Al-Qadisiya University

College of veterinary Medicine

Study on Common Disease of Pigeon in Iraq

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بسم الله الرحمن الرحيم

(وَقُــلْ رَبِّ زِدْنِــي عِلْــماً)

صدق الله العلى العظيم

طـه: من الآية(114)

Dedication

To my family

Alaa

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In the name of Allah most gracious most merciful, t

he first who deserves all thanks and appreciation for granting me with will, strength and help with which this research had been accomplished.

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Contents

1:Avian Influenza :

Avian influenza (AI) is an acute highly contagious and infectious viral disease of all species of birds in general, and poultry in particular worldwide in distribution (**Racnik *et al*.,2008)** . Influenza viruses belong to the family *Orthomyxoviridae according to their abilty to cause disease in poultry.* avian influenza viruses (AIV) are subclassified into two pathotype groups of highly pathogenic avian influenza (HPAI) viruses and low pathogenic avian influenza (LPAI) viruses (Martins,2102)**.** Transmission occurs directly or indirectly through aerosols, water, feed and other materials that have been contaminated by feces (**Kim *e al*.,2009)** .Pathogenicity of influenza viruses also depends on the role of surface HA antigen**(Peiris *et al*., 2007).** The incubation period of avian influenza viruses range from as short as a few hours in intravenously inoculated birds to 3 days in naturally-infected individual birds and up to 14 days in a flock depending on the subtype, the viral dose, the species and age of the birds and route of infection **(Abdu *et al*., 2005)** . Clinical signs in birds generally range from asymptomatic infection to drops in egg production and mild respiratory disease (**Capua and Alexander, 2004**) .The symptoms following infection with low pathogenic AIV may be as discrete as huddling ,ruffled feathers, depression, decrease activity, decreased feed and water consumption coughing, sneezing, rales, lacrimation and sinusitis also there is mild to moderate inflammation of the trachea, air sacs and conjunctiva (**Ladman *et al* ., 2008**) .

During the acute stages of infection in chicken there are severe lung congestion, hemorrhage and edema in dead chickens , such that excised tissue exuded serous fluid and blood (**Joannis *et al*., 2006)**. More varied visible lesions are seen in chickens surviving 3 to 5 days post infection , including congestion and /or cyanosis of the comb and wattles and swollen head, especially prominent from periorbital and intramandibular subcutaneous edema , the change in the comb and wattles progress to depressed areas of dark red to blue areas of ischemic necrosis as the result of vascular infarction (**Ellis *et al*., 2004).** Definitive diagnosis of AIV depends on various laboratory methods including indirect evidence of infection by detecting anti-influenza antibodies by serological methods and direct detection methods for either live virus , viral antigen, or viral nucleic acid (**Suarez *et al*., 2007**) .

2: Newcastle disease :

Newcastle disease caused by Newcastle disease virus (NDV), or avian paramyxovirus type 1, is an economically important disease of birds. Periodic outbreaks of Newcastle disease severely affect the poultry industry and, therefore, many countries rely on compulsory vaccination. NDV is classified in the genus Avulavirus of the family Paramyxoviridae (**Mayo, 2002**) and has a non-segmented negative-strand RNA genome consisting of six transcriptional units (**Lamb & Parks, 2007**). These encode at least six proteins: the nucleocapsid protein (NP), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin-neuraminidase (HN) and the polymerase (L) protein. During P gene transcription an additional, non-structural protein (V) is produced by means of mRNA editing and functions as an interferon antagonist (**Park et al., 2003**). The M, F and HN proteins are associated with the viral envelope, in which the M protein is involved in budding and morphogenesis, whereas F and HN mediate the entry and release of NDV. The virulence of NDV is mainly determined by the amino acid sequence of the proteasecleavage site of the F protein. Virulent NDV strains can be discriminated from low- or non-virulent strains by the presence of multiple basic amino acids at the proteolytic cleavage site of the F protein (**Ogasawara et al., 1992**). The NP protein encapsidates the RNA genome to form the nucleocapsid and associates with the P and L proteins. The P protein is essential for viral RNA synthesis and is involved in all of its aspects. The L protein is an RNA-dependent RNA polymerase that associates with the NP and P proteins, together constituting the viral replication complex (**Lamb & Parks, 2007**). This complex is responsible for transcription and replication of the viral genome. Pigeon paramyxovirus type 1 (PPMV-1) viruses are variant strains of NDV associated with infections of pigeons and have a

