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Chlamydia psittaci in garden birds in Sweden

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ABSTRACT

Increased numbers of human infections with *Chlamydia psittaci* have been associated with bird feeding activities in southern Sweden. Information on occurrence and genotype of *C. psittaci* in garden birds in Sweden is required to corroborate this finding but data are limited. Additionally, pathogenicity of *C. psittaci* for garden birds is poorly understood. In this study, *C. psittaci* infection was investigated in 275 garden birds representing 22 species submitted for wildlife disease surveillance between 2009 and 2019. PCR was used to detect *C. psittaci* DNA in liver and lung. Positive samples were genotyped, additional PCR was performed on feces, and tissues were examined microscopically. *C. psittaci* was found in six (2.2 %) birds; three great tits (*Parus major*), two feral (*Columba livia*) and one wood pigeon (*Columba palumbus*). Two great tits and the wood pigeon had inflammatory lesions associated with *C. psittaci*. In the great tits and wood pigeon, *C. psittaci* genotype A, the cause of most human cases, was detected. Genotype B, considered endemic in pigeons, was detected in the feral pigeons. Low incidence of *C. psittaci* in dead Swedish garden birds was similar to studies on apparently healthy Swedish birds. Pathological findings were consistent with *C. psittaci* being fatal in half of the positive birds, which also had higher bacterial loads in feces. This highlights the risk for human infection via infected garden birds, especially regarding great tits and pigeons.

1. Introduction

Chlamydia psittaci, a gram-negative bacterium, can infect mammals, including humans, but birds are the main host. Avian infections have been detected in at least 460 free-living or pet bird species [1]. *C. psittaci* is classified into different genotypes by sequencing the *ompA* gene. Nine different genotypes have been identified, of which seven appear in birds [2]. Certain genotypes are found more often in particular orders of birds. Genotype A has been detected most frequently in psittacine birds, B in pigeons and doves, C in ducks and geese and E in pigeons and ducks [3–6]. However, these data mainly come from farmed or captive birds. Less is known about genotypes in wild birds [1], although genotype A and, to a lesser extent, genotype E, have been found in passerine and columbiform garden birds in Britain [7]. All *C. psittaci* variants are potentially zoonotic but type A is the most commonly detected genotype in humans [8–10]. Type A was also detected in connection with outbreaks in humans in southern Sweden in 2013 when garden bird feeding and handling of bird feeders were identified as risk factors [11].

Infection can cause disease in birds and the severity of the illness depends on the host species, virulence of the strain and route of exposure [12]. Clinical signs in birds are usually nonspecific such as lethargy and anorexia [13], but can also include respiratory signs, mucopurulent nasal discharge, diarrhea and polyuria [12]. The incubation time varies from three days up to several weeks [13]. Gross pathological findings are not characteristic enough to differentiate chlamydiosis from other systemic diseases. Common pathologic lesions found associated with chlamydiosis are respiratory, enteric, and ocular disease. Infected birds can excrete the bacterium intermittently via feces and secretions from the respiratory tract. Stress, as well as concomitant infections can activate shedding of the bacterium [14].

Wild pigeons and doves appear to be an important reservoir of chlamydial infection across Europe and typically do not show clinical signs [15]. In contrast, the prevalence of *C. psittaci* appears to be more variable in wild passerines studied in Europe, ranging from 0 to 54%, and infection may or may not be associated with disease [2,7,15–18]. In Sweden, three studies have investigated *C. psittaci* in wild birds and only

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one included garden birds [19–21]. All birds were apparently healthy and a very low prevalence ranging from 1% in waterfowl and raptors to 2.9% in passerines was found [19–21].

Zoonotic *C. psittaci* infection became highly relevant in a human outbreak in southern Sweden in 2013, when a three times higher incidence of pneumonia caused by this bacterium was recorded [11]. Over half of the human cases diagnosed with chlamydiosis could be associated with feeding garden birds and contact with bird feeders [22]. Between 2014 and 2021 an average of 39 human infections with *C. psittaci* were detected per year. In 2019, *C. psittaci* was detected in more than 70 cases of human pneumonia, which was a significant increase compared to previous years. During 2020 and 2021, cases decreased to just above 50 human cases per year [23].

