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Prevalence of Antibody to Chicken Anaemia Virus (CAV) in Swedish Chicken Breeding Flocks Correlated to Outbreaks of Blue Wing Disease (BWD) in their Progeny

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Engström BE: Prevalence of antibody to chicken anaemia virus (CAV) in Swedish chicken breeding flocks correlated to outbreaks of blue wing disease (BWD) in their progeny. Acta vet. scand. 1999, 40, 97-107. – A serological survey for antibody to Chicken Anaemia Virus (CAV) was performed on broiler breeders as well as layer breeding birds in Sweden at the end of their rearing period. Grandparents (GP) of both types leaving quarantine were in 21 out of 26 cases free from antibody to CAV, but often became infected soon thereafter. A total of 10 outbreaks of blue wing disease (BWD) in 3 series were recorded in the broiler and layer parent generation, all of which were progeny of 3 late seroconverting GP-flocks. All but one of 22 layer parent flocks had been infected and had seroconverted during the rearing period. Subsequently BWD was not recorded from commercial layers. Broiler parent flocks were more protected from CAV infection during rearing. Eighteen out of 94 broiler parent flocks had not developed antibody to CAV before coming into lay. Outbreaks of BWD were reported in progeny flocks from all these broiler breeders, with the exception of those that had been vaccinated.

Good hygienic routines along with isolation of the birds delayed the seroconversion to CAV in broiler breeders and vaccination of these breeders protected their progeny from outbreaks of BWD. Broiler flocks in houses where BWD had occurred recently had always antibodies to CAV at slaughter. It was possible to eradicate the infection from the house and prevent the infection between flocks by proper cleaning and disinfection of the broiler houses.

breeders; layers; broilers; decontamination; hygiene procedures; vaccination.

Introduction

Blue wing disease (BWD) is an acute disease in young chickens (*Engström & Luthman* 1984), caused by chicken anaemia virus (CAV), formerly called chicken anaemia agent (*Engström* 1988). The disease BWD is characterised by high mortality in the flock between 12 to 20 days of age. The most prominent lesions are seen in the skin, with gangrenous dermatitis, and in the lymphoid tissue, with severe depletion of lymphocytes in thymus and bursa of Fabricius.

Horizontal infection with CAV in fully immune competent chickens does not cause any sign of disease. Even a subclinical CAV infection in broilers, however, may have an economical impact on the performance of a flock (*McNulty et al.* 1991).

CAV, which has a circular single-stranded DNA (*Noteborn & Koch* 1995), was first isolated from commercial chickens in Japan by *Yuasa et al.* (1979). CAV has so far been isolated from both healthy chickens and from chickens with BWD, anaemia-dermatitis or haemorrhagic syndrome in Japan (*Goryo et al.* 1985 and 1987, *Yuasa et al.* 1987, *Otaki et al.* 1987), in Taiwan (*Lu et al.* 1993), Europe (*Bülow et al.* 1983, *Chettle et al.* 1989, *McNulty et al.* 1990, *Farkas et al.* 1992, *Hoop et al.* 1992 and *Picault et al.* 1992), USA (*Goodwin et al.* 1989, *McNulty et al.* 1989, *Rosenberger & Cloud* 1989, *Lucio et al.* 1990, *Lamichhane et al.* 1991), in Australia (*Firth & Imai* 1990), New Zealand (*Stanislawek & Howell* 1994), Argentina (*Buscaglia et al.* 1994) and Brazil (*Brentano et al.* 1991).

Serological surveys have demonstrated the presence of CAV in commercial chickens in Japan (*Yuasa et al.* 1985), in Europe (*McNulty et al.*1988, *Jörgensen* 1990), in the USA (*McNulty et al.*1989, *Goodwin et al.* 1990, *Lucio et al.* 1990) in South Africa (*Wicht & Maharaj* 1993), in Israel (*Malkinson et al.* 1990) and in Chile (*Toro et al.* 1994). Most of these studies were done on broiler breeders of all ages. The prevalence of antibody to CAV was high in all countries.

