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Review article

A review article on avian Erysipelas infection: An occupational disease of one health importance

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Abstract

This review article was designed to spotlight on erysipelas infection of poultry regarding the disease history and nomenclature, the bacterium, virulence factors and pathogenicity, susceptibility, infection and transmission, pathology, human infection, laboratory diagnosis, and the prevention and control. Erysipelas is an acute emerging and occupational disease that affects a wide range of birds especially turkeys. The disease is caused by a bacterium, *Erysipelothrix rhusiopathiae (E. rhusiopathiae)*, which is a ubiquitous pathogen in the environment. Infection with *E. rhusiopathiae* is more common in adults than young birds and usually occur through mechanical skin injuries. Erysipelas in acute stage causes sudden death of infected birds, while some birds may show darkening of the skin or cyanosis in the head region and sharp drop in egg production. In post-mortem lesions, birds with *E. rhusiopathiae* show septicaemic picture, haemorrhages all over the body, valvular endocarditis, diffuse enlargement of internal organs, enteritis, and arthritis. The disease in human is known as erysipeloid and it mostly affects persons in direct contact with infected birds or contaminated poultry products. Strict biosecurity measures and treatment especially with penicillin derivatives are crucial for erysipelas control in infected birds. Living and inactivated vaccines for turkeys and other species of birds are used for prophylaxis.

Keywords: Avian, *Erysipelothrix rhusiopathiae*, Erysipeloid, Occupational

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INTRODUCTION

Erysipelas is a worldwide emerging bacterial disease which affects many species such as humans and domestic and wild animals (Bricker and Saif, 2013). It is an acute fulminating occupational disease of a wide range of domestic and free range wild avian species. Severe economic losses are incorporated with erysipelas infection especially for the turkeys industry (Pattison et al., 2008). The disease is characterized by an acute septicemic picture with vascular and degenerative changes, especially in the liver, spleen, and kidney (Bickford et al., 1978). The causative agent of erysipelas is a ubiquitous bacterium *Erysipelothrix rhusiopathiae* (*E. rhusiopathiae*) that is widely distributed in the environment (soil, water, sewage, etc.) all over the world (Opriessnig et al., 2020). Infection of birds with *E. rhusiopathiae* is usually occur through injuries of the skin and mucous membranes by biting insects, sharp objects, cannibalism, or during beak trimming and toe-clipping (Bricker and Saif, 1991).

The disease in humans is called erysipeloid. Infection with *E. rhusiopathiae* is occupationally related and occurs mostly in people who work closely with contaminated poultry or their products, wastes, and soil such as abattoir workers, butchers, veterinarians, and farmers (Romney et al., 2001). Infected people usually show skin lesions in the hands or fingers in the form of swollen violaceous zone with severe pain, besides lymphangitis, arthritis, and infective endocarditis (Wang et al., 2010).

Penicillin is the drug of choice for the rapid treatment of *E. rhusiopathiae* for both birds and humans (Schmitt et al., 2014). Living attenuated vaccine used for swine could be also used to prevent *E. rhusiopathiae* outbreaks in commercial cage-free layer chickens (Crespo et al., 2019). Inactivated vaccines also used for vaccination of turkeys and chickens (Eriksson et al., 2014; Wattrang et al., 2020).

Therefore, this review article was designed to spotlight on erysipelas infection of poultry regarding the disease history and nomenclature, the bacterium, virulence factors and pathogenicity, susceptibility, infection and transmission, pathology, human infection, laboratory diagnosis, and the prevention and control.

HISTORY AND NOMENCLATURE

The first detection of genus *Erysipelothrix* members was made by Koch in 1876, and a few years later, the bacterium was identified by Löffler as the causative agent of erysipelas in pigs (Stackebrandt et al., 2006). In 1884, the bacterium was associated with a disease condition in humans (Rosenbach, 1909). However, the first outbreak of erysipelas in poultry species was in turkeys in 1904, after which many reports of erysipelas in chickens were described (Beaudette and Hudson, 1936). The first name of the species was introduced by Trevisan in 1885 (Langford and Hansen, 1954). The author's described the bacterium as a spore-forming rod and named *Bacillus insidiosus*. However, in 1900, the name *Bacterium rhusiopathiae* was introduced by Migula (Stackebrandt et al., 2006). In 1909, the genus *Erysipelothrix* was

