Open Access

Berl Münch Tierärztl Wochenschr 127, 183–187 (2014) DOI 10.2376/0005-9366-127-183

© 2014 Schlütersche Verlagsgesellschaft mbH & Co. KG ISSN 0005-9366

Korrespondenzadresse: benjamin.schade@tgd-bayern.de

Eingegangen: 07.08.2013 Angenommen: 13.02.2014

Online first: 29.04.2014 http://vetline.de/open-access/ 158/3216/

Summary

Zusammenfassung

U.S. Copyright Clearance Center Code Statement: 0005-9366/2014/12705-183 \$ 15.00/0

Case report/Fallbericht

Bavarian Animal Health Service, Poing, Germany¹ National Institute of Animal Health, Tsukuba, Ibaraki, Japan²

Erysipelas in a free-range layer flock with conjunctival oedema as an unusual clinical sign

Rotlauf bei Legehennen in Freilandhaltung mit Lidödem als ungewöhnliches klinisches Symptom

Ferdinand Schmitt^{1*}, Benjamin Schade^{1*}, Brigitte Böhm¹, Yoshihiro Shimoji², Corinna Pfahler¹

*Both authors have contributed equally to this work.

Erysipelas was diagnosed in a free-range laying flock with a high mortality of up to 7% per day and a severe decrease in egg production to 45%. The disease had a short course and unusual clinical features for erysipelas, including swollen, lacrimating and encrusted eyes. Bacteriologically, trapped poultry red mites and affected animals were culture-positive for *Erysipelothrix rhusiopathiae*. Isolates from layers and mites were both serotype 1b. Histopathology revealed disseminated intravasal coagulopathy in conjunctival small vessels as the cause of the oedema of the eye adnexes. After treatment with penicillin, mortality and egg production returned to normal levels. Although erysipelas in laying hens is rarely reported, it can develop as an emerging disease in alternative rearing systems and should always be considered if mortality increases in an older flock, especially with a high infestation of poultry red mites.

Keywords: Conjunctival oedema, disseminated intravasal coagulopathy, poultry red mite, *Erysipelothrix rhusiopathiae*

In einer Legehennenherde mit Freilauf wurde Rotlauf diagnostiziert. Die tägliche Mortalität betrug bis zu 7 % und die Legeleistung sank auf 45 %. Die Tiere zeigten einen kurzen Krankheitsverlauf und ein unübliches Symptom für eine Rotlauferkrankung: Es konnten geschwollene, tränende und verkrustete Augen bei den Legehennen festgestellt werden. Bakteriologisch konnte in eingefangenen Roten Vogelmilben und verstorbenen Tieren kulturell Erysipelothrix rhusiopathiae nachgewiesen werden. Eine Serotypisierung ergab, dass es sich sowohl bei den Isolaten aus den Legehennen als auch bei denen aus den Milben um den Serotyp 1b handelte. Histopathologisch konnte eine disseminierte intravasale Gerinnung in kleinen Bindehautgefäßen als Ursache des Lidödems diagnostiziert werden. Nach einer Penicillinbehandlung normalisierten sich sowohl die Mortalität als auch die Legeleistung. Rotlauf bei Legehennen wird nur selten beschrieben, dennoch kann es durch die vermehrte Freilandhaltung zu einem häufigeren Auftreten kommen. Aus diesem Grund sollte Rotlauf differenzialdiagnostisch berücksichtigt werden, wenn die Mortalität in einer älteren Herde ansteigt und zeitgleich ein erhöhter Milbenbefall festgestellt wird.

Schlüsselwörter: Lidödem, disseminierte intravasale Gerinnung, Rote Vogelmilbe, *Erysipelothrix rhusiopathiae*

Introduction

In 2010, cages for laying hens were banned in Germany, and alternative housing systems replaced the cages. Today, furnished cages, aviaries and floor systems are the

predominant layer houses in Germany. In these systems, the birds are kept in variable sized groups, depending on the system, with a scratch area. A considerable number of flocks are free-range, which may expose the flock to various hazards, such as *Erysipelothrix* (E.) rhusiopathiae.

