

## Studies on the persistence and excretion of egg drop syndrome 1976 virus in chickens

Ursula Heffels , S.E.D. Khalaf & E.F. Kaleta

To cite this article: Ursula Heffels , S.E.D. Khalaf & E.F. Kaleta (1982) Studies on the persistence and excretion of egg drop syndrome 1976 virus in chickens , Avian Pathology, 11:3, 441-452, DOI: 10.1080/03079458208436116

To link to this article: <https://doi.org/10.1080/03079458208436116>



Published online: 03 Jan 2008.



Submit your article to this journal [↗](#)



Article views: 423



View related articles [↗](#)



Citing articles: 1 View citing articles [↗](#)

## STUDIES ON THE PERSISTENCE AND EXCRETION OF EGG DROP SYNDROME 1976 VIRUS IN CHICKENS<sup>1</sup>

HEFFELS, URSULA, S.E.D. KHALAF<sup>2</sup> and E.F. KALETA

*Klinik für Geflügel der Tierärztlichen Hochschule Hannover  
Bischofsholer Damm 15, D-3000 Hannover 1, W. Germany*

### SUMMARY

EDS virus strain 127 did not induce clinical signs or gross lesions in susceptible chicks of various age groups and in adult cocks. Virus persistence in various internal organs and the rate of excretion of virus by experimentally-infected chicken declined rapidly with increasing age. Virus 127 was detectable in organs of young chicks up to 5 weeks post-infection and in their faeces up to 2 weeks post-infection. In adult birds virus 127 persisted in tissues for about 3 weeks and was excreted with faeces for only 1 week post-infection.

Vaccination of adult cocks with an inactivated vaccine resulted in intermittent shedding of virus only within the first 4 days post-challenge; thus, in comparison to non-vaccinated chickens, vaccination reduced virus excretion in faeces to a major extent.

### INTRODUCTION

Since the first description of the egg drop syndrome 1976 (EDS 76) as a viral disease many reports have been published on the world-wide distribution of this condition in the domestic chicken (McFerran *et al.*, 1977; Badstue and Smidt, 1978; Baxendale, 1978; Meulemans *et al.*, 1978; Picault, 1978; Rampin *et al.*, 1978; Vielitz, 1978; Zsák and Bartha, 1979; Hwang *et al.*, 1980; Firth *et al.*, 1981; Yamaguchi *et al.*, 1981), duck and goose (Calnek, 1978; Villegas *et al.*, 1979; Schloer, G.M., 1980), and in various species of free-living birds (Kaleta *et al.*, 1980; Malkinson and Weisman, 1980). These findings indicate that EDS 76 is of both economical and ecological importance. Most of the work published to date has been on studies of the disease in fowl. The aetiological agent was identified as an avian haemagglutinating adenovirus (McFerran *et al.*, 1978a; Todd and McNulty, 1978; Adair *et al.*, 1979; Kraft *et al.*, 1979; McFerran, 1979) which is shed by carriers in the faeces but is also egg transmitted (McFerran, 1979; Baxendale, 1980; Darbyshire and Peters, 1980; van Eck, 1980).

---

Received 27 October 1981

Accepted 25 January 1982

<sup>1</sup>Supported in part by a grant of the Bundesministerium für Ernährung, Landwirtschaft und Forsten, Bonn

<sup>2</sup>Fellow of the German Academic Exchange Service from the University of Cairo, Egypt.

McFerran *et al.* (1978b) suggested that early (or vertically) infected chicks remain carriers of the virus during the growing period. The development of sexual maturity is accompanied by activation of the latent virus, which in turn results in shedding of infectious virus and laying of malformed eggs for several weeks at the beginning of the laying period. However, little information is available on the persistence of infectious virus following natural or experimental infection of susceptible or immune birds. Nevertheless, such information is of prime importance for an understanding of the epizootiology and pathogenesis of the disease and its consequences. A major advance in the control of EDS 76 was the development of inactivated vaccines which confer a satisfactory immunity following a single intramuscular injection (Baxendale *et al.*, 1978; Viaene *et al.*, 1979; Baxendale *et al.*, 1980; Khalaf *et al.*, 1980; Redmann *et al.*, 1981). This prompted us to investigate whether immunised birds excrete infectious virus soon after challenge. In this study, we tried to obtain information on the development of clinical symptoms, gross lesions, virus persistence and virus excretion in experimentally infected chickens of different ages. In addition, virus excretion by susceptible and immunised birds was examined.

