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Enterococcus cecorum, an opportunistic poultry pathogen: deeper understanding for better farm control

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■ ***Enterococcus cecorum*** **has become a major pathogen of poultry worldwide. This bacterium causes locomotor disorders that lead to mortality, increased use of antibiotics and economic losses, particularly in fast-growing broiler farms. This article summarises current epidemiological, zootechnical and basic knowledge with a view to improving disease control and prevention on farms.[1]**

Introduction

Diseases affecting poultry typically have multiple causes, including interactions between environmental conditions, genetics, husbandry practices, and the immune status or susceptibility of the animals. In the last five decades, broiler growth rate has increased five-fold between 29 and 35 days of age (Zuidhof *et al.*, 2014). This rapid increase in body mass exerts mechanical stress on the birds' skeletal systems, facilitating invasion and colonisation of joints by opportunistic bacteria. Such bone infections are most often associated with *Staphylococcus aureus*, *Escherichia coli* or *Enterococcus cecorum* (Wideman, 2016; Wijesurendra *et al.*, 2017). Over the last fifteen years, locomotor pathologies caused by *E. cecorum* have gradually reached a worrying level in broiler farms, affecting both animal health and welfare.

This review attempts to establish a link between current physiological and molecular knowledge of *E. cecorum* and pathology on farms. It also looks at possible modes of transmission, detection methods and current treatments, as well as preventive zootechnical and biosecurity measures to contain the spread of this bacterium within and between farms. Lastly, it discusses strategies of research and development for controlling this pathogen on farms (Figure 1).

1. An opportunistic pathogen in broiler farming

■ 1.1 *E. cecorum*, a commensal bacterial agent of the intestinal tract of poultry

E. cecorum was first described in 1983 in Belgium, where it was isolated from the caecal contents of a dead chicken, under the name *Streptococcus cecorum* (Devriese *et al.*, 1983). In 1989, it was reclassified within the *Enterococcus* genus, which includes more than 75 species of ubiquitous bacteria found in the intestinal microbiota of terrestrial animals, including birds (Parks *et al.*, 2020; Schwartzman *et al.*, 2024).

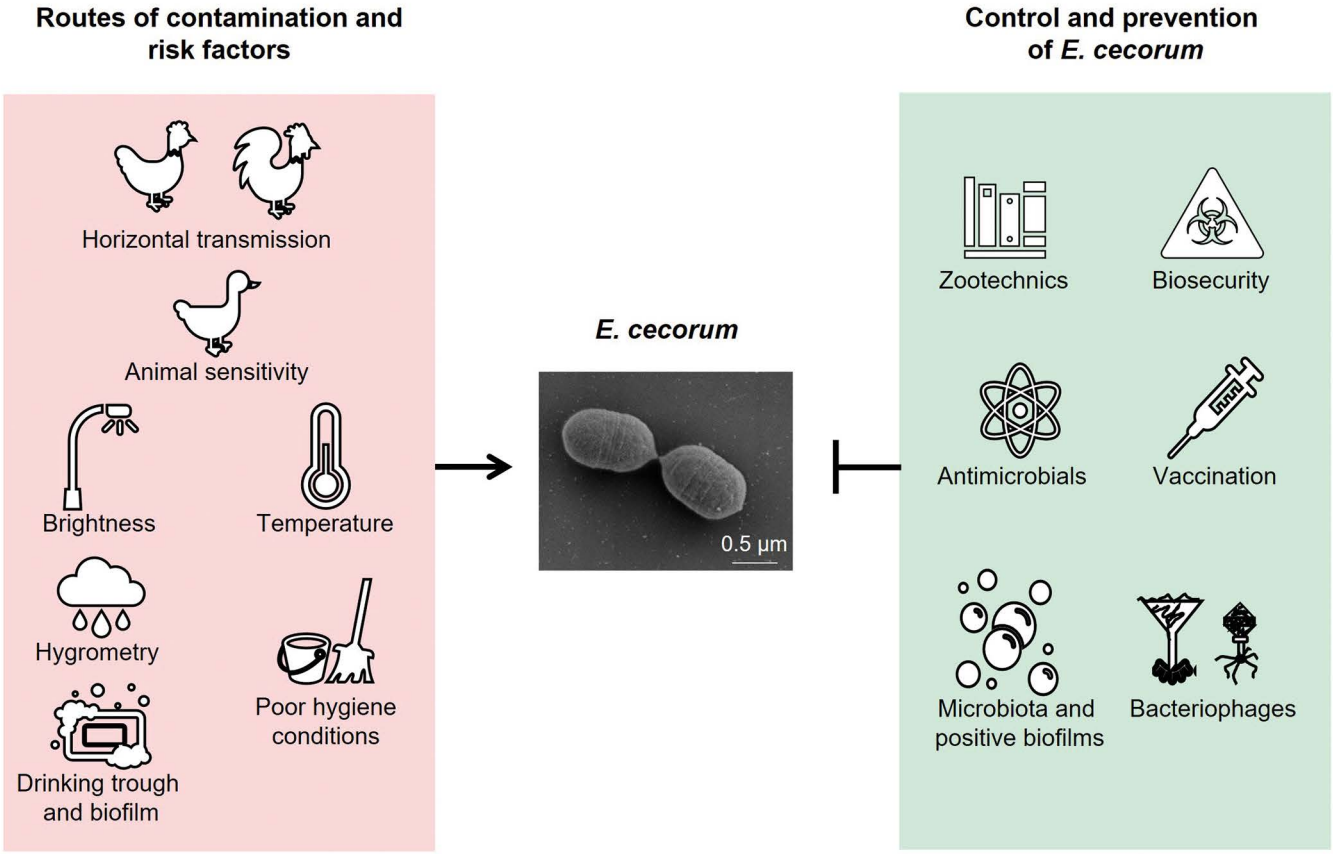
E. cecorum is a Gram-positive cocci-shaped facultative anaerobic and non-spore-forming bacterium (Jung *et al.*, 2018). It is a commensal bacterium in the intestinal microbiota of poultry, particularly chickens (Devriese *et al.*, 1991b). It is also found in the intestinal tract of other animals such as pigs, cattle, horses, ducks and turkeys (Devriese *et al.*, 1991a; Scupham *et al.*, 2008). *E. cecorum* has the ability to form biofilms on inert surfaces (Grund *et al.*, 2022; Laurentie *et al.*, 2023a), and is an opportunistic agent causing locomotor disorders in poultry (Jung *et al.*, 2018).

■ 1.2. Clinical signs

E. cecorum pathologies are primarily observed in chickens, although

1 This article was presented at the 15th Journées de la Recherche Avicole et Palmipèdes à Foie gras, 20-21 March 2024 in Tours (Souillard *et al.*, 2024).

Figure 1. ***Possible routes of contamination by*** **E. cecorum** ***and preventive measures.***



Routes of contamination and risk factors that may favour the presence of *E. cecorum* in broiler farms (left). Development and research strategies for controlling the pathogen (right). Scanning electron microscopy photo of *E. cecorum,* centre of figure (Photo: © T. Meylheuc and P. Serror, MIMA2-Micalis).

cases have also been reported in other poultry species, such as ducks and turkeys (Dolka *et al.*, 2017; Souillard *et al.*, 2022). The signs of the disease include reduced feed consumption, uneven growth, dehydration and increased mortality ranging from 7 to over 10% (Robbins *et al.*, 2012; Jung & Rautenschlein, 2014). An initial septicaemic phase results in lesions of pericarditis, fibrinous perihepatitis and splenomegaly (Jung & Rautenschlein, 2014). Locomotor disorders and lameness most often appear from three to four weeks of age (Stalker *et al.*, 2010; Jung & Rautenschlein, 2014; Borst *et al.*, 2017). The characteristic clinical sign of locomotor pathology in chickens infected with *E. cecorum* is birds sitting on their hocks (Jung *et al.*, 2018). Typical of enterococcal spondyloarthritis, this sign results from paralysis caused by the development of an inflammatory lesion in the free thoracic vertebra that compresses the spinal cord (Jung & Rautenschlein, 2014). Arthritis, synovitis and necrosis of the femoral heads have also been observed (Stalker *et al.*, 2010; Borst *et al.*, 2012; Jung & Rautenschlein, 2014). Sepsis and bone lesions result in carcass condemnations at slaughter, with rates reaching up to 9.75%. (Jung & Rautenschlein, 2014). Diseases caused by *E. cecorum* thus result in significant economic losses on farms.

