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Infections with Erysipelothrix rhusiopathiae in poultry flocks

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Severe disease outbreaks of erysipelas in poultry flocks are caused by infection with the bacterium *Erysipelothrix rhusiopathiae*. This disease occurs in a wide range of poultry, including turkey, broiler chicken, hen, goose, and pheasant flocks. Erysipelas usually follows an acute course in birds, in the form of sepsis. Infections with *E. rhusiopathiae* occur through damaged skin or mucous membranes. The symptoms that occur just before death include unsteady locomotion and a lack of coordinated movement. A chronic form of bacterial infection primarily occurs in turkeys that have been vaccinated against erysipelas, and, in addition to affecting the joints, endocarditis can occur. Usually, carcasses are in good condition and correctly muscled, although ecchymosis might be visible in subcutaneous fat and thigh muscles, while internal organs become enlarged, such as the liver, spleen and kidneys. Histopathological tests indicate major vascular changes in the parenchymatous organs. Points of necrosis related to the infarctions of the large vessels are rarely observed, whereas spilled degenerative changes of parenchymatous organs are more likely to be observed. Turkeys are the only species of poultry for which vaccines against erysipelas have been registered; however, this vaccination is also effective for other poultry. *E. rhusiopathiae* is mainly sensitive to penicillin, but also responds to cephalosporins, tetracyclines, quinolines, clindamycin, erythromycin, and piperacillin.

Keywords: bacteria; veterinary; disease; vaccination; turkey; hen; duck

Introduction

Erysipelas is a bacterial disease caused by *Erysipelothrix rhusiopathiae*, which could potentially occur in all vertebrate species. The disease of greatest prevalence and economic importance is swine erysipelas. The acute, sub-acute, and chronic forms of swine erysipelas may occur in sequence or separately. Mortality can range between 0-100%, with death occurring up to 6 days after the first signs of illness. Pigs are the most

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commonly affected species; however, cases have been reported in birds, sheep, fish, and reptiles (Imada *et al.*, 2004; Erikkson *et al.*, 2009; Wang *et al.*, 2010).

Erysipelas in birds is generally an acute, fulminating infection. It is a septicaemic disease; however the chronic form occasionally occurs after acute outbreaks. Erysipelas is of primary importance in turkeys, although outbreaks sometimes occur in other birds species. Outbreaks usually occur suddenly, with a few birds being found dead initially, followed by increasing levels of mortality on subsequent days. Mortality may range from <1% to 50%. (Bailie *et al.*, 1970; Brickford *et al.*, 1978; Boerner *et al.*, 2004; Opriessnig *et al.*, 2005) The regulation of this disease is important because *E. rhusiopathiae* may cause serious economic losses to farmers. Infection due to *E. rhusiopathiae* in humans is occupationally related, and principally occurs as a result of contact with sick animals, their products or wastes, or contaminated soil/bedding. Three forms of human disease have been recognised: (1) a localised cutaneous lesion form, termed 'erysipeloid'; (2) a generalised cutaneous form, and (3) a septicaemic form, often associated with endocarditis. Erysipeloid is the most common form of infection in humans, and while it has been suggested that the incidence of human infection might be declining due to technological advances in the animal industry, infection continues to occur in certain environments (Mutalib *et al.*, 1993; 1995; Veraldi *et al.*, 2009; Wang *et al.*, 2010).