## MATERIALS AND METHODS

### *Cell cultures*

Kidneys from 1 to 3-day-old SPF chicks were the source of chick kidney cells (CKC), while chick embryo liver cells (CELC) were cultivated from livers of 11 to 14-day-old SPF embryos. Cells were grown in Eagle's basal medium with Earle's salts containing 10% tryptose phosphate broth, antibiotics (sodium benzyl penicillin 200 iu/ml, streptomycin sulphate 200 µg/ml, nystatin 50 µg/ml) and 15% foetal calf serum (FCS). In the maintenance medium the FCS was reduced to 2%.

### *Virus*

Strain 127 of EDS 76 virus propagated in CKC or CELC contained  $10^{6.5}$  tissue culture infective doses<sub>50</sub> (TCID<sub>50</sub>)/ml. It was used for the experimental infections and for the serological tests.

### *Experimental birds*

All chicks were hatched from SPF eggs (Valo eggs, Lohmann, Cuxhaven, Germany) and maintained in individual cages or isolators. The experimental designs with details of number and age of birds, mode of infection and specimens examined are summarised in Table 1. All birds were observed daily for clinical symptoms and necropsied at the end of the experiments.

### *Vaccination*

For investigation of virus excretion of vaccinated chicken some adult cocks received an intramuscular injection of 0.5 ml of the inactivated vaccine "Nobi-Vac EDS 76" (VEMIE, Kempen, Germany) before experimental infection.

### *Specimens for virus reisolation*

*Faeces and intestinal content.* 1-3 g of faeces or content of duodenum, jejunum, caecum and cloaca were diluted 1:2 with nutrient broth containing antibiotics (sodium benzyl penicillin 200 iu/mg, streptomycin sulphate 200 g/ml, nystatin 50 g/ml), shaken vigorously, centrifuged for 5 min at 2500 g and stored at -20°C until required. After thawing and another low speed centrifugation cycle the supernatant was used for inoculation of cell cultures.

*Buffy coat.* Samples of 1-5 ml of heparinised blood were kept at room temperature for 3 hours for sedimentation to obtain the phase rich in leukocytes. This phase was

Table 1. Experimental designs: Chicken, route of infection and sample tested.

Experiment	EDS virus status	Age	Number of chicken per		Infection with virus 127		Sample examined
	at time of infection		Group	Sampling	Route	Log <sub>10</sub> <sup>a</sup> TCID <sub>50</sub> /bird	
A	Free	4 days	5	5	Intranasal	5.5	Droppings, (at 35th day p.i. <sup>b</sup> : liver, spleen, kidney, content of 4 parts of intestine)
		25 days	5	5	Intranasal	5.8	
		46 days	5	5	Intranasal	5.8	
		33 weeks	18	18-7 <sup>c</sup>	Oral	6.5	
B	Free	2 days	27	3	Intranasal	5.5	Buffy coat, brain, trachea, colon
		21 days	27	3		5.8	
		36 days	27	3		5.8	
C	Free	33 weeks	13	1	Oral	6.5	Liver, kidney, testes, content of 4 parts of intestine
D	1 w.p.vacc. <sup>d</sup>	33 weeks	3	3	Oral	6.5	Droppings
	2 w.p.vacc.	33 weeks	3	3			
	4 w.p.vacc.	33 weeks	3	3			

a Tissue culture infective dose<sub>50</sub>

b Post infection

c One bird was killed on each day of testing in order to obtain organ samples for virus reisolation (see Experiment C)

d Weeks post-vaccination

carefully transferred onto Ficoll Isopaque (density 1.077 g/ml) and centrifuged at 200 g for 5 min which led to the separation of the buffy coat cells from erythrocytes. After washing in sterile phosphate buffered saline (PBS) pH 7.2 and another centrifugation at 200 g for 5 min the buffy coat cells ( $10^6$ – $10^7$  cells/sample) were resuspended in 1 ml PBS and seeded immediately onto cell cultures.

*Internal organs.* Portions of the organs listed in Table 1 were homogenised, resuspended in 4 ml nutrient broth containing antibiotics, and stored at  $-20^\circ\text{C}$ . Before inoculation onto cell cultures the tissue samples were thawed and centrifuged at low speed to deposit gross particles.