E. rhusiopathiae may induce a septicaemic disease in different avian species, especially in turkeys (Meleagris gallopavo). Less often, valvulitis and arthritis are chronic outcomes of the disease (Bricker, 2008). Layers are rarely affected, but it is not known whether brown or white laying hens have different predispositions for erysipelas. Bisgaard reported that layers are rarely affected and that white Leghorns predominate (Bisgaard et al., 1980; Kurian et al., 2012). Kurian et al. (2012) and Mazaheri et al. (2005) reported that it is mostly older layer flocks that are affected by the disease. E. rhusiopathiae can survive and replicate in the soil under warm and alkaline conditions for a few weeks (Reboli and Farrar, 1989; Bricker, 2008), although the length of survival is not known. Recurrence of erysipelas in the succeeding flock is possible, especially if disinfection was not properly performed or the resting period between the two flocks was too short (Bisgaard et al., 1980; Mutalib et al., 1993; Hafez et al., 2001). It is hypothesised that the disease can be actively introduced into a flock by pigs, rodents and the poultry red mite (Dermanyssus gallinae) (Butcher and Panigrahy, 1985; Reboli and Farrar, 1989; Chirico et al., 2003; Mazaheri et al., 2006). Passive introduction into animal housing systems can occur from feed containing fish meal or contaminated soil, cages, clothes, shoes, drinkers, or manure (Butcher and Panigrahy, 1985; Reboli and Farrar, 1989; Bricker, 2008; Bender et al., 2010). After infection, laying hens show peracute death, reduced vigour, depression, diarrhoea and a drop in egg production (Bisgaard and Olsen, 1975; Bisgaard et al., 1980; Mutalib et al., 1993; Chirico et al., 2003; Bricker, 2008). The overall mortality rate in one cycle is generally low but can reach up to 25-50% (Bisgaard et al., 1980; Mazaheri et al., 2006; Bricker, 2008; Stokholm et al., 2010). The economic impact of this disease is under debate, and some authors consider erysipelas in layers to be a minor problem (Bisgaard and Olsen, 1975; Bricker, 2008). Hafez and colleagues described sporadic outbreaks of erysipelas in German layer flocks (Hafez et al., 2001).

Here, we describe an outbreak of erysipelas in a brown free-range layer flock that experienced a high daily mortality rate of up to 7%, with presumptive transmission by the poultry red mite and an unusual manifestation of this disease in the ocular region.

Case Details

The affected farm, situated in Southern Germany in Bavaria, has four layer flocks with a total of 2500 birds in an aviary system with free-range access for 6 hours per day. Two flocks are kept in a single-layer house; the other two flocks are situated under the same roof but in different compartments with separated ventilation, feeding and drinking supplies. The access to the layer compartments leads through a pig and horse compartment situated in the same building. The feed consists of a conventional layer ratio without fishmeal and is free of genetically modified organisms, especially soybeans. The water is supplied from the public water system. All flocks are supplemented with vitamins (Soluvit ADE Form, Alhorn, Germany), calcium phosphate (Koni CalPhos, Konipharm, Essen, Germany) and formic acid via the drinking water on a regular basis.