divided into three separate species-related host; *E. murisepticus* (mice), *E. porci* (pigs), and *E. erysipeloides* (human) (Rosenbach, 1909). By Buchanan in 1918 and in 1920, the name *E. rhusiopathiae* was introduced and designated as the type species (Winslow et al., 1920). The name *E. rhusiopathiae* originates from the Greek and literally translates as (rose, red skin), trix (hair), rhusius (reddening), and pathus (disease) or 'erysipelas thread of red disease' (Euzéby, 2013).

THE BACTERIUM

Erysipelothrix spp. are Gram-positive, non-spore-forming, and rod-shaped bacteria belonging to the Erysipelotrichaceae family (Phylum Firmicutes; class Erysipelotrichia; order Reysipelotrichales) (Verbarg et al., 2004). There are different species of *Erysipelothrix*, namely, *E. inopinata* (Verbarg et al., 2004), *E. larvae* sp. nov (Bang et al., 2016), *Erysipelothrix* sp. strain 1, *Erysipelothrix* sp. strain 2, *Erysipelothrix* sp. strain 3 (Takahashi et al., 2008); *E. tonsillarum* (Takahashi et al., 1992; Opriessnig et al., 2020), and *E. piscisicarius* sp. nov. (Pomaranski et al., 2020). However, *E. rhusiopathiae* is the only avian pathogenic species (Parte, 2018; Opriessnig et al., 2020). The genus *Erysipelothrix* has been divided into serotypes based on heat stable cell wall antigens (Kalf and White, 1963). At least 28 serotypes are known within the genus. Species of *E. rhusiopathiae* are belonging to serotypes 1a, 1b, 2, 4, 5, 6, 8, 9, 11, 12, 15, 16, 17, 19, 21, and N (Opriessnig et al., 2013). However, serotypes 1, 2, 4, 5, 6, 8, 15, 16, and 21 have been recorded in birds (Takahashi et al., 2000).

The bacterium is very resistant to environmental factors, meat curing, smoking, and salting. It has been reported that *E. rhusiopathiae* can survive and replicate in the soil and decayed organic matter for a few weeks under warm and alkaline conditions (Bricker, 2008). The long-term survival of *E. rhusiopathiae* in soil with a maximum survival time of 72 days has been reported (Chandler and Craven, 1980).

VIRULENCE FACTORS AND PATHOGENICITY

Erysipelothrix rhusiopathiae produces some virulence factors (Figure 1) that cause cellular damage, penetration of the host cells, and consequently induction of infection (Shimoji, 2000; Opriessnig et al., 2020). One of these, neuraminidase enzyme which splits α sialic acid moiety from sialo-glyco-conjugates and leads to damage of the vascular system and formation of the hyaline thrombus (Shimoji et al., 2002). The presence of coagulase and hyaluronidase (hydrolyzes matrix substance or hyaluronic acid) can facilitate bacterial spread. Moreover, the bacterial capsular antigen can resist the phagocytic action of macrophages and helps in the intracellular survival of the pathogen. Some virulence proteins such as adhesive surface protein (*Rsp*A, *Rsp* B and *Rsp*C) can bind and adhere to cellular surfaces of the hosts and formed biofilm (Ogawa et al., 2011) and heat shock protein (*dna*J) that participates actively in the response to hyperosmotic and heat shock by preventing the aggregation of stress-denatured proteins (Pattison et al., 2008). The bacterium also possesses a polysaccharide capsule that has a virulence-associated

function and resistance to phagocytosis (Shimoji, 2000). It has been found that *E. rhusiopathiae* can survive intracellularly inside murine macrophages which may be facilitated by several antioxidant factors and phospholipases (Ogawa et al., 2011). Makino et al. (1998) identified *E. rhusiopathiae* surface protective antigen (*Spa*) gene which is associated with protection against the disease (Ingebritson et al., 2010). The identified proteins were *Spa*A, *Spa*B1, *Spa*B2, and *Spa*C (Shen et al., 2010). Not all *E. rhusiopathiae* strains are similar in virulence and that the serotype does not accord with virulence (Wang et al., 2010).