Most outbreaks of BWD in Sweden have occurred in broilers that were progeny of certain young parents (*Engström & Luthman* 1984) suspected of spreading the disease. CAV can be transmitted by the egg from viraemic breeders causing BWD in their infected progeny. If the breeders have developed antibody to CAV before commencing to lay, the offspring is fully protected from the disease (*McNulty* 1991).

The aim of the present study is to correlate the prevalence of antibody to CAV in grandparents and parents of both egg-laying and meat-type breeds of chicken and in broilers in Sweden with outbreaks of BWD in the progeny.

Materials and methods

Birds

Grandparents (GP) of both egg-producing and meat-type of breeds were imported to Sweden as day-old chicks and kept in quarantine until 16 weeks of age. They were all tested for antibodies to CAV at 13-15 weeks of age, before leaving the quarantine. Some flocks were tested later as well (Table 1).

Both broiler parent flocks and layer parent flocks were tested before they were moved to egg-production houses. Some broiler parents lacking antibody to CAV on the first investigation were retested later. (Table 2).

Broilers were tested at slaughter (32-60 days of age). During 1992, broilers were tested in 4 slaughter houses and during 1993-94 in 2, see Table 3. In 1992, the samples were taken at random, but in 1993-1994 the samples were taken in areas where outbreaks of BWD occurred in 1993.

Outbreaks of BWD occurred on one occasion in 1992 and one occasion in 1993.

In order to find out how long CAV persisted in houses after outbreaks of BWD, 6 farms were selected for a special surveillance at the end of 1993 and the beginning of 1994. Three consecutive flocks from 10 different houses were tested for the presence of antibody to CAV at slaughter in the geographic area called A.

Biosecurity and hygienic measures in poultry rearing farms

Poultry rearing farms are generally well separated from each other in Sweden. The GP flocks were most isolated from other birds and the layer parent flocks least isolated at the time of this study.

The hygienic measures according to the Swedish prophylactic salmonella control programme (*Engström* 1994) are very strict in order to stop introduction of salmonella into the birds. The layer parent flocks were the only

Table 1. Serological survey for antibody to CAV in grandparent flocks of broilers and layers 1988-92 and occurence of series of outbreaks of BWD in progeny. Flocks found positive were not retested.

Establishment	Grand parents age (weeks)						Series of outbreaks of BWD in progeny
	13-16		20-25		30-31		
	No. flocks tested	No. positive	No. flocks retested	No. positive	No. flocks retested	No. positive	
Broilers							
A	6	0	5	5	0	–	0
B	7	1	3	0	3	3	2
Layers							
C	4	0	2	1	1	1	1
D	2	0	0	–	0	–	0
E	2	0	1	1	0	–	0
F	5	4	0	–	0	–	0

birds in this study not affiliated to the control programme.

Vaccination

Since 1991 a live vaccine (TAD Thymo vac) has been used in Sweden. Broiler breeders lacking antibody to CAV have been vaccinated before being moved from the rearing house at 18-20 weeks of age.

Sera

Sera used in this study represented different breeder and broiler flocks between 1980-1983 and 1988-1994 originating from all parts of Sweden.

Twenty sera were taken from each flock, which consisted of birds kept together in one house. If there were several houses on a farm all flocks were tested individually.

A serum neutralisation test was used until 1992. An ELISA test was subsequently used.

Serum neutralisation test

The serum neutralisation test (SN) used was the simplified method described by *Bülow et al.* (1988), with some modifications.Twenty sera from each flock were tested as 2 pooled samples and diluted 1:10 and 1:100 in PBS after heat-inactivation. Some sera were tested individually as well.

The MDCC-MSB1 cells were cultured in Dulbecko,s modified essential medium (DMEM) with 10% foetal calf serum, penicillin 50 IU/ml and streptomycin 50 μg/ml at 39.5° C in an atmosphere of 5% CO_2. 10^5 $TCID_{50}$/0.1 ml of the Swedish CAV- strain 1/80 (*Engström* 1988) was used in the test.

