with bacteria in agalactic sows (3).

CANINE CHLAMYDIAL INFECTION

Although little specific work has been done on the nature of chlamydial infections in dogs, chlamydiae have been incriminated in a number of canine diseases. Chlamydial infections do occur as serological surveys have demonstrated chlamydial antibodies in up to 50% of clinically normal dogs (122, 176, 180).

Chlamydiae were isolated from a dog with conjunctivitis (145) and implicated as the cause of superficial ketatitis in German Shepherds (179). An encephalitic syndrome in dogs was attributed to chlamydiae after organisms were isolated in eggs and antibodies were demonstrated in the serum of affected animals (50). Chlamydiae isolated from a dog with a similar syndrome produced cerebral and pulmonary lesions after intracerebral inoculation of mice (25).

Chlamydiae were also identified in a distemper-like systemic disease of dogs (50, 58). Acute, subacute and chronic forms were observed; young dogs were more severely affected. Fever, inappetence, bronchopneumonia, peritonitis, vomiting, diarrhea and skin lesions were seen. The curative effect of tetracycline and the development of rising chlamydial antibody titers were the criteria used to differentiate this syndrome from canine distemper. A similar disease was produced by the experimental intravenous inoculation of dogs with the agent of ovine polyarthritis (82, 191).

Fraser *et al* (42) described the occurrence of pneumonia in a dog that had access to an aviary in which more than 100 budgerigars died from chlamydiosis. Four people who also had contact with these birds developed psittacosis. Chlamydial agents were isolated from the feces of the affected dog and two contact dogs.

Overall there are few reports of chlamydial disease in dogs in spite of the apparent high rate of infection demonstrated in serological surveys. This may reflect a high level of resistance in this species, or once again, the failure of investigators to consider chlamydiae in the examination of illnesses.

FELINE CHLAMYDIAL INFECTION

Chlamydial psittaci was first isolated from cats with pneumonia by Baker, in 1942 (6). More recent investigators have found the organisms most frequently in cases of conjunctivitis (21, 140, 146, 187). These cats may present a unilateral mucoid or mucopurulent conjunctivitis, the opposite eye becoming involved in five to seven days. Chemosis is common; the cornea is not affected. The disease is transmissible by contact cats, probably by aerosol. Affected cats respond rapidly to tetracycline ointment locally, but will clear spontaneously if left untreated. These untreated cats tend to suffer recurring conjunctivitis every ten to 14 days. Latent infection occurs in some recovered cats, leading to enzootic infection in catteries. Kittens born to infected queens may show severe conjunctivitis at the time when their eyelids normally open. Typical chlamydial inclusions can be demonstrated in conjunctival scrapings from these kittens, but attempts to identify chlamydiae in the queens' cervix have been unsuccessful (22). Some of these kittens may fade away and die; necropsy occasionally reveals histological but subclinical pneumonia (71). A syndrome of this type in a litter of abandoned kittens was recently reported (15).

Cats which recover develop resistance to reinfection, but it is fragile (22). Vaccination with egg attenuated organisms tends to reduce the incidence and severity of the disease experimentally and is recommended in high risk situations (74, 97).

Apparent transmission of the feline organism to man has been reported, resulting in follicular conjunctivitis in the owner of several infected cats (140). It has been suggested that the feline organism was originally contracted from hunting birds or mice (160, 173). In this context it is worth noting that Baker first observed feline chlamydial pneumonia on farms where he was studying pneumonia of calves (160).

CHLAMYDIAL INFECTION IN RODENTS, LAGOMORPHS AND OTHER SPECIES

Many colonies and strains of *mice* have been found to be latently infected with chlamydial agents (72). Organisms were isolated after several blind passages, by the intranasal inoculation of lung material from apparently normal mice into normal susceptible mice (52, 100). Chlamydiae were also isolated from mice during continuous intraperitoneal passage of liver and spleen suspensions but the relationship of this organism to the mouse pneumonitis agent was not determined (48). Chlamydial organisms have also been recovered from healthy wild rodents: the white-footed mouse, the dusty-footed wood rat and the neotropical water rat (33, 93). It has been suggested that these may act as reservoirs for chlamydia in areas where epizootics in domestic animals occur sporadically.

Naturally occurring inclusion conjunctivitis has been described in guinea pigs (53, 73, 99). The disease predominates in four to eight week old animals and varies from slight hyperemia to severe conjunctivitis with profuse purulent discharge. Keratitis with pannus may develop. Inclusions can be demonstrated in conjunctival scrapings. While investigating the similarities between this disease (caused by *C. psittaci*) and inclusion conjunctivitis of man (*C. trachomatis*), it was discovered that guinea pigs with primary conjunctivitis were resistant to reinfection of the eyes or urethra. The immunological importance of this finding is presently being investigated (1, 64, 127). Chlamydia were also isolated by Storz (154) from guinea pigs in a herd

undergoing an epizootic with heavy death losses, especially among young stock. The animals experienced depression, incoordination and in some cases diarrhea. Serous peritonitis was found at necropsy; some livers had numerous grey pin-point foci. The disease was reproduced experimentally by intraperitoneal inoculation of guinea pigs with the isolate. It should be noted that this herd was housed on an experimental farm where sheep and cattle with proven chlamydial infections were kept.