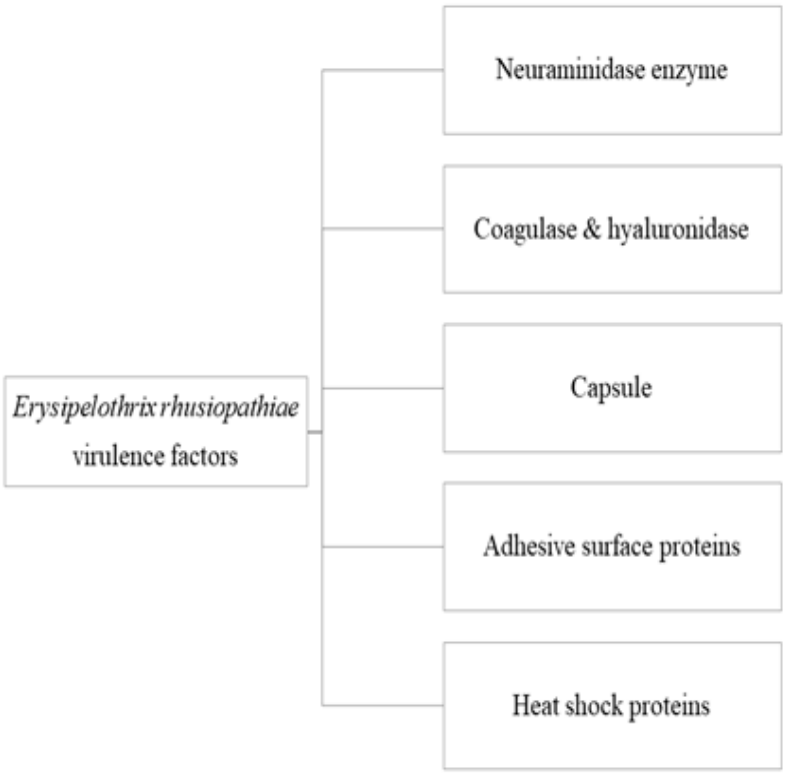

Figure 1 Different virulence factors of *Erysipelothrix rhusiopathiae*

SUSCEPTIBILITY

Although pigs are considered as the natural reservoir of *E. rhusiopathiae*, many other mammals (Griffiths et al., 1991), fish (Opriessnig et al., 2013), and birds (Forde et al., 2016) may harbor the bacterium. Amphibians, reptiles, insects, and humans (Yokomizo and Isayama, 1972; Veraldi et al., 2009) can be also infected.

Erysipelas affects a wide range of poultry species including domestic or free living birds. The first isolation of *E. rhusiopathiae* from a chicken was reported by Hausser in 1909 (Beaudette and Hudson, 1936). Commercial layer turkey (Hollifield et al., 2000; Health and Agency, 2013; Hoepers et al., 2019) and chicken flocks (Mazaheri et al., 2005; Eriksson et al., 2013) are highly susceptible to erysipelas infection. Erysipelas in broiler chickens and pullets is rare, with only a few reports of natural *E. rhusiopathiae* infection (Milne et al., 1997). Moreover, outbreaks of erysipelas have been also reported in ducks (Dhillon et al., 1980) and geese flocks (Bailie et al., 1970; Gunning and Morton, 1988; Bobrek et al., 2015).

Although *Erysipelothrix* infection in the wild birds is sporadic, some massive outbreaks have been reported in Eared Grebes of the Great Salt Lake (Jensen and Cotter, 1976). Free ranging wild and captive birds such as racing pigeons (Cousquer, 2005), quails (Mutalib et al., 1995), emus (Swan and Lindsey, 1998; Morgan et al., 2011), pheasants (Milne et al., 1997; Hennig et al., 2006), chukar partridge (Pettit et al., 1976), malleefowl (Blyde and Woods, 1999), parrots (Livingston et al., 2013), chukars (Butcher and Panigrahy, 1985), Hawaiian crow (Work et al., 1999), blue penguin (Boerner et al., 2004), endangered kakapo (Gartrell et al., 2005), laughing kookaburra (Opriessnig et al., 2005), takahe, kiwi, and black stilts (Alley and Gartrell, 2019) showed infections with *E. rhusiopathiae*.