Little is known about the occurrence of *C. psittaci* in garden birds in Sweden. Therefore, the aim of this study was to investigate the presence and pathological significance of *C. psittaci* in Swedish garden birds to better understand the impact on bird health and risks for zoonotic transmission.

2. Materials and methods

2.1. Animals

Within the framework of the wildlife disease surveillance program at the National Veterinary Institute, Sweden (SVA), 1440 garden birds representing 22 different species were examined from 2009 to 2019. The birds were found dead, or in a small number of cases, were moribund and euthanized for animal welfare reasons and all had mild to moderate post-mortem autolysis. Necropsies were performed according to standard procedures to prevent cross-contamination of samples. Following initial gross post-mortem examination, the majority of birds were diagnosed with some form of infectious disease or traumatic injury. Common main diagnoses included trauma (29%) seen in all orders, infections with salmonella (21%) mainly seen in finches and corvids, infections with trichomonas (15%) mainly seen in finches and parids and infections with pigeon paramyxovirus (9%) in pigeons.

From these 1440 cases, 350 were randomly selected. Of these, 275 had archived lung, liver and feces available and were therefore included in this study. These 275 cases were collected throughout Sweden with 111 from southern Sweden, 137 from mid-Sweden and 26 from the most northern regions. Birds were collected in all seasons: 77 from the winter (December-February), 55 from spring (March – May), 97 from the summer (July – August) and 50 birds were collected during autumn (September – November). The birds included in the study represented a total of 22 species from the following groups: finches (107), thrushes (52), corvids (50), pigeons (37), titmice/parids (21), woodpeckers (3) and weavers (5).

2.2. Real-time PCR

Frozen liver and lung tissue from all 275 animals was collected on sterile cotton swabs. DNA was extracted from the swabs using IndiMag Pathogen kit (Indical Bioscience, Leipzig, Germany) and analyzed by real-time PCR specific for *C. psittaci* genetic material as previously described [24]. Fecal samples from PCR positive birds were analyzed in the same way. Genotyping of positive samples was conducted by sequencing the *ompA* gene following methods described by [25,26].

2.3. Histology and immunohistochemistry

Histological examination and immunohistochemistry were performed on available organ samples from animals in which *C. psittaci* was detected by PCR to investigate the presence of any pathology, localize bacterial antigen in tissues and determine if inflammatory lesions were associated with the bacterium. Briefly, tissues previously fixed in 10% neutrally buffered formalin had been processed and embedded in paraffin. Three to four μm sections from these paraffin blocks were stained using Mayer's haematoxylin and eosin [27] for histological examination. For immunohistochemistry, 4 μm thick sections from the same paraffin blocks were deparaffinized and rehydrated. High temperature epitope retrieval at pH 9 was followed by blocking of endogenous peroxide activity (Dako REAL Peroxidase-blocking solution, ready-to-use, Agilent Technologies Sweden AB, Kista, Sweden) for 5 min at room temperature. Sections were then treated with 2% bovine serum albumin (BSA) for 20 min. Slides were incubated with 1:50 dilution of anti-Chlamydia mouse monoclonal antibody (Progen, Heidelberg, Germany) for 30 min at room temperature. Visualization of bound antibodies was performed by using the polymer detection system Dako EnVision® + System-HRP Labelled Polymer (anti-mouse) (Agilent Technologies Sweden AB, Kista, Sweden) for 30 min followed by application of the chromogen diaminobenzodine (DAB) (Dako Liquid DAB+ Substrate Chromogen System, Agilent Technologies Sweden AB, Kista, Sweden) for 10 min. Sections were then counterstained with Mayer's haematoxylin for five minutes. For the negative control, the same procedure was followed except the antibody was omitted and slides were incubated with 2% BSA.

3. Results

Of the 275 birds examined, six (2.2%) had detectable *C. psittaci* DNA in the pooled liver and lung samples (Table 1). The positive birds consisted of three great tits (*Parus major*), two feral pigeons (*Columba livia*) and one wood pigeon (*Columba palumbus*). Of the 21 great tits examined, 3 (14%) were positive (Table 1). Two of 24 (8%) feral pigeons and 1 of 11 (9%) wood pigeons examined were positive (Table 1). Positive birds originated from five different counties throughout the southern half of Sweden and all were found during the colder half of the year from October to March.