worldwide distribution (Alexander et al., 1985a). Several Newcastle disease outbreaks in chickens have been attributed to PPMV-1, which makes it a real threat to the poultry industry (**Irvine et al., 2009; Liu et al., 2006**). The F proteins of all PPMV-1 strains examined to date contain a poly-basic cleavage site motif, afeature of NDV generally associated with high virulence. However, some PPMV-1 strains cause only minimal disease and have a low intracerebral pathogenicity index (ICPI) in chickens (**Meulemans et al., 2002**). Nevertheless, they do have a virulence potential for chickens, which can emerge upon serial passages in these animals . Sequence analysis of such passaged viruses has mainly focused on the F gene, and it was concluded that the increase in virulence was not associated with changes in the F protein sequence (Kommers et al., 2003). This is in agreement with our own observation that replacement of the F gene of a virulent NDV strain by that of a non-virulent PPMV-1 strain resulted in a virulent chimeric virus, indicating that the non-virulent phenotype of the PPMV-1 strain is not caused by the F protein (**Dortmans et al., 2009**). By exchanging genes between a low-virulent PPMV-1 strain and a highly virulent NDV strain, we recently showed that virulence of NDV (and PPMV-1) is associated with the activity of the viral replication proteins . Consistently, the increase in virulence observed during passaging of PPMV-1 in chickens might also be caused by changes in these proteins. To test this hypothesis, we passaged the low-virulent PPMV-1 strain AV324 in chickens. We indeed observed an increase in virulence and here we show this to be due to the accumulation of mutations in the P and L proteins. These mutations resulted in more efficient virus replication both in vitro and in vivo, indicating that virulence of PPMV-1 for chickens is directlyrelated to the efficiency of virus replication **(Dortmans et al., 2010)**.

3: The pox

The poxviruses (family *Poxviridae*) cause illness characterized by generalized or localized cutaneous lesions, and most member viruses have broad host ranges. The overall broad host range of this family is demonstrated by the two subfamilies of the *Poxviridae*. The subfamily *Entomopoxvirinae* infects insects, and the subfamily *Chordopoxvirinae* infects vertebrates; the latter consists of eight genera, and other "unclassified chordopoxviruses." Theclassified genera are *Orthopoxvirus*, *Parapoxvirus*, *Avipoxvirus*, *Capripoxvirus*, *Leporipoxvirus*, *Suipoxvirus*, *Molluscipoxvirus*, and *Yatapoxvirus*.(Li *etal*., 2010).

Poxvirus infections of humans, cattle, sheep, goats, companion animals, birds, and zoo animals have been reported worldwide but in general represent an underappreciated cause of health care utilization(**Damon,** 2007). Poxvirus infections can be clinically confused with other cutaneous disease, and other poxviruses are emerging or reemerging infectionsin various parts of the world. The majority of human poxvirus infections are zoonotic. Poxvirus infections are also a significant burden to agricultural communities. Poxviruses represent one of largest viruses known and replicate in the cytoplasm of the infected cell and encode most enzymes for their life cycle. The genomes of poxviruses are a linear double-stranded DNA genome in the range of 134 kb (*Parapoxvirus*) to 330 kb (*Avipoxvirus*) and encode more than 130 genes. The central region, comprised of nearly 100 genes which encode viral gene expression, DNA replication, and virion formation, has a structural arrangement that is conserved in most chordopoxviruses. Between genera of chordopoxviruses, host specificity and genome sequence have diverged.(Li *etal*., 2010).