The diseased flock consisted of 900 layers (Lohmann Brown Classic and Lohmann Selected Leghorn, LTZ,

Cuxhaven) and was located in the poultry house with an adjacent flock. This flock was purchased in May 2012 at 18 weeks from a local pullet farm and both lines were kept in the same house. The pullets were vaccinated against Marek's disease, Newcastle disease, Avian encephalomyelitis, infectious laryngotracheitis, infectious bronchitis, Mycoplasma gallisepticum, Salmonella enterica subsp. enterica serovar Enteritidis and infectious bursitis. Total mortality was unremarkable (below 0.5% per month). During the ninth month laying period, the egg production rate was 77% for the mixed flock, 10% less than the egg production curve recommended by Lohmann Tierzucht. This was mainly due to very small hens that were delivered by the pullet breeder and a light programme that was not adjusted to the fact that the birds were too small. Litter was replaced regularly and deposited on a dung heap approximately 500 metres away from the poultry houses. Eggs were collected by an egg belt and sorted outside of the compartments in the poultry house. Before entering each poultry compartment, the human attendants changed their shoes, but their clothes and hands were not washed or disinfected. Environmental conditions (e.g., ammonia, dust) were appropriate.

In the first week of March 2013, increasing mortality and a severe drop in egg production were observed, predominantly affecting brown layers. The white layers showed similar clinical signs but these were less pronounced, and only a few birds were affected. The dead birds were scattered throughout the aviary, particularly near nest boxes. During flock inspection, approximately 20% of the chickens showed reduced vigour, ruffled feathers, mild diarrhoea and signs of exsiccosis. In addition, a considerable infestation with the poultry red mite was noted in this flock.

Initially, the mortality was low (0 to 3.2%). Because enteritis was considered to be the cause, the flock was treated with neomycin in the drinking water for five days (30 mg neomycinsulfate per kg body weight, Neomycinsulfat, Klat Chemie, Alhorn, Germany). On the third day of treatment, the daily mortality rate had increased to 7%, and the egg production declined in the following six days to 45% (Fig. 1). At this time, several brown layers showed uni- or bilateral swollen periorbital regions as well as lacrimation and encrustation of the eyes (Fig. 2A). Signs of diarrhoea with large amounts of urate crystals were evident. Due to the high mortality, the vicinity to a lake with a considerable population of wild waterfowl, and the presence of respiratory signs (swelling of the sinus and conjunctiva and lacrimation) and diarrhoea, veterinary health authorities were informed. The farm was shut down due to a daily mortality above 2%. This mortality rate and its consequences (setting up strict biosecurity measures, initiating epidemiological studies and exclusion of avian influenza and Newcastle disease) are regulated by Geflügelpest-Verordnung 2009 (EU-Directive 92/40/EWG). Another antibacterial treatment was permitted and applied due to the persisting high mortality and the new clinical sign of swollen sinuses and eyes (100 mg/kg body weight Tylosin, 5 days, Tylan soluble, Elanco Animal Health, Bad Homburg, Germany), which had only minimal effect.

A post-mortem examination was performed at the Bavarian Animal Health Service and the Bavarian Health and Food Safety Authority on 23 layers (7 Lohmann Selected Leghorns and 16 Lohmann Browns) on two days

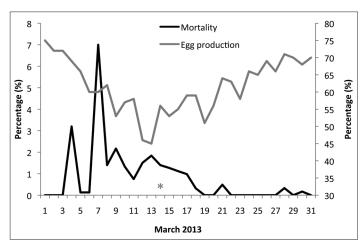


FIGURE 1: Mortality and egg production Mortality rate and egg production in March 2013 of affected barn no. 3 (left X-axis for mortality, right X-axis for egg production). Note the severe increase in mortality in the first week and a corresponding drop in egg production. The asterisk indicates the first penicillin treatment.

(n = 10, 07.03.2013 and n = 13, 08.03.2013) to determine the cause of death and, in particular, to exclude infection with Avian Influenza Virus and Newcastle Disease Virus.