Infection in poultry with *E. rhusiopathiae* may occur at any age, but reported cases have usually concerned older ages as layers (Kurian et al., 2012). More than 67% of erysipelas outbreaks occurred in Sweden flocks were from 60 weeks of age and upwards (Wattrang et al., 2021).

INFECTION AND TRANSMISSION

Erysipelas can be actively introduced into poultry flocks through pigs, rodents, and the poultry red mite (*Dermansyssus gallinae*) (Chirico et al., 2003; Mazaheri et al., 2006). Indirect contact of commercial poultry with pigs, sheep, or other animals may contribute to infection (Acha and Szyfres, 2003). Contaminated feed as fish meal, as well as contaminated soil, cages, clothes, shoes, drinkers, or manure may be important sources of *E. rhusiopathiae* infection for commercial poultry flocks (Bender et al., 2010). It has been found that asymptomatic recovered birds may be carriers for many weeks after *E. rhusiopathiae* infection and disseminated the bacteria in surroundings via droppings (Pattison et al., 2008). Mechanical transmission through biting flies, toe-clipping, beak trimming, and sharp edges may predispose to skin injury and erysipelas infection. Cannibalism of *E. rhusiopathiae* infected dead carcass may pose a risk for the flock infection (Bricker, 2008). As *E. rhusiopathiae* is a soil-associated pathogen, so ingestion of any decomposed or decayed materials in the farm may help in the spread and transmission of infection (Cousquer, 2005). Moreover, non-impervious bedding floors may be a prompting factor, besides the behavioral factors as fighting within the flock (Eriksson et al., 2014). *E. rhusiopathiae* is not a vertically or egg transmitted pathogen and the infection of layer breeder chickens has no adverse impact on the quality of hatching eggs in terms of increased embryo mortality (Mazaheri et al., 2006).

CLINICAL SIGNS AND PATHOLOGY

In acute *E. rhusiopathiae*, sudden death of infected birds can occur (Mutalib et al., 1993). However, some birds may show darkening of the skin, diarrhea, depression, emaciation, and sharp drop in egg production up to 45% (Bisgaard et al., 1980; Schmitt et al., 2014). Specific severe cyanosis with irregular reddish-purple colored head region or snood of infected turkeys was observed. Moreover, waterfowl may show congestion of foot-web. Dark red crusty patches on the back of laying chicken hens have been also seen (Mutalib et al., 1993). Unusual ocular manifestations in terms of swollen, lacrimating, and encrusted eyes have been reported in a free range layer chicken flock

(Schmitt et al., 2014). Arthritis with unsteady walking and a lack of coordinated movement could be seen in chronic stages (Gunning and Morton, 1988).

Although mortality rate of erysipelas is generally low, but it can reach up to 25-50% (Stokholm et al., 2010). Mortality may range from less than 1% to more than 50% according to the organism's virulence, the host's susceptibility and immune response, and the presence of other complicating factors (Brickford et al., 1978; Opriessnig et al., 2005). Schmitt et al. (2014) diagnosed erysipelas cases in a free-range laying flock with a high mortality of up to 7% per day.

Some lesions of *E. rhusiopathiae* infection are presented in Figure (2). Dead birds with *E. rhusiopathiae* may be septicemic with congestion and hemorrhages on the internal organs, particularly on skeletal muscles, coronary and abdominal fat, myocardium, pancreases, and pleura (Bobrek et al., 2013). Valvular endocarditis as well as regression and discoloration of the ovary were recorded in laying hens (Stokholm et al., 2010). In addition, hepatomegaly with sub-capsular hemorrhagic and mottled necrotic liver were noticed (Mazaheri et al., 2005). Spleenomegally with focal necrosis was seen (Brickford et al., 1978). The kidneys became edematous and congested (Takahashi et al., 1994). The lungs were edematous and engorged with a little ecchymosis in the parenchyma (Bobrek et al., 2015). Enteritis and ulceration of the caecal mucosa may be seen. Ecchymosis was observed on the surface of the pancreas (Bailie et al., 1970). Arthritis and purulent synovitis as well as vegetative endocarditis have been reported (Hollifield et al., 2000). Cellulitis was also reproduced after experimental infection of broiler chickens with *E. rhusiopathiae* (Derakhshanfar et al., 2004).