Etiology

Erysipelothrix rhusiopathiae is a gram-positive bacillus that is resistant to environmental factors, and does not produce endospores. This bacterium may occur as a pathogen or commensal organism in many species of wild and domestic mammals, in addition to birds and fish (Bricker and Saif, 1997; Wang *et al.*, 2010). The bacteria was first isolated in 1876 by Koch from the blood of mice with symptoms of septicaemia, hence the original name *Erysipelothrix murisepticemia*. The pathogen was later isolated from a variety of vertebrate species, and was called, among others names, *Erysipelothrix insidiosa* (Trevisan 1885), *Bacillus rhusiopathiae suis* (Kitt 1893), *Bacterium rhusiopathiae* (Migula 1900), *Erysipelothrix porci* (Rosenbach 1909), and *Erysipelothrix erysipeloides* (Rosenbach 1909). In the published literature, 36 different names have been recorded for *Erysipelothrix spp.* and the present name of *Erysipelothrix rhusiopathiae* was accepted in 1966. The name of the bacteria was created from Greek words erysipelas (rose, red skin), trix (hair), rhusius (reddening), and pathus (disease). The comparison with hair originated from a specific image of the bacterium on a slide that presented the rough colonies from chronic form of the disease. On this slide, long filiform cells that resemble hair are visible (Woodbine, 1950). The cell morphology of *E. rhusiopathiae* is variable. The mentioned rough form of the colony (R) was isolated on blood agar from animal tissues with the chronic form of the disease. Colonies of the rough form are about 1-2 mm in size, have irregular edges, and are flat and dry (Bricker and Saif, 1997). Bacterial cells may be arranged in small chains that form long threads. Such cell morphology also occurs in the older colonies of the smooth form. The rough form grows well at a temperature of 37°C and on media with a pH of 7. Smooth colonies (S) are small, with a diameter of 0.3-1.5 mm, and are round with a slightly convex surface. These colonies tend to grow from the acute form of the disease, forming small colonies with a zone of alpha haemolysis. Staining shows that the bacteria are gram-positive, short, slightly bent bacilli. The smooth form grows well at a temperature of 33° C, and on media with a pH of 7.6-8.2 (Wang *et al.*, 2010).

The genus *Erysipelothrix* includes *E. tonsillarum*, however this species is considered non-pathogenic, which has been confirmed with the experimental intracutaneous

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infection of swine that had been previously subjected to immune-suppressive medicines (Harada *et al.*, 2011). In addition, chickens that were inoculated with the strain *E. tonsillarum* did not show any symptoms of disease (Takahashi *et al.*, 1994). There was only one case where *E. tonsillarum* have been isolated from a dog with endocarditis (Takahashi *et al.*, 1993). *Erysipelothrix sp.* strains are classified based on serological properties, which are determined by a heat-stable antigen, namely, the peptydoglycan of the cell wall. Serotypes 1, 2, 4, 5, 6, 8, 9, 11, 12, 15, 16, 17, 19, 21, and N belong to the species *E. rhusiopathiae*, while serotypes 3, 7, 10, 14, 20, and 22 belong to the species *E. tonsillarum*. Due to a slight homology in the DNA of *E. rhusiopathiae* and *E. tonsillarum*, some authors differentiate *Erysipelothrix sp.* as strain 1 (serotype 13) and *Erysipelothrix sp.* as strain 2, (serotype 18) (Bricker and Saif, 1997; Takeshi *et al.*, 1999; Bender *et al.*, 2009). The serotypes recorded in birds to date include 1, 2, 4, 5, 6, 8, 15, 16, and 21. In swine, the serotypes that most frequently cause the disease are serotypes 1, 2, and 21, while in sheep and goats, the most frequent serotypes are 1, 2, 5, 15, and 21 (Takahashi *et al.*, 1992; Imada *et al.*, 2004; Eriksson *et al.*, 2009). Chooromoney *et al.* (1994) analysed the different strains using multilocus enzyme electrophoresis (MEE), and established that serotyping was not related to the epidemiological properties of the strains. The apathogenicity of *E. tonsillarum* probably results from the lack of an ability to produce neuraminidase, which is the enzyme responsible for the decomposition of sialic acid, leading to cell damage. The amount of enzyme that is produced increases at the moment that the bacterium proliferates. Strains of lower virulence tend to produce less of the enzyme (Wang *et al.*, 2005; 2010; Harada *et al.*, 2011). Coagulase and hialuronidase are additional factors of virulence, as these enzymes facilitate the penetration of host cells. It is suspected that different bird species exhibit different levels of sensitivity to *E. rhusiopathiae* infection (Bailie *et al.*, 1970; Dhillon *et al.*, 1980; Bricker and Saif, 1997; Hollifield *et al.*, 2000; Derakhshanfar *et al.*, 2004; Gartrell *et al.*, 2005).

Source of infection

Infections with *E. rhusiopathiae* occur through damaged skin or mucous membranes, and infection *per os* (orally) has been confirmed experimentally. The dosage required to cause death in turkeys ranged from 10^4 to 10^6 cells of the bacterium given subcutaneously or *per os*. Mortality of turkeys occurred at 44-120 hours after exposure to the disease. Symptoms occurred later and mortality was lower when administering the infection *per os* (Bricker and Saif, 1997). The amount of the bacteria in the blood, when the first symptoms of the disease were documented, ranged from 10^7 to 10^8/ml (Brickford *et al.*, 1978).