At necropsy, the animals were in good body condition with active ovaries. The lungs, livers and spleens were congested and the kidneys were moderately swollen. Five out of thirteen chickens examined on 08.03.2013 at the Bavarian Animal Health Service also had a moderate periorbital swelling (Fig. 2A), and swollen and reddened conjunctiva with injected scleral vessels. In addition, the mucosa of the nasal cavity and sinus, as well as the comb and wattle, were reddened. Two chickens had multifocal to coalescing fibronecrotic foci in the liver (Fig. 2D) and spleen. Microscopically, 11 out of 13 animals showed fibrin thrombi in liver sinusoids and small vessels of the liver, spleen, lung and brain. The conjunctiva was oedematous with dilated and congested vessels with multifocal intravascular fibrin thrombi (Fig. 2B, C). Conjunctivitis was not present. Mild to severe acute multifocal necrotising hepatitis (Fig. 2E) and splenitis was detected in eight hens.

Bacteriologically, E. rhusiopathiae was isolated from liver, spleen, lung and brain after 24 h of aerobic incubation at 37°C on Columbia Agar supplemented with 5% sheep blood. The results showed typical growth of small colonies with alpha-haemolysis and the species was determined by biochemical identification with VITEK2compact (bioMerieux, Nürtingen, Germany). Serotyping was carried out by agar gel precipitation tests with autoclaved cell extracts and rabbit antisera against formalin-killed cells of reference strains as previously described, and the isolates from the layers were serotyped as 1b (Takahashi et al., 1996). After the diagnosis of erysipelas, penicillin at a dose of 68 mg/kg body weight was given via the drinking water for five days (Phenoxypen WSP 325 mg/g, Dopharma Research B.V., Raamsdonksveer, The Netherlands). Mortality returned to normal levels. In the first week of April, mortality increased again and another penicillin treatment was needed. The total mortality was 34% at the end of the period. Egg production did not rise above 78% for the rest of the cycle.

To exclude other pathogens, swabs taken from the conjunctiva, trachea and sinus were analysed on Colombia Agar using the methods described above. Bacteriologically, Staphylococcus chromogenes was found in the conjunctiva and sinus, and Gallibacterium spp. in the trachea and nasal cavity. Cultures were negative for Ornithobacterium rhinotracheale, and Mycoplasma gallisepticum could not be detected by polymerase chain reaction (PCR). Avian metapneumovirus viral RNA could not be detected. Avian influenza virus and Newcastle disease virus were excluded by PCR according to the "Official Collection of Methods for the Sampling and Investigation of Materials of Animal Origin for Notifiable Animal Diseases (Method Collection)" by the Bavarian Health and Food Safety Authority (accessible at www.fli.bund. de).

To reveal the potential source of infection, poultry red mites were trapped according to Chirico et al. (Chirico et al., 2003). Additionally, conventional traps were used to trap rodents. The two pigs on the farm were examined for signs of erysipelas and revealed no clinical signs; erysipelas had never been detected in any pigs on that farm in the past. The pigs were only kept for a short period and were not vaccinated against erysipelas. Bacteriologically, the liver, spleen, kidney, lung and intestine from a trapped rat (Rattus norvegicus) were culturenegative for E. rhusiopathiae. In contrast, the poultry red mites that were collected at the onset of increased mortality and two weeks later were culture-positive for E. rhusiopathiae and the isolates were serotyped as 1b. The farmer was instructed to use a Permethrin product (Intermitox[®], Interhygiene GmbH, Cuxhaven, Germany) to reduce mite populations in the poultry house. During the service period, another mite treatment was applied, and thorough cleaning and disinfection with a Cresol product (Neopredisan 135-1, Norderstedt, Germany) was performed. The outdoor pen was dug over, and quick lime was incorporated into the soil. The pen was left unused for four weeks.

The flock adjacent to the affected herd consisted of 780 Lohmann Brown Classics and Lohmann Selected Leghorns purchased from the same pullet breeder with the same vaccination protocol. Mortality and egg production in this flock and in the other two flocks were not affected.