Serum samples were considered free of neutralising antibodies when cells were destroyed simultaneously with the virus controls or within one subculture. Serum neutralising (SN) titres of 1:10 were considered positive, which indicated that one or more of the birds had been infected and seroconverted in the flock, while a titre of 1:100 indicated that more individual sera in the pool would be positive to CAV.

Enzyme linked immunosorbent assay (ELISA) test

A commercial antibody ELISA-kit was used (Flockscreen CAV antibody test, Guildhay Ltd England). The test was performed according to

Table 2. Serological survey for antibody to CAV in parent flocks of broilers (A-E) and layers (F-I) and occurence of series of outbreaks of BWD in progeny. Flocks found positive were not retested.

Establishment and year	Parent flocks			Series of outbreaks of BWD in progeny
	Number tested	Seroconverted at age (weeks)		
		13-20	27-31	
Broilers				
A				
1983	7	4	NT[1)]	3
1989-91	18	16	NT	2
1993	12	9	NT	0
B				
1989-91	14	10	2[2)]	4
1993	7	4	NT	0
C				
1983	2	1	1	1
1989-90	7	7	–	0
1993	6	4	NT	2[3)]
D				
1989-90	5	5	–	0
E				
1980-82				
House 1 A[4)]	3	3	–	0
House 2 A	2	2	–	0
House 3 A	4	4	–	0
House 3 B[5)]	3	1	2	2
House 4 A	2	2	–	0
House 4 B	2	1	1	1
Layers				
1980-92				
F	4	4	–	0
G	7	6	NT	0
H	2	2	–	0
I	9	9	–	0

[1)] Not tested.
[2)] Only 2 out of 4 flocks retested.
[3)] Parents not vaccinated.
[4)] Chickens reared in another district.
[5)] Chickens reared in isolation near the hatachery. All birds moved to the farm at 18 weeks of age.

the manufacturer's instructions. Twenty sera from each flock were tested. If one or more of the sera was clearly positive, the flock was judged to be infected with CAV.

Registration of outbreaks of blue wing disease

All outbreaks of BWD with significant mortality were reported to our laboratory. The clinical diagnosis was confirmed by post-mortem ex-

Table 3. Serological survey for antibody to CAV in broilers at slaughter at 38-60 days of age.

Abattoir (area)	Year	No. flocks tested	No. flocks seroconverted	Presence of BWD (–/+)
A	1992	10	0	–
	1993	31	20	+
	1994	45	0	–
B	1992	10	0	–
	1993	13	4	–
	1994	3*	0	–
C	1992	9	0	–
D	1992	14	4	+

* Flocks in these houses were CAV seropositive in 1993.

amination (*Engström & Luthman* 1984). Typical lesions are haemorrhages in the skin and depletion of precursor cells in thymus and sometimes in the bone marrow.

Outbreaks of BWD occurred consistently in a number of broiler flocks during one period. Each series of outbreaks of BWD in progeny of the same parent flock was recorded in the Tables as series of outbreaks of BWD.

Results

Breeders

Antibody to CAV was detected in grand parent and parent flocks of both broilers and layers, but the prevalence varied between the different categories. The results of the serological surveys and the occurrence of BWD in the offspring is presented in Tables 1-2.

In general, GPs (Table 1) were diagnosed as free of antibody to CAV, when they were released from the quarantine at 16 weeks of age. The birds from one quarantine station (Table 1;F), however, were often found to be infected. BWD was only registered once in the offspring of layer GP (Table 1;C). This GP-flock was moved from the quarantine to a breeder house with new battery cages. The flock was retested at 32 weeks of age and found positive to CAV. Outbreaks of BWD with 1%-3% mortality were later reported from layer parent flocks originating from this GP flock.

In the broiler breeder establishment A (Table 1), the GP flocks seroconverted before coming into lay and no outbreak of BWD subsequently occurred amongst the offspring.

In the other broiler breeder establishment (B, Table 1), 3 GP flocks had not seroconverted at 25 weeks of age but they had all seroconverted at 31 weeks of age. Series of outbreaks of BWD were reported from progeny flocks from 2 of these GP flocks. The outbreaks in the parent flocks containing 85% females resulted in very low mortality. From the same hatch, however, mainly males of the female-line and a few females from the male-line were delivered to broiler farms, which experienced severe outbreaks of BWD with high mortality.