Broiler chickens infected intramuscularly with a dosage of $6.2 \times 10^8 - 1.3 \times 10^9$ of the bacterium exhibited typical symptoms of the disease, including depression, asthenia, fluffing of the feathers, and the lowering of the wings. The infection resulted in 25% mortality in infected birds (Takahashi *et al.*, 1994). In the case of laying hens, symptoms occurred 2-5 days after initial infection. Many authors suggest that, among different age classes of hens, older birds are significantly more sensitive to infection. In field cases of the disease, the highest mortality occurred in hens during the period of egg laying, with 31% mortality of the flock being recorded at the end of the production cycle and 6.5% mortality being recorded in the 34th week of life. For birds up to the 20th week of life (point of lay), death only occurred occasionally. Mortality is probably related to the high burden of the organism, which leads to a decline in bird immunity. Despite the infective dosage of the pathogen being unknown in field cases, certain authors reported an

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exacerbation in the course of the disease in older birds (Mazaheri *et al.*, 2005; 2006; Brannstrom *et al.*, 2010; Kurian *et al.*, 2012). A clear decrease in age-related sensitivity to infection has not been observed in turkeys (Bricker and Saif, 1997).

Milne *et al.* (1997) suggested that pheasants are the least sensitive bird species to the infection, reporting that *E. rhusiopathiae* only weakly expressed pathological symptoms and internal body changes in pheasants compared to turkeys. Another example of the disease, resembling *E. rhusiopathiae* was observed by Brickford *et al.* (1978) in turkeys. In this instance, *post mortems* showed the presence of numerous ecchymosis in muscles and parenchymatous organs.

Opinions differ with respect to the level of sensitivity of waterfowl to infections with *E. rhusiopathiae*. According to Janowska *et al.* (1978), incidence is higher in ducks compared to geese, which indicates that geese have lower sensitivity to infection with *E. rhusiopathiae*. However, personal observations indicate that the incidence of this disease in geese causes significant economic losses due to high mortality, and it seems that younger geese are more sensitive to infection (Bailie *et al.*, 1970). Dhillon *et al.* (1980) observed a similar percentage of deaths in farmed ducks. The course of the disease probably depends on level of exposure to the infective agents.

The source of infection with *E. rhusiopathiae* depends on the poultry husbandry system. Birds are more exposed to infection when they are in contact with the external environment, such as on domestic or ecological farms, in aviaries or with access to an outdoor run. This is due to the increased possibility of contact with carriers, such as wild fowl, rodents, and insects (Morgan *et al.*, 1994; Mazaheri *et al.*, 2006; Eriksson *et al.*, 2009). Contact with the soil is an important infection vector, as *Erysipelothrix* may survive in soil for several months. The bacterium primarily enters the soil via the faeces of sick animals. Herds of swine and sheep kept in the vicinity are most frequently mentioned as a source of infection (Bailie *et al.*, 1970; Janowska *et al.*, 1978). In addition, fish and badly prepared fishmeal may be a source of *Erysipelothrix* (Panigrahy and Hall, 1977; Boerner *et al.*, 2004). Turkeys and geese are most commonly exposed to this infection, with the exposure of geese being related with access to runs and significant contact with the surrounding environment. The spread of the disease is supported by the occurrence of cannibalism in flocks of turkeys.

Poultry that are housed in closures with a limited possibility of contact with other birds are least exposed to infection. Outbreaks of *E. rhusiopathiae* may occur in laying hens kept in cages in the event of infestation with the mite *Dermansyssus gallinae*. This parasite may carry both viruses and bacteria, including *E. rhusiopathiae*, which has been confirmed by the results from tests of *Erysipelothrix* isolates obtained from hens, in which *D. gallinae* was documented on the same farm. Tests using molecular biology techniques indicated that the isolates are identical (Eriksson *et al.*, 2009). Despite the fact that it is not possible to cause the experimental infection of hens during blood taking by infected parasites, it is likely that *Erysipelothrix* is one of several bacteria that are carried by *D. gallinae* (Brannstrom *et al.*, 2010).