Discussion

As in the case presented here, the first indicator for erysipelas in a layer flock is an increased mortality in older leghorn hens scattered throughout the house, a few unspecific clinical signs in the surviving chickens and an increased infestation with the poultry red mite (*Dermanyssus gallinae*) at the end of the laying cycle (Bisgaard and Olsen, 1975; Bisgaard et al., 1980; Mutalib et al., 1993; Mazaheri et al., 2005; Bricker, 2008; Stokholm et al., 2010). Notably, in contrast to previous reports, primarily brown layers were affected (Bisgaard et al., 1980). An unusual feature in this case was a high daily mortality with 2 to 7% per day for at least four days without any other concurrent infection.

At necropsy, beside the known unspecific macroscopic lesions, such as a general hyperaemia and splenomegaly, an unusual symptom of swollen eyes with injected conjunctival vessels was determined in this case, which

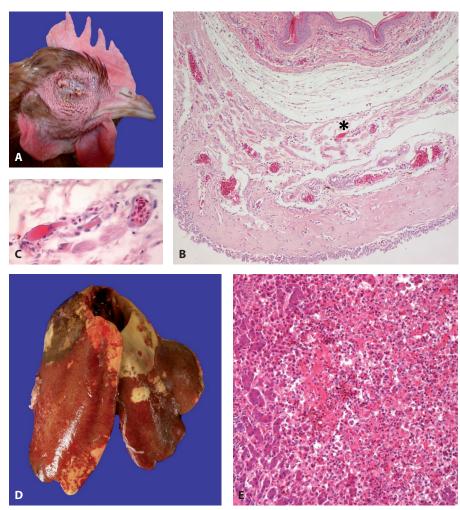


FIGURE 2: Pathological and histopathological findings. A: Brown layer. Swollen eye region with encrustation and lacrimation. B: Conjunctiva. Hyperaemia and oedema; the asterisk indicates fibrin thrombus in a small vessel, HE, 100x. C: Conjunctiva. Higher magnification of fibrin thrombus from Figure 2B, HE, 400x. D: Liver. Severe fibronecrotic hepatitis. E: Liver. Acute necrosis with fibrin exudation and mild granulocytic infiltration, HE, 200x.

has not been described in other cases of erysipelas in poultry before. Histopathology revealed DIC in conjunctival small vessels as the cause of the oedema of the eye adnexes. Also other authors described thrombi within small vessels of poults as a strong indicator of erysipelas since DIC is uncommon in poultry (Bickford et al., 1978; Shibatani et al., 1997; Bricker, 2008). Therefore, erysipelas should be included as an important differential diagnosis to Avian influenza, Newcastle Disease and Pasteurellosis sharing the symptome of conjunctival oedema and high daily mortality in older layer flocks.

Factors that lead to an outbreak and contribute to the severity of an outbreak are numerous and often multifactorial. In the case described here, the weather was cold and rainy for a few weeks. Cold, rainy weather is thought to increase outbreaks (Bricker, 2008), but Bisgaard did not observe this correlation (Bisgaard et al., 1980). Additionally, lacerations due to toe-clipping, beak trimming, sharp edges, fights and insects can pave the way for erysipelas. Other factors include hysteria, cannibalism, moult and bad hygienic practices, such as the rare removal of cadavers (Bisgaard et al., 1980; Butcher and Panigrahy, 1985; Bricker, 2008).

Although several potential vectors were tested, we failed to determine the route of infection of the flock. A trapped rat was negative for E. rhusiopathiae. We cannot exclude definitely a transmission of E. rhusiopathiae from the pigs to the nearby poultry house, but the pigs were clinically healthy all the time. Interestingly, collected poultry red mites from the affected flock repeatedly cultured positive for E. rhusiopathiae. In the adjacent flock, mite infestation was very low and the flock was younger. Several case reports of erysipelas described a high infestation of poultry with mites in affected flocks (Bisgaard et al., 1980; Chirico et al., 2003; Mazaheri et al., 2005; Eriksson et al., 2010). It therefore seems plausible to assume that red mites may have contributed to a mechanical transmission of the bacterium between chickens in the flock.