■ 1.3 History and epidemiology

E. cecorum emerged as a poultry pathogen in the early 2000s. The first cases were reported in Scotland (Wood *et al.*, 2002) and the Netherlands (Devriese *et al.*, 2002), then more widely in other European countries (Makrai *et al.*, 2011; Szeleszczuk *et al.*, 2013; AMCRA, 2021) and North America (Stalker *et al.*, 2010; Borst *et al.*, 2012). In France, there has been a noticeable increase in pathologies associated with *E. cecorum* in poultry over the last 15 years. Data from RNOEA, the French poultry epidemiological surveillance network, shows a significant emergence of this bacterium. In 2006, *Enterococcus* represented only 0.4% of all pathogens reported, whereas this proportion had risen to 12.9% by 2020 (Souillard *et al.*, 2022). Most diseases associated with *Enterococcus* were found in broilers. Furthermore, locomotor disorders, which were caused by *E. cecorum*, increased from 6% in 2017 to 14.8% in 2022. Among all transmitted pathogens in broilers reported to the network in 2022 for this production (n = 4 289), *E. cecorum* was second only to *E. coli*, accounting for 16.3% and 63.6%, respectively. This emergence can be explained in part by increased vigilance on the part of veterinarians in the field and improvements in laboratory diagnostic methods since the 2010s (Dolka *et al.*, 2017; Karunarathna *et al.*, 2017; Suyemoto *et al.*, 2017; Tessin *et al.*, 2024). However, *E. cecorum* pathology is now a major disease in broiler production worldwide.

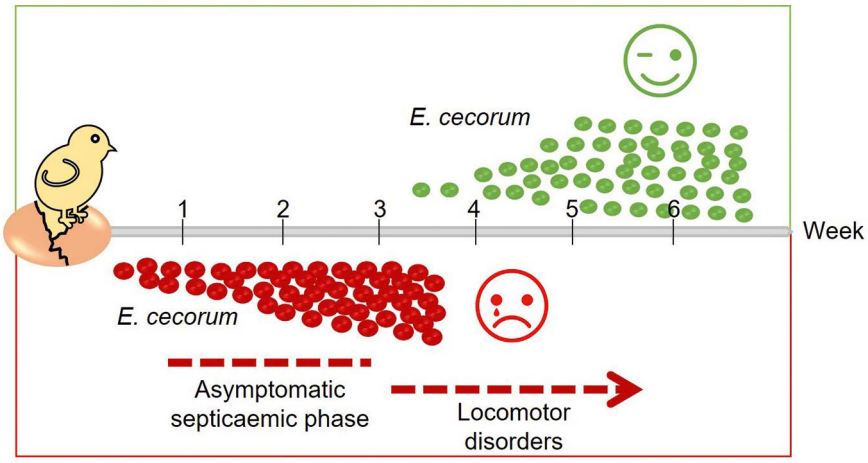
■ 1.4. Pathogenesis

Clinical strains of *E. cecorum* appear to be adapted to colonise the gut from the first days of life, whereas commensal isolates do not appear to colonise the gut at a detectable level until the third week of life (Devriese *et al.*, 1991b; Borst *et al.*, 2017) (Figure 2). Indeed, Borst *et al.* (2017) showed that in farms with clinical episodes of *E. cecorum,* the bacterium was detected in the intestinal contents as early as the first week of life in 60% of animals. Conversely, *E. cecorum* was only detectable from the third week of life in 30% of animals in farms without an infectious episode (Borst *et al.*, 2017). According to the current model, the bacterium enters the bloodstream after colonising the intestine and crossing the intestinal mucosa, which explains why it is detected in organs such as the heart, liver and spleen during the early phase of infection (Figure 3). During this septic phase, *E. cecorum* is thought to spread to skeletal sites, including the thoracic vertebrae, femoral heads and joints, causing inflammation that leads to lameness and paralysis (Borst *et al.*, 2017). The resistance of clinical isolates of *E. cecorum* to high concentrations of lysozyme could give them an ecological advantage in the early colonisation of chicks (Manders *et al.*, 2024).

■ 1.5. Virulence or opportunism

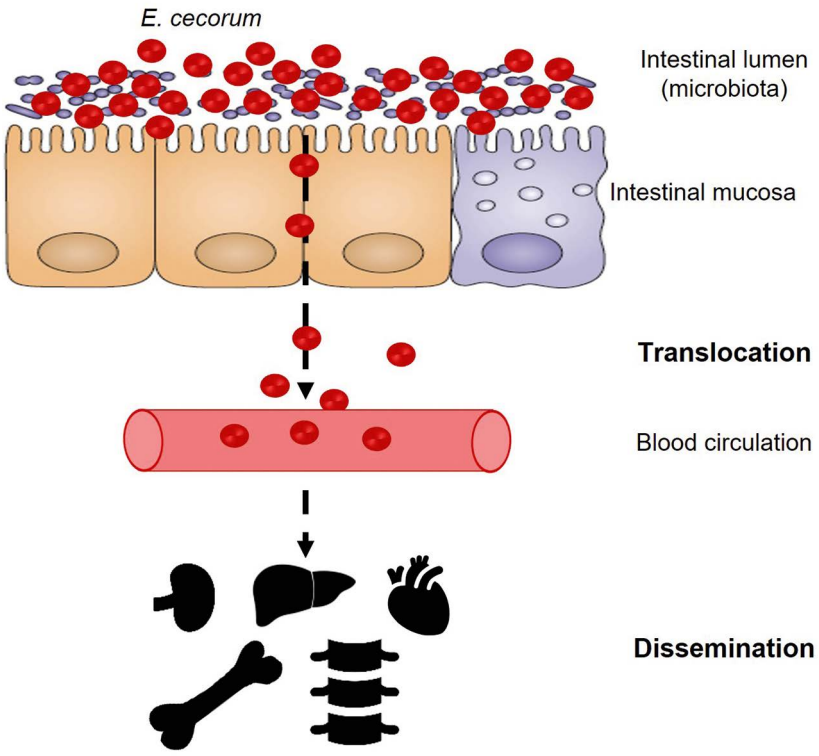
Several molecular epidemiology studies have used pulsed-field electrophoresis (PFGE) to profile commensal and clinical isolates. Results from the USA, Canada, Belgium, the Netherlands, Germany and Poland have shown that commensal isolates are more diverse than clinical isolates, suggesting the evolution of specific clones with higher pathogenic potential (Kense & Landman, 2011; Boerlin et al, 2012; Borst *et al.*, 2012; Robbins *et al.*, 2012; Huang *et al.*, 2023). Genomic analysis of approximately one hundred avian clinical isolates collected in France between 2007 and 2017 confirmed the clonal nature of avian clinical isolates, which are part of a phylogenetic clade found in the United States and Europe (Laurentie *et al.*, 2023a). More recently, a phylogenetic study of about thirty clinical strains from American farms, isolated from septicaemic animals during the first three weeks of life, seems to raise doubts concerning the clonal nature of the clinical isolates responsible for lameness by revealing a septicaemia-associated clone (Rhoads *et al.*, 2024). Mutations identified in seven genes conserved across all strains appear to reflect host adaptation. At present, we cannot rule out the emergence of a new clone responsible for septicaemia. It would be interesting to assess the infectious potential of these strains and determine their ability to induce lameness.

Figure 2. ***Temporal association between the presence of* E. cecorum *and clinical signs.***



E. cecorum colonises the intestine of healthy chickens at a detectable level from the third week. In clinically-affected flocks, *E. cecorum* is detected as early as the first week in the intestines of chicks.

Figure 3. ***Model of* E. cecorum *infection in chicks.***



Early colonisation of the intestine by *E. cecorum* (during the first week) allows the bacteria to cross the intestinal mucosa and spread throughout the body. *E. cecorum* then reaches the organs (liver, heart, kidney) and bone sites that cause the inflammatory lesions responsible for lameness and paralysis.