The avian pox viruses form a subgroup of viruses of varying cross relationship degrees. They affect a wide range of sixty bird species of various families (Bolte *et al.*, 1999). The infection could be natural or artificial in cutaneous or diphtheric/ pharyngeal forms, or both.

Sometimes viruses produce a protein similar to an epidermal growth factor (Moss, 1996). They multiply easily in cell cultures and on the chorioallantoic membrane of embryonated eggs and form type A cytoplasmic inclusions in the cells of these culture systems (Murphy *et al.*, 1995). The genome of the fowl pox virus is more than 100 kb larger than the orthopoxvirus vaccinia, indicating that the avipox viruses have the potential to code for more proteins than other groups of poxviruses (Coupar *et al.*, 1990). Host specificity is considered to be one of the important criteria for differentiation of avian poxviruses. Vaccines of fowl pox or pigeon pox virus origin have been routinely used for more than half acentury to prevent fowl pox in commercial poultry in endemic areas. However, inrecent years, outbreaks of fowl pox have occurred in previously vaccinated flocks due to the emergence of variant strains of fowlpox virus (FPV) and to the fact that the novel FPV exhibited enhanced virulence after integration of avian reticuloendotheliosis virus (REV) into their genomes (Singh *et al.*, 2000). Mortality and morbidity related to pigeon poxvirus infection may be very high in pigeons (Tripathy, 1991). During the course of this study, a reference strain could not be procured in spite of repeated attempts. In order to overcome the problem, the identity of the field pigeon pox virus (PPV) isolates was compared with field fowl pox virus (FPV) isolates through *in vivo* cross infections of natural hosts such as pigeons and chickens, respectively. Interpretation of host specificity of agents was based on the formation of atypical pox lesions, generalized, dispersed or local (focal) infection, or death of experimental hosts(Siddique *etal*.,2011).

4: Chlamydiosis:

Chlamydiosis is an important infectious disease of people and many animals, including birds. It is caused by the Gram-negative obligatory intracellular bacterium Chlamydia spp., mostly by C. psittaci in birds (**Vanrompay 2013**). The disease caused by this pathogen in birds is known as avian chlamydiosis and it can cause diseases in humans (**Rehn *et al*. 2013**). The groups of people in close contact with birds, like avian breeders, veterinarians, workers of poultry farms, etc., are particularly exposed to Chlamydia infections. There are few reports of cases of human-to-human psittacosis (**Wallensten *et al*. 2014**). The Chlamydia spp. can cause an important disease of birds that is most commonly found in parrots (Psittaciformes) and pigeons (Columbiformes). The outcome of the illness is mostly chronic with conjunctivitis or is asymptomatic, but it might also appear as an acute disease. Depending on the chlamydial strain and the avian host, chlamydiae cause pericarditis, air sacculitis, pneumonia, lateral nasal adenitis, peritonitis, hepatitis, and splenitis.
Generalized infections result in fever, anorexia, lethargy, diarrhea, and occasionally shock and death. Chlamydiosis is a very common chronic infection of psittacine birds. Infections cause conjunctivitis, enteritis, air sacculitis, pneumonitis, and hepatosplenomegaly (Harkinezhad et al. 2009). The asymptomatic birds can play a role in the transmition of Chlamydia in the environment, and thereby might be significant in epidemiology of chlamydiosis. C. psitaci has been isolated frommore than 460 avian species in the wild (**Kaleta and Taday 2003**); however, little is known about the prevalence of other species of Chlamydia among wild bird population worldwide. Some reports suggest that avian infections might be caused only by C. psittaci (**Harkinezhad *et al*. 2009**), but others have confirmed infections caused by other species of Chlamydia such as C. pecorum and C. trachomatis(**Sachse *et al*. 2012**), C. abortus (**Pantchev *et al*. 2009**), and C. ibidis (**Vorimore *et al*. 2013**).

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