The drug of choice still is penicillin, but oxytetracyclin, tylosin and fluorchinolons can be used as well. Resistance to these antibiotics has not been described and minimal inhibitory concentrations for the most common antibiotics are still very low (Reboli and Farrar, 1989; Eriksson et al., 2009; Wang et al., 2010). Some authors recommend not treating affected flocks (Bisgaard et al., 1980) because infection is self-limiting and relapses can occur (Bisgaard et al., 1980; Wang et al., 2010). We used penicillin treatment success-

fully because mortality and egg production remained stable with normal mortality and egg production.

Because E. rhusiopathiae is sensitive to most disinfectant hygienic measures, thorough cleaning and disinfection are important to prevent subsequent outbreaks of erysipelas (Bricker, 2008; Wang et al., 2010). Treatment with an acarizide against poultry mites is also recommended, due to the probable transmission of E. rhusiopathiae through the mites (Chirico et al., 2003). Additionally, vaccination against *E. rhusiopathiae* can be considered, although vaccine failure can occur due to different serotypes (Bisgaard et al., 1980; Chirico et al., 2003) and sometimes even if the serotypes do not differ (Bisgaard et al., 1980; Hafez et al., 2001; Bricker, 2008; Eriksson et al., 2010; Stokholm et al., 2010). A possible explanation is that not all strains of *E. rhusiopathiae* are similarly virulent and that the serotype does not coincide with virulence (Bisgaard et al., 1980; Takahashi et al., 1994; Bricker, 2008; Wang et al., 2010). In our case, the owner declined vaccination of the following flock due to high vaccine production costs for small flock numbers. To date, no signs of erysipelas have been noted, and the flock performs well.

As alternative keeping systems become more prevalent, diseases of minor importance in caged layer operations reappear and cause a substantial financial impact on egg production. In free-range layer productions, a disease on the increase is erysipelas (Bisgaard and Olsen, 1975; Bisgaard et al., 1980; Stokholm et al., 2010; Eriksson et al., 2013). There are different opinions of financial importance, ranging from high impact on costs to nearly none (Bisgaard and Olsen, 1975; Bricker, 2008; Wang et al., 2010). In the case reported here we presume that a very pathogenic E. rhusiopathiae strain induced a high daily mortality. The overall mortality was 34% and egg production was severely reduced for the rest of the laying period. This resulted in a significant financial loss. Although erysipelas in laying hens is rarely reported in the literature, it can develop to an emerging disease and should always be considered if mortality increases in an older flock, especially with a high infestation of poultry red mites.

Acknowledgement

We thank Astrid Nagel, Birgit Isele-Rüegg, Fritz Fassler and Thomas Wagner for excellent technical assistance. Furthermore we are grateful for broad information provided by the farmer and critical revision of the manuscript by Philipp Olias. Parts of this work were financially supported by the Free State of Bavaria and the Bavarian Joint Founding Scheme for the Control and Eradication of contagious Lifestock Diseases.

Conflict of interest: The corresponding author confirms that there are not any conflicts of interest influencing this study.

References

- Bender JS, Shen HG, Irwin CK, Schwartz KJ, Opriessnig T (2010): Characterization of Erysipelothrix species isolates from clinically affected pigs, environmental samples, and vaccine strains from six recent swine erysipelas outbreaks in the United States. Clin Vaccine Immunol 17: 1605–1611.
- **Bickford AA, Corstvet RE, Rosenwald AS (1978):** Pathology of experimental erysipelas in turkeys. Avian Dis 22: 503–518.
- **Bisgaard M, Olsen P (1975):** Erysipelas in egg-laying chickens: clinical, pathological and bacteriological investigations. Avian Pathol 4: 59–71.
- **Bisgaard M, Norrung V, Tornoe N (1980):** Erysipelas in poultry. Prevalence of serotypes and epidemiological investigations. Avian Pathol 9: 355–362.
- Bricker JM (2008): Erysipelas. In: Saif YM, Fadly AM, Glisson JR, McDougald LR, Nolan LK, Swayne DE (eds.), Diseases of Poultry. Blackwell Publishing, Singapore.
- **Butcher G, Panigrahy B (1985):** An outbreak of erysipelas in chukars. Avian Dis 29: 843–845.
- Chirico J, Eriksson H, Fossum O, Jansson D (2003): The poultry red mite, *Dermanyssus gallinae*, a potential vector of *Erysipelo-thrix rhusiopathiae* causing erysipelas in hens. Med Vet Entomol 17: 232–234.
- Eriksson H, Jansson DS, Johansson KE, Baverud V, Chirico J, Aspan A (2009): Characterization of Erysipelothrix rhusiopathiae