Despite these advances, distinguishing between clinical and commensal strains remains a challenge. The identification of genes preferentially found in clinical isolates could help to distinguish them from non-clinical isolates (Borst *et al.*, 2015; Huang *et al.*, 2023; Laurentie *et al.*, 2023a). For example, the capsule protects bacteria from phagocytosis and the prevalence of certain capsule genes in clinical isolates suggests a role in virulence (Huang *et al.*, 2023; Laurentie *et al.*, 2023a). Other genes more frequently found in the genomes of clinical isolates may contribute to virulence by encoding functions that are likely to provide alternative metabolic capacities for survival and reproduction within the host (Borst *et al.*, 2015; Laurentie *et al.*, 2023a; Rhoads *et al.*, 2024). Currently, there is no infection model to distinguish clinical from non-clinical isolates or to study the impact of specific genes on virulence. The lethality test on embryonated chicken eggs has shown significant variations in mortality and lesions between isolates, but its relevance and reliability need to be consolidated due to disparities between studies (Borst *et al.*, 2014; Ekesi *et al.*, 2021; Dolka *et al.*, 2022; Huang *et al.*, 2023; Laurentie *et al.*, 2023a). Oral inoculation in two-week-old birds was found to be more effective than intravenous inoculation or air sacs in reproducing spinal lesions, as evidenced by macroscopic and microscopic examination for spinal lesions (Martin *et al.*, 2011). The team of A. Jung's team in Germany described an oral infection model in day-old chicks that was as close as possible to field conditions. Although this model reproduced the septic phase and late clinical signs in up to 20% of infected birds, only one of the two clinical isolates tested proved virulent (Schreier *et al.*, 2021). Recently, an *in ovo* infection model at 18 days of embryonic development detected the bacterium in the femoral head and free thoracic vertebra in over 60% of birds (Arango *et al.*, 2023). However, as the presence of *E. cecorum* was not tested in the intestinal microbiota, it is not possible to determine whether the bone infection resulted from the initial infection or from early intestinal colonisation. Distinguishing between commensal and clinical isolates of *E. cecorum* remains challenging due to the lack of reliable molecular or phenotypic tools.

■ 1.6. Antibiotic resistance

Several North American and European studies show a high prevalence of tetracycline resistance (>70%) in clinical and non-clinical isolates of *E. cecorum,* while macrolide resistance (erythromycin, spiramycin and tylosin) is more common in clinical isolates (Jung *et al.*, 2018; Laurentie *et al.*, 2023b). Conversely, lincomycin resistance is more prevalent in isolates of non-clinical origin, which have more extensive antibiotic resistance profiles than clinical isolates (Boerlin *et al.*, 2012; Borst *et al.*, 2012; Jackson *et al.*, 2015; Laurentie *et al.*, 2023b). In France, 43.3% of isolates appear to be resistant to more than three classes of antibiotics, compared to 1.4% of isolates sensitive to the twenty or so molecules tested (Laurentie *et al.*, 2023b). Nevertheless, the majority of isolates of clinical origin remain sensitive to the antimicrobials authorised for use in poultry. Similarly, it is reassuring to note that resistance to antimicrobials of critical importance in human medicine such as vancomycin, gentamicin, tigecycline, linezolid and daptomycin is not widespread in *E. cecorum* (Laurentie *et al.*, 2023b) (Figure 4). These trends are validated by the distribution of resistance genes in genomes (Sharma *et al.*, 2020; Laurentie *et al.*, 2023b; Huang *et al.*, 2024). Tetracycline resistance genes (*tet(M)* and *tet(L)*), macrolide resistance genes (*erm(B)*) and, to a lesser extent, bacitracin resistance genes (*bcr* operon) are the most common. Chromosomal mutations in the *gyrA* and *parC* genes explain more than 65% of quinolone resistance (Laurentie *et al.*, 2023b) and a *pbp2* gene could contribute to ampicillin resistance (Huang *et al.*, 2024). While no plasmids have been described in *E. cecorum,* the most common resistance genes are carried by complex genetic elements that have all the characteristics of mobile elements.

■ 1.7 Predisposing factors in animals

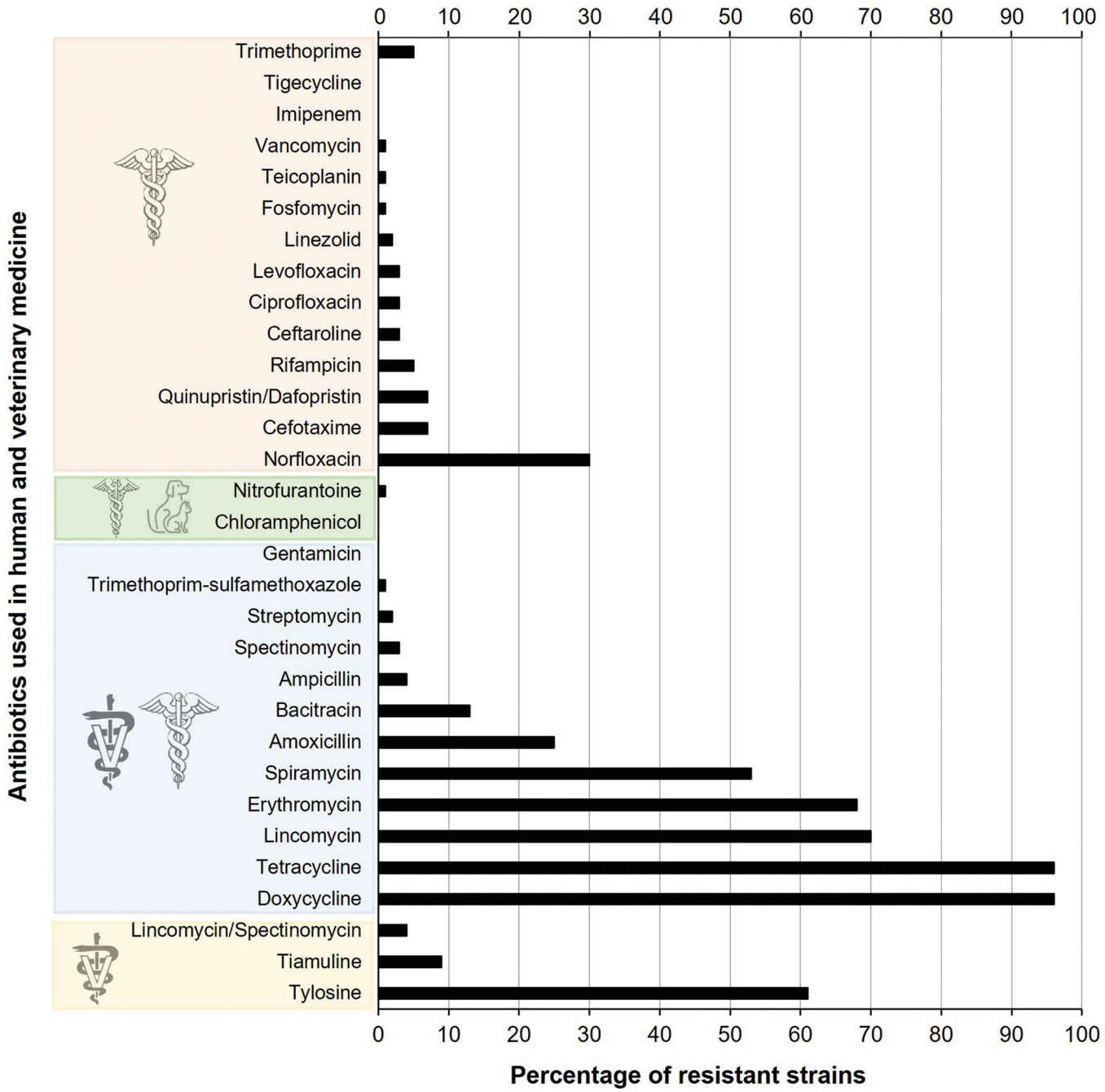
Animal sensitivity can influence the development of locomotor pathologies caused by *E. cecorum*. Increased body weight in poultry can lead to mechanical stresses on the locomotor system, leading to bone lesions with colonisation by various opportunistic bacteria (Wideman, 2016). Abnormal development of the locomotor system, such as early osteochondrosis lesions, could also predispose poultry to *E. cecorum* infections (Borst *et al.*, 2017). However, no comparative studies on predisposition to infection as a function of lineage growth rate have been published thus far. A reduction in animal immunity or concomitant infections could also play a role in the development of the disease. The hypothesis of an alteration in the intestinal barrier favouring translocation of the bacteria has been raised in relation to intercurrent infections (*E. coli* or *Eimeria* parasites) or changes in the microbiota (Borst *et al.*, 2017). However, co-infection of *E. cecorum* with a mixture of three *Eimeria* spp. species, including *Eimeria tenella*, has recently been associated with a decrease in the incidence of *E. cecorum* bacteraemia and the severity of spondyloarthritis (Borst *et al.*, 2019). This result could be explained by a change in the intestinal microbiota, especially as a strain of *E. tenella* has been shown to contribute to the diversification of the caecal microbiota and the strengthening of the intestinal mucosa (Zhou *et al.*, 2020). Heat stress, which causes changes in the composition of the intestinal microbiota, could also alter the integrity of the intestinal mucosa and promote *E. cecorum* translocation (Schreier *et al.*, 2022b). While none of these factors is sufficient to trigger infection by *E. cecorum,* their combination could contribute to additive risk.