3.1. Pathology

Suitable tissues for histopathological examination were only available from five of the six PCR positive birds. Two out of three great tits and the wood pigeon showed gross and histopathological changes in the form of inflammation in the air sacs, pancreas and on serosal membranes in the body cavity. Chlamydial antigen could be visualized in these inflammatory lesions by immunohistochemistry (Fig. 1). The third great tit was severely autolyzed and was difficult to assess, but it did not show any obvious pathological changes that could be associated with chlamydia infection. The only lesions observed in the feral pigeons were inflammatory changes (multifocal, lymphoplasmacytic interstitial inflammation) in the kidneys and pancreas consistent with pigeon paramyxovirus infection. These birds did not show lesions typical for *Chlamydia* infection and Chlamydial antigen could not be visualized in the renal or pancreatic inflammation by immunohistochemistry.

3.2. Bacterial load and genotyping

Fecal samples from the positive birds were also positive by real-time PCR with the same or lower Ct-values (Table 2). CT-values in the PCR analyses of liver and lung samples ranged from 23 to 32 in the three birds with pathological lesions associated with *C. psittaci* (Table 2). CT-values were higher, corresponding to a lower bacterial load, in the three birds that did not have *C. psittaci*- associated lesions (Table 2).

C. psittaci detected in the three great tits and the wood pigeon was typed to genotype A. *C. psittaci* genotype B was detected in both feral pigeons.

4. Discussion

This study found *Chlamydia psittaci* in great tits and pigeon species in Sweden. The detection of zoonotic genotype A in great tits and a wood

Table 1
Total number of tested birds from each taxonomic group alongside the number and % of positive cases.

Taxonomic group	Species	No. positive/ No. tested/	Percent positive
Finches (Fringillidae)		0/107	
	Bullfinch (*Pyrrhula pyrrhula*)	0/42	
	Greenfinch (*Chloris chloris*)	0/27	
	Common redpoll (*Carduelis flammea*)	0/26	
	Chaffinch (*Fringilla coelebs*)	0/7	
	Hawfinch (*Coccothraustes coccothraustes*)	0/5	
Thrushes (Turdinae)		0/52	
	Common blackbird (*Turdus merula*)	0/39	
	Song thrush (*Turdus philomelos*)	0/8	
	Fieldfare (*Turdus pilaris*)	0/4	
	Redwing (*Turdus iliacus*)	0/1	
Corvids (Corvidae)		0/50	
	Western jackdaw (*Coloeus monedula*)	0/40	
	Eurasian magpie (*Pica pica*)	0/7	
	Common raven (*Corvus corax*)	0/1	
	Hooded crow (*Corvus corone* cornix)	0/1	
	Rook (*Corvus frugilegus*)	0/1	
Pigeons (Columbidae)		3/37	8%
	Feral pigeon (*Columba livia*)	2/26	8%
	Wood pigeon (*Columba palumbus*)	1/11	9%
Titmice/Parids (Paridae)			
	Great tit (*Parus major*)	3/21	14%
Weavers (Ploceidae)		0/5	
	House sparrow (*Passer domesticus*)	0/4	
	Eurasian tree sparrow (*Passer montanus*)	0/1	
Woodpeckers (Picidae)		0/3	
	Great spotted woodpecker (*Dendrocopos major*)	1/1	
	European green woodpecker (*Picus viridis*)	1/1	
	Eurasian three-toed woodpecker (*Picoides tridactylus*)	1/1	
Total		**6/275**	**2 %**

pigeon corroborates previous epidemiological findings that linked human infections to garden bird feeding. Additionally, three of four birds infected with genotype A had associated pathological lesions, indicating that this genotype also can cause disease in birds.