#### *Virus reisolation*

The buffy coat suspensions and the supernatants of the tissue and faeces samples were inoculated onto CELC, termed "primary cultures", and passaged three times in CKC at 4 to 8 days intervals before being discarded as negative. At the end of each passage the presence of EDS 76 virus in the medium was confirmed by specific haemagglutination (HA), and positive samples were not passaged further.

#### *Serology*

In order to study the experimental infections blood samples from all birds were collected at the day of termination and the sera were tested for specific antibodies to EDS 76 virus in the haemagglutination inhibition (HI) test performed by the  $\beta$ -method using 4 HA units and 1% chicken erythrocytes.

## RESULTS

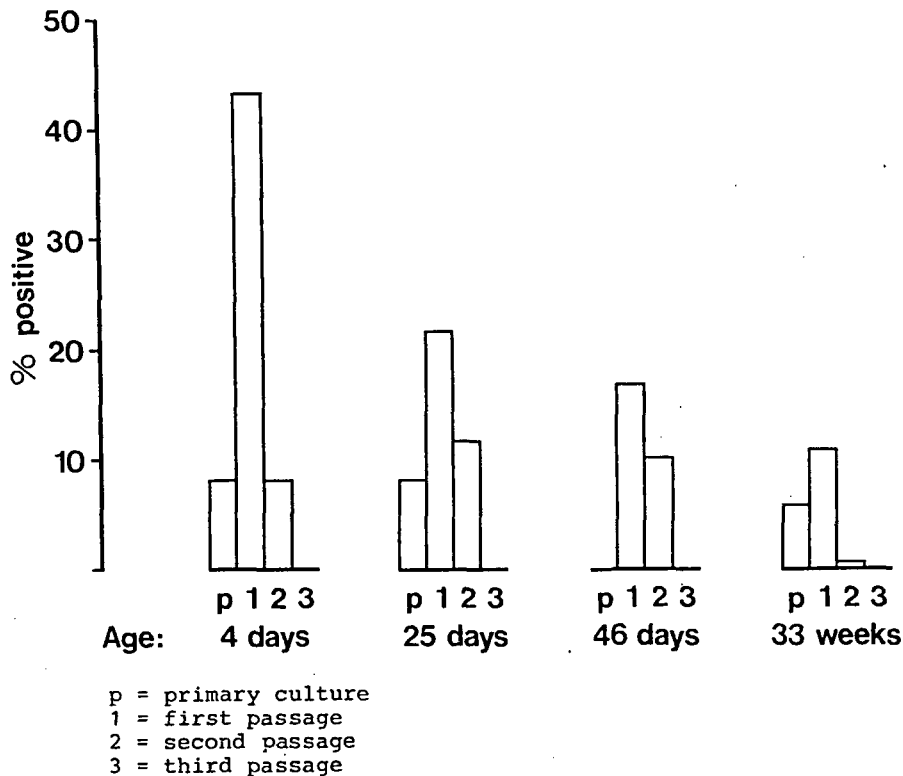
### *Clinical and pathological findings*

The EDS virus strain 127 did not induce clinical signs of disease in any chickens of the age groups under study. In addition, at necropsy none of the experimental birds showed gross lesions which could be attributed to the inoculated virus. Microscopic examinations of semen samples obtained from infected adult cocks did not indicate any alterations of the morphology and viability of spermatozoa.

### *Influence of number of passages in cell cultures on the reisolation rate of virus 127*

The first question on virus reisolation concerned our methodology. Our question was: How many passages in chick kidney cell cultures are necessary to recover even small quantities of virus 127 from faecal samples? To answer this question, we used birds which were at the time of inoculation 4, 25, 46 days and 33 weeks old (Experiment A). The number of samples per group taken over the whole examination period was 60 from each group of young birds and 150 from the adult birds.

It is quite clear from the data presented in Text-fig.1 that the rate of virus detection in primary cultures is relatively low when compared with the first passage. The first passage yielded the highest rates of reisolation for all age groups. In the second passage additional samples became positive, but none did so in the third passage. The results indicate that at least two passages are necessary to detect most virus 127 in the samples of faecal material. Similar results were obtained when tissue samples were the primary inocula (Experiment B and C).