All layer parents (Table 2, F-I), with exception of one flock, had antibody to CAV at 13-20 weeks of age and there was no report of BWD in progeny flocks.

Eighteen out of 94 broiler parents (Table 2, A-E) had not seroconverted before they were transferred from their rearing houses at 18-20 weeks of age. Series of outbreaks of BWD were reported from progeny flocks from all parents

that seroconverted later than 20 weeks of age to CAV.

No outbreak of BWD was reported in progeny from vaccinated parent flocks.

A retrospective study of establishment E (Table 2) showed that CAV was only transmitted from houses 3 and 4, which had received 18-week-old birds from 2 different rearing houses, placed in 2 different pens in the same house. The parents from one rearing house (B) seroconverted late on 3 occasions during 1981-82. These flocks transmitted CAV to their progeny for long periods and many outbreaks of BWD were reported. The flock reared in house B in 1980 seroconverted early and no BWD was reported from the offspring.

Broilers (Table 3)

After outbreaks of BWD in a house, antibody to CAV was always detected in the consecutive flocks. In establishments with several broiler houses close together, antibody to CAV was only detected in the houses where BWD had occurred. During periods when no outbreaks of BWD were registered in 1992 and 1994, no serum antibody to CAV was found in broiler flocks at slaughter. In 1993 4 flocks in area B had seroconverted to CAV at slaughter, although no outbreaks of BWD had occurred in these houses during that period.

In 10 houses in area A, where outbreaks of BWD occurred in 1993, 3 consecutive flocks in these houses were tested for antibody to CAV at slaughter in order to find out if the houses were sanitised and free from CAV. In all houses, the first flock after an outbreak of BWD had antibody to CAV, but in the second crop only 6 out of 10 flocks had seroconverted. In the third consecutive flock of birds, however, the chickens in all 10 houses were free from antibody to CAV at slaughter. Flocks from 3 farms in area B, where seropositive flocks were detected in 1993, were also found seronegative in 1994.

Discussion

Most outbreaks of BWD in Sweden have occurred in broilers that were progeny of certain young parents (*Engström & Luthman* 1984) suspected of spreading the disease.

The present study shows that all broiler parents that did not seroconvert to CAV before start of lay produced broilers that experienced BWD due to vertical transmission. Four of these parent flocks were retested at 30-32 weeks of age and all had started to seroconvert at that age.

These results are in accordance with previous studies showing that outbreaks of BWD/chicken anaemia can be traced back to broiler breeders that seroconverted at a late stage to CAV. (*Vielitz & Landgraf* 1988, *Chettle et al.* 1989, *Jörgensen* 1991, *McIlroy et al.* 1992). Although the CAV infection is widespread, flocks of some categories may be protected from infection during the rearing period.

Imported GP flocks were kept in well isolated quarantine buildings, and in most cases they did not become infected until after release from quarantine but prior to the point of lay. The offspring of the late-infected broiler GP-flocks showed prominent differences in the prevalence of BWD depending on the type of production they were delivered to. The chickens destined to be parents had a very low mortality rate caused by BWD, while the chickens sent to broiler farms experienced high mortality. On an earlier occasion, 1800 out of 2000 chickens, derived from the same broiler GP flock, died in a broiler house, but hardly any mortality was registered in the parent house (unpublished observation). The most obvious difference between the 2 categories is that there were 80%-90% females in the parent house, but 80%-90% males in the broiler house. A theory that males could be more susceptible to disease caused by CAV finds support in *Goryo et al.* (1987), who found a 10 times higher mortality rate in males than in females. *Engström & Luthman* (1984), on the

other hand, found that both sexes were equally affected. Since 1983, new hybrids of broilers have been imported to Sweden. The susceptibility of birds of different sex may vary in the different hybrids. On the other hand, there may be a higher risk for other infections that enhance the mortality rate after a CAV infection in a broiler house compared with that of a breeder house.