Knowledge about *C. psittaci* in wild Swedish birds is limited. Previous studies indicate a relatively low general prevalence among healthy birds of various orders. In a study of fecal samples from 312 apparently healthy migratory passerines of 18 different species, *C. psittaci* was detected in 9 birds (2.9%) of six species (tree pipit (*Anthus trivialis*), meadow pipit (*Anthus pratensis*), robin (*Erithacus rubecula*), song thrush (*Turdus philomelos*), fieldfare (*Turdus pilaris*) and great tit (*Parus major*) [21]. Within these respective species, the prevalence varied between 7 and 10%. Occurrence of *C. psittaci* within positive species in our study was similar, ranging from 8 to 14%. Examination of cloacal swabs from approximately 300 live nestling peregrine falcons (*Falco peregrinus*) and white tailed eagles (*Haliaeetus albicilla*) and 20 dead adult white tailed eagles detected *C. psittaci* DNA in 1.3% of the birds using real time PCR [19]. In a study of cloacal swabs from 496 migratory wetland birds of 22 species where the majority were mallards (*Anas platyrhychos*) [20], *C. psittaci* was detected by real-time PCR in four wild birds (1.0%) of two species (one common tern, *Sterna hirundo*, and three mallards). International studies have, in some species including parids and pigeons, shown both lower and higher occurrence of *C. psittaci* [7,16,17]. Some of the studies investigated apparently healthy birds [17], while others selected birds based on a suspicion of potential disease [7,16].

The current study focused on garden birds i.e. those that the public may come into close contact with in the garden environment, for example during bird feeding. The examined birds were found dead or moribund and in many cases were diagnosed with infectious diseases. Because increased shedding of *C. psittaci* is associated with concurrent disease or stress, these sick or injured garden birds were hypothesized to be at a higher risk of carrying and shedding *C. psittaci* compared to healthy birds that were investigated in previous Swedish studies. However, general occurrence of *C. psittaci* in these birds was at about the same level as in previously studied Swedish birds. Of the 22 bird species included in the study, *C. psittaci* was detected only in great tits and pigeons. These are the same species identified in previous studies, sometimes with high prevalence of the bacterium [7]. In these species, about one in ten birds was infected with the bacterium and in half of the infected birds (3/6), the chlamydia infection was considered to have been the cause of death. Lesions in these birds corresponded to those described in other studies [7].

The birds that showed pathological changes caused by the chlamydia infection mostly had lower Ct-values during PCR examination of intestinal samples (Table 1), corresponding to higher bacterial loads that are subsequently shed in feces [12].

In this study, the great tits and the wood pigeon were infected with *C. psittaci* genotype A and feral pigeons were infected with genotype B. Genotype A has been repeatedly isolated from psittacine birds, but also from pigeons [7]. Genotype A was also the predominant genotype found in infected passerines in Britain [7]. Type B is described as endemic in pigeons but has been detected in several bird species [6,28]. Both types are said to be able to cause both acute and chronic infections with intermittent spread of bacteria via the feces and secretions from the respiratory tract.

All *C. psittaci* variants are potentially zoonotic but type A is the most commonly detected genotype in humans [8–10]. Type A was also detected in connection with outbreaks in humans in southern Sweden in 2013 when garden bird feeding and handling of bird feeders were identified as risk factors [11]. When considering only great tits in our study, 14% had detectable infection and all were infected with genotype A. Tits are also one of the most common birds at Swedish bird feeders. More in-depth investigations focusing on *C. psittaci* in tits is therefore warranted to better understand the degree of risk for zoonotic transmission.

5. Conclusions

The presence of *C. psittaci* in the investigated group of garden birds found dead or moribund is similar to what has previously been reported in healthy, wild Swedish birds. Across all species investigated, occurrence was low. However, when species are considered separately, the occurrence of the bacterium was relatively high in infected species in this study. In half of the six birds in this study that carried the bacterium, cause of death was attributed to the chlamydia infection. These birds also had a higher bacterial load in the gut than the three other birds. Infected birds were found during the colder months of the year from October to March and came from five different counties in the southern half of the country.