Hen eggs were excluded as a potential source of the infection. It was suspected that in the event of bacteremia, the pathogen might be delivered to the oocyte and the egg. This phenomenon may lead to the higher mortality of embryos, carrier-state, or development of the disease in chicks. Eggs laid and incubated from hens with confirmed *E. rhusiopathiae* exhibited no increase in embryonic mortality, despite the lack of disinfection of shells that could have allowed infection. Furthermore, the hatchling chicks from infected hens did not show any symptoms of the disease. *Erysipelothrix rhusiopathiae* has not been isolated from cultured eggs, dead embryos, or chicks killed on the 10th and 20th day of life (Mazaheri *et al.*, 2006). Hence, it can be concluded that

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the health of the progeny is not directly affected by *E. rhusiopathiae* in infected flocks of parent stock.

Clinical symptoms

Erysipelas usually has an acute course in birds. Despite the fact that the disease does not occur in the form of an epidemic, and is rather subject to repeat endemics in a given area, its outbreak may bring about significant losses. In the case of turkey flocks, the disease tends to occur suddenly, and has the course in the form of sepsis. Symptoms that occur just before death include unsteady walking and a lack of coordinated movement (Janowska *et al.*, 1978; Gunning and Morton, 1988). In general, before the first deaths occur, irregularities in the flock are not visible. Sometimes limited or uncharacteristic symptoms occur, such as the fluffing of the feathers or a decline in feed consumption (Takahashi *et al.*, 1994).

Erysipelothrix rhusiopahiae may cause spots to appear on the skin of birds. Significant dark red crusty patches on the back of laying hens kept in cages have been observed (Mutalib *et al.*, 1993). The snood (head area) of turkeys may appear pale with oedema (swelling), along with dark rough patches on the head (Milne *et al.*, 1997; Derakhshanfar *et al.*, 2004). After experimental subcutaneous infection, the affected area exhibited oedema after several hours, with the skin of the birds becoming blue, with small amounts of serum-fibrinous effusion. Necrosis of the skin and hypoderma was observed (Brickford *et al.*, 1978; Derakhshanfar *et al.*, 2004). In addition, the reddening of the skin of the toes in 2-to-4-day-old turkeys after toe-trimming has been observed. The toe joints were oedematous; however, no effusion was observed in any of the turkeys, although cultures prepared from the surface of the joint indicated the presence of *E. rhusiopahiae* (Hollifield *et al.*, 2000). Arthritis is a characteristic in the chronic form of the disease. The chronic form occurs most frequently in turkeys that have been vaccinated against erysipelas. In addition to changes in the joints, endocarditis may be present (Bricker and Saif, 1997).

Excluding the case of natural infection in hens with *E. rhusiopahiae*, clear symptoms of the disease may not be observed. Problems tend to occur suddenly, with farmers first suspecting that the flock has been poisoned (Mutalib *et al.*, 1993; Milne *et al.,* 1997). However, egg production clearly declines during the production period of flocks. In the case of experimental infection symptoms appear around the third day after inoculation, including a decline in appetite, asthenia, and paleness of the cap occurred. The disease usually lasts about three weeks in hen flocks (Bricker and Saif, 1997; Brannstrom *et al.*, 2010)

Erysipelothrix rhusiopahiae is able to cause cellulitis, which can be an economic cost in terms of exclusion at slaughter, and has been confirmed experimentally in broiler chickens. The generation of clear necrosis changes, with oedema and infiltration of heterophils, can be observed in birds that survive longer than 24 hours from the moment of infection. In birds that die within 24 hours as a result of sepsis in the histological preparation, a slight infiltration of inflammatory cells is normally observed (Derakhshanfar *et al.*, 2004).