In most countries with an intensive poultry production, broiler breeders are probably infected with CAV very early. *McNulty et al.* (1988) showed that broiler parents had often seroconverted at 8-9 weeks of age in the UK.

Some of the parent flocks in the present study that seroconverted late were reared in geographically isolated houses, which may have delayed the point of infection. A second factor that may have contributed to the late exposure could be the strict hygienic procedures enforced by the Swedish prophylactic salmonella control programme (SPSCP) on these flocks. Inevitably, the CAV infection reaches the birds later in life, probably due to a more frequent transport of both equipment and eggs during the egg-production period. Isolation and strict hygiene have been reported as a reason for late seroconversion to CAV in breeders from other countries. After a new salmonella control programme was introduced in Denmark in 1989, the number of outbreaks of BWD increased dramatically (*Bisgaard* personal communication, 1991). *Jörgensen et al.* (1995a) showed that the percentage of Danish broiler breeders that had seroconverted to CAV at 12-15 weeks of age declined significantly between 1991-1993 due to improved standards of housing and hygiene.

Stricter hygiene used in attempts to control Salmonella in the UK has also resulted in occasional late seroconversion to CAV and subsequent BWD outbreaks in progeny (*McIlroy et al.* 1992).

Vielitz & Landgraf (1988) also referred to strict hygienic measures as a reason for delayed CAV infection of broiler breeders in Germany.

In order to prevent transmission of CAV from broiler breeders to their progeny, vaccination with a live vaccine has been used in Sweden since 1991, as it is not possible to prevent a CAV infection during the entire egg-production period. No outbreaks of BWD have occurred in offspring from vaccinated broiler breeders. During 1992, some broiler parent flocks were not vaccinated due to lack of vaccine and outbreaks of BWD were reported in the offspring of these parent flocks during 1992 and 1993.

All but one of the layer parent flocks had seroconverted before being moved to the egg-production house and no case of BWD appeared in commercial layers during this period. Occasional outbreaks of BWD have, however, previously occurred in commercial layers in Sweden (*Engström & Luthman* 1984).The earlier exposure to CAV and seroconversion of layer parent flocks may be explained by the fact that the rearing houses for layer parents in Sweden are not as well-isolated as those for broiler parents and were not affiliated to the SPSCP during this period. Early infection in layer parents was also reported by *Jörgensen* (1990), who found that 4 out of 5 layer parent flocks in Denmark, 8-24 weeks of age, had CAV antibody.

The prevalence of antibody to CAV in Swedish broiler flocks was low in this study and antibody to CAV was mostly detected in periods when outbreaks of BWD had occurred in the area.

In 1992-1993 several flocks in houses that previously experienced outbreaks of BWD had seroconverted after a subclinical CAV-infection. They had probably become infected in a CAV-contaminated house.

In Denmark in 1989-1990, *Jörgensen* (1990) found that 20/27 broiler flocks of 5-6 weeks of age had seroconverted. In the UK and Canada,

7/13 and 15/23 had antibody to CAV at slaughter at an age of 5-7 weeks (*McNulty et al.* 1989). The SPSCP, introduced in 1970, has gradually improved the standard of hygiene, resulting in a very low prevalence of salmonella in broilers (*Engström* 1994). After further improvements of the SPSCP in 1987, it also showed an effect on the prevalence of campylobacter in broilers, reducing it from 75% to only 15% in 2 years (*Berndtsson,* personal communication, 1996). These results indicate that the transmission of pathogens from the surroundings can be limited using strict rules of hygiene. In Denmark a similar control programme resulted in a decrease in the prevalence of subclinical CAV infection to 20% in 1992 (*Jörgensen et al.* 1995a). In accordance with SPSCP, all farms must apply all-in/all-out management involving immediate removal of manure from the house and the farm, strict cleaning and disinfection of the houses between each batch of birds. During this study, however, it was not possible to decontaminate the houses immediately after an outbreak of BWD, but after 1-2 crops of birds the procedure succeeded.