2. Which transmission route(s)?

■ 2.1. Vertical transmission

How poultry are contaminated and how *E. cecorum* is introduced into farms is still poorly understood. The

Figure 4. ***Distribution of antibiotic resistance in*** **E. cecorum** ***strains.***



Out of 208 strains of *E. cecorum*, 43.3% are resistant to more than three classes of antibiotics and a large majority are resistant to tetracyclines (>90%) and macrolides (>60%) (according to Laurentie *et al.*, 2023b).

possibility of vertical transmission has been studied, with inconclusive results. Indeed, genomic and metabolomic patterns of *E. cecorum* from breeding birds and the clinical isolates from their offspring have not shown any correlation and the bacterium has not been found in the hatchery environment (Kense & Landman, 2011; Robbins *et al.*, 2012). Furthermore, experimental infection of breeder chickens failed to detect the presence of the bacterium in eggs (Thofner & Christensen, 2016). Although vertical transmission of *Enterococcus* species has been demonstrated, this does not seem to involve *E. cecorum* (Shterzer *et al.*, 2023). Current research is limited, and further investigation is required to explore the possibility of vertical transmission of the bacterium in broiler farms.

■ 2.2. Horizontal transmission

Horizontal transmission can result from direct contact between animals or indirectly, via contaminated equipment. Birds excrete *E. cecorum* in their droppings, and can thus become infected via the faecal-oral route (Borst *et al.*, 2017). In addition, the bacterium could be transmitted by inhalation of

contaminated dust from farm houses (Jung & Rautenschlein, 2014). The frequency of recurrences on farms, as reported in Belgium (Herdt *et al.*, 2008) and France (Potier et al, 2024) suggests that there may be biological reservoirs such as darkling beetles, rodents, etc., or environmental reservoirs in areas of persistence difficult to clean and disinfect, such as feeding lines, ventilation and heating systems, as well as drinking pipettes, feeders and cracks in the floor (Luyckx *et al.*, 2015; Tessin *et al.*, 2024). A recent study shows a higher prevalence in summer (Dunnam *et al.*, 2023). The bacterium survives on different substrates (litter, dust, plastic) at different temperatures and humidity levels, particularly on litter at 15°C and 32% humidity (Grund *et al.*, 2021). In this study, the two clinical strains showed prolonged survival compared with the commensal strain. Although *E. cecorum* has not been isolated from the environment of farms affected by the bacterium (Robbins *et al.*, 2012; Grund *et al.*, 2022), the presence of DNA from the 16S gene of *E. cecorum* was detected in the drinking systems (Grund *et al.*, 2022) and also after cleaning in the air admission circuit and the anteroom (Tessin *et al.*, 2024). Further research is therefore needed to provide a better understanding of the *E. cecorum* contamination routes and of persistence areas on the farms.

3. Diagnosis and treatment

■ 3.1. Diagnosis of the disease

Lameness, with potential paralysis characterised by a "sitting on the hocks" position, increased mortality and arthritic lesions, necrosis of the femoral heads or spondyloarthritis are clinical signs suggestive of *E. cecorum* infection. Other bacterial agents, such as *E. coli,* can also cause these clinical signs and lesions in poultry. Bacteriological analysis is therefore necessary to confirm the diagnosis of *E. cecorum* infection. Isolation of *E. cecorum* from lesions is classically performed in the presence of CO_2 (5%) on a blood agar medium to which colistin and nalidixic acid may be added to inhibit the growth of Gram-negative bacteria. Identification is currently carried out using MALDI-TOF (matrix-assisted laser desorption-ionisation time-of-flight) mass spectrometry, which is easier and more reliable than PCR identification (Karunarathna *et al.*, 2017). This development has certainly contributed to the increase in the identification of *Enterococcus* and *E. cecorum* species in avian pathology since 2006 (Souillard *et al.*, 2022).

■ 3.2. Treatment

Antibiotic susceptibility tests for *E. cecorum* are performed according to *E. faecalis* or *E. faecium* standards (Borst *et al.*, 2012; Jackson *et al.*, 2015; Dolka *et al.*, 2016). Epidemiological cut-offs (ECOFFs) have been provisionally established for about twenty antibiotics in *E. cecorum* (Laurentie *et al.*, 2023b). These require independent validation by other laboratories on a larger collection of strains before they can be adjusted and approved by the European Committee for Antimicrobial Susceptibility Testing (EUCAST). A better assessment of the antibiotic resistance of strains should help to improve the effectiveness of treatments and thus better control the use of antibiotics in livestock farming.

Currently, if an *E. cecorum* infection is diagnosed, early antibiotic treatment must be implemented rapidly to limit the spread of infection within a poultry flock. Treatment is ineffective on birds already exhibiting lesions and paralysis. If the choice of antibiotic is based on the results of the antibiogram, penicillin derivatives, in particular amoxicillin, are commonly used (Devriese *et al.*, 2002; Herdt *et al.*, 2008; Jung *et al.*, 2018), sometimes requiring repeated treatments within a flock. However, due to the frequent recurrence of the disease on certain farms and its severity, antibiotic treatment during the first week of life has been suggested to control the disease, using amoxicillin and/or tylosin (Herdt *et al.*, 2008) or lincospectin (Schreier *et al.*, 2022a). In accordance with Regulation (EU) no 2019/6, which regulates the use of antibiotics in animals to reduce antibiotic resistance, the use of antimicrobial medicinal products in a poultry flock for prophylactic purposes must remain exceptional "when the risk of infection or infectious disease is very high and the consequences are likely to be serious" (Article 107 of Regulation (EU) no 2019/6). It is essential to control the circumstances under which *E. cecorum* infections occur, in order to implement preventive measures based on farm management and sanitary measures.

4. Possible measures to implement on poultry farms to prevent the spread of infection

■ 4.1. Farm management to limit risk

Various zootechnical measures have been proposed to limit the risk of *E. cecorum* pathologies (Figure 1). Among these, adjusting lighting programmes to slow animal growth during the first few weeks of life could limit the impact of the disease (Jung *et al.*, 2018). Insufficient darkness in buildings has been identified as a risk practice. Less than six hours of light cut-off at ten days of age would constitute an aggravating practice (Remiot *et al.*, 2019). In addition, increasing skeletal strength and limiting mechanical stress on the musculoskeletal system could help reduce the risk of bone lesions and *E. cecorum* infections. Optimising early rearing conditions with good litter quality, sufficient air ventilation and temperature control could also limit the risk of disease (Remiot *et al.*, 2019; Schreier *et al.*, 2022b). Disease prevention also involves preventing digestive disorders and intercurrent infections that could promote infection, such as coccidiosis (Borst *et al.*, 2017) or immunosuppressive diseases such as Gumboro disease (Sary *et al.*, 2018). Finally, the frequency of recurrences suggests that *E. cecorum* persists in the environment, including farm buildings, even though the presence of viable bacteria has not been demonstrated due to the lack of a selective culture medium (Tessin *et al.*, 2024). The presence of *E. cecorum* in watering systems is still a matter of debate (Grund *et al.*, 2022; Tessin *et al.*, 2024). It has also been shown that inadequate cleaning

and disinfection could be a risk factor (Remiot *et al.*, 2019) and that *E. cecorum* DNA could be detected after the cleaning/disinfection of houses, in the anteroom, pipes and air circuits (Tessin *et al.*, 2024). The potential environmental persistence of *E. cecorum,* evidenced by detection even after cleaning and disinfection, underscores the need to reinforce biosecurity (compliance with the anteroom and protocols for changing clothes and washing hands, disinfection of equipment) and hygiene practices (disinfection of houses and water pipes) to better prevent the disease.

An alternative way of preventing the proliferation of undesirable bacteria on farms is to use mixtures of bacteria to form a protective biofilm on buildings before chick arrival (Guéneau *et al.*, 2022a) (Figure 5). The application of a mixture of *Bacillus* spp. and *Pediococcus* spp. strains could limit the development of enterobacteria and enterococci (Guéneau *et al.*, 2022b). This measure could contribute to better environmental hygiene management for birds. The rotation of disinfection agents (biocides) and bacterial biofilms between two flocks should reduce the risk of emergence of resistant strains.