Autopsy and anatomical-pathological changes

From the moment that natural cases of erysipelas occurs in birds, symptoms indicate the presence of generalised sepsis (Bricker and Saif, 1997). In all species of birds for which

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cases of erysipelas have been described so far, autopsy indicated similar internal anatomical changes. Most frequently, dead birds were in good condition and correctly muscled, although ecchymosis may be visible in the subcutaneous fat and muscles of the thighs (Mutalib *et al.*, 1993; Gartrell *et al.*, 2005). The internal organs become engorged, swollen, and congested, including the liver, spleen and kidneys. Often, greyish-yellow points of necrosis the size of a pin head occur in the liver (Panigrahy and Hall, 1977; Brickford *et al.*, 1978; Gunning and Morton, 1988; Morgan *et al.*, 1994; Takahashi *et al.*, 1994), and hepatic mottling and sub-capsular ecchymosis may be present (Panigrahy and Hall, 1977; Milne *et al.*, 1997; Gartrell *et al.*, 2005). Ecchymosis is also visible in the pericardial fat and on the cardiac muscle (Brickford *et al.*, 1978; Janowska *et al.*, 1978) and sometimes it is possible to observe fluid in the pericardial sac. Points of necrosis may be found in the spleen (Brickford *et al.*, 1978). The lungs are usually engorged with a little ecchymosis in the parenchyma (Brickford *et al.*, 1978; Boerner *et al.*, 2004).

The intestines become congested, with various levels of inflammation occurring, ranging from catarrh to haemorrhagic inflammation; however, it is not a pathognomic symptom (Panigrahy and Hall, 1977; Brickford *et al.*, 1978; Janowska *et al.*, 1978; Gartrell *et al.*, 2005; Opriessnig *et al.*, 2005). Ecchymosis is usually observed on the surface of the pancreas (Bailie *et al.*, 1970). The kidneys become oedematous, and both may become congested (Takahashi *et al.*, 1994) and pale (Pettit *et al.*, 1976; Brickford *et al.*, 1978). Changes in the reproductive system were not observed (Mutalib *et al.*, 1993; 1995). The chronic form is characterised by oedema of the joints. After the joints have been cut, effusion can be visible (Panigrahy and Hall, 1977; Hollifield *et al.*, 2000).

When conducting histopathological testing of parenchymatous organs, vascular changes predominate. Conglomerates of bacteria surrounded by thrombocytes are visible in the capillaries and smaller arterial and venous vessels. Points of necrosis related to infarctions regarding large vessels are rarely observed, with degenerative changes of parenchymatous organs being more likely to be observed. The destruction of the parenchymatous cells of organs in the acute form of the disease is clearly visible in the liver, spleen, and kidneys.

Atrophy of the lymphatic tissue, often leading to a complete lack of lymphocytes, may be observed in the spleen. In extreme cases, the pulp of the spleen is completely destroyed, with only a vascular layer and hypertrophic reticular cells remaining. Ecchymosis and oedema of the cells are particularly visible in the heart and lungs. Infiltration of the mononuclear cells in the endocardium and pericardial sac usually occurs several days after the onset of infection. Infiltration of inflammatory cells in the synovium is observed in the joints.

Immunisation of birds

The only species of poultry for which vaccines against erysipelas have been registered are turkeys. The most frequently used vaccines are based on bacterial cells that have been inactivated with formalin (Bricker and Saif, 1997). Tests have confirmed that vaccines applied widely in swine husbandry are also effective on turkeys (Adler and Nilson, 1952; Bricker and Saif, 1988). In Poland, research on the effectiveness of the vaccines against erysipelas for turkeys was carried out by Krasnodębska-Depta and Janowska (1980) and Krasnodębska-Depta and Koncicki (1988). These researchers established that a double intravenous administration of both life vaccines and inactivated vaccines gave full protection of turkeys against infection with *Erysipelothrix sp*. In addition, the administration of vaccines for application *per os* obtained a satisfactory level of protection. However, the double administration of the vaccine with drinking water

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resulted in lower level of antibodies compared to injection (Krasnodębska-Depta and Koncicki, 1988). Bricker and Saif (1988) assessed the application of the vaccine against erysipelas administered *per os*, and found that an appropriate level of protection may be obtained only after a double administration of the vaccine has been given at an appropriate dosage. The minimal dosage (administered twice to birds to fully protect them) is 4 x 10^9 bacteria.