The histopathology of layer chickens had *E. rhusiopathiae* revealed disseminated intravascular coagulopathy in conjunctival small vessels (Shibatani et al., 1997; Schmitt et al., 2014). Conglomerates of bacteria surrounded by thrombocytes were seen in blood capillaries and small arteries and veins. Thrombi within small vessels were also noticed in turkeys (Bickford et al., 1978; Bricker, 2008). Necrosis related to infarctions of blood vessels with degenerative changes of parenchymatous organs were observed. Generalized congestion of hepatic sinusoids with hyalinization of the walls, massive areas of necrosis, and fibrin thrombi were also seen (Mutalib et al., 1993). The pulp of the spleen was destroyed, with only a vascular layer and hypertrophic reticular cells remaining, and the pancreas showed necrosis (Bobrek et al., 2015). Ecchymosis and edema were visible in the heart and lungs (Bobrek et al., 2015). Infiltration of the mononuclear cells in the endocardium and pericardial sac was detected. Moreover, infiltration of inflammatory cells in the synovium was seen in joints. Lymphocytic infiltration with necrosis and shortening of the intestinal villi were observed (Bobrek et al., 2015). Massive congestion and lymphocytic infiltration were also detected in kidneys (Bobrek et al., 2015).

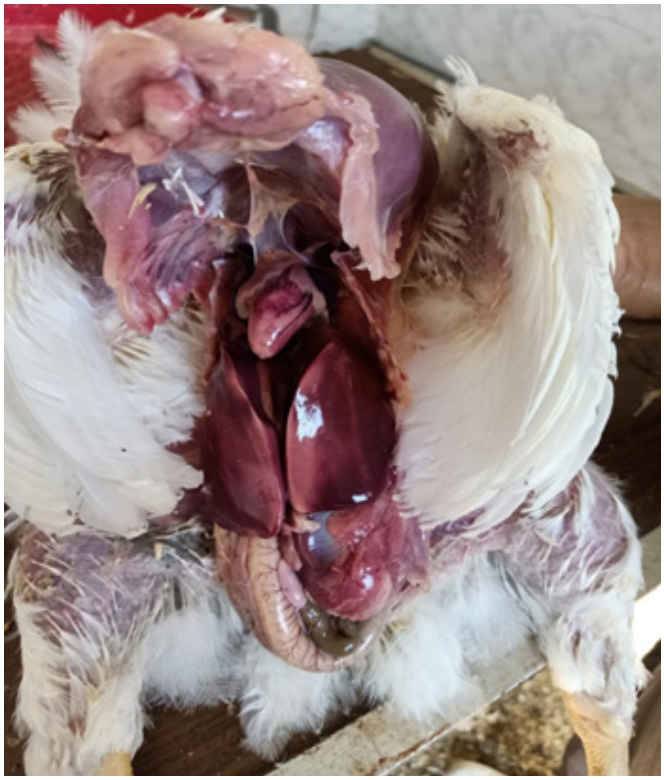

Figure 2 Severely congested muscles, congested heart with hemorrhages on the coronary fat, and severely congested liver with sub-capsular hemorrhages

HUMAN INFECTION

The first description of human's erysipeloid was in 1884, when Rosenbach isolated *E. rhusiopathiae* from a patient. Erysipeloid cases were recorded during the period 1957-1960 at University College Hospital in London. Although *E. rhusiopathiae* infection is not common in humans, it may be under-diagnosed due to its similarity to other bacteria and its diagnostic difficulties (Brooke and Riley, 1999).