*Text-fig. 1. Influence of age of birds and number of passages in cell cultures on rate of re-isolation of virus 127 from faeces.*

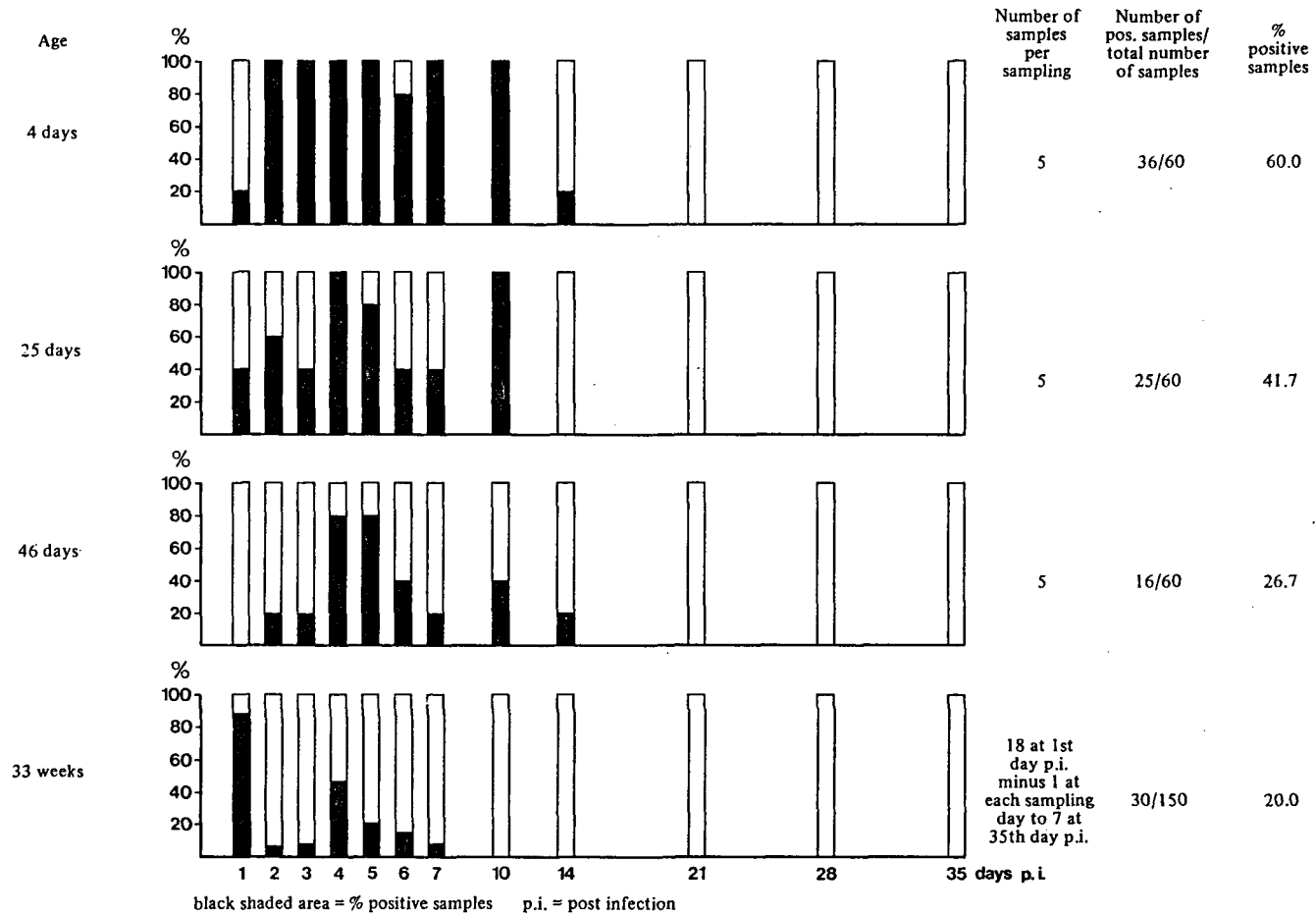
*Reisolation of virus 127 from faeces of chicken of various ages*

Results of examination of faecal samples for virus 127 (Experiment A) taken at different times post-infection are shown in Text-fig. 2. The duration of virus excretion in the group of youngest birds extended from the 1st to the 14th day post-inoculation. Quite similar results were obtained with the chickens which were 25 and 46 days old at time of infection. The 33-week-old cocks excreted virus only for the first 7 days post-inoculation.