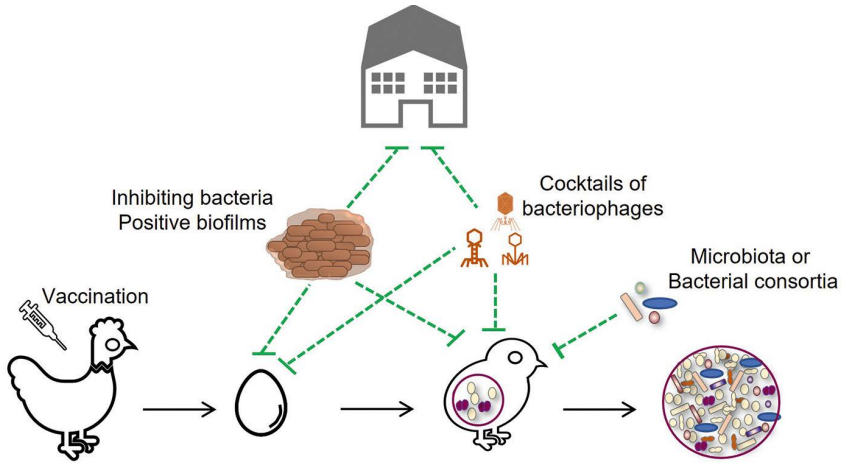
■ 4.2. Strategies for prevention

Vaccination of poultry could be a valuable tool for controlling *E. cecorum* diseases on farms (Figure 1). However, there is currently no commercial vaccine available, although research is ongoing. In particular, it has been shown that vaccination of breeding hens with a polyvalent inactivated vaccine against *E. cecorum* did not prevent the disease in chicks (Borst *et al.*, 2019). Serological ELISA methods have yet to be developed to assess serological responses to *E. cecorum* infection and vaccination in breeding hens and chicks (Jung & Rautenschlein, 2020; Silberborth *et al.*, 2024).

Modern poultry farming practices disrupt the natural vertical transmission of chicken microbiota by preventing contact between hens and chicks. As a result, the animals acquire a poorly diverse and ill-defined microbiota from their environment. This early disruption of colonisation can lead to an imbalance in the microbiota, or dysbiosis, and/or a lack of maturation of the intestinal mucosa and immune system, which can affect production performance and resistance to pathogens (Rubio, 2019; Rychlik, 2020). The colonisation of the initial microbiota is essential for inducing a good immune response (Rodrigues *et al.*, 2021). While the role of the intestinal microbiota in the infectious potential of *E. cecorum* has not been established, the intestinal detection of clinical isolates in the first two weeks and the efficacy of early treatment with lincospectin in preventing infection suggest a window of opportunity for intervention to promote the ecological barrier of the microbiota and the mucosal barrier (Hankel *et al.*, 2021; Schreier *et al.*, 2022a) (Figure 5). Among the various strategies considered in livestock farming to stimulate the ecological barrier of the intestinal microbiota, the use of probiotic strains such as *Bacillus* for their antagonistic activity is one of the oldest (Cutting, 2011). Various *Bacillus* strains have been isolated for their inhibitory activity against *E. cecorum* (Medina Fernández *et al.*, 2019; Penaloza-Vazquez *et al.*, 2019; Sandvang *et al.*, 2024). Their efficacy depends on the probiotic strains and the *E. cecorum* isolate targeted. Further work is needed to study their efficacy *in vivo* and characterise the inhibiting molecules. Understanding the role of the microbiota in the pathogenesis of *E. cecorum* is a prerequisite for developing new preventive strategies that could be based on the transfer of healthy microbiota or the implantation of *consortia* of avian probiotic or commensal bacteria.

Cocktails of bacteriophages are also available in some European countries and North America as food supplements or probiotics (Bacteriophage. news., n.d.; Gigante & Atterbury, 2019; Abd-El Wahab *et al.*, 2023). Phages lyse bacteria with the advantage of having a limited host spectrum, reducing collateral effects in the targeted microbiota. Their use in cocktails also reduces the risk of resistant strains emerging. To date, virulent phage specific to *E. cecorum* have not been described, but this approach is worth considering because of the genetic similarity of clinical strains. Administration of these phages during the first two weeks of rearing could help prevent or delay early colonisation of infectious isolates. However, whatever alternative methods are considered, it is essential to assess their environmental consequences on human and animal health, environmental microbial ecology and the risk of emergence of resistant strains.

Figure 5. ***Research strategies to prevent the proliferation of*** **E. cecorum** ***in the poultry houses and the chick environment.***



In addition to vaccinating breeding flocks against *E. cecorum,* two biological control methods need to be developed: bacterial consortia and the use of bacteriophage cocktails. These two methods are not mutually exclusive. They can be used to treat the chick's environment, eggs and poultry houses. They can also be used to protect chicks from intestinal colonisation by the pathogen via a "barrier effect" of the bacterial consortia and/or the elimination of by virulent bacteriophages.

Conclusion

Like many opportunistic pathogens, the virulence of *E. cecorum* is multifactorial. Its relatively recent emergence suggests a direct correlation with changes in rearing practices, which may have led to the selection of a clone capable of infecting fast-growing broilers chickens. It is essential to improve our zootechnical, epidemiological and fundamental knowledge of the physiology and pathogenesis of *E. cecorum* in order to implement effective measures for the prevention and control of the disease. To effectively manage this pathogen, it is essential to develop rapid diagnostic methods capable of distinguishing clinical from non-clinical strains, thus clarifying routes of transmission and farm contamination. The development of a robust animal model reproducing at least the early phase of infection is necessary to understand the interaction of *E. cecorum* with the microbiota and its host, using global approaches (metagenomics, metabolomics and proteomics). This knowledge should enable preventive strategies to be defined to limit the use of antibiotics and the associated selection pressure, as well as reducing the clinical impact, animal suffering and economic losses on farms.

Authors' contributions

All the authors took part in writing the article, coordinated and supervised by Pascale Serror.

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References

Abd-El Wahab, A., Basiouni, S., El-Seedi, H. R., Ahmed, M. F. E., Bielke, L. R., Hargis, B., Tellez-Isaias, G., Eisenreich, W., Lehnherr, H., Kittler, S., Shehata, A. A., & Visscher, C. (2023). An overview of the use of bacteriophages in the poultry industry: Successes, challenges, and possibilities for overcoming breakdowns. *Frontiers in Microbiology*, *14*, 1136638. https://doi.org/10.3389/fmicb.2023.1136638

AMCRA. (2021). *Mesures pour un bon usage des antibiotiques lors d'un traitement de groupe chez la volaille* (Rapport). Centre de connaissances antimicrobial consumption and resistance in animals. https://amcra.be/swfiles/files/Advies_Groepsbehandelingen_Pluimvee_Finaal_FR_goedgekeurd-RvB_finaal.pdf

Arango, M., Forga, A., Liu, J., Zhang, G., Gray, L., Moore, R., Coles, M., Atencio, A., Trujillo, C., Latorre, J. D., Tellez-Isaias, G., Hargis, B., & Graham, D. (2023). Characterizing the impact of *Enterococcus cecorum* infection during late embryogenesis on disease progression, cecal microbiome composition, and early performance in broiler chickens. *Poultry Science*, *102*(11), 103059. https://doi.org/10.1016/j.psj.2023.103059

Bacteriophage.news. (s. d.). *Updated bacteriophage product database* [Base de données]. Retrieved from https://www.bacteriophage.news/phage-products/

Boerlin, P., Nicholson, V., Brash, M., Slavic, D., Boyen, F., Sanei, B., & Butaye, P. (2012). Diversity of *Enterococcus cecorum* from chickens. *Veterinary Microbiology*, *157*(3-4), 405-411. https://doi.org/10.1016/j.vetmic.2012.01.001

Borst, L. B., Suyemoto, M. M., Robbins, K. M., Lyman, R. L., Martin, M. P., & Barnes, H. J. (2012). Molecular epidemiology of *Enterococcus cecorum* isolates recovered from enterococcal spondylitis outbreaks in the southeastern United States. *Avian Pathology*, *41*(5), 479-485. https://doi.org/10.1080/03079457.2012.718070