In 1965, a chlamydia was identified as the cause of spontaneous abortion in rabbits (114), later chlamydial pneumonia was described in domestic rabbits (139). Uveitis was produced in rabbits after intravenous inoculation of *C. psittaci* (67, 68). The role of intraocular persistence of organisms was considered in the maintenance of latent infection in various species.

A systemic chlamydial infection appeared to be the cause of a cataclysmic die off among snowshoe hares and muskrats in Saskatchewan in 1961 (149, 150). In stricken areas, the entire population of these two animal species died. Chlamydia were isolated from the blood and spleens of dead animals. Livers were congested and had small whitish-yellow necrotic lesions on the surface. Spleens were usually enlarged. Mice, guinea pigs and rabbits died six to eight days after inoculation with chlamydial agents from this epizootic. Seropositive muskrats were identified not only in Saskatchewan, but also in the Yukon, the Northwest Territories and the State of Wisconsin. It was suggested that the relatively high rate of subclinical chlamydial infections in man in northern Canada may be attributed in part to intensive trapping of these species (185, 186). However, this may also be connected with chlamydial infection in fur seals, which in one serological survey was reported in 30% of the population (32).

Both the common and woolly opossum have been shown to develop fatal chlamydial infection. This was characterized by the development of hindlimb paralysis and convulsive seizures prior to death (131, 132).

Chlamydia psittaci has also been isolated from the eyes of free living koalas in Australia (24). There are reports that the disease has occurred since 1887 (173). The degree of conjunctivitis and chemosis may be extreme, leading to impaired vision, diminished food intake and death.

ZOONOSES

The agent of chlamydial infections in mammals and birds, *Chlamydia psittaci*, does not appear to be very host specific. There are numerous reports of chlamydial diseases occurring simultaneously in different species of animals (55, 93, 141, 160).

The transmission of *C. psittaci* from birds to man is universally recognized. Parrots, other psittacine birds, chickens, ducks, turkeys, seagulls, gamebirds and wild and domestic songbirds have all been incriminated (19, 20, 117, 160). Forty to fifty human cases of psittacosis are reported annually in the U.S.A. (19). The disease in man may vary from inapparent infection to severe pneumonitis with septicaemia and death. Most frequently a transient influenza-like syndrome is observed with nausea, fever, vomiting, myalgia, chills, headache and malaise. Trachoma-like follicular conjunctivitis may be the only sign. Infection is usually acquired by inhalation of dust from infected droppings, exudates, down or other contaminated particles.

There are few well documented cases of human chlamydial infection with chlamydia of mammalian orgin. Although this may reflect the infrequent occurrence of such zoonoses, it is also possible that these infections go unrecognized or unreported. Several serological surveys of persons in close contact with chlamydia infected animals suggest that these animals may be a source of human infection (141, 160). One study found chlamydial antibodies in serum samples from sheep herders in the northwestern U.S (47) and chlamydial infections were subsequently identified in sheep in this area (190). A chlamydial agent was isolated from the cerebrospinal fluid of a febrile child exposed to sheep experiencing chlamydial abortion (49). Infectious agents with chlamydial properities were also isolated from an aborted human fetus and the placentas of several aborting women who had contact with sheep, goats or cattle suffering from chlamydial pneumonia or abortion (51, 130). Cattle affected with chlamydial bronchopneumonia have been incriminated in several reports of human infection (38, 126, 137). Accidental laboratory infection with the agent of ovine abortion has also been reported (10, 11).

Infected fur seals, snowshoe hares and muskrats were suggested as the source for high chlamydial antibody titers in Canadian eskimos (93, 150). Experimentally infected muskrats are known to shed organisms in their feces (67). In addition, it is interesting to note that the first human cases in a psittacosis epidemic in Louisiana in 1943, were a husband and wife who had processed animal pelts, chiefly muskrat (102).

Human follicular conjunctivitis occurred as a result of laboratory infection with a chlamydial agent that was most probably of psittacine origin (139). A similar syndrome was reported in the owner of several cats suffering from chlamydial conjunctivitis. The human isolate in this case produced conjunctivitis in experimental kittens (107, 140).

Chlamydia infected birds present a significant health hazard to humans. It is also possible that chlamydia infected mammals present a similar threat. Therefore persons involved in animal care and food processing should practice careful hygiene and sanitation procedures when handling potentially infected animals or carcasses.

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