Although the study was limited, it shows that feces and respiratory secretions from wild garden birds, especially great tits and pigeons, pose a risk to people who may come into contact with them, for example

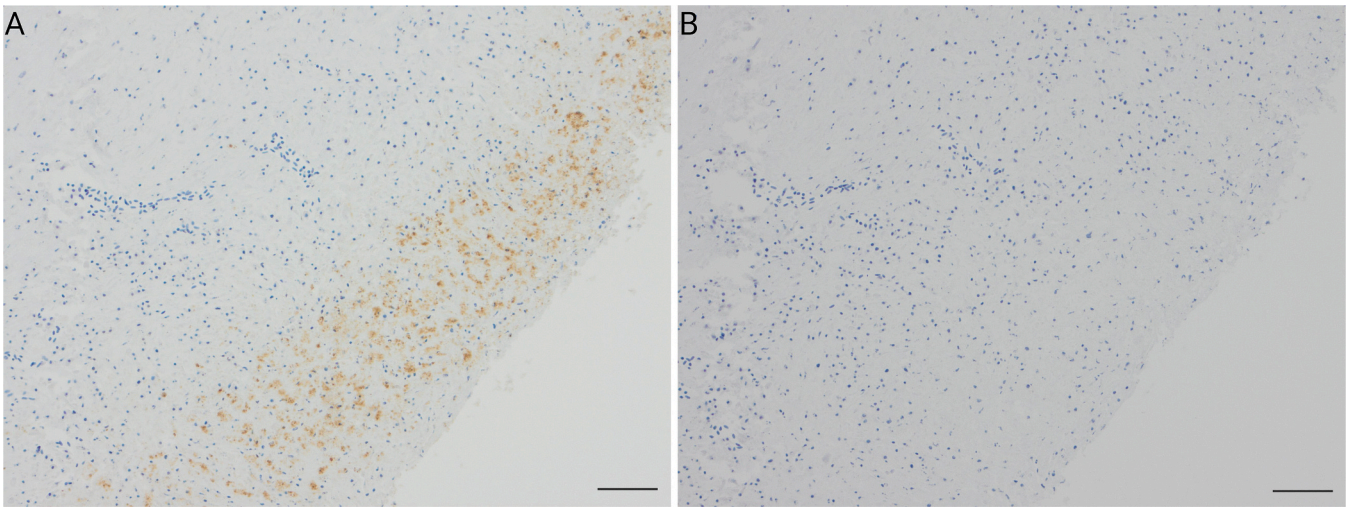

Fig. 1. A. Immunohistochemical labelling of *Chlamydia* lipopolysaccharide antigen within the serosal inflammation on the duodenum of a great tit (*Parus major*) with *C. psittaci* infection confirmed using PCR. Brown, punctate areas of antigen are seen within a wide zone along the duodenal surface. B. Negative control of the same tissue section in which the antibody was omitted. Scale bar is 50 μm. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 2
Results of molecular analyses of garden birds infected with *Chlamydia psittaci* in Sweden. Cycle threshold (Ct) values of real time PCR analyses are presented by tissue and genotypes are derived from sequencing of the *ompA* gene.

		Ct value for *Chlamydia psittaci* real time PCR analysis		
Specimen number	**Species**	**Liver and lung**	**Intestine**	**Genotype (*ompA* gene)**
VLT 858/2013	Great tit *(Parus major)*	38.2	33.2	A
VLT 5/2014*	Great tit *(Parus major)*	32.1	22.4	A
VLT 288/2018*	Great tit *(Parus major)*	29.9	22.5	A
VLT 565/2019	Feral pigeon (*Columba livia*)	38	26.8	B
VLT 2785/2019	Feral pigeon (*Columba livia*)	37.7	37.1	B
VLT 2483/2019*	Wood pigeon (*Columba palumbus*)	22.9	23.4	A

* Birds with pathological changes associated with *C. psittaci* infection.

during bird feeding. The results underline the importance of good hygienic practices when feeding birds, of using bird feeders that are designed to minimize contamination and of avoiding inhalation of dust when handling bird feeders.

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CRediT authorship contribution statement

Ellinor Spörndly-Nees: Formal analysis, Writing – original draft, Writing – review & editing, Visualization. **Henrik Uhlhorn:** Conceptualization, Methodology, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Project administration. **Tomas Jinnerot:** Methodology, Investigation, Writing – original draft, Writing – review & editing. **Aleksija Neimanis:** Conceptualization, Methodology, Writing – review & editing, Visualization, Supervision, Funding acquisition.

Declaration of Competing Interest

The authors declare no conflict of interest." The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

Data availability

Data will be made available on request.

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