Borst, L. B., Suyemoto, M. M., Keelara, S., Dunningan, S. E., Guy, J. S., & Barnes, H. J. (2014). A chicken embryo lethality assay for pathogenic *Enterococcus cecorum*. *Avian Diseases*, *58*(2), 244-248. https://doi.org/10.1637/10687-101113-Reg.1

Borst, L. B., Suyemoto, M. M., Scholl, E. H., Fuller, F. J., & Barnes, H. J. (2015). Comparative genomic analysis identifies divergent genomic features of pathogenic *Enterococcus cecorum* including a type IC CRISPR-cas system, a capsule locus, an *epa*-like locus, and putative host tissue binding proteins. *PLoS ONE*, *10*(4), e0121294. https://doi.org/10.1371/journal.pone.0121294

Borst, L. B., Suyemoto, M. M., Sarsour, A. H., Harris, M. C., Martin, M. P., Strickland, J. D., Oviedo, E. O., & Barnes, H. J. (2017). Pathogenesis of enterococcal spondylitis caused by *Enterococcus cecorum* in broiler chickens. *Veterinary Pathology*, *54*(1), 61-73. https://doi.org/10.1177/0300985816658098

Borst, L. B., Suyemoto, M. M., Chen, L. R., & Barnes, H. J. (2019). Vaccination of breeder hens with a polyvalent killed vaccine for pathogenic *Enterococcus cecorum* does not protect offspring from enterococcal spondylitis. *Avian Pathology*, *48*(1), 17-24. https://doi.org/10.1080/03079457.2018.1536819

Cutting, S. M. (2011). *Bacillus* probiotics. *Food Microbiology*, *28*(2), 214-220. https://doi.org/10.1016/j.fm.2010.03.007

Devriese, L. A., Dutta, G. N., Farrow, J. A. E., Van De Kerckhove, A., & Phillips, B. A. (1983). *Streptococcus cecorum*, a new species isolated from chickens. *International Journal of Systematic and Evolutionary Microbiology*, *33*(4), 772-776. https://doi.org/10.1099/00207713-33-4-772

Devriese, L. A., Ceyssens, K., & Haesebrouck, F. (1991a). Characteristics of *Enterococcus cecorum* strains from the intestines of different animal species. *Letters in Applied Microbiology*, *12*(4), 137-139. https://doi.org/10.1111/j.1472-765X.1991.tb00524.x

Devriese, L. A., Hommez, J., Wijfels, R., & Haesebrouck, F. (1991b). Composition of the enterococcal and streptococcal intestinal flora of poultry. *Journal of Applied Bacteriology*, *71*(1), 46-50. https://doi.org/10.1111/j.1365-2672.1991.tb04661.x

Devriese, L. A., Cauwerts, K., Hermans, K., & Wood, A. M. (2002). *Enterococcus cecorum* septicemia as a cause of bone and joint lesions resulting in lameness in broiler chickens. *Vlaams Diergeneeskundig Tijdschrift*, *71*(3), 219-221. https://www.scopus.com/inward/record.uri?eid=2-s2.0-0036558345&partnerID=40&md5=f1dea35caef06ce66a8742ba10a7bbef

Dolka, B., Chrobak-Chmiel, D., Makrai, L., & Szeleszczuk, P. (2016). Phenotypic and genotypic characterization of *Enterococcus cecorum* strains associated with infections in poultry. *BMC Veterinary Research*, *12*(1), 129. https://doi.org/10.1186/s12917-016-0761-1

Dolka, B., Chrobak-Chmiel, D., Czopowicz, M., & Szeleszczuk, P. (2017). Characterization of pathogenic *Enterococcus cecorum* from different poultry groups: Broiler chickens, layers, turkeys, and waterfowl. *PLoS ONE*, *12(9), e0185199*. https://doi.org/10.1371/journal.pone.0185199

Dolka, B., Czopowicz, M., Dolka, I., & Szeleszczuk, P. (2022). Chicken embryo lethality assay for determining the lethal dose, tissue distribution and pathogenicity of clinical *Enterococcus cecorum* isolates from poultry. *Scientific Reports*, *12*(1), 10675. https://doi.org/10.1038/s41598-022-14900-9

Dunnam, G., Thornton, J. K., & Pulido-Landinez, M. (2023). Characterization of an Emerging *Enterococcus cecorum* outbreak causing severe systemic disease with concurrent leg problems in a broiler integrator in the Southern United States. *Avian Diseases*, *67*(2), 137-144. https://doi.org/10.1637/aviandiseases-D-22-00085

Ekesi, N. S., Hasan, A., Parveen, A., Shwani, A., & Rhoads, D. D. (2021). Embryo lethality assay as a tool for assessing virulence of isolates from bacterial chondronecrosis with osteomyelitis in broilers. *Poultry Science*, *100*(11), 101455. https://doi.org/10.1016/j.psj.2021.101455

Gigante, A., & Atterbury, R. J. (2019). Veterinary use of bacteriophage therapy in intensively-reared livestock. *Virology Journal*, *16*(1), 155. https://doi.org/10.1186/s12985-019-1260-3

Grund, A., Rautenschlein, S., & Jung, A. (2021). Tenacity of *Enterococcus cecorum* at different environmental conditions. *Journal of Applied Microbiology*, *130*(5), 1494-1507. https://doi.org/10.1111/jam.14899

Grund, A., Rautenschlein, S., & Jung, A. (2022). Detection of *Enterococcus cecorum* in the drinking system of broiler chickens and examination of its potential to form biofilms. *European Poultry Science*, *86*. https://doi.org/10.1399/eps.2022.346

Guéneau, V., Plateau-Gonthier, J., Arnaud, L., Piard, J. C., Castex, M., & Briandet, R. (2022a). Positive biofilms to guide surface microbial ecology in livestock buildings. *Biofilm*, *4*, 100075. https://doi.org/10.1016/j.bioflm.2022.100075

Guéneau, V., Rodiles, A., Frayssinet, B., Piard, J. C., Castex, M., Plateau-Gonthier, J., & Briandet, R. (2022b). Positive biofilms to control surface-associated microbial communities in a broiler chicken production system - a field study. *Frontiers in Microbiology*, *13*, 981747. https://doi.org/10.3389/fmicb.2022.981747

Hankel, J., Bodmann, B., Todte, M., Galvez, E., Strowig, T., Radko, D., Antakli, A., & Visscher, C. (2021). Comparison of chicken cecal microbiota after metaphylactic treatment or following administration of feed additives in a broiler farm with enterococcal spondylitis history. *Pathogens*, *10*(8), 1068. https://doi.org/10.3390/pathogens10081068

Herdt, P., Defoort, P., Steelant, J., Swam, H., Tanghe, L., Goethem, S., & Vanrobaeys, M. (2008). *Enterococcus cecorum* osteomyelitis and arthritis in broiler chickens. *Vlaams Diergeneeskundig Tijdschrift, 78*(1), 44-48. https://doi.org/10.21825/vdt.87495

Huang, Y., Eeckhaut, V., Goossens, E., Rasschaert, G., Van Erum, J., Roovers, G., Ducatelle, R., Antonissen, G., & Van Immerseel, F. (2023). Bacterial chondronecrosis with osteomyelitis related *Enterococcus cecorum* isolates are genetically distinct from the commensal population and are more virulent in an embryo mortality model. *Veterinary research*, *54*(1), 13. https://doi.org/10.1186/s13567-023-01146-0

Huang, Y., Boyen, F., Antonissen, G., Vereecke, N., & Van Immerseel, F. (2024). The Genetic landscape of antimicrobial resistance genes in *Enterococcus cecorum* broiler isolates. *Antibiotics*, *13*(5), 409. https://doi.org/10.3390/antibiotics13050409

Jackson, C. R., Kariyawasam, S., Borst, L. B., Frye, J. G., Barrett, J. B., Hiott, L. M., & Woodley, T. A. (2015). Antimicrobial resistance, virulence determinants and genetic profiles of clinical and nonclinical *Enterococcus cecorum* from poultry. *Letters in Applied Microbiology*, *60*(2), 111-119. https://doi.org/10.1111/lam.12374

Jung, A., & Rautenschlein, S. (2014). Comprehensive report of an *Enterococcus* cecorum infection in a broiler flock in Northern Germany. *BMC Veterinary Research*, *10*, 311. https://doi.org/10.1186/s12917-014-0311-7

Jung, A., Chen, L. R., Suyemoto, M. M., Barnes, H. J., & Borst, L. B. (2018). A review of *Enterococcus cecorum* infection in poultry. *Avian Diseases*, *62*(3), 261-271. https://doi.org/10.1637/11825-030618-Review.1