The reisolation rate of virus 127 was plotted for each day of examination, and is indicated by the height of the black-shaded areas in the columns of Text-fig. 2. In addition, the numbers and percentages of virus-positive samples over the whole period of testing are given. From this graph, we conclude that the rate of virus excretion in faeces markedly decreased with increasing age of the young chicks in the first three groups. Virus shedding by the 33-week-old cocks was found to be even lower than the shedding rate of the 46-day-old chickens.

*Reisolation of virus 127 from different tissues of chickens of various ages*

Experiment B was performed to follow the persistence of virus 127 in internal organs of young chickens for a period of 42 days. Three birds per group and time were examined for virus 127. The results are presented in Table 2. Virus isolations were obtained from all age groups and from all tissues examined during the 1st week post-



Text-fig. 2. Age dependence of excretion of virus 127 with faeces.

Table 2. Reisolation of virus 127 from tissues of chickens of different ages following experimental infection.

Age at time of infection	Tissue	Days post-infection									
		3	5	7	9	11	13	18	35	42	
2 days	Buffy coat	0/3 <sup>a</sup>	2/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Brain	0/3	3/3	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Trachea	0/3	3/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Colon	0/3	2/3	2/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
21 days	Buffy coat	2/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Brain	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Trachea	3/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Colon	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
36 days	Buffy coat	1/3	0/3	0/3	1/3	0/3	0/3	0/3	0/3	1/3	0/3
	Brain	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Trachea	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	Colon	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3

a Number of positive samples/total number of samples.



inoculation. However, in buffy coat sporadic isolates were obtained up to the 35th day post-infection. Attempts to reisolate EDS 76 virus on the 35th day post-infection from liver, kidney, spleen and intestine of the young chickens which were previously examined for virus excretion in faeces (Experiment A) were not successful.

In Experiment C, adult cocks were examined for persistence of virus following oral infection. The results are presented in Table 3. Although virus excretion in faeces of this age group could only be detected up to the 7th day post-infection, virus persisted in internal organs up to 21 days post-infection. Virus was isolated from all four parts of the intestine, i.e. duodenum, jejunum, caecum and cloaca, as well as from liver, kidney, testes and caecal tonsils. The frequency of isolations did not seem to differ much between the examined tissues.

*Reisolation of virus 127 from faeces of immunised versus non-immunised adult cocks*  
To investigate whether immunised chickens still shed detectable amounts of virus 127 in their faeces post-infection, adult cocks were vaccinated 1, 2 and 4 weeks prior to challenge (Experiment D). The results were compared with those of the experimental infection of adult, unvaccinated cocks (Experiment A) (Table 4). Virus could be reisolated from unvaccinated birds on each day during the 1st week post-infection but not later. In contrast, vaccinated birds shed sporadically at irregular intervals during the first 4 days post-challenge.

*Immune response*

At the end of experiment A the sera of all birds were found to be positive with HI antibody titres between 4 log<sub>2</sub> and 6 log<sub>2</sub> in all age groups. In experiment B and C, specific HI antibodies were first detected in sera of birds killed 7 days post-infection in all age groups except those from the youngest group which developed antibodies beginning on the 9th day post-infection. Sera from chickens killed subsequently were all positive for EDS virus. The HI antibody titres ranged between 3 log<sub>2</sub> and 6 log<sub>2</sub>.

In experiment D, birds in the first group had not developed HI antibodies at the time of challenge, while sera from cocks in the second and third group were positive at this time, with HI titres of 2 log<sub>2</sub> and 5 log<sub>2</sub>, respectively. At the end of experiment the sera of all birds contained HI antibodies with titres between 5 log<sub>2</sub> and 6 log<sub>2</sub>.

Table 3. *Reisolation of virus 127 from tissues of 33-week-old cocks following experimental infection.*

Tissue	Virus reisolations at days post-infection <sup>a</sup>												
	1	2	3	4	5	6	7	10	14	21	28	35	70
Duodenum						+		+		+			
Jejunum						+	+	+	+				
Caecum	+ <sup>b</sup>					+							
Caecal tonsils	+	+	+	+	+	+	+						
Cloaca						+			+				
Liver					+				+				
Kidney	+		+	+		+			+	+			
Testes			+						+	+			

<sup>a</sup> One bird per sampling day

<sup>b</sup> Reisolation of virus 127

Table 4. Reisolation of virus 127 from faeces of immunised and non-immunised adult cocks following experimental infection.