The human disease is known as erysipeloid, especially the cutaneous form of the infection (Veraldi et al., 2009). The disease is regarded as an occupational zoonosis and it is reported to have gained entry in human through wound or aberration of the skin (Opriessnig et al., 2020). Thus, it affects particularly animal handlers, fish processors, butchers, cooks, housewives, and farmers (Salamah, 1988; Wang et al., 2010; Eriksson et al., 2014). The main source of human's infection with *E. rhusiopathiae* is the contact with infected animals or their products. In Japanese studies, slaughter chickens bi-products from 83% of farms were positive for *Erysipelothrix* sp. (Nakazawa et al., 1998a), and that 30% of chicken meat samples were positive for *Erysipelothrix* sp. (Nakazawa et al., 1998b) by culture methods and in both studies the majority of positive isolates were *E. rhusiopathiae*. Takahashi et al. (2000) in Japan, reported that laying hens at slaughter showed 5.5% positive chickens for *E. rhusiopathiae*, while Eriksson et al. (2013) in Sweden, showed that laying hens at slaughter were 100% positive. A report on chickens of different ages and production categories in New Zealand revealed overall 40% positive chickens for *E. rhusiopathiae* (Kurian et al., 2012).

Localized lesions of erysipeloid in fingers that eventually spread to the lymph nodes have been documented in caretakers handled with dead erysipelas infected layer chickens (Mutalib et al., 1993). Similarly, skin lesions of erysipeloid were seen in workers engaged in quail processing unit where an outbreak of erysipelas had been found among these birds (Mutalib et al., 1995). Infection with different species of *Erysipelothrix* induces erythema serpens, Rosenbach's erythema, and fish handler's disease, etc. (Reboli and Farrar, 1989; Brooke and Riley, 1999). Three forms of erysipeloid in human include; a mild or acute cutaneous form which differs from (true erysipeloid), the diffuse or generalized cutaneous form, and the systemic form which is rare, unusual, and may result in septicemia, infective endocarditis, and encephalitis in immune defects people (Silberstein, 1965; Wang et al., 2010; Kozdruń et al., 2015). Erysipeloid is often localized in the hands or fingers as a well-defined swollen violaceous zone with severe pain. Fever, lymphangitis, lymphadenopathy, vesicles, and arthritis in an adjacent joint may also occur (Reboli and Farrar, 1989; Brooke and Riley, 1999).

LABORATORY DIAGNOSIS

Samples can be collected from liver, spleen, heart blood, joint, bone marrow, or lymph nodes (Acha and Szyfres, 2003). Conventional isolation and identification of the bacterium are still used to confirm the field diagnosis.

Several media such as modified blood azide, Bohm's, and Parker's are used for selective isolation of *E. rhusiopathiae* under facultative anaerobic condition and temperatures between 5 and 42°C. Rough colonies are about 1-2 mm in size and have irregular or flat edges (Bricker and Saif, 1997). However, smooth colonies are small round, with a diameter of 0.3-1.5 mm, and slightly convex with α zone of hemolysis.

Following isolation, tentative microscopic and biochemical tests are used for further confirmation. *E. rhusiopathiae* is a Gram positive, non-spore-forming, non-acid-fast, and non-motile bacilli (Brooke and Riley, 1999). The bacterium is negative for oxidase, catalase, and methyl red, but positive for H_2S production and sugar fermentation (Pattison et al., 2008).

Fluorescent antibody test and mouse protection assay can be also applied for more identification. Recent molecular techniques such as polymerase chain reaction are widely used for the identification of *E. rhusiopathiae* and they are targeting 16S rRNA gene (Hennig et al., 2006; Harada et al., 2009; Kurian et al., 2012).

SEROLOGY

Experimental infection of chickens with *E. rhusiopathiae* can elucidate antibodies that detected by a growth agglutination test (Takahashi et al., 1994). Moreover, serum samples from laying hens revealed presence of antibodies which suggested that exposure to *E. rhusiopathiae* was common (Takahashi et al., 2000). High prevalence of antibodies to *E. rhusiopathiae* was demonstrated in serum of laying hens using enzyme linked immuno-sorbent assay (Kurian et al., 2012). Subtyping of *E. rhusiopathiae* isolates by pulsed field gel electrophoresis (PFGE) using the restriction enzyme *Sma*I is a suitable method (Okatani et al., 2001; Opriessnig et al., 2004). It has been found that this test is one of the most suitable methods for the characterization of *E. rhusiopathiae* isolates and a suitable fingerprinting method for epidemiology. Serotyping is

a less suitable method for subtyping of *E. rhusiopathiae* than PFGE (Okatani et al., 2000). Serotyping is a traditional method for subtyping isolates of *E. rhusiopathiae*, however, this method is of limited use for epidemiological studies of erysipelas outbreaks. In the study of Eriksson et al. (2013), serotypes 1a, 1b, 1ab, 4, and 6 were the predominant among laying chickens. Strains of *E. rhusiopathiae* vary in virulence and there is no relation between the virulence and serotype (Bisgaard et al., 1980, Wang et al., 2010). Besides, serological surveys of healthy chickens revealed relatively high levels of antibodies to *E. rhusiopathiae* in positive birds (Kurian et al., 2012; Eriksson et al., 2013).