Jung, A., & Rautenschlein, S. (2020). Development of an in-house ELISA for detection of antibodies against *Enterococcus cecorum* in Pekin ducks. *Avian Pathology*, *49*(4), 355-360. https://doi.org/10.1080/03079457.2020.1753653

Karunarathna, R., Popowich, S., Wawryk, M., Chow-Lockerbie, B., Ahmed, K. A., Yu, C., Liu, M., Goonewardene, K., Gunawardana, T., Kurukulasuriya, S., Gupta, A., Willson, P., Ambrose, N., Ngeleka, M., & Gomis, S. (2017). Increased incidence of enterococcal infection in nonviable broiler chicken embryos in Western Canadian hatcheries as detected by matrix-assisted laser desorption/ionization-time-of-flight mass spectrometry. *Avian Diseases*, *61*(4), 472-480. https://doi.org/10.1637/11678-052317-Reg.1

Kense, M. J., & Landman, W. J. M. (2011). *Enterococcus cecorum* infections in broiler breeders and their offspring: Molecular epidemiology. *Avian Pathology*, *40*(6), 603-612. https://doi.org/10.1080/03079457.2011.619165

Laurentie, J., Loux, V., Hennequet-Antier, C., Chambellon, E., Deschamps, J., Trotereau, A., Furlan, S., Darrigo, C., Kempf, F., Lao, J., Milhes, M., Roques, C., Quinquis, B., Vandecasteele, C., Boyer, R., Bouchez, O., Repoila, F., Le Guennec, J., Chiapello, H., … Serror, P. (2023a). Comparative genome analysis of *Enterococcus cecorum* reveals intercontinental spread of a lineage of clinical poultry Isolates. *mSphere*, *8*(2), e0049522. https://doi.org/10.1128/msphere.00495-22

Laurentie, J., Mourand, G., Grippon, P., Furlan, S., Chauvin, C., Jouy, E., Serror, P., & Kempf, I. (2023b). Determination of epidemiological cutoff values for antimicrobial resistance of *Enterococcus cecorum*. *Journal of Clinical Microbiology*, *61*(3), e01445-22. https://doi.org/10.1128/jcm.01445-22

Luyckx, K. Y., Van Weyenberg, S., Dewulf, J., Herman, L., Zoons, J., Vervaet, E., Heyndrickx, M., & De Reu, K. (2015). On-farm comparisons of different cleaning protocols in broiler houses. *Poultry Science*, *94*(8), 1986-1993. https://doi.org/10.3382/ps/pev143

Makrai, L., Nemes, C., Simon, A., Ivanics, É., Dudás, Z., Fodor, L., & Glávits, R. (2011). Association of *Enterococcus cecorum* with vertebral osteomyelitis and spondylolisthesis in broiler parent chicks. *Acta Veterinaria Hungarica*, *59*(1), 11-21. https://doi.org/10.1556/AVet.59.2011.1.2

Manders, T., Benedictus, L., Spaninks, M., & Matthijs, M. (2024). *Enterococcus cecorum* lesion strains are less sensitive to the hostile environment of albumen and more resistant to lysozyme compared to cloaca strains. *Avian Pathology*, *53*(2), 106-114. https://doi.org/10.1080/03079457.2023.2286985

Martin, L. T., Martin, M. P., & Barnes, H. J. (2011). Experimental reproduction of enterococcal spondylitis in male broiler breeder chickens. *Avian Diseases*, *55*(2), 273-278. https://doi.org/10.1637/9614-121410-Reg.1

Medina Fernández, S., Cretenet, M., & Bernardeau, M. (2019). In vitro inhibition of avian pathogenic *Enterococcus cecorum* isolates by probiotic *Bacillus* strains. *Poultry Science*, *98*(6), 2338-2346. https://doi.org/10.3382/ps/pey593

Parks, D. H., Chuvochina, M., Chaumeil, P. A., Rinke, C., Mussig, A. J., & Hugenholtz, P. (2020). A complete domain-to-species taxonomy for Bacteria and Archaea. *Nature Biotechnology*, *38*(9), 1079-1086. https://doi.org/10.1038/s41587-020-0501-8

Penaloza-Vazquez, A., Ma, L. M., & Rayas-Duarte, P. (2019). Isolation and characterization of *Bacillus* spp. strains as potential probiotics for poultry. *Canadian Journal of Microbiology*, *65*(10), 762-774. https://doi.org/10.1139/cjm-2019-0019

Potier, P. H., Rigomier, P., Moalic, P. Y., & Bourgeon, F. (2024). *Étude préliminaire de la diversité moléculaire d'Enterococcus cecorum dans les élevages bretons* [Communication]. 15e Journées de la Recherche Avicole et Palmipèdes à Foie Gras, Tours.

Remiot, P., Panaget, G., Chataigner, E., & Chevalier, D. (2019). *Enterococcus cecorum chez le poulet de chair : enquête en élevage pour identifier des pratiques zootechniques à risque* [Communication]. 13e Journées de la Recherche Avicole et Palmipèdes à Foie Gras, Tours. https://www.itavi.asso.fr/publications/Enterococcus-cecorum-chez-le-poulet-de-chair-enquete-en-elevage-pour-identifier-des-pratiques-zootechniques-a-risque?search=Enterococcus%20cecorum%20chez%20&order=score

Rhoads, D. D., Pummill, J., & Alrubaye, A. A. K. (2024). Molecular genomic analyses of *Enterococcus cecorum* from sepsis outbreaks in broilers. *Microorganisms*, *12*(2), 250. https://doi.org/10.3390/microorganisms12020250

Robbins, K. M., Suyemoto, M. M., Lyman, R. L., Martin, M. P., Barnes, H. J., & Borst, L. B. (2012). An outbreak and source investigation of enterococcal spondylitis in broilers caused by *Enterococcus cecorum*. *Avian Diseases*, *56*(4), 768-773. https://doi.org/10.1637/10253-052412-Case.1

Rodrigues, D. R., Wilson, K. M., & Bielke, L. R. (2021). Proper immune response depends on early exposure to gut microbiota in broiler chicks. *Frontiers in Physiology*, *12*, 758183. https://doi.org/10.3389/fphys.2021.758183

Rubio, L. A. (2019). Possibilities of early life programming in broiler chickens via intestinal microbiota modulation. *Poultry Science*, *98*(2), 695-706. https://doi.org/10.3382/ps/pey416

Rychlik, I. (2020). Composition and function of chicken gut microbiota. *Animals*, *10*(1), 103. https://doi.org/10.3390/ani10010103

Sandvang, D., Capern, L. C., Meuter, A., & Betz, S. (2024). *Évaluation in vitro de la capacité d'inhibition d'un probiotique triple souche de* Bacillus *sur des isolats cliniques d'*Enterococcus cecorum [Communication]. 15e Journées de la Recherche Avicole et Palmipèdes à Foie Gras, Tours, 595-599.

Sary, K., Brochu-Morin, M. E., & Boulianne, M. (2018). Enterococcus cecorum *chez la volaille. Une maladie en émergence au Québec* (Bulletin zoosanitaire). RAIZO. https://cdn-contenu.quebec.ca/cdn-contenu/adm/min/agriculture-pecheries-alimentation/sante-animale/surveillance-controle/raizo/reseau-aviaire/Bulletinzoosanitaire_Enterococcuscecorum_volaille_MAPAQ.pdf

Schreier, J., Rautenschlein, S., & Jung, A. (2021). Different virulence levels of *Enterococcus cecorum* strains in experimentally infected meat-type chickens. *PLoS ONE*, *16*(11), e0259904. https://doi.org/10.1371/journal.pone.0259904

Schreier, J., Karasova, D., Crhanova, M., Rychlik, I., Rautenschlein, S., & Jung, A. (2022a). Influence of lincomycin-spectinomycin treatment on the outcome of *Enterococcus cecorum* infection and on the cecal microbiota in broilers. *Gut Pathogens*, *14*(1), 3. https://doi.org/10.1186/s13099-021-00467-9

Schreier, J., Rychlik, I., Karasova, D., Crhanova, M., Breves, G., Rautenschlein, S., & Jung, A. (2022b). Influence of heat stress on intestinal integrity and the cæcal microbiota during *Enterococcus cecorum* infection in broilers. *Veterinary Research*, *53*(1), 110. https://doi.org/10.1186/s13567-022-01132-y