Ex-periment	Vaccination pre-infection (weeks)	Days post-infection													
		1	2	3	4	5	6	7	10	14	21	28	35	42→70	
D	1	0/3 <sup>a</sup>	1/3	0/3	1/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	2	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3			
	4	2/3	3/3	1/3	0/3	0/3	0/3	0/3	0/3	0/3					
A	No vaccination	15/18	1/17	1/16	7/15	3/14	2/13	1/12	0/11	0/10	0/9	0/8	0/7	0/26	

<sup>a</sup> Number of positive samples/total number of samples.

## DISCUSSION

The absence of clinical signs and gross lesions (apart from effects on egg production and shell quality) was described previously (McCracken and McFerran, 1978; Hiruma *et al.*, 1981). Similarly, the present data give no indication of clinical signs or macroscopically visible changes in young or adult male chickens following experimental infection with EDS 76 virus.

The degree of virus excretion in faeces was distinctly dependent on age. Although the adult cocks were inoculated orally with a higher dose of virus than were the younger age groups, the rate and the duration of virus excretion was definitively lower than from the newly-hatched chicks. The longest time interval between experimental infection and virus recovery from faeces was 2 weeks. Cook and Darbyshire (1980), also, were unable to recover EDS virus from pharyngeal and cloacal swabs from adult hens for longer than 3 weeks. In spite of these results, Cook and Darbyshire (1981) could demonstrate virus transmission from infected chicks to susceptible contact chicks during the whole growing period.

Persistence of virus was demonstrated by reisolation in the young chickens up to the 5th week and in the adult birds up to the 3rd week post-infection. Virus reisolations were successful from peripheral white blood cells and from different tissue homogenates, which might have contained white blood cells. Consequently, we have no information which proves the persistence of virus in organ-specific cells, e.g. hepatocytes, since the possibility exists that the virus isolations were from residual blood cells and not from organ-specific cells.

Further studies are in progress to clarify the question of localisation and persistence of EDS virus in latently infected chicks during the growing period, which might lead to an outbreak of EDS 76 at the beginning of the laying period (McFerran *et al.*, 1978b).

Vaccination of susceptible adult cocks reduced the rate and duration of virus excretion. This reduction was evident on challenge 1 week post-vaccination and remained at similar levels on challenge 2 and 4 weeks post-vaccination. Our results agree with the findings of Baxendale (1980), who was unable to reisolate virus 127 from vent swabs and buffy coat samples taken from adult layers at weekly intervals for 4 weeks post-challenge. Thus, vaccination not only prevents drop in egg production and the formation of malformed eggs (Khalaf *et al.*, 1980), but also reduces to a major extent excretion and subsequent dissemination of EDS virus in poultry flocks.

## REFERENCES

- Adair, B.M., McFerran, J.B., Connor, T.J., McNulty, M.S. and McKillop, E.R. (1979). Biological and physical properties of a virus (strain 127) associated with the Egg Drop Syndrome 1976. *Avian Pathology*, 8: 249-264.
- Badstue, P.B. and Smidt, Brita. (1978). Egg-drop Syndrome 76 in Danish Poultry. *Nordisk Veterinärmedicin*, 30: 498-505.
- Baxendale, W. (1978). Egg drop syndrome 76. *Veterinary Record*, 102: 285-286.
- Baxendale, W. (1980). Recent research on egg drop syndrome 76 (EDS 76). *Proceeding of 29th Western Poultry Disease Conference, Acapulco, Gro, Mexico, April 1980*, p.206-211.
- Baxendale, W., Lütticken, D., Hein, R. and Orthel, F.W. (1978). Vaccine against the egg drop syndrome 76 of chickens. *International Virology IV 119. Abstracts of the fourth International Congress for Virology, The Hague*.
- Baxendale, W., Lütticken, D., Hein, R. and McPherson, I. (1980). The results of field trials conducted with an inactivated vaccine against the egg drop syndrome 76 (EDS 76). *Avian Pathology*, 9: 77-91.