PREVENTION AND CONTROL

Strict biosecurity measures and good hygienic practices are important for prevention of erysipelas. *E. rhusiopathiae* is sensitive to most common disinfectant, thus thorough cleaning and disinfection are very crucial to prevent subsequent outbreaks of erysipelas (Wang et al., 2010). Recurrence of the disease in the succeeding flock may be possible, particularly if hygienic practices were not properly performed or the resting period between the successive flocks was too short (Bisgaard et al., 1980; Hafez et al., 2001). Moreover, eradication of red mite using acarizides is also recommended (Chirico et al., 2003).

Penicillin, cephalosporins, and erythromycins are suitable for the treatment of *E. rhusiopathiae* infection (Veraldi et al., 2009; Schmitt et al., 2014). As well, oxytetracyclin, tylosin, and fluorchinolons can be used for treatment. No reported resistance to these antibiotics and their minimal inhibitory concentrations are still very low (Reboli and Farrar, 1989; Wang et al., 2010). However, *E. rhusiopathiae* infected cases may be not treated due to self-limiting nature of infection and relapses may occur (Wang et al., 2010).

Living attenuated *E. rhusiopathiae* vaccines or bacterins are commercially available and offer immunity for 6-12 months in turkeys (Bricker and Saif, 1988; Eriksson et al., 2014). Previously affected pullets farms were vaccinated at placement with a single dose of inactivated *E. rhusiopathiae* vaccine as a prophylactic measure to prevent further outbreaks (Eriksson et al., 2014). Vaccination of laying hens flocks was effective (Mutalib et al., 1993). Intramuscularly vaccinated layer chickens with inactivated *E. rhusiopathiae* vaccine showed increasing the levels of immunoglobulins Y earlier after infection compared with chickens in the control non vaccinated group (Wattrang et al., 2020). Double intravenous vaccinations of turkeys with both live and inactivated vaccines gave full protection against infection with *Erysipelothrix* sp. (Krasnodębska-Depta and Janowska, 1980; Krasnodębska-Depta and Koncicki, 1988). However, double vaccinations in the drinking water gave low antibody titers (Krasnodębska-Depta and Koncicki, 1988). Emus vaccinated subcutaneously at 4-8-week-old using 0.5 ml/ bird showed no symptoms of the disease after experimental infection with *E. rhusiopathiae* (Swan and Lindsey, 1998). However, vaccine failure may happen due to presence of different serotypes and even if the serotypes do not differ (Hafez et al., 2001; Stokholm et al., 2010). Outbreaks of erysipelas in vaccinated flocks have been detected (Eriksson et al., 2014). For instance, analysis of erysipelas in Sweden between 2010 and 2019 revealed that 30% of outbreaks occurred in vaccinated flocks (Wattrang et al., 2021).

To avoid zoonosis in humans, wearing of protective cloths and other necessary protective gears are essential during handling with suspected dead birds. Moreover, persons in close contact to the wild birds should take extra precautions to avoid *E. rhusiopathiae* infection via any damage of the skin (Sheng et al., 2000).

CONCLUSION

As erysipelas is an emerging disease affecting different commercial poultry species and it can be transmitted to human, thus such infection should be given in consideration in our flocks. Moreover, *E. rhusiopathiae* can be withstood in the environment for a long time and the birds showed titers of antibodies against infection. Accordingly, more research work and studies on the surveillance of erysipelas are the must.

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AUTHOR CONTRIBUTIONS

WAA: Collected the literature, drafted and revised the manuscript, and approved the final manuscript.

CONFLICT OF INTEREST

The author has not declared any conflict of interest.

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