The vaccination of bird species for which the vaccine was not registered has produced satisfactory results. One example is emus, which were vaccinated subcutaneously in the fourth and eighth week of life with the dosage recommended for turkeys (0.5 ml/ bird), with the subsequent experimental infection with *E. rhusiopathiae* producing no symptoms of the disease (Swan and Lindsey, 1998). In vaccinated flocks of emu, erysipelas caused by the strain belonging to serotype 21 occurred. Despite the fact that the applied vaccine contained both serotypes 1 and 2, which tend to be isolated from cases of erysipelas in swine, the birds showed full immunity to infection with *E. rhusiopahiae* after infection. The existence of cross-immunity among individual serotypes of *E. rhusiopahiae* has been indicated through observations from mice, swine, and dolphins (Takahashi *et al.*, 1984; Sawada and Takahashi 1987; Redhead *et al.*, 1998; Lacave *et al.*, 2001). Mutalib *et al.* (1993) has suggested the effectiveness of vaccines in flocks of laying hens.

Our own observations in geese indicate the possibility of the safe application of vaccines developed for swine and turkeys in flocks threatened by the occurrence of erysipelas (data not published).

Treatment

It has been assumed that *E. rhusiopahiae* is mainly sensitive to penicillin, but it also responds to cephalosporins, tetracyclines, quinolines, clindamycin, erythromycin, and piperacillin. It is resistant to vancomycin, chloromycetin, gentamycin, polymyxin B, streptomycin, and sulphametaxasol. The selected medicines for treatment include penicillins and cephalosporins (Veraldi *et al.*, 2009), with penicillins being most frequently used in animals.

Conclusions

Erysipelas is likely to occur in a wide range of avian hosts. In most species, the disease occurs as a septicaemia, characterised by ecchymosis on the surface of the heart and pancreas, and internal organs become enlarged, including the liver, spleen, and kidneys. Chronic erysipelas may appear in both urticarial and endocardial forms. The internal anatomical changes can prove useful for diagnosis; however, full diagnosis must be based on microbiological examination of organ samples (liver, heart, and joints). The isolation and identification of *E. rhusiopathiae* is necessary for definitive diagnosis, and the antibiotic of choice for treating *E. rhusiopathiae* is penicillin.

References

ADLER, H. and NILSON, M. (1952) Immunisation of Turkeys against Swine Erysipelas with Several Types of Bacterins. *Canadian Journal of Comparative Medicine and Veterinary Science* **16**: 390-393.

https://doi.org/10.1017/S0043933913000822 Published online by Cambridge University Press

BAILIE, W., BURY, R., BICKNELL, E. and KNUDTSON, W. (1970) *Erysipelothrix* infection in goslings. *Avian Diseases* **14**: 555-556.

BENDER, J., KINYON, J., KARIYAWASAM, S., HALBUR, P. and OPRIESSNIG, T. (2009) Comparison of conventional direct and enrichment culture methods for *Erysipelothrix spp.* from experimentally and naturally infected swine. *Journal of Veterinary Diagnostic Investigation* **21**: 863-868.

BOERNER, L., NEVIS, K., HINCKLEY, L., WEBER, E. and FRASCA, S. (2004) Erysipelothrix septicaemia in a little blue penguin (*Eudyptula minor*). *Journal of Veterinary Diagnostic Investigation* **16**: 145-149.

BRANNSTROM, S., HANSSON, I. and CHIRICO, J. (2010) Experimental study on possible transmission of the bacterium *Erysipelothrix rhusiopathiae* to chickens by the poultry red mite, *Dermanyssus gallinae*. *Experimental Applied Acarology* **50**: 299-307.

BRICKER, J. and SAIF, Y. (1997) Erysipelas, in: CALNEK, B.W. (Ed.) *Diseases of poultry*, pp. 302-312 (Iowa State University Press).

BRICKER, J. and SAIF, Y. (1988) Use of live oral vaccine to immunize Turkeys against erysipelas. *Avian Diseases* **32**: 668-673.

BRICKFORD, A., CORSTVET, R. and ROSENWALD, A. (1978) Pathology of experimental erysipelas in turkeys. *Avian Diseases***22**: 503-518.

CHOOROMONEY, K., HAMPSON, D., EAMENS, G. and TURNER, M. (1994) Analysis of *Erysipelothrix rhusiopathiae* and *Erysipelothrix tonsillarum* by multilocus enzyme electrophoresis. *Journal of Clinical Microbiology* **32**: 371-376.

DERAKHSHANFAR, A., GHANBARPOUR, R. and YAZDANI, S. (2004) A pathologic study on experimental *Erysipelothrix rhusiopathiae* cellulitis in broiler chickens. *Veterinarski Arhiv* **74**: 217-224.