- Calnek, B.W. (1978). Hemagglutination inhibition antibodies against an adenovirus (virus 127) in white pekin ducks in the United States. *Avian Diseases*, 22: 798-801.
- Cook, Jane K.A. and Darbyshire, J.H. (1980). Epidemiological studies with egg drop syndrome 1976 (EDS 76) virus. *Avian Pathology*, 9: 437-443.
- Cook, Jane K.A. and Darbyshire, J.H. (1981). Longitudinal studies on the egg drop syndrome 1976 (EDS 76) in the fowl following experimental infection at 1-day-old. *Avian Pathology*, 10: 449-459.
- Darbyshire, J.H. and Peters, R.W. (1980). Studies on EDS-76 virus infection in laying chickens. *Avian Pathology*, 9: 277-290.
- Firth, G.A., Hall, M.H. and McFerran, J.B. (1981). Isolation of a haemagglutinating adeno-like virus related to virus 127 from an Australian poultry flock with an egg drop syndrome. *Australian Veterinary Journal*, 57: 239-242.
- Hiruma, M., Hidaka, A., Takai, S., Sasaki, T. and Higashihara, M. (1980). Pathological lesions of the oviducts in fowls experimentally infected with avian adenovirus strain H-162 associated with egg drop syndrome-1976. *Japanese Veterinary Poultry Association: English Summaries of the Studies on Poultry Diseases in Japan*, p.13-15.
- Hwang, M.H., Lamas, J.M., Hipolito, O. and Silva, E.M. (1980). Egg drop syndrome-1976, a serological survey in Brazil. *Proceedings of the VIth European Poultry Conference WPSA, Hamburg, Vol.II*, p.313-320.
- Kaleta, E.F., Khalaf, S.E.D. and Siegmann, O. (1980). Antibodies to egg drop syndrome 76 virus in wild birds in possible conjunction with egg-shell problems. *Avian Pathology*, 9: 587-590.
- Khalaf, S.E.D., Kaleta, E.F., Siegmann, O. and Lüders, H. (1980). Untersuchungen zur Schutzimpfung von Legehennen gegen das Egg drop Syndrom 1976 (EDS 76). *Proceedings of the VIth European Poultry Conference WPSA, Hamburg, Vol.II*, p.408-415.
- Kraft, V., Grund, S. and Monreal, G. (1979). Ultrastructural characterisation of isolate 127 of egg drop syndrome 1976 virus as an adenovirus. *Avian Pathology*, 8: 353-361.
- Malkinson, M. and Weisman, Y. (1980). Serological survey for the prevalence of antibodies to egg drop syndrome 1976 virus in domesticated and wild birds in Israel. *Avian Pathology*, 9: 421-426.
- Meulemans, G., Froyman, R., Peeters, J. and Halen, P. (1978). Causes of drop in egg production in Belgian laying flocks. *Vlaams Diergeneeskundig Tijdschrift*, 47: 292-298.
- McCracken, R.M. and McFerran, J.B. (1978). Experimental reproduction of the egg drop syndrome 1976 with a haemagglutinating adenovirus. *Avian Pathology*, 7: 483-490.
- McFerran, J.B. (1979). Egg drop Syndrome, 1976 (EDS '76). *Veterinary Quarterly*, 1: 176-180.
- McFerran, J.B., Rowley, Hlene M., McNulty, M.S. and Montgomery, Linda, J. (1977). Serological studies on flocks showing depressed egg production. *Avian Pathology*, 6: 405-413.
- McFerran, J.B., Connor, T.J. and Adair, B.M. (1978a). Studies on the antigenic relationship between an isolate (127) from the egg drop syndrome 1976 and a fowl adenovirus. *Avian Pathology*, 7: 629-636.
- McFerran, J.B., McCracken, R.M., McKillop, Eileen R., McNulty, M.S. and Collins, D.S. (1978b). Studies on a depressed egg production syndrome in Northern Ireland. *Avian Pathology*, 7: 35-47.
- Picault, J.P. (1978). Chutes de ponte associées à la production d'oeufs sans coquille ou à coquille fragile. *L'Aviculteur*, 379: 57-60.
- Rampin, T., Enice, F. and Mandelli, G. (1978). Anticorpi contro l'antigene "B/C14" in ovaiole e riproduttori di allevamenti italiani. *Clinica Veterinaria*, 101: 265-272.
- Redmann, T., Khalaf, S.E.D., Lüders, H., Kaleta, E.F. and Siegmann, O. (1981). Feldbeobachtungen zur Epizootologie und Prophylaxe des Egg Drop Syndrom 1976 (EDS 76). *Deutsche Tierärztliche Wochenschrift*, 88: 125-128.
- Schloer, G.M. (1980). Frequency of antibody to adenovirus 127 in domestic ducks and wild waterfowl. *Avian Diseases*, 24: 91-98.
- Todd, D. and McNulty, M.S. (1978). Biochemical studies on a virus associated with Egg Drop Syndrome 1976. *Journal of General Virology*, 40: 63-75.
- Van Eck, J.H.H. (1980). Egg transmission of egg drop syndrome 1976 virus in fowl (1980). *Veterinary Quarterly*, 2: 176-178.
- Viaene, N., Spanoghe, L., Bijmens, B., Devos, A. and Sierens, G. (1979). Vaccination against egg drop syndrome 1976 with an inactivated vaccine, strain B.C. 14 in oil adjuvans. *Vlaams Diergeneeskundig Tijdschrift*, 48: 163-168.
- Vielitz, E. (1978). Adeno-Virusinfektion des Huhnes. *Lohmann Information März/April* p.9-12.
- Villegas, P., Kleven, S.H., Eidson, C.S. and Trampel, D. (1979). Isolation of a hemagglutinating adenovirus serologically related to adenovirus 127. *Avian Diseases*, 23: 507-514.