- isolates from poultry, pigs, emus, the poultry red mite and other animals. Vet Microbiol 137: 98–104.
- Eriksson H, Brannstrom S, Skarin H, Chirico J (2010): Characterization of *Erysipelothrix rhusiopathiae* isolates from laying hens and poultry red mites (*Dermanyssus gallinae*) from an outbreak of erysipelas. Avian Pathol 39: 505–509.
- **Eriksson H, Nyman AK, Fellstrom C, Wallgren P (2013):** Erysipelas in laying hens is associated with housing system. Vet Rec 173: 18.
- Hafez HM, Mazaheri A, Prusas C, Böhland K, Pöppel M, Schulze D (2001): Aktuelle Geflügelkrankheiten bei Legehennen im Zusammenhang mit alternativen Haltungssystemen. Tieraerztl Prax 29: 168–174.
- **Kurian A, Neumann EJ, Hall WF, Marks D (2012):** Serological survey of exposure to *Erysipelothrix rhusiopathiae* in poultry in New Zealand. N Z Vet J 60: 106–109.
- **Mazaheri A, Lierz M, Hafez HM (2005):** Investigations on the pathogenicity of *Erysipelothrix rhusiopathiae* in laying hens. Avian Dis 49: 574–576.
- Mazaheri A, Philipp HC, Bonsack H, Voss M (2006): Investigations of the vertical transmission of *Erysipelothrix rhusiopathiae* in laying hens. Avian Dis 50: 306–308.
- Mutalib AA, King JM, McDonough PL (1993): Erysipelas in caged laying chickens and suspected erysipeloid in animal caretakers. JVet Diagn Invest 5: 198–201.
- **Reboli AC, Farrar WE (1989):** *Erysipelothrix rhusiopathiae*: an occupational pathogen. Clin Microbiol Rev 2: 354–359.
- Shibatani M, Suzuki T, Chujo M, Nakamura K (1997): Disseminated intravascular coagulation in chickens inoculated with *Erysipelothrix rhusiopathiae*. J Comp Pathol 117: 147–156.
- **Stokholm NM, Permin A, Bisgaard M, Christensen JP (2010):**Causes of mortality in commercial organic layers in Denmark. Avian Dis 54: 1241–1250.
- Takahashi T, Takagi M, Yamaoka R, Ohishi K, Norimatsu M, Tamura Y, Nakamura M (1994): Comparison of the pathogenicity for chickens of *Erysipelothrix rhusiopathiae* and *Erysipelothrix tonsillarum*. Avian Pathol 23: 237–245.
- Takahashi T, Nagamine N, Kijima M, Suzuki S, Takagi M, Tamura Y, Nakamura M, Muramatsu M, Sawada T (1996): Serovars of Erysipelothrix strains isolated from pigs affected with erysipelas in Japan. J Vet Med Sci 58: 587–589.
- Wang Q, Chang BJ, Riley TV (2010): Erysipelothrix rhusiopathiae. Vet Microbiol 140: 405–417.

Address for correspondence:

Dr. Benjamin Schade Bavarian Animal Health Service Senator-Gerauer-Straße 23 85586 Poing Germany benjamin.schade@tgd-bayern.de