DHILLON, A., WINTERFIELD, R., THACKER, H. and RICHARDSON, J. (1980) Erysipelas in domestic white pekin ducks. *Avian Diseases* **24**: 784-787.

ERIKSSON, H., JANSSON, D., JOHANSSON, K., BAVERUD, V., CHIRICO, J. and ASPAN, A. (2009) Characterisation of *Erysipelothrix rhusiopathiae* isolates from poultry, pigs, emus, the poutry red mite and others animals. *Veterinary Microbiology* **137**: 98-104.

GARTRELL, B., ALLEY, M., MACK, H., DONALD, J., MCINNES, K. and JANSEN, P. (2005) Erysipelas in the critically endangered kakapo (*Strigops habroptilus*). *Avian Pathology* **34**: 383-387.

GUNNING, R. and MORTON, B. (1988) Outbreak of erysipelas in farmer geese. *The Veterinary Record* **20**: 191.

HARADA, K., MURAMATSU, M., SUZUKI, S., TAMURA, Y., SAWADA, T. and TAKAHASHI, T. (2011) Evaluation on the pathogenicity of *Erysipelothrix tonsillarum* for pigs by immunosuppression with cyclophosphamide or dexamethasone. *Research in Veterinary Science* **90**: 20-22.

HOLLIFIELD, J., COOPER, G. and CHARLTON, B. (2000) An outbreak of erysipelas in 2-day old poults. *Avian Diseases* **44**: 721-724.

IMADA, Y., TAKASE, A., KIKUMA, R., IWAMARU, Y., AKACHI, S. and HAYAKAWA, Y. (2004) Serotyping of 800 strains of *Erysipelothrix* isolated from pigs affected with erysipelas and discrimination of attenuated live vaccine strain by genotyping. *Journal of Clinical Microbiology* **42**: 2121-2126.

JANOWSKA, I., KRASNODĘBSKA-DEPTA, A. and BIESZKE, R. (1978) A case of erysipelas in geese. *Medycyna Weterynaryjna* **8**: 471-472.

KRASNODĘBSKA-DEPTA, A. and JANOWSKA, I. (1980) Immunogenic properties of some strains of *Erysipelothrix rhusiopathiae* for turkeys. *Medycyna Weterynaryjna* **6**: 331-333.

KRASNODĘBSKA-DEPTA, A. and KONCICKI, A. (1988) Oral immunisation of Turkey against red fever by means of the Orvac vaccine. *Medycyna Weterynaryjna* **12**: 720-721.

KURIAN, A., NEUMANN, E., HALL, W., MARKS, D. (2012) Serological survey of exposure to *Erysipelothrix rhusiopathiae* in poultry in New Zealand. New Zealand Veterinary Journal **60**: 106-109.

LACAVE, G., COX, E., HERMANS, J., DEVRIESE, L. and GODDEERIS, B. (2001) Induction of cross-protection in mice against dolphin *Erysipelothrix rhusiopathiae* isolates with a swine commercial vaccine. *Veterinary Microbiology* **80**: 247-253.

MAZAHERI, A., LIERZ, M. and HAFEZ, H. (2005) Investigation of the patogenicity of *Erysipelothrix rhusiopathiae* in laying hens. *Avian Diseases* **49**: 574-576.

MAZAHERI, A., PHILIPP, H., BONSACK, H. and VOSS, M. (2006) Investigation of the vertical transmission of *Erysipelothrix rhusiopathiae* in laying hens. *Avian Diseases* **50**: 306-308.

MILNE, E., WINDSOR, R., ROGERSON, F. and PENNYCOT, T. (1997) Concurrent infection with enteric protozoa and *Erysipelothrix rhusiopathiae* in chicken and pheasant flocks. *The Veterinary Record* **141**: 340-341.

MORGAN, M., BRITT, J., COCKRILL, J. and EITEN, M. (1994) Erysipelothrix rhusiopathiae infection in an emu (Dromaius novaehollandiae) *Journal of Veterinary Diagnostic Investigation* **6**: 378-379.