CAV is a very stable virus resistant to various chemical and physical treatments. *McNulty et al.* (1991) and *Yuasa* (1992) showed that CAV in organic material was difficult to sanitise properly; however, we have demonstrated that decontamination, though difficult, is possible in practice.

The strict hygiene routines in the Swedish broiler industry were of great benefit for the health of the birds but ironically resulted in a large number of BWD-outbreaks in broiler flocks as early as 1972. The outbreaks were due to vertical transmission from breeders infected during egg-production.

After outbreaks of BWD in broilers, it was difficult to sanitise the houses from CAV to prevent horizontal infection in the consecutive flock. The importance of avoiding a subclinical CAV-infection is not clear, but *McNulty et al.* (1991) found that subclinical infection of CAV in broilers may have an economical impact on the performance of a flock. Broiler flocks seronegative to CAV at slaughter achieved 13% greater net income compared to flocks with CAV-antibodies. *Jörgensen et al.* (1995b) on the other hand, found no difference in performance between CAV positive and negative broiler flocks in Denmark. *McConnell et al.* (1993) found during an experiment that 3-week-old chickens, exposed to CAV by a natural route resulting in a subclinical infection, became immunosuppresed.

Further studies are needed to elucidate the importance of subclinical CAV infections in both younger and older chickens.

Conclusions

The prevalence of antibody to CAV varied between the different categories of breeders during the rearing period prior to the start of laying eggs.

Breeders with the best biosecurity and sanitising programmes for empty houses were best protected from CAV-infection during the rearing period, but the infection seems to reach the birds later in life during the egg production period. GP-flocks were best protected in their quarantines, while parents of layers were not well- protected. Parents of broilers were not infected with CAV in 18 out of 94 tested flocks.

Breeders infected with CAV during the egg-laying period, as demonstrated by the occurrence of seroconversion, caused outbreaks of BWD in their progeny due to vertical transmission of the virus. A controlled infection by vaccination of the breeders before leaving the rearing house, however, protected the progeny from BWD.

The prevalence of antibody to CAV in broiler flocks was relatively low. Broilers kept in houses where outbreaks of BWD had occurred were horizontally infected without showing any signs of disease. It was difficult to decontami-

nate the houses from CAV, but after 2-3 crops the broilers were seronegative to CAV at slaughter.

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Sammanfattning

Förekomst av antikroppar mot chicken anemia virus (CAV) i svenska avelshönsflockar korrelerat till utbrott av blåvingesjuka hos deras avkomma.

En serologisk undersökning av förekomsten av antikroppar mot chicken anaemia virus (CAV) genomfördes på avelsflockar för såväl slaktkyckling som

värphöns under slutet av uppfödningsperioden. Far- och morföräldrar (GP) av båda typer saknade antikroppar mot CAV när de lämnade karantänen i 21 av 26 undersökta flockar. Tre GP-flockar, som smittades först sedan de börjat lägga befruktade ägg, producerade 3 serier av utbrott i sammanlagt 10 föräldraflockar som drabbades av blåvingesjuka (BWD).
Tjugotvå värphönsföräldraflockar, som testades efter uppfödningen hade alla utom en smittats och bildat antikroppar mot CAV. Slaktkycklingföräldrarna var mer skyddade mot CAV-smitta under uppfödningen; 18 av 94 flockar hade inte bildat antikroppar när de lämnat uppfödningshusen. Alla sent smittade föräldrar spred CAV till avkomman, resulterande i ett stort antal utbrott av BWD.
God hygien och isolerad uppfödning kunde skydda aveldjuren från att bli smittade av CAV så att de inte blev immuna under uppfödningen. En kontrollerad infektion i form av ett vaccin utvecklade immunitet och skyddade avkomman mot BWD.
Vid serologisk undersökning av slaktkycklingflockar på slakterier sågs antikroppar mot CAV framför allt i flockar som var uppfödda i hus där det nyligen förekommit utbrott med blåvingesjuka. Det gick att sanera smittan från husen med noggrann rengöring och desinfektion, men det var svårt.

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