Schwartzman, J. A., Lebreton, F., Salamzade, R., Shea, T., Martin, M. J., Schaufler, K., Urhan, A., Abeel, T., Camargo, I. L. B. C., Sgardioli, B. F., Prichula, J., Frazzon, A. P. G., Giribet, G., Tyne, D. V., Treinish, G., Innis, C. J., Wagenaar, J. A., Whipple, R. M., Manson, A. L., … Gilmore, M. S. (2024). Global diversity of enterococci and description of 18 previously unknown species. *Proceedings of the National Academy of Sciences of the United States of America*, *121*(10), e2310852121. https://doi.org/10.1073/pnas.2310852121

Scupham, A. J., Patton, T. G., Bent, E., & Bayles, D. O. (2008). Comparison of the cecal microbiota of domestic and wild turkeys. *Microbial Ecology*, *56*(2), 322-331. https://doi.org/10.1007/s00248-007-9349-4

Sharma, P., Gupta, S. K., Barrett, J. B., Hiott, L. M., Woodley, T. A., Kariyawasam, S., Frye, J. G., & Jackson, C. R. (2020). Comparison of antimicrobial resistance and pan-genome of clinical and non-clinical *Enterococcus cecorum* from poultry using whole-genome sequencing. *Foods*, *9*(6), 686. https://doi.org/10.3390/foods9060686

Shterzer, N., Rothschild, N., Sbehat, Y., Dayan, J., Eytan, D., Uni, Z., & Mills, E. (2023). Vertical transmission of gut bacteria in commercial chickens is limited. *Animal Microbiome*, *5*(1), 50. https://doi.org/10.1186/s42523-023-00272-6

Silberborth, A., Schnug, J., Rautenschlein, S., & Jung, A. (2024). Serological monitoring of *Enterococcus cecorum* specific antibodies in chickens. *Veterinary Immunology and Immunopathology*, *269*, 110714. https://doi.org/10.1016/j.vetimm.2023.110714

Souillard, R., Laurentie, J., Kempf, I., Le Caër, V., Le Bouquin, S., Serror, P., & Allain, V. (2022). Increasing incidence of *Enterococcus*-associated diseases in poultry in France over the past 15 years. *Veterinary Microbiology*, *269*, 109426. https://doi.org/10.1016/j.vetmic.2022.109426

Souillard, R., Laurentie, J., Repoila, F., Kempf, I., & Serror, P. (2024). Enterococcus cecorum*, un agent pathogène opportuniste des volailles : mieux le connaitre pour une meilleure maitrise en élevage* [Communication]. 15e Journées de la Recherche Avicole et Palmipèdes à Foie Gras, Tours. https://hal.inrae.fr/hal-04547075v1

Stalker, M. J., Brash, M. L., Weisz, A., Ouckama, R. M., & Slavic, D. (2010). Arthritis and osteomyelitis associated with *Enterococcus cecorum* infection in broiler and broiler breeder chickens in Ontario, Canada. *Journal of Veterinary Diagnostic Investigation*, *22*(4), 643-645. https://doi.org/10.1177/104063871002200426

Suyemoto, M. M., Barnes, H. J., & Borst, L. B. (2017). Culture methods impact recovery of antibiotic-resistant enterococci including *Enterococcus cecorum* from pre- and postharvest chicken. *Letters in Applied Microbiology*, *64*(3), 210-216. https://doi.org/10.1111/lam.12705

Szeleszczuk, P., Dolka, B., Zbikowski, A., Dolka, I., & Peryga, M. (2013). First case of enterococcal spondylitis in broiler chickens in Poland. *Medycyna Weterynaryjna*, *69*(5), 298-303. https://www.scopus.com/inward/record.uri?eid=2-s2.0-84877952513&partnerID=40&md5=58c76d2664df507d9d3b7c1b11fb18f5

Tessin, J., Jung, A., Silberborth, A., Rohn, K., Schulz, J., Visscher, C., & Kemper, N. (2024). Detection of *Enterococcus cecorum* to identify persistently contaminated locations using faecal and environmental samples in broiler houses of clinically healthy flocks. *Avian Pathology*, *53*(4), 312-320. https://doi.org/10.1080/03079457.2024.2334682

Thofner, I., & Christensen, J. P. (2016). *Investigation of the pathogenesis of* Enterococcus cecorum *after intravenous, intratracheal or oral experimental infections of broilers and broiler breeders* [Conférence]. VETPATH, Prato (Italy).

Wideman, R. F. (2016). Bacterial chondronecrosis with osteomyelitis and lameness in broilers: A review. *Poultry Science*, *95*(2), 325-344. https://doi.org/10.3382/ps/pev320

Wijesurendra, D. S., Chamings, A. N., Bushell, R. N., Rourke, D. O., Stevenson, M., Marenda, M. S., Noormohammadi, A. H., & Stent, A. (2017). Pathological and microbiological investigations into cases of bacterial chondronecrosis and osteomyelitis in broiler poultry. *Avian Pathology*, *46*(6), 683-694. https://doi.org/10.1080/03079457.2017.1349872

Wood, A. M., MacKenzie, G., McGillveray, N. C., Brown, L., Devriese, L. A., & Baele, M. (2002). Isolation of *Enterococcus cecorum* from bone lesions in broiler chickens [2]. *Veterinary Record*, *150*(1), 27. https://www.scopus.com/inward/record.uri?eid=2-s2.0-0037021879&partnerID=40&md5=f90786da573d92519c7acc5bf77457af

Zhou, B. H., Jia, L. S., Wei, S. S., Ding, H. Y., Yang, J. Y., & Wang, H. W. (2020). Effects of *Eimeria tenella* infection on the barrier damage and microbiota diversity of chicken cecum. *Poultry Science*, *99*(3), 1297-1305. https://doi.org/10.1016/j.psj.2019.10.073

Zuidhof, M. J., Schneider, B. L., Carney, V. L., Korver, D. R., & Robinson, F. E. (2014). Growth, efficiency, and yield of commercial broilers from 1957, 1978, and 2005. *Poultry Science*, *93*(12), 2970-2982. https://doi.org/10.3382/ps.2014-04291

Abstract

In less than 20 years, *Enterococcus* cecorum has become one of the major pathogens in broiler farms, distributed worldwide. This opportunistic bacterium is a commensal of the poultry gut, responsible for locomotor disorders that can lead to flock mortality. *E. cecorum* is a cause of poor animal welfare, increased use of antibiotics and economic losses. This review aims to establish a link between *E. cecorum* infections and the current physiological and molecular knowledge of the bacterium. Genomic analyses indicate that clinical isolates of *E. cecorum* are adapted to intestinal colonisation of chicks from the very first days of life. Conversely, commensal strains colonise the intestine later, at the earliest during the third week of life. The contamination routes of farms and the factors that could favour *E. cecorum* infection are most likely multiple: vertical and horizontal transmissions, zootechnical parameters, biosecurity practices and animal susceptibility. This review also presents a number of ideas under development to improve the control of *E. cecorum* on farms and prevent infections.

Résumé

Enterococcus cecorum*, un agent pathogène opportuniste des volailles : mieux le connaître pour une meilleure maîtrise en élevage*

En moins de 20 ans, Enterococcus cecorum *est devenu l'un des agents pathogènes majeurs dans les élevages de poulets de chair et de répartition mondiale. Cette bactérie opportuniste, commensale de l'intestin des volailles, est responsable de pathologies locomotrices pouvant entraîner de la mortalité dans les élevages. Elle est à l'origine d'une dégradation du bien-être animal, d'une utilisation accrue d'antibiotiques et de pertes économiques. Cette revue tente d'établir un lien entre les pathologies à* E. cecorum *et les connaissances actuelles physiologiques et moléculaires de la bactérie. Les analyses génomiques indiquent notamment que les isolats cliniques de* E. cecorum *seraient adaptés à la colonisation intestinale du poussin dès les premiers jours de vie. À l'inverse, les souches commensales coloniseraient l'intestin plus tardivement, au plus tôt durant la troisième semaine de vie. Les voies de contamination des élevages et les facteurs de risque qui pourraient favoriser l'infection à* E. cecorum *sont probablement multiples : transmission verticale et horizontale, paramètres zootechniques, pratiques de biosécurité et sensibilité des animaux. Cette revue présente également quelques pistes en cours de développement pour mieux maîtriser* E. cecorum *au sein des élevages et prévenir les infections.*

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