MUTALIB, A., KEIRS, R. and AUSTIN, F. (1995) Erysipelas in quail and suspected erysipeloid in plant employees. *Avian Diseases* **39**: 191-193.

https://doi.org/10.1017/S0043933913000822 Published online by Cambridge University Press

MUTALIB, A., KING, J. and McDONOUGH, P. (1993) Erysipelas in caged laying chickens and suspected erysipeloid in animals caretakers. *Journal of Veterinary Diagnostic Investigation* **5**: 198-201.

OPRIESSNIG, T., VANCE, R. and HALBUR, P. (2005) *Erysipelothrix rhusiopathiae* septicaemia in a Laughing kookaburra (*Dacelo novaeguineae*). *Journal of Veterinary Diagnostic Investigation* **17**: 497-499.

PANIGRAHY, B. and HALL, C. (1977) An outbreak of erysipelas in coturnix quails. *Avian Diseases* **21**: 708-710.

PETTIT, J., GOUGH, A. and TRUSCOTT, R. (1976) *Erysipelothrix rhusiopathiae* infection in chukar partridge (Alectoris gracea).*Journal of Wildlife Diseases* **12**: 254- 255.

REDHEAD, K., PUGH, C., JENSEN, N. and HOUGHTON, S. (1998) Cross protection against *Erysipelothrix rhusiopathiae* serotype 10 induced by a serotype 1 and 2 vaccine. *Veterinary Record* **142**: 612.

SAWADA, T. and TAKAHASI, T. (1987) Cross protection of mice and swine given live-organism vaccine against challenge exposure with strains of *Erysipelothrix rhusiopathiae* representing ten serovars. *American Journal of Veterinary Research* **48**: 81-84.

SWAN, R. and LINDSEY, M. (1998) Treatment and control by vaccination of erysipelas in farmed emus (*Dromaius novohollandiae*) *Australian Veterinary Journal* **76**: 325-327.

TAKAHASHI, T., FUJISAWA, T., TAMURA, Y., SUZUKI, S., MURAMATSU, M., SAWADA, T., BENNO, Y. and MITSUOKA, T. (1992) DNA relatedness among Erysipelothrix rhusiopathiae strains representing all twenty-three serovars and *Erysipelothrix tonsillarum. International Journal of Systematic Bacteriology* **42**: 469-473.

TAKAHASHI, T., TAKAGI, M., SAWADA, T. and SETO, K. (1984) Cross protection In mice immunised with live erysipelas vaccine to challenge exposure with strains of *Erysipelothrix rhusiopathiae* of various serotypes. *American Journal of Veterinary Research* **45**: 2115-2118.

TAKAHASHI, T., TAKAGI, M., YAMAOKA, R., OHISHI, K., NORIMATSU, M., TAMURA, Y. and NAKAMURA, M. (1994) Comparison of the patogenicity for chickens of *Erysipelothrix rhusiopathiae* and *Erysipelothrix tonsillarum*. *Avian Pathology* **23**: 237-245.

TAKAHASHI, T., TAMURA, Y., YOSHIMURA, H., NAGAMINE, N., KIJIMA, M., NAKAMURA, M. and DEVRISE, L. (1993) *Erysipelothrix tonsillarum* isolated from dogs with endocarditis in Belgium. *Research in Veterinary Science* **54**: 264-265.

TAKESHI, K., MAKINO, S. and IKEDA, T. (1999) Direct and rapid detection by PCR of *Erysipelothrix sp.* DNA s prepared from bacterial strains and animal tissues. *Journal of Clinical Microbiology* **37**: 4093-4098.

VERALDI, S., GIRGENTI, V., DASSONI, F. and GIANOTTI, R. (2009) Erysipeloid: a review. *Clinical and Experimental Dermatology* **43**: 859-862.

WANG, Q., CHANG, B. and RILEY, V. (2010) *Erysipelothrix rhusiopathiae*. *Veterinary Microbiology* **140**: 405-417.

WANG, Q., CHANG, J., MEE, J. and RILEY, V. (2005) Neuraminidase production by *Erysipelothrix rhusiopathiae*. *Veterinary Microbiology* **107**: 265-272.

WOODBINE, M. (1950) *Erysipelothrix rhusiopathiae*. Bacteriology and chemotherapy. *Bacteriology Review* **14**: 161-178.

https://doi.org/10.1017/S0043933913000822 Published online by Cambridge University Press

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