- Zsák, L. and Bartha, A. (1979). Isolation of an adenovirus associated with egg drop syndrome (EDS) in laying hens in Hungary. *Magyar Állatorvosok Lapja*, 30: 691-693.
- Yamaguchi, S., Imada, T., Kawamura, H., Taniguchi, S., Saio, H. and Shimanatsu, K. (1981). Outbreaks of egg-drop syndrome-1976 in Japan and its etiological agent. *Avian Diseases*, 25: 628-641.

## RESUME

### Etude de la persistance et de l'excrétion du virus EDS-76 chez le poulet

La souche 127 de virus EDS n'a pas induit de signes cliniques et de lésions macroscopiques chez des poulets sensibles de différents groupes d'âge et chez des coqs adultes. La persistance du virus dans différents organes et le taux d'excrétion du virus par les poulets infectés expérimentalement ont diminué rapidement au fur et à mesure de l'augmentation de l'âge. Le virus 127 était détectable dans les organes de jeunes poulets jusqu'à 5 semaines après l'infection et dans les fécès jusqu'à 2 semaines après l'infection. Chez les animaux adultes le virus 127 persistait dans les tissus pendant 3 semaines, et était excrété dans les fécès pendant 1 semaine après infection.

La vaccination des coqs adultes avec un vaccin inactivé a résulté en une excrétion intermittente du virus seulement pendant les 4 jours postérieurs au challenge; en conséquence, en comparaison des poulets non vaccinés il y a eu réduction importante de l'excrétion virale dans les fécès des poulets vaccinés.

## ZUSAMMENFASSUNG

### Untersuchungen über die Persistenz und Ausscheidung des Virus des Egg Drop Syndrom 1976 bei Hühnern

Der EDS Virusstamm 127 induziert bei empfänglichen Küken verschiedener Altersgruppen und bei erwachsenen Hähnen keine klinischen oder pathologisch-anatomischen Veränderungen. Die Persistenz des Virus in verschiedenen inneren Organen und die Ausscheidungsrate des Virus nimmt bei experimentell infizierten Küken mit zunehmendem Alter rasch ab. Das Virus 127 konnte in Organen von kleinen Küken bis zu 5 Wochen post infectionem und in ihren Faeces bis zu 2 Wochen p.i. nachgewiesen werden. Bei erwachsenen Tieren persistierte der Stamm 127 in den Geweben ca. 3 Wochen und wurde mit den Faeces nur 1 Woche p.i. ausgeschieden. Nach einer Impfung erwachsener Hähne mit einem inaktivierten Impfstoff erfolgte eine intermittierende Virusausscheidung nur innerhalb der ersten 4 Tage nach einer Testinfektion; im Vergleich zu nichtgeimpften Tieren, wird die Virusausscheidung mit dem Faeces durch die Impfung in erheblichen Umfang reduziert.